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SINGLE CASES BOOK

11th Congress of the
European Federation
of Internal Medicine
(EFIM)

XXXIII
National Congress of
the Spanish Society
of Internal Medicine
(SEMI)

Palacio Municipal de Congresos
Campo de las Naciones



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SC-1 A CASE OF ANAPLASTIC ASTROCYTOMA PRESENTING WITH RHABDOMYOLYSIS

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Summary: Rhabdomyolysis is a syndrome characterized by muscle necrosis and release of intracellular muscle contents to circulation. Astrocytoma is one of the primary central nervous system tumors composed of neuroglial cells and provides a variety of symptoms according to lesion size and location. Here we present a 25-year-old male requiring mechanical ventilation that came with loss of muscle strength on the left extremity, suffered rhabdomyolysis due to clonus and diagnosed as anaplastic astrocytoma after the brain lesion biopsy.

Introduction: Rhabdomyolysis is a syndrome characterized by muscle necrosis and release of intracellular muscle contents to circulation. The classic presentation is with myalgia, myoglobinuria with red to brown color urine and elevated muscle enzymes. The disease spectrum may involve life-threatening illness with electrolyte imbalance or acute renal failure or may be with asymptomatic elevations of muscle enzymes. Astrocytoma is one of the primary central nervous system tumors composed of neuroglial cells and provides a variety of symptoms like headaches, seizures, memory loss, motor weakness or speech disorder according to the size and location of the lesion. Here we present a 25-year-old male requiring mechanical ventilation that came with loss of muscle strength on the left extremity, suffered rhabdomyolysis due to clonus and diagnosed as anaplastic astrocytoma after the brain lesion biopsy.

Case report: 25-year-old male patient applied with complaints of left upper and left lower extremity weakness and numbness to neurology outpatient clinic. The tomographic examination revealed hypodense areas on bilateral centrum semiovale, compression on the right lateral ventricle frontal horn and midline shift to the left. With a possible ADEM (acute disseminated encephalomyelitis) diagnosis, patient was given 2 times of pulse steroid therapy and 5 sessions of plasmapheresis. 4 days later, there was a decrease in the left lower and upper extremity strength. On neurological examination, left peripheral facial paralysis, left upper limb spastic hemiparesis, left hemihypoesthesia, left Babinski (+), and clonus (+) was found. 3 days later, the patient was intubated after the cease of the spontaneous breathing. On the same day, the blood biochemistry were as follows; urea 221 mg / dl, creatinine 5.51 mg / dl, sodium 146 mmol / l, potassium, 6.08 mmol / l, LDH 10 200 U / L, ALT 6870 U / L, AST 3690 U / l, uric acid, 21.6 mg / dl, indirect bilirubin, 3.38 mg / dl, total bilirubin, 6.44 mg / dl, INR 4.58 and aPTT was determined as 61.8. CK was 58750 U / l, viral hepatitis markers were HBsAg, anti-HIV, anti-HCV, HCV RNA negative, serological markers were ANA, anti-ds DNA, AMA, anti-LKM-1, anti-A SS anti-SS, B, anti-Scl 70, anti-Jo-1, P-ANCA-negative, the C-ANCA positive and viral TORCH group markers were negative. With current laboratory and physical examination findings, patient was considered as rhabdomyolysis due to clonuses. Towards clonus, he was started atracurium 0.4 mg / kg with an initial bolus followed by IV infusion of 12 mcg / kg / min in maintenance dose. He was started hemodialysis with a Integra brand of bedside hemodialysis machine and adequate hydration was maintained. Clonuses ended, at the 10th day, the blood biochemistry was as follows: urea:191 mg / dl, creatinine 4.66 mg / dl, sodium 135 mmol / l, potassium, 4.09 mmol / l, LDH 583 U / L, ALT 25 U / L, AST 32 U / L, total bilirubin, 1.28 mg / dl, and INR was 1.08. The patient was treated with diuretics after the urine output per day increased up to 2000 cc. During the follow up creatinine fell down to 1.12 mg / dl. The patient's clinical picture was re-evaluated. The supratentorial brain MR series revealed multiple brain lesions. Biopsy was taken from areas of the lesion. Anaplastic astrocytoma grade 3 was reported from the pathology. Due to the existing intubation and brain involvement, surgery, radiotherapy and chemotherapy could not be applied. Rhabdomyolysis declined. After the patient's spontaneous breathing came back, 5-day high-dose radiotherapy was started with the support of O₂. Clonuses disappeared and atracurium was stopped. On the neurological examination, verbal responses to verbal stimulation was achieved. He is still alive with continuation of the treatment of astrocytoma.

Conclusion: Seizures are among the non-traumatic causes of rhabdomyolysis and may occur in 56 % of the anaplastic astrocytoma cases. Our patient suffered rhabdomyolysis due to extensive clonuses derived from the brain tumor. He was treated with hydration and hemodialysis treatment, all the medications were carefully selected and potentially hepatotoxic drugs were stopped. Therefore, in order to prevent clonuses, the neuromuscular blocking agent atracurium without renal or hepatic excretion degraded by non-enzymatic Hofmann elimination was simultaneously started with the treatment of rhabdomyolysis. Drugs were used until the end of radiotherapy and drug therapy. The drug was discontinued after the completely disappearing of clonuses. Therefore, especially in patients with clonuses due to space-occupying lesions of the brain, rhabdomyolysis should not be ignored and atracurium should be remembered to be highly effective without hepatotoxic or nephrotoxic side effects.

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SC-2 A CASE OF DIFFUSE LARGE B CELL LYMPHOMA ON THE BASIS OF A CHRONIC MYELOPROLIFERATIVE DISORDER

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Abstract: Interlineage switch from myeloid to lymphomateous malignancies is a rare phenomenon. Progression from a chronic myeloproliferative disorder to large diffuse B cell lymphoma has been reported in very few cases. We describe a case of a 73-year-old woman who developed large diffuse B cell lymphoma six years after the diagnosis of an unclassifiable chronic myeloproliferative disorder, which had been treated with hydroxyurea for over six years.

Introduction: Interlineage switch is a rare phenomenon in hemotologic disorders. More than 99 % of all cases of acute myeloid leukemia show relapses of acute leukemia within the myeloid lineage. Progression of myelodysplastic syndrome and of BCR-ABL negative chronic myeloproliferative disorders to AML is a frequent phenomenon, occurring in 30% and 10% of cases, respectively. In contrast, progression of myelodysplastic syndrome to large diffuse B cell lymphoma is only occasionally reported and seems to be extremely rare in chronic myeloproliferative disorder.

Case report: A 73-year-old female patient had complaints of fatigue, weight loss and excessive sweating for six years. In physical examination, we recognized splenomegaly. In complete blood count, white blood cells were 112000/mm³, hemoglobin was 9 g/dl, platelet count was 650000/mm³. Peripheral blood smear showed basophilia and granulocytocis with neutrophils and immature granulocytocis. Bone marrow aspiration and biopsy were performed, bone marrow smear showed granulocytic hyperplasia. Pathology of the bone marrow was compatible with the chronic myeloproliferative disease. For Philadelphia chromosome analysis, we performed a genetic test and BCR-ABL was negative. So we diagnosed her as BCR-ABL negative chronic myeloproliferative disease. Since the disease diagnosis-nearly for six years, patient had been monitored periodically and given treatment of hydroxyurea. Patient this time came again with the complains of red-purple painful lesions on skin of left hip for 6 months. In complete blood count, white cells were 11000/mm³, hemoglobin was 9.6 g/dl, platelet count was 331000/mm³. In physical examination, we recognized a red-purple, painful, 5 or 6 centimeters in diameter palpable lesions on skin of left hip and sacrum. A biopsy from the lesions was done. Pathology reported these lesions as diffuse large B cell lymphoma infiltration. Metastatic lesions were reported in underskin tissues at right atrium, mediastinal lymph nodes, right iliac bone and bilateral surrenal glands by PET-BT. Neoplastic cells were marked with CD 20 strongly. R-CHOP chemotherapy was planned for 6 cures as first line therapy with the suggestion of NCCN (National Comprehensive Cancer Network). After the treatment, skin lesions and metastasis disappeared but nearly 2 or 3 months later occurred again. 2 cures -21 days between cures- of R-GDP therapy were planned as second line therapy when new skin metastasis and L5 vertebra metastasis were found in periodical controls. Patient is alive and still coming for treatment.

Conclusion: In the literature, to our knowledge, progression of chronic myeloproliferative disease to large diffuse B cell non-hodgkin lymphoma is a rare condition. New lesions of our patient responded to chemotherapy but again occurred very quickly. In such a patient, monitored for a long time with a BCR-ABL negative chronic myeloproliferative disease and had palpable skin lesions, we should remember the progression to another hemotologic malignancy.

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SC-3 DABIGATRAN AS A THERAPEUTIC POSSIBILITY IN HEPARIN-INDUCED THROMBOCYTOPENIA

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Case report: Dabigatran, a new anticoagulant drug, is a reversible direct thrombin inhibitor approved for use in the prophylaxis of deep vein thrombosis in cases of total knee and hip replacement, as an anticoagulant treatment for arrhythmia derived from atrial fibrillation (AF) (1) and is pending European Medication Agency approval for the treatment of venous thromboembolism. Lepirudin and argatroban administered by parenteral route are already approved and widely used to treat heparin-induced thrombocytopenia (HIT). In theory dabigatran with oral administration could be used in the treatment of HIT. In our experience, we present the first case of HIT type II treated with dabigatran to be reported in published medical literature. We present a 67-year old patient, with recurrent thrombosis, admitted by diagnosed portal vein thrombosis probably related to JAK2-positive myeloproliferative syndrome. Treatment was started with unfractionated heparin (UFH) in view of the possible need for surgery for mesenteric ischaemia; it was replaced on day 4 by enoxaparin 1 mg/kg/12 hours. On the seventh day after the start of UFH and the third day after initiating LMWH, platelet levels started to progressively fall from one million to 500,000, until the twelfth day where it stabilised at 123,000, and was



associated with dyspnea, hypoxemia and bilateral pleural effusion. Angio-CT scan of the chest was performed with bilateral pulmonary thromboembolism (PT). PF4/heparin antiplatelet-antiheparin antibody analysis was ordered (ID-PaGIA Heparin/PF4 antibody test / ID-Card). The test was positive with a high probability score of 7-8, in the "The 4 Ts". (2) Treatment was begun with lepirudin, which was replaced with dabigatran after 36 hours with the patient's informed consent. Her evolution was favourable, with disappearance of the pleural effusion. The platelet count rose to close to 300,000 in 48 hours, with the PT resolved on an angio-CT scan performed two weeks after the start of the treatment. HIT usually occurs from the 5th to the 10th day after starting heparin treatment (3) with a global incidence of 2.6%. To date, treatment consists ceasing exposure to any type of heparin, anticoagulant treatment with direct thrombin inhibitors: lepirudin, bivalirudin, argatroban and after recuperation of platelet start low doses of VAK together with the anticoagulant being administered. In our case, the existence of recurring thrombosis despite correct anticoagulation with VKA (probably secondary to JAK-2 positive myeloproliferative neoplasm) as well as in subsequent development of HIT-II, where the same myeloproliferative neoplasm could be a contributing factor. This scenario led us to consider different therapeutic options including the possibility of prolonged use of a direct thrombin inhibitor administered orally, at the same dose used in prior published studies RECOVER and RELAY (150 mg/12 hours) with an excellent response. We believe that dabigatran will be a drug to be considered in the treatment of HIT-II thanks to its rapid onset of action, ease of use, oral administration minimization of risk of anaphylaxis. Our experience and results should be confirmed by further studies.

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SC-4 A CASE OF ACUTE PSYCHOSIS DUE TO VITAMIN B12 DEFICIENCY

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Objectives: Patients with vitamin B12 deficiency may present with hematologic, neuropsychiatric, and cardiovascular symptoms. A clinical picture of acute psychosis can be seen in rare cases and if not corrected, vitamin B12 deficiency can lead to permanent damage.

Case: A sixty-year-old male patient had numbness and tingling in both hands for two months and an impaired sleep pattern, a decreased attention and auditory hallucinations emerging last week. The patient had no known previous neuropsychiatric disorders, alcohol abuse or history of vegetarian life. On physical examination, the patient had cachectic appearance, pale skin and conjunctivas, and hemihypoesthesia, especially at the the upper extremities. The patient had loss of time orientation, reduced attention and difficulty to achieve their goal. Tangentially from time to time, with an euthymic mood, memory was hypoamnesic; he had auditory hallucinations, impaired judgment of subject and object, desorganised behavior and impaired self-care. The patient was confirmed to have acute psychosis after the psychiatric consultation. Laboratory examination was as follows; hemoglobin: 8.8gr/dL (13-17), hematocrit: 24.8% (39.5-50.3), white blood cell count: 4200/mm³ (4000-10500), MCV: 133.1fL (80-99), vitamin B12: 85pg/mL (175-885), and folate: 17.14ng/mL (7-31.4), respectively. Hypersegmented normochromic macrocytic erythrocytes and neutrophils were observed in peripheral blood smear. Colonoscopy in order to explain the pathological weight loss revealed deep ulcers at the terminal ileum, the largest 1 cm in size. In addition to vitamin B12 replacement, haloperidol 10 mg per day was started. During the second week the antipsychotic treatment was discontinued and a half month later after the discharge, hemoglobin was 13.2gr / L and acute psychosis clinical picture was resolved. Hypoesthesia of hands and feet were partly continued complaints.

Conclusion: The diagnosis and treatment of vitamin B12 deficiency is quite simple. In order not to lead to persistent psychiatric symptoms, regardless of the presence of anemia, it would be appropriate to check blood levels of vitamin B12 in patients admitted for psychiatric problems.

SC-5 MALE OSTEOPOROSIS POSSIBLY DUE TO HASHITOXICOSIS: A CASE REPORT

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Objectives: Hashimoto's disease and osteoporosis are less frequent in males. The disease starts with hyperthyroidism called hashitoxicosis in approximately 5 % of patients. Causes of male osteoporosis are hypogonadism, alcoholism, hyperthyroidism and thyroxine usage. We here present an osteoporotic male patient possibly due to hashitoxicosis.

Case: A 49-year old male patient was admitted with diffuse back pain. Thoracal and lomber x-ray radiography revealed reduced density on vertebral bodies. The diagnosis of osteoporosis was made by a T score of -3.2 on DEXA at lumbar spine (L2). For secondary causes of osteoporosis, after questioning revealed the use of large amounts of alcohol (wine) per day; the thyroid, parathyroid, testosterone, free testosterone, osteocalcin and urinary hydroxyproline, calcium and creatinine tests were performed. Thyroid stimulating hormone (TSH): 8.23 mIU / ml (0.35-5.5), free T3: 2.8 pg / ml (2.3-4.2), free T4: 0.6 ng / dl (0.89-1.76), anti-peroxidase antibody (anti- TPO): 754.4 IU / ml (0-12), anti-troglobulin antibody (anti-TG): 78.8 IU / ml (0-34) was found in the other investigation results. Ultrasonography revealed thyroiditis. Hashimoto's disease diagnosis was made as the TSH receptor antibodies were positive. After finding the coexistence of osteoporosis and Hashimoto thyroiditis, we deepened the patients history and she expressed that she had sometimes palpitations, sweating and fever which started about three years ago, but she did not go to hospital. In this patient caught at the hypothyroid phase of Hashimoto's disease, hashitoxicosis related complaints at the beginning of the disease suggest us think that an earlier hyperthyroid phase should be the reason of developing osteoporosis. He is still on follow up for the treatment of osteoporosis and hypothyroidism.

Discussion: In Hashimoto's disease, antiperoxidase and antithyroglobulin antibodies; and in Graves' disease, thyroid stimulating immunoglobulins (TSIs)'s are often positive. TSIs have been blamed for the coexisting hyperthyroidism and Graves' disease in Hashimoto's disease. As a result, we think that Hashimoto's disease, which has the characteristics of Graves' disease because of the hashitoxicosis period, can be the cause osteoporosis.

SC-6 STEROID INDUCED MULTIFOCAL OSTEONECROSIS MIMICKING SEPTIC ARTHRITIS IN A PATIENT WITH AUTOIMMUNE HEPATITIS: HYPERBARIC OXYGEN TREATMENT IS USEFUL

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Objectives: Glucocorticoid treatment is a common factor among the nontraumatic causes of osteonecrosis (ON). To our knowledge, a case of ON associated with steroid treatment in a patient with autoimmune hepatitis has not been reported. Here we aimed to present a case of ON with multifocal involvement mimicking septic arthritis in a patient with autoimmune hepatitis and to report the usefulness of hyperbaric oxygen (HBO) therapy.

Case: A 48-year-old female patient had complaints of pain and swelling of the ankle and knee joints which began two months ago. She had a diagnosis of autoimmune hepatitis for seven months and two weeks ago, was diagnosed with steroid-induced ON of both knees and ankles in another healthcare center. On physical examination, there were mild erythema, effusion, warmth, and diffuse tenderness to palpation on bilateral knees and ankles. There was pain in both knees with flexion and extension. The patient's other examination findings were normal. The admission laboratory investigation was as follows: hemoglobin: 9.9 g / dl, ESR: 97 mm / h, aspartate aminotransferase: 18 IU / L, alanine aminotransferase: 8 IU / L, gamma glutamyl tranferase: 22 IU / L, alkaline phosphatase: 126 IU / L. HBO therapy was begun to the patient. On the 5th day of hospitalization, high fever (38.4°C) once a day together with pain, effusion and significant increase in complaints of heat and redness in the the right knee occurred. Septic arthritis (SA) was the consideration and the joint fluid sample was taken for cell count, direct microscopic examination, gram stain and microbiological culture. There was no identifiable factor, so magnetic resonance imaging examination of the knee joint (MRI) was performed. Magnetic resonance imaging showed signal intensity changes consistent with osteonecrosis of the femur and tibia, an increase of intra-articular fluid of the knee, mild hypertrophic sinovia and edema at the surrounding muscle groups and fatty tissue of the knee joint. The pre-diagnosis of septic arthritis was discarded as microbiological samples and magnetic resonance imaging were not consistent. Antibiotic treatment was stopped, and HBO therapy for a total of 20 sessions at 2.5 ATA for 2 hours a day was continued. After the treatment, the pain improved significantly. She did not accept any surgical intervention, thereafter physical therapy exercises were started. The patient is still under medical treatment in terms of hypothyroidism and autoimmune hepatitis.



Conclusions: Our case is rare and first in the literature as a female having multifocal arthritis and applying with a clinical picture similar to septic arthritis. In conclusion, although rare, cases of ON may present with multifocal septic arthritis-like joint involvement and the beneficial effects of HBO therapy in treating the disease should be kept in mind.

SC-7 PARANEOPLASTIC LIMBIC ENCEPHALITIS RELATED TO RENAL CELL CARCINOMA

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Case report: Paraneoplastic limbic encephalitis is a rare paraneoplastic syndrome characterized with changes in mental status and behaviour abnormalities. Although small cell carcinoma is the most common, it has been identified development of limbic encephalitis with other kinds of carcinoma. Our patient is 66 years old male, who has had nephrectomy for renal cell CA in 2007 and brain operation for year the metastasis in the same. Through the clinical follow up; fluctuations of consciousness, confusion, lethargy, reductions in speech and limitation of orientation and cooperation from time to time have been observed. At beginning; it had been resolving spontaneously within a few hours or days; but later continued for days and weeks developing restrictions of daily activities, walking, somnolence and nourishment. Brain tomography has taken as etiological research. Hemogram and routine biochemistry were normal. At this time; the patient's complaints increased and malnutrition has started. Supportive treatment and total parenteral nutrition have been started. At this period immunohistochemical evaluation has made and Anti-N-metil D-aspartate receptor antibody found positive. Because of this the patient has diagnosed with Paraneoplastic Limbic Encephalitis related to Renal Cell Carcinoma despite its rareness. The treatment has changed to pulse steroid of 1000 mg/day. After the first dose of steroid, consciousness came back in a significant way. In the second day the patient began to move in bed and speak. In the fifth day patient has become to require some help, but can take care of most personal requirements. In cerebrospinal fluid, there can be high values of proteins and lymphocytes. In our case; protein levels were increased. So antineural antibody tests has planned and anti-NMDA receptor antibody detected as positive and Paraneoplastic Limbic Encephalitis Related To Renal Cell Carcinoma diagnosed. At the article of Graus et.al; the positive antineural antibodies support the diagnosis. Finally; in our case of Paraneoplastic Limbic Encephalitis Related To Renal Cell Carcinoma; we exhibit that the treatment is successful for ending the symptoms and Limbic Encephalitis is a rare but possible complication for renal cell carcinoma. This is the first case of Limbic Encephalitis Related To Renal Cell Carcinoma in the literature.

SC-8 HYPEREOSINOPHILIC SYNDROME PRESENTING WITH ACUTE HEART FAILURE

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Objective: We report involvement of the heart in patient with hypereosinophilic syndrome (HES).

Case: 43-year-old female patient had a chest pain, progressing for a month and increasing exertional, shortness of breath, fatigue, and a history of 10 kg weight loss. Because of these complaints patient admitted to the cardiology outpatient clinic. In echocardiography, pericardial effusion, left ventricular ejection fraction 38% and stage 3 diastolic dysfunction was detected. In coronary angiography for the etiology of cardiomyopathy, vascular pathology was not seen. In complete blood count, white blood cell count were 11100/mm³ and eosinophil were 30%. And also in peripheral blood smear cells 22% eosinophils were seen. In the bone marrow aspiration-biopsy examination eosinophils were 20% of the myeloid progenitor cells. No significant findings were observed for leukemic infiltration. Cardiac examination for Advanced Magnetic Resonance Imaging was performed. In the delayed enhancement MRI all subendocardial surface and surface of the interatrial septum facing left atrium had late enhancement areas. Also late enhancement areas was seen at the mid cardiac section of inter ventricular septum. The patient with Hipereosinophilia ongoing, the other causes of height eosinophils were ruling out and cardiac MRI findings as non-ischemic heart involvement was evaluated as cardiac involvement in HES.

Conclusion: As a result, when patient who has acute heart failure with eosinophilia as in our case report, the cardiac involvement in HES should be considered in the differential diagnosis.

SC-9 TORSADÉ DE POINTS IN HIV PATIENT WITH ANTIRETROVIRAL THERAPY

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Abstract: Tenofovir is an nucleotide analogue reverse transcriptase inhibitor used for antiretroviral treatment. We described a case of a 42-year-old woman with a polymorphic ventricular tachycardia due to low potassium serum levels with metabolic acidosis and normal anion gap secondary to tenofovir treatment.

The clinical relevance of renal failure by tenofovir is low, but big enough to carry out a frequent evaluation of renal function in those patients who need treatment with this drug. Clinical presentation: A 42-year-old woman was admitted by loss of consciousness. Past medical history was significant because for C3 HIV with last CD 4 count of 396 cells/mm³, pyelonephritis and HVC co- infection. Usual treatment included Emtricitavine/Tenofovir, Ritonavir, Reyataz, Isoniazid, methadone 130, Quetiapine 100, Diazepam 5, Escitalopram 10. She related malaise with intense asthenia and weakness in lower limbs in the 10 days before. During transfer in the ambulance she suffered two episodes of polymorphic ventricular tachycardia, keeping pulse and constants, which reverted spontaneously. At physical inspection, she was conscious, but sleepy. BP 105/57, SO₂ 95%, HR 86 bpm. Not fever. Cardiac system, pulmonary auscultation, abdominal and neurological examination were normal. Admission laboratory showed. Na 133; K 1.1; bicarbonate 15.2; Cl 11; Cr 1.41; Urea 23; uric acid 1.1; P 1.7; Ca 7.7; serum pH 7.21; troponin T 0.29. 24 hours urinalysis showed glycosuria 11'65, uric acid 346, K 49, Na 252, Cl 210. Single urine test showed Uric Acid 9'9. Basal serum cortisol, parathormone, and thyroids hormones level, were normal as well the renal ultrasound.

Comments: After completing analytical study of the metabolic acidosis with normal anion gap, alkaline urine and persistence of low bicarbonate despite intravenous support with alkaline substances (with high protein and glucose urine levels and low uric acid and P serum levels) we concluded the patient suffered from proximal tubular acidosis as the cause of Polymorphic ventricular tachycardia with low potassium serum levels. After ruling out the alternative etiological we attributed the clinical symptoms to the use of Tenofovir and we discontinued it. The proximal renal tubule is the largest site for excretion of xenobiotics including the tenofovir (which is secreted actively through organic anion transporters in the tube). The proximal tubule dysfunction leads to the loss of substances in the urine which are usually reabsorbed in the tubule (such as proteins of low molecular weight, phosphate or glucose). Tenofovir renal tubular toxicity often occurs in animals treated with this drug, but at concentrations higher than the ones are used in human therapy. The mechanism by which this drug alters the tubular function is still unknown. The clinical relevance of renal failure by tenofovir is low and occurs mostly in the first months of treatment. The incidence, severity and reversibility of nephrotoxicity by tenofovir, seems to be related to the dose and duration of therapy.

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SC-10 LEPTOMENINGEAL CARCINOMATOSIS IN A KRUKENBERG TUMOR ASSOCIATED SIGNET-RING CELL GASTRIC CANCER

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Single case: A 28-year-old woman was admitted to Department of Oncology of Gülhane School of Medicine in Ankara, Turkey, with persistent headache, nausea, vomiting, vertigo and diplopia. Her complains had lasted approximately for 30 days and strengthened last week. In patient's medical history, it's learned that she was diagnosed as signet-ring cell gastric cancer 22 months ago and total gastrectomy was performed. 6 cycles of 5-fluorouracil (5-FU)/folic acid and 45 Gy of radiotherapy was administered in follow. 14 months later from the diagnose date, 17 cm right ovarian mass detected in her routine follow-up and she had a surgery of right salpingo oophorectomy. She was diagnosed as Krukenberg tumor and 6 cycles of docetaxel, cisplatin, and 5-FU (DCF) chemotherapy was planned but because of the side effects of the therapy she was discontinued her therapy after the first cycle. In her physical examination her weight was 52 kg and height was 173 cm. She was oriented to time, place and person. Her vital signs were as follows: a blood pressure of 110/70 mmHg, a pulse rate of 88 beats/min and a respiration rate of 17 breaths/min. She had neck stiffness but doesn't have fever or skin rash. She had temporary spontaneous nystagmus and disarthric speech. Psychomotor slowing was detected as a sign of an alteration on her mental status. Both lower extremities had paresis with preserved deep tendon reflexes. Plantar responses were flexor. Sensory examinations were normal. The breathing sounds were clear and the heart sounds were regular without a murmur. Hepatosplenomegaly was not noted. In her laboratory tests no abnormality was detected except low



hemoglobin levels of 9,6 g/dL (10,7 – 13,0 g/dL) and highly elevated CA 19-9 levels from 156,6 U/ml to 1901,6 U/ml (0 – 35 U/ml) in 3 months. Magnetic resonance imaging (MRI) study of brain with intravenous contrast administration was performed. Linear appearance of contrast enhancement in cerebellar fissures and around cranial nerves was showed. A lumbar puncture and analysis of the cerebrospinal fluid (CSF) was performed. Gram stain and culture were negative. The results showed an elevated protein concentration with normal glucose content and to be hypercellular with many carcinoma cells. Radiological diagnosis of LMC was confirmed by these results and the patient was treated with intrathecal administration of 15 mg methotrexate, once a week, for 3 times. Additional whole-brain irradiation of 20 Gy was performed. By this therapy she had dramatic relief from headaches, nausea, vomiting and diplopia. She was enabling oral intake of food and she was discharged by DCF systemic chemotherapy plan of 6 cycles.

Discussion: In summary, LCM is a rare complication of gastric cancer but recent studies imply that it occurs more than expected but usually might be misdiagnosed. Cytology of the CSF is the gold standard although to avoid from false-negative results, combination of enhanced MRI and CSF cytology be used for accurate diagnosis. Prognosis is poor and worsened if not treated. But, clinical studies are required for standardizing therapy. In conclusion, if the patient who treated for the gastric cancer presenting with neurological symptoms, should be excluded for LCM by clinician.

SC-11 BILATERAL FACIAL PALSY IN YOUNG IMMUNOCOMPETENT PATIENT

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Case report: We present the case of a 31-year-old man, who has acute onset of left facial paralysis. Was treated with prednisone for suspected Bell's palsy, but five days later, there is also right peripheral facial palsy and low back pain of mechanical properties. Hospitalization is recommended to extend the study. Personal medical history; pharyngeal infection treated with amoxicillin three weeks ago, allergy to cephalosporins, viral meningitis when he was three years old without neurological sequelae, professional painter, hunting on weekends, no medication regularly consumed, active smoking, no recent travel abroad and no risk sexual practices. The first day of hospitalization in the Internal Medicine Service, the patient had an acceptable overall situation. Eupneic at rest with saturation by pulse oximetry baseline of 98%. Afebrile. Normotensive. No neck stiffness or other meningeal signs. Bilateral peripheral facial palsy mainly on the left. Pupils isochoric. Fotomotores reflexes preserved. No alteration of other cranial nerves. Retained strength and sensitivity. Preserved and symmetrical deep tendon reflexes. No dysmetria or ataxia. Bilateral flexor plantar cutaneous reflex. No cardiac murmurs or rubs. Breath sounds with no noise added. No submandibular, lateral cervical, supraclavicular, axillary or inguinal lymphadenopathy. Normal abdominal palpation. No skin lesions. No arthritis or signs of phlebitis. The study was completed at that time with electrocardiogram showing sinus rhythm without repolarization abnormalities. Chest and column radiograph without pathological findings. Brain CT with contrast unaltered. The blood count, biochemistry and basic coagulation profile showed no remarkable changes. The inflammatory markers were not elevated. Negative mononucleosis heterophile antibodies. It was decided to perform lumbar puncture, obtaining a crystal clear liquid, with an opening pressure horizontally about 17 cm H₂O, with the following cyto-biochemical profile; Leukocytes 40 cells/mm³ (100% mononuclear), glucose 41 mg / dl, Protein 2.03 g / l, LDH less than 30, ADA less than 3 U / L, lactic acid 2.7 mmol / l. Gram stain and Ziehl Neelsen negative. Latex agglutination antigen detection of Streptococcus pneumoniae, Haemophilus influenzae and Neisseria meningitidis negative. CSF serology is requested for Mycoplasma pneumoniae, Coxiella burnetii, Coxsackie A9 and B, Brucella, Borrelia burgdorferi, Leptospira, syphilis, Listeria monocytogenes, Epstein-Barr virus, measles virus, Echovirus, Varicella zoster, cytomegalovirus, mumps virus, herpes Simplex I and II, cultures of bacteria and fungi as well as PCR study of bacteria (Borrelia burgdorferi), mycobacteria (tuberculosis and bovis) and viruses (herpes Simplex I and II), Varicella zoster, Epstein-Barr virus, Cytomegalovirus and Echovirus,. With the diagnosis of bilateral facial paralysis with clear fluid meningitis was treated empirically with ampicillin, acyclovir and doxycycline, given the history of allergy to cephalosporins. The study was completed with the request of protein electrophoresis and immunoglobulins, thyroid hormones, cryoglobulins, ANA, anti DNA, ANCA, tumor markers, ACE, complement C3 and C4, mantoux and serologies for hepatitis A, B and C, Toxoplasma, Leishmania, HSV I and II, HIV 1 +2, Syphilis, Brucella, Borrelia burgdorferi, Rickettsia Connorii, Campylobacter jejuni, Epstein-Barr virus, Varicella zoster, measles, Mycoplasma pneumoniae, Coxiella burnetii, Chlamydia and cytomegalovirus). Ophthalmoscopy shows no sign of uveitis or alterations in the retina. With background EEG tracing within normal limits. The 6th day of hospitalization, the patient reported self-limited episodes of diplopia, facial and abdominal dysesthesia, severe back pain and weakness in limbs that impede progress. Binocular diplopia. Corneal reflex abolished. Decreased trigeminal sensitivity in travel V2 and V3. No visual field defects. Loss of strength by Medical Research Council scale at the level of proximal lower limb muscles (3/5) and distally (4/5). No clonus. Dysaesthesia and decreased sensation in earlier metameric tour D4 to D11 and a feeling of numbness in fingers clear metameric unrelated. Bilateral hyporeflexia on achilles and patellar level. Bilateral flexor plantar cutaneous reflex. Walks with difficulty without

clear ataxia. Magnetic resonance imaging with contrast of brain and spinal cord, shows affection of multiple cranial nerves bilaterally (III, V, VI and VII), enhancement is also observed in multiple roots at the conus medullaris and cauda equina. Since there is no parenchymal or meningeal involvement, given the neurological outcome, is suspected of possible atypical presentation of Guillain-Barré syndrome (Miller Fisher syndrome). Therefore, treatment is initiated with intravenous immunoglobulins pending results of serological tests, PCR and blood and CSF cultures. Complementary tests are requested; GQ1b antibody, anti-GD1a, anti-Hu, anti-Yo, anti-CVZ (which will be negative) and electroneurogram, which reports diffuse axonal involvement. After five consecutive daily doses of immunoglobulin (400 mg / kg / day), the patient had mild improvement in neurological symptoms and disappearance of pain in the lumbar region, so he starts rehabilitation. It remains empiric antibiotic and antiviral treatment while awaiting test results. The 16th day of hospitalization, the patient has permanent diplopia and left ptosis. Lumbar puncture was repeated, obtaining clear looking liquid, with opening pressure of 20 cmH₂O, and with the following characteristics: glucose 26 mg / dl, protein 5.13 g / l, leucocytes 82 cells/mm³ (100% mononuclear). It is requested cytological and microbiological study. Given the significant increase in cellular protein associated with the hypoglycorrhachia and no other definitive diagnosis, after clinical session commented on, we decided to start tuberculosis (TB) treatment empirically with isoniazid, rifampicin, pyrazinamide, ethambutol, and dexamethasone. After two days of TB treatment, the patient worsens clinically, even being unable to get itself out of bed. It was decided to discuss the case with neurosurgery, to request meningeal biopsy, given the torpid evolution and lack of response to treatments. At that time, we receive the report of the Pathology Department of cerebrospinal fluid (CSF) cytology sample extracted from the 16 th day of admission, showing the appearance of immature lymphocytes. Given this finding we come in contact with the Hematology Department of our hospital. Lumbar punctures was repeated in order to send an urgent sample for flow cytometry to our referral hospital, since lack of this technique in our institution. Confirms the occurrence of a proportion of 80% B cell clonal CD 19 +, CD 20 +, CD 38, kappa-and lambda +. The cytogenetic analysis confirmed the presence of c-myc rearrangement and translocation t (8; 14). The study is completed by CT / PET without pathological findings and bone marrow biopsy does not detect infiltration by lymphomatous cells lineage. Was treated with 6 cycles as Burkimab scheme with triple intrathecal therapy associated with disappearance of malignant cells in CSF after the second session of chemotherapy. At 7 months after diagnosis the patient is in complete remission, presenting a residual mild left facial paralysis and weakness in left lower extremity.

Discussion: Burkitt lymphoma is an aggressive neoplasm of B lymphocytes. Have been described three clinical forms, endemic (mainly in Africa and associated with Epstein-Barr virus with mandibular bone involvement), sporadic (abdominal masses in young patients) and associated with immunodeficiencies (mainly HIV). In total represents less than one percent of all lymphomas, mainly affecting males under 35 years in the sporadic form. It is usually presented in the form of clinical involvement of lymph nodes, intestine, stomach, kidney, ovary, testis, lung, bone marrow and blood, is exceptional, especially the lethal primary involvement of CNS. What makes this case unique is the striking clinical presentation of extramedullary Burkitt lymphoma, with important primary involvement of multiple nerve roots and cranial nerves, as well seen in the MRI study, without meningeal enhancement. Secondly, it is remarkable that it was a patient without a known history of immunosuppression or HIV, which makes this case an extremely rare presentation of Burkitt lymphoma. The differential diagnosis was made initially with diseases caused by pathogens commonly implicated in the clear liquid meningitis predominantly mononuclear (mainly viruses) and neuroborreliosis, neurosarcoidosis, tuberculosis, myasthenia gravis and botulism. We believe that the pathogenesis of neuropathy is based on direct infiltration of nerve roots by lymphoma cells, rather than a paraneoplastic phenomenon. Despite not having histological study that could confirm our hypothesis, there are several observations that lead us to suspect this possibility. First we find a rapid clinical improvement after starting chemotherapy, even to the disappearance of malignant cells in cerebrospinal fluid after completing the second course of treatment. Second, the striking enhancement after administration of gadolinium and cranial nerve roots affected, described in the study by MRI, suggesting this finding breaking the blood-brain barrier by lymphoid cells. Third, the absence of antibodies usually become positive in case of paraneoplastic neuropathies. After reviewing the literature, a few cases of lymphoma with primary presentation to Burkitt nervous system, much less with a clinical and radiological involvement as evident at the peripheral nervous system. As mentioned, most of those affected are associated states of immunosuppression, mainly HIV. In immunocompetent found several cases with involvement at different levels of central nervous system parenchyma cerebral, third ventricle, and hypothalamus, pituitary gland. We found only one case of primary leptomeningeal involvement by Burkitt lymphoma in an immunocompetent child. In conclusion, and derived from the analysis of this case, it may recommend that all patients with suspected Miller Fisher syndrome and after completing conventional treatment, present clinical worsening, assessing the possibility of lymphomatous infiltration of the nervous system. After reviewing the literature, we found two cases of patients suspected of Miller Fisher syndrome treated with immunoglobulins, in which, after a certain stabilization of the neurological clinic, are finally diagnosed with peripheral nervous system infiltration by aggressive B lymphoma. To this end, the cytology remains the gold standard, but due to the morphological similarity between benign and malignant cells, has low sensitivity and high false negative rate. That is why we suggest the possibility of seeking a highly sensitive and specific test, such as flow cytometry and the study of molecular biology in the cerebrospinal fluid.



SC-12 THE PHYSICAL EXAMINATION, THE DETERMINING STEP FOR CLINICAL DIAGNOSIS

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Case report: A 71 year old white male with not known drug allergies, was admitted to the emergency room of our hospital for general malaise. The patient has a stable history of plasma cell dyscrasia MGUS type IgG lambda and arterial hypertension treated with telmisartan, hydrochlorothiazide and amlodipine. The patient was independent for daily living activities and only urinary incontinence. The patient reports one month history of sleep disturbance in the form of day-time somnolence, episodes of disorientation and lack of concentration. Facial flushing appeared with blood pressure rise. He has been seen in the Emergency Room (ER) on several occasions without definitive diagnosis. In the physical exam the initial blood pressure was 153/78 mmHg and there was a significant difference in blood pressure between both arms (SBP 130 mmHg in the left arm, SBP 160mmHg in the right arm) with a heart rate of 73 beats per minute and measured oxygen saturation by pulse oximetry 96%. Cardiac auscultation was rhythmic and no murmurs or extratonos were heard. Pulmonary auscultation with preserved breath sounds. Abdominal examination was normal. A 1-2cm right axillary painless adenopathy was palpable. There were normal and symmetrical peripheral pulses. The chest x-ray showed cardiomegaly with elongated and calcified aorta and radiological signs of COPD. The EKG showed sinus rhythm at 74 beats per minute, left axis and no alterations in repolarization. No blood chemistry alterations were observed. In one of the ER visits a cranial CT was performed showing hypodensities in periventricular white matter compatible with small vessel disease and hypodense lesion in right insular region of 6mm compatible with lacunar infarct. Right axillary lymphadenopathy was detected by ultrasound without signs of malignancy. Due to significant blood pressure differences in the upper extremities a chest CT with contrast was performed yielding a 70-75% stenosis in the proximal segment of the left subclavian artery. Subsequently, Doppler of supra-aortic trunks was carried out demonstrating blood flow reversal in the left vertebral artery. The diagnosis impression was left subclavian artery stenosis with steal syndrome. Arterial angiographic with therapeutic stent placement relieved the patients' symptoms with complete clinical remission. Now that we have reached a diagnosis, we must reflect on the major lessons of this case, that is, physical examination. The key to the diagnosis of this patient was the measurement of blood pressure in both arms, something basic and simple. An accurate medical history and physical examination should be the bases of our clinical experience as internists.

SC-13 HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS SYNDROME

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Introduction: The hemophagocytic lymphohistiocytosis syndrome (HLH) is characterized by systemic proliferation of benign macrophages with prominent hemofagocytaria activity.

Case description: We report the case of a 67 year old woman that was admitted to ICU with fever and hypotension. As a medical history of interest is hypertension, autoimmune axonal neuropathy (treated with plasmapheresis and poliglobin) and autoimmune primary hypothyroidism. The patient was admitted to the Internal Medicine Department because of fever and jaundice of one week duration. A Physical examination revealed jaundice of skin and mucous membranes and painful hepatomegaly of 3 cm. Analytics: leucopenia, thrombocytopenia and progressive coagulopathy. X- ray: bilateral diffuse infiltrates. Normal ECG. CT cervical-thoracic-abdomino-pelvic: hepatosplenomegaly. Positive serology for HBV (anti-HBs and anti HBc). Beta2 microglobulin: 7.45 and CA19.9: 41.2. Mantoux and microbiological studies negatives but a positive blood culture for *Staphylococcus hominis hominis*. Bone marrow biopsy: cytopenias in relation to process toxic-drug-infectious. Treated from the start with broad spectrum antibiotics. At 15 days after admission begins with gradual impairment of liver function tests at the expense of cholestasis. In abdominal ultrasound shows homogeneous hepatosplenomegaly, with intra and extrahepatic bile duct of normal caliber, gallbladder wall slightly edematous, with minimal amount of pericholecystic fluid. On echocardiogram are unremarkable. ICU admission was decided due to the gradual deterioration, where he developed severe pancytopenia and progressive respiratory failure requiring IOT and VM connection. Suspecting hemophagocytic lymphohistiocytosis is made new bone marrow biopsy confirmed the diagnosis and treatment is initiated, as HLH-2004 protocol, dexamethasone, cyclosporin A and etoposide. Within 24 hours the patient evolves and develops unfavorably hepatic coagulopathy, renal failure and ARDS and she died.

Discussion: The HLH includes a broad group of entities characterized by hemophagocytosis with cytopenia of at least two of the three series in the examination of peripheral blood, increased levels of cytokines and serum ferritin. May occur associated with infectious diseases, cancer and autoimmune diseases. Syndrome is rare and its presentation is nonspecific diagnosis difficult to have a mortality of 60% in some form. His presentation can be similar to sepsis, fever, splenomegaly, liver dysfunction, lymphadenopathy,

rash, neurological disorders, anemia and cytopenia. From the histopathological point of view, the existence of hemophagocytosis in the bone marrow, spleen and / or lymph nodes and absence of malignant findings are sufficient to confirm the diagnosis. The goals of treatment are to exclude other causes and / or infectious agents that may require specific management and establish the extent of the disease. Is used to treat corticosteroid, etoposide, and cyclosporine A.

SC-14 IMMUNOCOMPETENT PATIENT WITH DISSEMINATED INFECTION BY MYCOBACTERIUM BOVIS

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Introduction: Tuberculosis is an infectious disease caused by several species of the genus Mycobacterium, all of them belonging to the Mycobacterium tuberculosis complex.

Case description: We report the case of a 77years-old man (without medical or surgical history of interest) that was admitted with fever and disorientation. Physical examination highlighted a temperature of 39 ° C and disorientation to time, place and person with episodes of psychomotor agitation and incoherent speech to the neurological examination. In the initial complementary tests, no evidence of any biochemical alteration. The CBC, red and white series unaltered, with 304,000 platelets, ESR of 39 and normal coagulation. The gases disclosed a partial respiratory failure and the chest radiograph was reported as diffuse micronodular interstitial pattern with greater involvement of upper lobes. With these results we performed a chest CT which showed a micronodular pattern with random distribution and greater involvement of upper lobes where nodules were larger, one cavitated. Small peripheral consolidation with air bronchogram inhomogeneous anterior segment of LSI and lingula. Moreover we had a positive Mantoux (21 mm). In view of the results, a lumbar puncture was performed, in wich there was a clear looking liquid, glucose 29 (simultaneous capillary glucose 140), total protein 144, WBC 36 (62% monocytes and 38% polymorphonuclear) and ADA 13. With the findings of nodular and interstitial pattern, predominantly lymphocytic meningitis glucose consumption is a diagnosis of disseminated tuberculosis, initiating treatment with isoniazid, rifampicin, pyrazinamide and streptomycin, and dexamethasone. With this treatment the patient developed satisfactorily, being the high very close to its baseline, with good tolerance to medication in clinical and analytical controls performed. At 6 weeks after admission was the result of culture for mycobacteria in urine, CSF, and duodenal aspirate remained positive for Mycobacterium bovis.

Discussion: Mycobacterium bovis is a slow-growing mycobacteria, aerobic and the cause of bovine tuberculosis. Related to M. tuberculosis, the mycobacterium that causes tuberculosis in humans, M. bovis can also infect and cause tuberculosis in humans (3% of TB cases, 0.5% in immunocompetent patients). The genome sequence of M. bovis has more of a 99.95% overlap with M. tuberculosis, so the disease caused by M. bovis in humans is virtually indistinguishable from that caused by M. tuberculosis, both clinically and histologically. In the case of M. bovis is recommended to treat the infection with four TB: isoniazid, rifampicin, streptomycin and ethambutol. It is advisable to carry out sensitivity studies with the aim of identifying the strain, and to exclude resistance.

SC-15 FAILING THE FALLEN

Clinical case chosen for the presentation at the Young Internists' Day-Clinical Gymnasium

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Introduction: We present the inpatient journey of an elderly frail 'faller' to demonstrate the complexities of an otherwise simple 'fall'.

Description: A 72 year old Caucasian lady presented to the acute medical admissions unit (MAU) having sustained a fall from her bed at her residence and was found to be confused. She had a background of Rheumatoid Arthritis (RA), chronic kidney disease, Hypertension, Bronchiectasis, Hypothyroidism and total knee replacement. She was a non-smoker and lived in a house with her husband who looked after her. Functionally she was dependent for most activities of daily living (ADL) and had limited mobility with the help of a zimmer frame (ZF). Clinically she was confused, but her observations were stable and systemic examination was unremarkable apart from a few bi-basal crepitations, and stigmata of RA. Neurological examination was grossly normal with the ability to move all four limbs. Initial blood tests and preliminary chest X-ray suggested a diagnosis of mild delirium secondary to lower respiratory tract infection (LRTI) on the background of bronchiectasis. To elucidate the cause of confusion and given the history of fall, a computerised axial tomography (CAT) scan of the head was done which revealed a right parietal infarct. She was transferred to



the stroke unit and further clinical evaluation on day 10 revealed motor weakness of all four limbs - both upper limbs (grade 0/5) and both lower limbs (grade 3/5). The deep tendon reflexes were exaggerated in all four limbs with bilaterally extensor plantars, although sensation was intact. A magnetic resonance imaging (MRI) of the cervical spine revealed superior migration of C2 vertebrae into the foramen magnum consistent with basilar invagination resulting in severe spinal canal stenosis at the level of foramen magnum. Further examination (and X-rays) on subsequent days revealed a fracture of the shaft of the right humerus. The findings were discussed with the local tertiary referral centre and imagings were obtained to exclude a pathological fracture. This entailed another CAT scan of the chest, abdomen and pelvis which revealed bilateral pulmonary embolisms (PE). An inferior vena caval filter was inserted given that she had an episode of melaena. After detailed discussions the consensus opinion was to undergo an operation in the future when stable.

Discussion: Despite little hope of significant neurological recovery even after surgery, she is keen and optimistic about her future.

SC-16 THREE CAUSES OF ANEMIA: A SINGLE CASE

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Case report: A seventy years old woman, with hypertension, mitral and aortic valve fibrocalcification, kidney stones, depression and degenerative osteoarthritis was referred by her General Practitioner for anaemia 7.4g/dL on routine analysis. Previous admission two months before revealed iron deficiency anaemia, possibly associated with non steroid anti-inflammatory drugs. Upper endoscopy revealed peptic ulcers and hiatus hernia. She was discharged medicated with omeprazol and iron. Present admission complained of easy fatigue, anorexia and weight loss (about 12kgs in 6 months). She had no visible blood losses, although she did have very dark faeces possibly related with oral iron intake. On examination, she presented with discolored skin and mucous membranes. Nasogastric intubation provided no additional information. Blood work showed: Hb 6.9g/dL, MCV 64,9fL, HGM 18,6pg. She was admitted after receiving a pack of red blood cells (Hb 8.8g/dL). Biopsies from a previous upper endoscopy were positive for *H. pylori* infection and eradication therapy was performed accordingly. Additional laboratory tests showed 2.0% reticulocytes, iron 177ug/dL, transferrin saturation 44%, total iron binding capacity 406ug/dL, normal cobalamin and folic acid levels, protein electrophoresis with a $\beta 2$ peak (2.59 g /dL = 35%). Immunofixation showed elevated IgG and K, and no Bence Jones proteinuria. Tumor markers (α FP, CEA, CA 19-9, CA 125 and CA 15-3) were within normal range. Second upper endoscopy revealed ulcer healing and colonoscopy revealed diverticulosis and caecum neoplasia. She was submitted to a right hemicolectomy. Subsequently an immunoelectrophoresis was performed showing a monoclonal peak IgG K (2,7g/dL and 2,89g/dL respectively) as well as restriction of IgA (0,035g/dL) and λ (0,164g/dL). Bone marrow biopsy revealed 11% plasmocytes suggesting the diagnosis of multiple myeloma IgG K. The patient was therefore referred to an Haemathologist for further therapy and follow up. This case report is an example of how multiple factors can contribute to the development of anaemia and how important is it's investigation.

SC-17 RADIOLOGY AS A FRIEND. ADENOPATHIES RESEARCH IN A 57-YEAR-OLD MAN

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Reason for consulting: adenopathies research.

Personal data: 40 pack years smoking history. Hypercholesterolemia. Prostate adenoma (benign prostate hyperplasia). Rectal adenocarcinoma pT1 N0 (Rectal low anterior resection in July 2011). Treatment: dutasteride/tamsulosin 0.4/0.5 mg, atorvastatin 10 mg/d.

Clinical history: 57-year-old man, asymptomatic. No peripheral lymphadenopathies. No temperature. No perspiration. No pruritus. Control CT scan (August 2011): multiple hilar and mediastinal adenopathies with millimetric lung nodules; peripancreatic, celiac axis and liver hilum adenopathies. Normal bronchoscopy. Mediastinal adenopathies Fine Needle Aspiration Cytology (FNAC) showed compact agrupations of epithelioid histiocytes in granulomatous pattern without necrosis.

Physical examination: Height: 1.64 m. Weight: 75 Kg. BMI: 28 Kg/m². Blood pressure: 116/76 mmHg. Heart rate: 65 bpm. Respiratory rate: 16 bpm. Blood oxygen saturation 97% (FiO₂ 0.21). No adenopathies, no skin lesions. Normal cardiopulmonary examination. No mass in abdomen. Normal sized liver and spleen. No alterations in the rectum examination. Normal joints. No peripheral edema.

Tests: Blood count: WBC 5860/ μ L (N 3090, L 1760), Hg 13.5 g/dL, MCV 88 fL, platelets 262.000/ μ LESR: 9 mm (0-13). Coagulation test: normal. Chemistry: urea 62 mg/dL (21-50), creatinine 1.3 mg/dL (0.6-1.2), glucemia, Na, K, Cl, uric acid, albumin, proteins, Ca, P, HCO₃, ALT (GPT), AST (GOT), GGT, alkaline phosphatase, LDH, bilirubin, total cholesterol, HDL-C, LDL-C, triglycerides and TSH normal. PSA: 1.61 ng/ml (0.0-4.0). ACE: 67 U/L (20-70). Beta-2 microglobulin: 3.29 mg/L (1.1-2.5). Urine test: normal. Mantoux: negative (< 5 mm). Serology: RPR (*T. pallidum*) -. *Coxiella*. Chest X-ray: bilateral hilar adenopathies, right ribs fractures. Scintigraphy (Gallium): normal. Positron emission tomography (PET): high concentration of the radiotracer in mediastinal and abdominal lymph nodes (especially subcarinal and liver hilum), 4th right rib and millimetric lung nodules. CT- Guided Biopsy: technically impossible. Peripheral blood flow cytometry: normal. High NK cells (CD56+). Bone marrow biopsy: normal. BAL: lymphocytes 47.27 %, macrophages 37.90 %, neutrophil granulocytes 8.19 %. CD4/CD8 increased.

Radiological differential diagnosis: Unsharp interstitial nodules are found in sarcoidosis and subacute phase of hypersensitivity pneumonitis. Perilymphatic lung nodules are subpleural in location and tend to be nonuniform and patchy in distribution. They are most frequently found in sarcoidosis, lymphangitic carcinomatosis, and silicosis. Upper parts of the lungs are typically involved in granulomatous diseases (eg, sarcoidosis and hypersensitivity pneumonitis), Langerhans cell histiocytosis, and pneumoconioses, such as silicosis and coal workers' pneumoconiosis, but not asbestosis. The combination of right paratracheal and bilateral hilar lymph node enlargement in chest X-ray is often considered a reliable sign of sarcoidosis and has been called "1-2-3 sign" or "pawnbroker's sign".

Definitive tests: Videomediastinoscopy and biopsy of mediastinal adenopathies. Results of Anatomical Pathology: noncaseating granulomatous lymphadenitis, consistent with SARCOIDOSIS.

Diagnosis: Sarcoidosis stage II (bilateral hilar adenopathies and lung nodules).

Evolution: Acute mediastinitis (*Streptococcus pyogenes*) 5 days after surgery. He entered the operating room and received intravenous penicillin + clindamycin for one month. He was discharged and continued oral antibiotics (amoxicillin plus clavulanic acid). No other drugs were prescribed. 5 months later he was asymptomatic: No dyspnea, no temperature, no weakness, no anorexia. Weight gain. Normal cardiopulmonary examination. Respiratory rate: 16 bpm. Blood oxygen saturation: 96% (FiO₂ 0.21). Chemistry (April 2012): Creatinine 1.2, Urea 58, Calcium 9.9, LDH 276, ACE 82 U/L (20-70). Calciuria 171 mg/24 h (100-300). Beta-2 microglobulin 3.2. Fundoscopy: normal. Pulmonary function test: pending test. Chest X-ray: decrease of right paratracheal node enlargement. Persistent node bilateral hilar enlargement. Persistent major fissure enlargement. CT scan: stability of thorax and abdominal adenopathies and lung nodules.

SC-18 PARANEOPLASTIC MUSCULAR WEAKNESS

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Introduction: The lung tumours have been associated with paraneoplastic syndromes. We present a patient with microscopic polyangiitis vasculitis.

Clinical case: We report a 68 year old man who was admitted to the hospital because of myalgias. He was smoker and he was diagnosed of a small cell lung cancer 18 years ago. He has had revisions without evidence of disease until 2003. A month before admission the patient began with a progressive weakness predominantly in lower extremities, progressive muscular pain, difficulty in getting up and walking. On examination we confirmed proximal muscle weakness in lower extremities. In complementary tests he had anemia, the erythrocyte sedimentation rate was 91 mm and creatinine kinase was normal. Autoimmunity was positive for P-ANCA with title of 1/1280. The chest radiograph showed a reticular pattern in bases and a mediastinal widening. The electromyogram was normal and quadriceps muscle biopsy showed signs of small vessel vasculitis. Because of the patient's personal history, we request a chest computed tomography, which reported a thickening of bronchial wall. The bronchoscopy reported a neof ormation in carina whose biopsy was compatible with carcinoma composed of small cell carcinoma and in situ invasive squamous carcinoma. After the diagnosis of microscopic polyangiitis vasculitis (PAM) as paraneoplastic syndrome secondary to pulmonary neoplasm, a 1mg/kg/day prednisone and chemotherapy was established. After 4 cycles there was a clear hepatic and pulmonary progression, with exacerbation of muscular symptoms unresponsive to an increase in steroid dose. The patient died 11 months after diagnosis.

Discussion: Lung cancer is usually associated with paraneoplastic syndromes, less common with vasculitic manifestations, normally diagnosed in patients with haematological malignancies. Out of all the types of that vasculitis, MAP is one of the rarest. The symptoms may appear before the diagnosis of neoplasia. However, due to the patient's history, the high suspicion and a very closely monitoring it was possible to detect a neoplasm. Clinical involvement is generally focused on the kidney and lung, but these weren't observed in our patient. They may also be muscular symptoms. A review of the evidence, show no more than twenty cases in patients with solid tumors. Vasculitis evolution is related to the neoplasm course. Sometimes it is necessary to use immunosuppressant, although, in the case of cancer patients this is controversial. Up to 80% of the clinical symptoms are resolved when cancer is radically treated. In any case, it is necessary to think of a recurrence if the clinical vasculitis reappears. Because of this association, especially in older patients, we recommend screening for occult malignancy when response to conventional therapy is not adequate.



SC-19 MILIARY TUBERCULOSIS IN ONE PATIENT TREATED WITH INSTILLATIONS OF BACILLUS CALMETTE-GUERIN (BCG)

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Medical record: Male 77 years old. Bladder tumor and type cT1-G3 bladder paraganglioma treated by transurethral resection and instillations with BCG without evidence of recurrence. Two weeks after last BCG instillation, consulted with symptoms of malaise, weakness, fever, sweating at night and nonproductive cough.

Physical examination: Extremely uncomfortable, Blood Pressure: 145/70, Pulse: 112 bpm, Temperature 39°C, eupneic. Minimal crackles right lung base.

Diagnostic tests: Laboratory tests highlights creatinine 1.4 mg/dL, AST 80 IU/L, ALT 67 IU/L, GGT 729 IU/L, LDH 307 IU/L, ALP 358 IU/L, CRP 81 mg/L. Chest radiograph showed miliary pattern. Abdominal ultrasound showed calcified granulomas in the right hepatic lobe. Mantoux test was positive (11 mm). Blood and urine samples were negative for mycobacterial and bacterial culture. CT scan showed a diffuse bilateral micronodular pulmonary pattern. Transbronchial biopsy were negative.

Diferential diagnosis: Miliary pattern on chest radiograph should make think in mycobacterial infection, less frequent are septic emboli, hydatidosis and histoplasmosis. Noninfectious etiologies can be metastases, pulmonary infarction, sarcoidosis, rheumatoid nodules, Wegener disease and pneumoconiosis.

Evolution: Miliary tuberculosis and granulomatous hepatitis by *M. bovis* was suspected, starting tuberculostatic drugs, with improvement in general condition, and liver function. Evaluations at months 2 and 5 after discharge, showed normalization of transaminases and CRP, with slight improvement in miliary pattern in CT scan after 6 months.

Discussion: Systemic complications are fever and less frequent rash, granulomatous pneumonitis, hepatitis, arthralgia, renal abscess, cytopenia and sepsis. Samples should be obtained for staining for acid-fast bacilli, culture, and PCR testing for mycobacterial DNA when disseminated BCG infection is suspected. Therapy may be started when clinical suspicion is high. Antituberculous drugs should be used when acute severe symptoms or those that persist beyond 48 hours. When symptoms of moderate to severe cystitis a fluorquinolone or isoniazid should be used. If symptoms progress or fail to resolve up to 2 weeks, isoniazid should be continued or substituted and rifampin may be added. In disseminated infection, should be isoniazid, rifampin and ethambutol for the first 2 months, followed by 7 months of isoniazid and rifampin, as *M. bovis* is inherently resistant to pyrazinamide. Corticosteroids therapy is recommended. Instillations with BCG should be discontinued.

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SC-20 WOMEN 50 YEARS OF BLOOD AND BLUE LIPS

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Case report: Female patient, 50 years of age without allergies Known to medicines, posing as a personal history: Complex Congenital Heart Cardiopathy: single left ventricle with transposition of great vessels and hypoplasia of the pulmonary artery with congenital atrioventricular block, that needs bleedings of 300 ml, with a frequency of approximately 1month for polyglobulia importantly He is in treatment with torasemide and allopurinol. His basal situation is IABVD. School teacher until 2 years ago. 2 pillow orthopnea. He lives with his mother, with great family support. Admitted to our hospital from where comes the urge to present progressive increase in dyspnea usual to be done in the past few days with minimum effort and in the last 24 hours standby. Coinciding with this symptomatology increased edema in the lower limbs to ankles and abdominal distension. On physical examination performed on admission include a saturation of 36% with cyanosis of mucous important, a midsystolic murmur tricuspid and pulmonary III / VI with increase of 2 ° pitch, an increase of PVY, clubbing and edema in legs to ankles. Laboratory tests on admission are objectively a metabolic acidosis, increased creatinine with respect to previous probable cause azotemia and decreased total protein and albumin. The ECG shows a lock Complete AV and Plung. The echocardiogram confirmed the presence of left single ventricle with pulmonary hypoplasia transarterial pulmonary gradient of 117 mm Hg. While in rose plant is diuretic dose, you hydrated with fluid therapy and the albumin was recovered with protein intakes. He did notspecify oxygen. Today is stable, walks down the aisle without dyspnea,

transpulmonary gradient decreased with respect to income and remains hospitalized pending completion of Magnetic Resonance to assess cardiac status and pulmonary artery surgery and to ask that you lengthen the survival.

Discussion: All these people with single ventricle lacking half of the heart so that the left ventricular constitutes a single ventricle must pump blood both to deoxygenated as yes it is, mixing both in the cavity. Part of the group of cyanotic congenital heart disease and those are few cases that reach adulthood even with surgical procedures performed even in childhood, but even more rare, not reaching the ten worldwide, as our cases patient reach 50 with a more or less active without having been involved. This heart is characterized by cyanosis increases include reduced hemoglobin, crisis hypoxaemic sudden decrease in pulmonary flow, clubbing and polycythemia that is because the kidney to detect decrease in oxygen saturation that releases EPO stimulates the bone marrow and thus increased red blood cells. The drawback of this increase in red blood cells is the increase in viscosity that can have devastating consequences for the patient as strokes and brain abscesses caused by microemboli that can become infected.

SC-21 GRANULOMATOUS PERITONITIS SECONDARY TO A RUPTURE OF TERATOMA IN A PREGNANT WOMAN

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Objective: To describe an unusual case of granulomatous peritonitis reaction mimicking peritoneal carcinomatosis or tuberculosis due to the rupture of an ovarian teratoma in a pregnant woman.

Case report: A 36 years-old pregnant women at 11 weeks gestation, presented to the emergency department with a sudden severe abdominal pain located in her low right quadrant. A bedside ultrasonography showed anechogenic mass in her right ovary suggesting a torsion of corpus luteum cyst. An urgent laparoscopic exeresis of the ovarian cyst was performed. Unfortunately during the procedure, the cyst was ruptured, so the peritoneal cavity was contaminated with sebum and hairs. Meticulously, an intensive washing of peritoneal cavity was done. Pathologic examination showed an ovarian mature cystic teratoma. After four days, she was admitted again to hospital because she presented abdominal pain and elevated fever. Despite of being treated with intravenous wide range antibiotics, she didn't improve. So two weeks after her first operation, she was re-laparotomized and the exploration of peritoneal cavity revealed purulent material with rest of teratoma content. Bacteriological cultures were negative. At 14 weeks of gestation, as a result of possible damages over the fetus, the patient decided to interrupt her pregnancy voluntarily. Two weeks latter, the patient developed again abdominal distension, weight loss and fever. Inflammatory markers persisted elevated and microbiologic samples were negative. An abdominal computed tomography (TC) scan showed marked thickening of the omentum, peritoneum and mesentery. Histology of previous specimen confirmed granulomatous peritonitis without malignancy or tubercle bacilli. Therapy with prednisone was started and the patient's condition improved.

Conclusion: Cases of granulomatous peritonitis has been reported as a consequence of infections, malignancy and foreign body reactions. Although a rupture of a dermoid cyst is a rare complication, it could be added to the list of causes of granulomatous peritonitis.

SC-22 IMPAIRED MENTAL STATUS AS A CLINICAL PRESENTATION OF HYPOPHOSPHATEMIA

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Case report: A 64 year old woman presented to the emergency department with ten days history of diarrhea associated with fever and cramping diffuse abdominal pain. She had a medical history of low malignant stromal mucosecretor adenocarcinoma infiltrating behind the rectum, four months ago. The patient underwent surgery and one month later started chemotherapy with xeloda and after finishing it, she received adjuvant radiotherapy. On physical examination, the patient was awake and oriented, her vital signs were BP 155/95, T° 36,5°C, HR 96 and a RR 16. She had moderate dehydration. Her abdomen was soft but minimally tender and her bowel sounds were increased. Laboratory reveals a hemoglobin of 14.9 g/dl, low WBC count of $1.1 \times 10^3/\mu\text{L}$ with neutropenia of $7 \times 10^3/\mu\text{L}$. Na 125 meq/l, K 3 meq/l, Ca 10 mg/l. The rest of basic chemistry panel were unremarkable. She was admitted and underwent a MRI of the pelvis which ruled out a fistula. On day 3 of admission, she continued having watery loose stools. She had become increasingly sleepy, lethargic and confused; she appeared dehydrated and his abdomen becoming progressively distended. She underwent an upper and lower GI series seeing a dilated small bowel with no passage of barium to the large bowel, mechanical obstruction were ruled out, being diagnosed of paralytic ileus. Next day, the patient worsened her mental status, presented with more lethargy, sleepiness and disorientation with poor answer, becoming stuporous in a few hours. RBC and WBC count was within normal range, a



normal Na and K with high Mg of 3.3 mg/dl, phosphate (P) 1.3 mg/dl, corrected Ca 8.2 mg/dl, total proteins 3.6 g/dl, albumin 1.8, total cholesterol 34 mg/dl. Other test showed a PTH level of 557 pg/ml, urine Ca of 10, 24 hour urine Ca of 270 (normal range), punctual urine P 3.5, 24 hour urine P of 94.5 and a vitamin D level of 20.7 nmol/l. The patient was diagnosed of hypophosphatemia and vitamin D deficiency with secondary hyperparathyroidism due to malabsorption syndrome because of severe diarrhea and paralytic ileus, so a nasogastric tube was placed and started central parenteral nutrition with supplemental phosphates, oral vitamin D and intravenously sodium monophosphate. One day after that, the patient improved her mental status. Her paralytic ileus presented complete resolution and on the next day she started oral food intake. One week later, the patient almost had complete recovery so we performed a new laboratory test with a P of 3.6 mg/dl, corrected Ca 8.8 mg/dl and PTH of 66 pg/ml. Due to the good evolution, the patient was discharged and then controlled one month later in the outpatient. She was completely recover with normal physical exam and her laboratory test showed a normal P 4.1 mg/dl and PTH of 26 pg/ml.

SC-23 MALE PATIENT WITH ACUTE RESPIRATORY FAILURE AND MULTIPLE LUNG NODULES

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Case report: A 20 year-old-man was referred from his reference hospital because of fever, cough and dyspnea of sudden onset accompanied by severe hypoxemia and bilateral nodular lung infiltrates on chest Rx. He had not relevant medical history until six months before admission, when he begins to show signs of asthenia and an unquantified weight loss due to, seemingly, excessive work and an unbalanced diet. He asked his primary care physician, the sole finding being iron deficiency anemia, so iron supplementation was started. A blister accompanied by showy inflammatory signs appeared in his right foot two months later. Treatment with NSAID and oral amoxicillin-clavulanate was started. The response was poor requiring hospital admission because of right lower extremity cellulitis. Cutaneous infection resolved after administration of iv antibiotic therapy, however, low-grade-fevers persisted with the occasional appearance of high fever peaks associated to episodes of profuse sweating. The involuntary weight loss continued. On arrival at our hospital, physical examination revealed pallor, tachycardia, tachypnea and profuse sweating. The temperature was 39°C. Blood pressure, heart and respiratory rates, were 120/60 mm Hg, 130 bpm and 24 rpm respectively. Lung auscultation showed bilateral crepitant rales. He presented a striking hepato- splenomegaly without peripheral lymph nodes at any level. The main blood test results were: Hb 11 g/dL, ferritin 1535 ng/mL, 4700 leukocytes, LDH 413, plasma albumin 2.6, total protein 3.7, PCR 7.5 mg / dL, ESR 8 mm / h. Arterial blood gas (FiO₂ 1.0) showed pH 7.45, pCO₂ 33.8 mm Hg, pO₂ 47.5 mm Hg, HCO₃ 24.3 mmol / L. Broad-spectrum antibiotherapy and hemodynamic and respiratory support were started. CT of chest and abdomen revealed multiple bilateral nodules, homogeneous hepatomegaly and a significant splenomegaly. The retroperitoneal area was occupied by bulky adenopathic conglomerates, so, a biopsy was done. The echocardiogram was normal. Immunological and microbiological (virus, fungi, bacteria, M. Tuberculosis) tests were all negative. Blood immunoglobulins values were IgG 65, IgA 25 and IgM <20 consistent with Common Variable immunodeficiency. Replacement with i.v Ig was started. The patient developed progressive respiratory distress requiring mechanical ventilation. Two little subcutaneous nodules on the anterior abdominal wall, not present on admission, were also biopsied. Morphological and immunohistochemical studies of retroperitoneal adenopathy and skin biopsy showed large groups of phenotype B lymphoid cells with a high proliferative index and a pattern of angiocentric affectation with large areas of necrosis consistent with Lymphomatoid Granulomatosis. Studies of bone marrow were normal. Treatment with CHOP-R was started but the patient suffered severe neutropenia complicated by sepsis due to *P. aeruginosa* and died finally in ICU.

SC-24 IDIOPATHIC SYSTEMIC CAPILLARY LEAK SYNDROME: A RARE IDENTITY WITH IMPORTANT CLINICAL IMPLICANCE

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Case report: 63 year old woman with multiple episodes of shock with generalized edema. She was admitted to the hospital with circulatory shock in January 2010, and followed until she died in December 2011. She has a previous long history of non specific pre-syncope, studied in internal medicine, neurology, cardiology and allergy with uncertain diagnosis, in all cases vagal etiology was considered. She had a monoclonal gammopathy of uncertain significance. She reported repeated episodes of conscience loss, preceded by dizziness, with spontaneous recovery. Her symptoms had a very abrupt onset. When she was first examined she presents with abnormal consciousness and hypotension, tachycardia, and low oxygen saturation. The only physical examination

finding was generalized edema. It was performed blood test, Chest X-ray and ECG. Chest X-ray was normal. ECG: tachycardia sinus, without other findings. Laboratory finding: Hemoglobin > 20 g/dl, total leukocyte count > 40.000/ml, platelet count > 300.00/ml, serum creatinine > 2 mg/dl, normal serum ions and hypoalbuminemia. She was diagnosed of sepsis. She had a complete fast recover with intense intravenous fluids administration. The edema was exacerbated by fluid resuscitation. Blood test, confirmed a monoclonal protein IgG kappa. No other biochemical or hormonal abnormality. Abdominal ultrasonography, Body-scan, Echocardiogram and upper endoscopy were normal. Second admission was in May 2011, with the same symptoms. At this point the differential diagnosis included sepsis, unusual para neoplastic syndromes, systemic mastocytosis and carcinoid tumors. Blood, urine and pleural fluid for bacteria, fungi and mycobacterium were sterile. Serological tests for HIV 1&2, leptospira, dengue and rickettsiae were negative. ANA, ANCA, Anti-GBM were negative, and levels of C3 and C1 esterase inhibitor levels were normal. IgE showed no alteration. Serum cortisol and response to ACTH stimulation were normal. Plasma levels of lactate, pyruvate and CPK were normal. Serum tryptase was not elevated. Bone marrow biopsy was normal. A diagnosis of idiopathic systemic capillary leak syndrome (Clarkson's disease) was made, it is a ultimately a diagnosis of exclusion that is made when a patient manifests with one or more episodes of intravascular hypovolemia, generalized edema, and the diagnostic triad (hypotension, hemoconcentration, hypoalbuminemia) in the absence of an identifiable alternative cause. The keys for the diagnosis in this case were the exacerbated edema after treatment and the absence of other diagnosis; a monoclonal gammopathy strongly supports the suspicion. A second tomography was performed, and showed a pancreatic tumor, it is often a paraneoplastic syndrome; SCLS diagnosis implies exhaustive study for a possible hidden neoplasia.

SC-25 CEFALOTHORACIC ACQUIRED PARTIAL LIPODYSTROPHY: BARRAQUER-SIMONS SYNDROME

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Introduction: Acquired partial lipodystrophy (APL) is a chronic progressive condition in which adipocytes lyse in several characteristic body areas, leading to increase of unaffected fat ('lipohypertrophy'). A female predominance exists (3:1), and usually a family history is lacking. Complement alternative pathways have been advocated in the fat loss. No specific treatment exists.

Material and method: A case of a 43-year-old man was remitted to an Internal Medicine office because of arthralgias and bone radiologic abnormalities since 6 years ago. One sister had a mesangial proliferative glomerulonephritis which had progressed to end-stage chronic kidney disease in haemodialysis. In his medical record, a gouty arthritis and three resections of 'lipomas' in the deltoid and upper back had been elicited. The patient referred a progressive increase of volume of upper part of arms, buttocks and thighs from adolescence, that had been more evident after he augmented weight of 6 kg. in the previous months. He had mechanic type arthralgias in hips and knees mainly after walking. Physical examination revealed absent facial, anterior chest and abdominal subcutaneous fat, thin forearms and increase of buttocks, thighs and upper arms fat. Audition was normal. No synovitis was present. General biochemical and haematological analyses showed an elevated GGT (82 U/L) and urate level of 11.6 mg/dl. Reactive phase proteins were slightly elevated (CRP 7.3 mg/L). Lipid profile, complement C3 and C4 levels, hypothalamic-hypophysary axis hormones, somatomedin C, basal insulinemia, growth hormone, ACTH and 24 hour urine cortisol, hydroxyproline, piridinoline, deoxypiridinoline, calcium and phosphate were normal. HIV 1 and 2 serology were negative. Antinuclear antibodies, reumatoid factor, anti-neutrophil citoplasmic antibodies, antiphospholipid antibodies and C3NeF were repeatedly negative. An abdominal ecography revealed a diffuse liver steatosis. Bone scintigraphy showed signs of diffuse costovertebral hyperostosis.

Results: Medical personal and familial history, physical examination and complementary tests allowed the diagnosis of an acquired lipodystrophy, subtype cephalothoracic (Barraquer-Simons syndrome), with no associated autoimmune or metabolic disorders nor nephropathy. Repeated C3NeF, complement levels, autoantibodies and rheumatoid factor were negative during a 4 year follow-up. Diet treatment and exercise were recommended, with a 14 kg. of weigh loss and improvement of his arthralgias.

Conclusions: Lipoatrophy of the face, upper part of trunk and arms are the main features of this type of partial lipodystrophy and absence of familial history makes the differential diagnosis with generalised lipodystrophy and partial hereditary Köbberling-Dunnigan very easy. Symptoms usually begin during childhood or adolescence, but may begin as late at the 3rd or 4th decades of life. It may be associated to insuline resistance, systemic autoimmune diseases and mesangiocapillary glomerulonephritis associated to the presence of nephritic fator 3 IgG (C3NeF) and low C3 levels. This severe nephropathy may appear one decade after the debut of symptoms, and determination of C3NeF helps to early diagnosis. Treatment consists of physical exercise, antihyperglycemic medication directed to insulin resistance (whenever indicated), and cosmetic therapy.



SC-26 A 16 YEAR-OLD MALE WITH MOYA-MOYA DISEASE

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Case report: A 15 year-old male was admitted into hospital due to difficulty on language articulation and worsening of right hemiparesis he suffered since he was a child. His psychomotor development had been normal till the age of six, when right hemiparesis appeared suddenly, without study in his country, Bolivia. Because of this, he was seen in Paediatrics at his arrival, finding in a MRI right parietal cortico-subcortical ischemic changes. He had been asymptomatic until this new episode. At this admission a complete study was performed. An urgent CT showed a residual area of hypodensity in the right parietal zone. Blood test, thrombophilia study, anti-nuclear and anti-double stranded DNA antibodies, EKG, thorax x-rays... were normal. In the MRI there was a small left parietal acute ischemic lesion, the previous great right parietal ischemic sequelae and multiple small ones in both hemispheres. Brachiocephalic and brain angioRMI with gadolinium showed progressive narrowing of the cerebral arteries at the base of the brain involving the intracerebral portion of the internal carotid causing a "cigarette smoke" aspect. These findings suggested the diagnosis of Moya-Moya disease. A year later he suffered a new ischemic event and at this admission he was derived to our reference hospital for a neurosurgical revascularisation. Despite this, a month after it he suffered a new ischemic event, with no sequelae. Since then, he has been asymptomatic (6 months).

Discussion: Moyamoya disease is produced by the spontaneous, progressive occlusion of the circle of Willis with the simultaneous appearance of natural intracranial and extra-intracranial collaterals. Both children and adults can be affected and it is to be found not only in Asia but also in Europe and America, although the frequency is unknown. Its primary lesion is a thickening of the tunica interna in the distal segment of the internal carotids, initially within the anterior portion of the circle of Willis. This results in critical reduction in regional cerebral blood flow. The most common clinical manifestation is motor-type ischemic episodes, although it may also be accompanied by intracerebral haemorrhages. It can be diagnosed by means of suitable morphological and functional studies, such as cerebral pan angiography and xenon-CT, in patients with a founded clinical suspicion. Neurosurgical revascularisation is to date the most widely accepted treatment and can bring about a permanent clinical cure.

SC-27 TUMOR NECROSIS FACTOR- ALPHA ANTAGONISTS AND DEMYELINATING DISEASE

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Case report: A 55-year-old woman with long lasting rheumatoid arthritis, treated with low-dose methotrexate for over 13 years, had an increased activity of her RA that was not controlled by therapy with high dose MTX, leflunomide, and low dose prednisone. Therapy with Adamilumab, plus low dose MTX was commenced on November 2008, with excellent clinical response. By April 2009, the patient presented with a wide base ataxia, hyperreflexia, and lost of vibratory and proprioceptive sensation in the lower limbs, with bilateral plantar extensor responses. Adamilumab was discontinued. Levels of B12 vitamin, folates, complement, CRP, rheumatoid factor, tumor markers, anti-nuclear and anti-double stranded DNA antibodies were all normal. The biochemical and cytological characteristics of the CSF were normal, and the PCR for tuberculosis and JC virus were negative. Magnetic resonance of the spine showed diffuse involvement from the upper cervical levels to the first thoracic level. Hyperintense flair signals with gadolinium enhancement were observed in the posterior part of the spine from C7 to T1. Other lesions were observed in the brain, some with enhancement too. Visual evoked potenciales were negative. Treatment with high dose metilprednisolone was commenced, with short term clinical improvement. Six months later, the MR showed the lack of gadolinium enhancement, and the disappearance of some of the lesions.

Discussion: Demyelination is one of the potential side-effects of the therapy with TNF α antagonists. The mechanisms by which therapy with anti-TNF α promotes or unmasks a previously unknown demyelinating disease have not been established. RA is associated with many autoimmune diseases. Multiple sclerosis and RA share common pathogenic pathways. High levels of TNF α had been observed in the CSF of patients with MS. In chronic progressive MS, CSF levels of TNF α correlate well with disability and the rate of neurological deterioration. These observations suggested that, patients with MS may benefit from anti-TNF α therapy. Nevertheless, an update from the US Food and Drug Administration on patients receiving etanercept therapy, observed an incidence of demyelinating disease of 31 per 100000 patient-years of exposure, compared to 4 to 6 per 100000 per year for the general population. It has been speculated that, anti-TNF α treatment unmasks previously existing multiple sclerosis in patients with RA and a genetic propensity to enhanced demyelination by anti-TNF.

SC-28 PANCREATIC MASS WITH LIVER METASTASIS

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Introduction: Neuroendocrine tumors (NET) are a heterogeneous group of neoplasms. They are originated in neuroendocrine cells, with an annual incidence around 5/100,000 cases.

Clinical case: We report a 49 year old man who was admitted to internal medicine service to study an abdominal mass. He was ex-smoker. He had not standard treatment. A six weeks before admission, the patient began with a history of anorexia, fatigue, tendency to constipation and abdominal pain. He lost 5 kg of weight. He went to his primary care center, where abdominal ultrasound showed a big epigastric mass that includes pancreas, with multiple hepatic lesions suggestive of metastases. On examination, the patient was hypertensive and afebrile. On his chest, we could observe an erythematous rash. Abdomen was depressible, with large painful mass in the epigastrium and left hypochondrium. All other exploration was normal. In complementary tests: blood count and coagulation was normal. In biochemistry: alkaline phosphatase of 342 UI/L, calcium 15.3 mg/100, ferritin 552 ng/ml, enolase 18.4 mcg/L and beta 2 microglobulina which was 4.48 mg/L. The rest of parameters were normal. We found a Chromogranin A 429 ng/ml, with acid vanilmandelic, catecolaminas and 5-hidroxy-indolacetic normal in urine analysis. The rest hormonal study was normal. Abdominal. Computed tomography showed a big mass (13x10x10cm), located between the stomach and pancreatic body, unable to define their origin. The mass infiltrated splenic artery. Multiple hepatic lesions were suggestive of metastases. Gastroscopia indicated an antrum compression, with normal biopsy. The pancreatic nuclear magnetic resonance (MRI) showed a bulky mass that completely includes body and tail of pancreas. Stomach and splenic artery were infiltrated and there was a thrombosis of splenic vein. There were multiple hepatic lesions suggestive of metastasis. In the mass biopsy, pathologist informed as a low-grade neuroendocrine tumor, and strikingly express receptors for synaptophysin. After diagnosis of neuroendocrine tumor and paraneoplastic hypercalcemia, the patient underwent surgery, with pancreatoduodenectomy and splenectomy. Surgical margin was free of tumor. After surgery, liver metastases were treatment with chemoembolization. In this moment, the patient received an octreotide treatment, with favourable evolution.

Discussion: Neuroendocrine tumors include gastroenteropancreatic endocrine tumors (TEGEP), neuroendocrine tumors of unknown primary catecholamine-secreting tumors (pheochromocytoma, paraganglioma, ganglioneuroma, ganglioneuroblastoma, simpatoblastoma, neuroblastoma), medullary thyroid carcinoma, tumors adenohypophyseal, pulmonary neuroendocrine tumors and Merkel cell tumor. TEGEP are the most common. These are usually classified in two groups: pancreatic neuroendocrine tumors (NPT) and carcinoid tumors (CT). Diagnosis can be done with high-resolution MRI, with isotopic studies, ultrasound-endoscopy and hormonal tests. Liver is the most common site of NET spread. The best treatment if it is possible is surgery with chemotherapy and ablative action on liver dominant lesions with radiofrequency, radio-surgery and chemoembolization. The hormonal treatment achieved effective symptoms palliation and increased of total survival. Liver transplantation is considered in special situations where the primary lesion was resected, if the patient has a dominant liver disease or symptoms caused by tumor mass or hormone syndrome.

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SC-29 ACUTE MONOBLASTIC LEUKEMIA WITH SKIN INVOLVEMENT AS SKIN INFILTRATION

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Context: Acute myeloid leukemia presents skin involvement up to 13% of cases, being more often if there is a monocytic or myelomonocytic component. We present a case report of a male patient with acute monoblastic leukemia that presented with pancytopenia and multiple skin lesions.

Case report: An eighty-year-old male patient was admitted for a 2 month clinical history of undetermined weight loss, rectal bleeding and fatigue. He had a previous medical history of past heavy smoker, well-controlled type 2 diabetes, hypertension and non-small-cell lung carcinoma stage Ib treated with lobectomy and adjuvant chemotherapy with no evidence of disease up to the date. Physical examination revealed papular erythematous rash affecting thorax and both upper and lower limbs. The laboratory evaluation showed pancytopenia with $2000 \times 10^3/\mu\text{L}$ leucocytes, 8.5gr/dL of haemoglobin and $64.000 \times 10^3/\mu\text{L}$ platelets, a mean corpuscular volume of 98 fL and 1 erythroblast per 100 cells. The bone marrow examination was consistent with the diagnosis of acute monoblastic leukemia with massive invasion of the bone marrow. The histopathologic examination of the skin lesion biopsy revealed skin infiltration by monoblasts. The patients received 1 cycle of chemotherapy developing a severe pancytopenia complicated with urinary sepsis resulting in death 2 weeks after the diagnosis was made.



SC-30 WALDENSTRÖM'S MACROGLOBULINEMIA PRESENTING AS DYSPHAGIA

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Context: Waldenström's macroglobulinemia is a rare hematologic condition affecting 3:1000.000 people per year. The involvement of the gastrointestinal tract is rare and may be due to deposit of IgM monoclonal protein, malignant cells or amyloid.

Case report: An eighty-six-year-old female patient is admitted with a month history of dysphagia for solid food, 10kg weight loss and productive cough. She had a past medical history of hypertension, atrial fibrillation and urinary incontinence. The clinical examination revealed arrhythmic cardiac tones and a discrete bilateral pleural effusion. The laboratory exams showed an elevated erythrocyte sedimentation rate (104 mm/h) and an IgM monoclonal band in the serum protein electrophoresis with a 4050mg IgM count. The pleural effusion drainage revealed an exudate failing to demonstrate malignancy. The simple chest x-ray film showed multiple bilateral infiltrates and the total body CT scan confirmed the existence of multiple bilateral lung infiltrates suggestive of bronchial aspiration pneumonia, bilateral pleural effusion and a diffuse thickening of the esophagus. We performed an upper gastrointestinal endoscopic examination that showed a diffuse esophagic extrinsic compression and the esophagic biopsy revealed a subepithelial lymphoid cell infiltrate with CD20, CD19 and IgM positive cells confirming the clinical diagnosis of Waldenström's macroglobulinemia with upper gastrointestinal tract involvement. The bone marrow examination showed early infiltration by lymphoplasmocytic lymphoma with Waldenström immunophenotype.

SC-31 TETANUS IN THE HENHOUSE

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Case report: We report a 81 year old woman with hypertension treated with chlorthalidone who attended the emergency room for deviation to the left corner of the lips with difficulty opening the mouth. A physical examination revealed incisive wound sutured-bruised left front with signs of infection that occurred following accidental fall 5 days earlier in a henhouse. Valued primarily for Maxillofacial Surgery for suspected pathology of the temporo-mandibular joint, a facial CT scan was performed showing no abnormalities, and treatment with NSAIDs was started. The patient re-consulted by increasing symptoms 2 days later, with clear eating and breathing difficulties. The symptoms in the emergency evolve to intermittent generalized stiffness with acute respiratory failure needing urgent tracheotomy and mechanical ventilation, entering ICU. Suspecting tetanus, she was treated with anti-tetanic immunoglobulin, midazolam, fentanyl and metronidazole. Subsequently cisatracurium and enteral feeding were initiated. Episodes of hypertension alternating with hypotension from dysautonomia, especially early after admission, were treated with ACE inhibitors and fluid therapy. Loss of several teeth was associated with trismus. Among the complications occurred during admission include a ventilator associated pneumonia, ileus, anasarca and polyneuropathy resulting from her prolonged stay in ICU. 60 days after admission and progressive reduction of relaxation and sedoanalgesia, extubation was achieved. The patient initiated rehabilitation and finally moved to conventional hospital ward for continuity of care.

SC-32 A YOUNG WOMAN WITH FEVER AND CERVICAL LYMPHADENOPATHY

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Introduction: Kikuchi-Fujimoto's disease is a benign and self-limited disorder, characterized by regional cervical lymphadenopathy with tenderness, usually accompanied with mild fever and night sweats. Less frequent symptoms include weight loss, nausea, vomiting and sore throat. The aetiology is unknown.

Case report: A previously healthy 23-year-old woman presented to the emergency department with enlarged laterocervical adenopathy for two months accompanied with fever to 38°C, asthenia and nocturnal sweating. No other symptoms were referred. She had not recently travelled outside Spain and had not been exposed to animals. She is admitted to the Hospital with the suspicion of lymphoma. Physical examination revealed a healthy appearing young woman, with significant 4 cm left laterocervical node and 3 cm supraclavicular bilateral adenopathies tender to palpation, non erythematous and without surrounding cellulitis. Laboratory data revealed high ERS at 87 mm/h, mild leukopenia of 2,600/mm³ (neutrophils 47.4%, lymphocytes 41.3%), with antinuclear antibody

screen positive at 1:160 (negative antiDNA) and Beta-2-microglobulin levels were 2.1 mcg/mL. The tuberculin skin test (PPD) was non reactive. Main viral serologic tests were negative. Toracoabdominal CT described no systemic lymphadenopathy or other abnormalities. A surgical node biopsy was performed. Pathologic examination described a necrotizing paracortical lymphadenitis, consistent with Kikuchi's disease. The patient was treated with nonsteroidal anti-inflammatory drugs and corticosteroids with good evolution.

Discussion: Kikuchi-Fujimoto's disease is an extremely rare condition known to have a worldwide distribution with higher prevalence among asiatic individuals. It's more frequent in young women, presenting with painful laterocervical and supraclavicular adenopathy. In addition to lymphadenopathy, 30 to 50% of the patients with KFD may have fever. Leukopenia can be found in up to 50% of the cases. Light thrombocytopenia, neutropenia, hypertransaminasemia with elevation of lactic acid dehydrogenase may also be seen. Extranodal involvement is uncommon. Kikuchi Fujimoto's disease is generally diagnosed on the basis of an excisional biopsy of affected lymph nodes. Treatment is symptomatic (analgesics-antipyretics, non-steroidal anti-inflammatory drugs and, rarely, corticosteroids). Spontaneous recovery occurs in 1 to 4 months. Approximately 3-4% of the patients have recurrence of the disease.

SC-33 FEVER OF UNKNOWN ORIGIN IN A YOUNG MAN

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Introduction: Sphingomonas paucimobilis is a glucose-nonfermenting gram-negative bacillus. S. paucimobilis infections are rare and typically occur in immunocompromised patients. We describe a case of fever and hepatitis in a young immunocompetent man secondary to Sphingomonas paucimobilis bacteraemia.

Case report: A 33-year-old man with a medical history of morbid obesity and altered basal glycaemia is evaluated in an Internal Medicine consult. He refers one week evolution of fever up to 40 degrees Celsius without focus. There are no significant findings on physical examination. Initial blood tests highlight hypertransaminasemia with AST levels of 114 U/L, ALT of 156 U/L, GGT 180 U/L, FA 131 U/L, LDH 691 U/L and normal bilirubin levels. The CBC shows lymphocytosis (42%) and light thrombocytopenia. Image tests show a normal chest X-Ray, abdominal ultrasound, and echocardiography without signs of endocarditis. Blood cultures and serological studies were obtained. The first serology determination was negative and after 14 days of blood culture quinolone-sensitive gram-negative bacilli were obtained. We started treatment with levofloxacin 500 mg once a day for 1 week. On day 24th of blood culture, the gram negative bacilli were finally identified as Sphingomonas paucimobilis. The patient remained afebrile after levofloxacin initiation and hepatic enzymes were normalized. A second serology determination obtained 3 weeks after the first analysis was also negative.

Discussion: In our environment the most frequently causes of fever of unknown origin in young immunocompetent patients are infectious mononucleosis and Q fever, and it is also recommendable to dismiss primary HIV infection. Sphingomonas paucimobilis, is an organism of low virulence, and recovery from infections is the rule, even in debilitated hosts. It has been associated with a variety of infections, including ventilator-associated pneumonia, intravascular catheter-related blood stream infections, meningitis, osteomyelitis, septic arthritis, pleural empyema, splenic abscesses and biliary tract infections. S. paucimobilis bacteraemia occurs infrequently, especially in outpatients without underlying morbidity, but it has been encountered with increasing frequency in clinical settings.

SC-34 LIMBIC ENCEPHALITIS DUE TO NMDA AUTO-ANTIBODIES: A FATAL, TREATABLE AND REVERSIBLE DISEASE

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Case report: Anti glutamate receptor (NMDA) antibody encephalitis is a fatal but treatable and potentially reversible disease as long as it is promptly diagnosed and the adequate treatment is initiated. First reported in 2007 in association with ovarian tumors, it presents itself by stages, progressing from psychosis to memory deficits, convulsive disorders and later on dysautonomy with respiratory and cardiovascular instability which lead to death if left untreated. A 32 year old is brought to the emergency department due to behavioral changes, with auditory and visual hallucinations. Past medical history was relevant for ovarian cyst which had been removed 10 year earlier. Her blood work and head CT scan on admission were unremarkable and she was admitted to the psychiatric ward with an inaugural psychotic event. During the first day of hospital stay she developed oscillating mental status (Glasgow coma



scale 8-14), with psychomotor agitation which was difficult to control and was transferred to the intermediate care unit. Blood work was again unremarkable as was head CT with associated venogram; lumbar puncture with 17 cells, and no relevant change in the biochemical properties; Acyclovir and valproic acid were begun. Patient maintained psychomotor agitation alternating with profound somnolence associated with altered breathing patterns and facial dyskinesia. EEG in day 3 showed diffuse brain dysfunction mainly in the frontal region which could have an infectious cause but also auto-immune an cause. Brain MRI showed no changes; thyroid function was normal; Pelvic CT scan showed a large right ovarian mass with mixed characteristics which was suggestive of a teratoma. An lumbar puncture was performed to search for auto-antibodies and the patient was started on immunoglobulin (added to the corticosteroids she was already doing) for the possibility of paraneoplastic limbic encephalitis. She was taken to the OR on day 5 where the large teratoma was removed. The antibodies search in the patient's CSF were positive for NMDA antibodies. She remained in intensive care for 50 days with positive outcome. She was discharged home with no behavioral changes and with amnesia for the psychotic symptoms she experienced before admission, but remaining memory intact. Paraneoplastic limbic encephalitis is many times underdiagnosed, particularly when treating young women with *de novo* psychiatric manifestations and history of gynecologic tumors. Its treatment mainstay resides in the rapid removal of the tumor and the prompt initiation of corticotherapy and immunoglobulin.

SC-35 IMMUNOSUPRESSED PATIENT WITH DERMAL INFECTION BY MYCOBACTERIUM CHELONAE

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Case report: We report the case of a 80 years old woman with past medical history of hypertension, chronic kidney failure, polymyalgia rheumatic and pelvic fracture surgically repaired. She was being treated with furosemide, enalapril, omeprazole, calcium, D-vitamine, transdermic buprenorfine, acetylsalicylic acid and prednisone for 18 months (>10 mg/Kg/day). Two weeks prior to admission, she noticed fever and progression of multiple papules and pustules on both lower extremities. She denied having any fatigue, coughing or weight loss. She gave no personal or family history of tuberculosis. Physical examination revealed moon face, abdominal obesity and muscular atrophy of extremities. On both lower extremities there were numerous maculopustular lesions, many of them suppurated. The cardiopulmonary auscultation was normal, there were no palpable lymphadenopathies and other physical findings were also normal. The results of the blood analysis revealed: urea 153 mg/dl, creatinine 2 mg/dl, uric acid 7.7 mg/dl, lactate dehydrogenase 553 U/l, 12700 leukocytes/mm³, and normocytic anemia (hemoglobin 10 g/dl). The chest X-ray and the EKG were normal. The smear of one pustule demonstrated the presence of acid-fast bacillus with irregular morphology (Ziehl-Neelsen staining). Using Polymerase Chain Reaction (PCR), it was identified as *M. chelonae*. The sputum smear was positive. The urine smear and the blood cultures were negative. The biopsy of one of the lesions revealed subcutaneous cellular tissue infiltrated by histiocytes and polymorphonuclear cells and granulomas with central abscess (hematoxylin-eosin staining). The Ziehl-Neelsen staining revealed the presence of acid-fast bacillus. With the diagnosis of infection by *M. chelonae*, a treatment with clarithromycin and ciprofloxacin was started. The dose of prednisone was reduced progressively. The fever disappeared and the dermal lesions improved. Rapidly growing mycobacteria (RGM) are ubiquitous in nature and widely distributed in water, soil and animals. During the past three decades an increment of infections caused by RGM has been detected, both localized and disseminated, as well as nosocomial outbreaks of contaminated medical equipment. The microbiological diagnosis of RGM infections includes direct microscopic observation and culture. The taxonomic identification is performed by phenotypic, biochemical, chromatographic and molecular biology techniques. The treatment differs from that of other mycobacteriosis like tuberculosis, owing to the variable in vitro susceptibility of the species of this group. The RGM are resistant to conventional antituberculous drugs, but can be susceptible to broad spectrum antimicrobial agents(1). *M. chelonae* is one of the most frequent mycobacteria in immunosuppressed patients and has developed resistance to many antibiotics. The dermal infection is the most common target of this mycobacteria, sometimes with dissemination. It also can cause infection associated to traumatic lesions, surgical wounds, prosthetic materials and endovascular catheters (1).

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SC-36 ANEMIA SECONDARY A DUODENAL INFILTRATION LIKE PRESENTATION OF RENAL CELL CARCINOMA

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Summary: A woman 62 years old was admitted to our hospital to study constitutional syndrome and anemia. During last 3 months before admission, she presented progressive impairment, with constitutional syndrome and back pain. The abdominal CT scan showed large mass in lower pole of right kidney, which spans local and distal to the duodenum, liver and inferior vena cava. Also several lymph nodes with malignant aspect. An upper endoscopy revealed a bleeding duodenal mass with neoplastic appearance, which was biopsied. Bone scan was negative for metastasis and duodenal biopsy shows infiltration of clear cell renal carcinoma. This is a case of delayed diagnosis of renal carcinoma, with little semiology until advanced stages, eventually appears like constitutional box and anemia due to gastrointestinal bleeding presumably due duodenal infiltration.

Keywords: Renal cell carcinoma. Duodenal infiltration. Anemia. Constitutional symptoms.

Introduction: Renal cell carcinoma is characterized by lack early semiology, which determines high proportion of cases with various systemic clinical manifestations and metastasis (1). For these peculiarities, it has been called the internist's tumor (2). Represents approximately 2% -3% of all tumors. Anemia and gastrointestinal bleeding are expression of it. Manifestations of renal cell carcinoma like metastasis or infiltration of the upper digestive tract contiguity has been described, although very occasionally. Then we had a case of a woman with anemia secondary to gastrointestinal bleeding demonstrated by endoscopy and duodenal biopsy infiltration. Next to it, it is shown a wide local extension and metastasis in different images techniques.

Case: Female 62 years old, who was admitted for evaluation of iron deficiency anemia of 6 months duration, associated with constitutional framework over the past 3 months (unintentional weight loss of 9 kg), and recent onset of pain at the lumbar level of mechanical properties. She complained of chronic constipation and emission of dark stools, hard consistency, after treatment with iron. Tests for blood in stool, made in ambulatory care, for specific technique for human hemoglobin were positive. Her regular medication consisted of ferrous sulphate, omeprazol and analgesia with tramadol and paracetamol. Clinical examination revealed a good overall appearance. We observed, mucocutaneous pallor with normal vital signs. No palpable lymph nodes, heart sounds were rhythmic and controlled heart rate around 60-70 bpm. Globular abdomen was palpable, not distended, with no collateral circulation, or mass or organ enlargements, with peristalsis present. No masses or melena were observed DRE and the rest of the physical examination was normal. Analytic notes anemia (9.98 g / dl) with ferritin and low transferrin saturation. Renal function, liver, thyroid and tumor markers were normal. In urine was observed microhematuria. Upper gastrointestinal endoscopy showed a tumor-like lesion with erosions and active bleeding located in second's duodenal portion, which biopsy was taken. The CT of the abdomen showed a large mass of heterogeneous and irregular 12.9x12.3x11.8 cm in the lower half of right kidney, which pressured the duodenum anteromedially, infiltrating the posterior and lateral wall thereof, without radiological signs of occlusion. In the liver, the imagen revealed multiple focal lesions suggestive of metastases. There was infiltration of the light of the inferior vena cava, abundant subcutaneous collateral circulation, pelvic and retroperitoneal, as well as, vein azygos and hemiazzygos were dilated. Likewise showed multiple retroperitoneal lymphadenopathy centimeter, most of them of 3 cm pre-aorticocava location, the latter of clearly malignant. The bone scan was negative for metastasis. In the pathological examination of the duodenal biopsy was described ulcerated tumor composed of solid cell groups separated by a fibrovascular rich plot, cells with clear cytoplasm with hyaline eosinophilic granules, PAS positive and diastase resistant. The nuclei were ovoid, fine chromatin, and many nucleolados. The mitotic index was low (2 mitoses per 10 high power fields). The immunophenotype was positive for cytokeratin (AE1-AE3), CD10, and Bcatenina alfa1antitripsina and negative for CK7, CK20, Pax5, chromogranin and TTF1. Given these findings, cytological and supported by imaging tests, we established the diagnosis of clear cell carcinoma in advanced stage, considering the primary tumor mass and renal and duodenal infiltration as well expression of extension by contiguity or metastasis. After the diagnosis, she was followed by the oncology and palliative medicine.

Discussion: This case is another example of the late diagnosis which is printed on intrinsic characteristics of renal tumors. Precisely, its ability to remain silent in the initial stages of its natural history, makes more than 50% of renal tumors are diagnosed in advanced stages or casually. Also, some of them (6-10%) have the classic triad of flank pain, hematuria and abdominal mass. In the case reported only low back pain was the manifestation of the triad well defined, although the distended abdomen and microhematuria could be considered a posteriori as an expression of the triad. These peculiarities of the renal tumor and its anodyne presentation determine in most cases a diagnostic challenge. However, it seems that the presentation of clear cell renal carcinoma and anemia secondary to gastrointestinal bleeding, either externalized or not, has been documented in some cases, so; it might be reasonable to introduce this partnership in the search for differential pathologies patients with anemia and constitutional syndrome.

SC-37 THALLIUM POISONING IN A 12-YEAR OLD GIRL

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Introduction: Thallium intoxication is extremely rare. Since its banning as a component of rodenticides in 1991 in Europe, intoxication is mostly due to poisoning. It is potentially mortal and a challenge for its diagnose if the exposure isn't suspected. We present the case of a 12 year old girl who was victim of thallium poisoning.

Clinical case: 12 year old girl, from Pakistan is brought to the ER for a clinic without any trigger of a few hours of pain in lower limbs, with difficult on the walk. The pain started in both feet being ascendant, arriving to both hips. She also had loss in strength and paresthesias. The previous night she had experienced a vomit, frontal headache and perioral paresthesias that had ceased. The lab results show: liver cytolysis GOT > 200 UI/L and normal bilirubin levels. She was admitted for study at the paediatric service. During her stay the father told the medical team, that his wife and mother of the patient had experienced similar symptoms 5 months earlier, for which she had returned to Pakistan and was suspected for thallium poisoning. Thallium levels were therefore performed in the girl resulting in: (urine 1900 microg/l and blood 80 microg/l) see table 1, confirming toxic levels. It was suspected to be intentional poisoning. Treatment with Prussian blue was then initiated, as an initial dose of 500g every 8 hours, as well as forced diuresis with potassium supplements. 72 hours after starting treatment muscle weakness was increased, as well as disesthesias, and appearance of disfagia and desaturation. An arterial gasometry was performed being; pH 7,39; PaCO₂ 52; HCO₃ 31,5, PaO₂ 76. She was then admitted in the intensive care unit, intubated, and mechanical ventilation was initiated. She required 5 hemodialysis sessions. During her stay in intensive care lower eyelid hyperpigmentation was evidenced, as well as telogen effluvium that progressed to total alopecia. She was always tendent to hypertension and tachycardia, probably because of vegetative affectation. She improved slowly as thallium levels decreased, she was successfully extubated 12 days after her admission on intensive care. After sedation was removed, we were able to examine the neuropathy, resulting in a severe sensitive and motor neuropathy, predominantly of superior limbs. She was admitted in a rehabilitation clinic, being able (one month after discharge from the intensive care) to walk with an antiequine orthosis, with bad tolerance due to neuropathic pain.

Discussion: Thallium poisoning is a rare entity, but potentially mortal. It's early diagnose and treatment can avoid aftermath in affected patients. Our patient had a severe thallium intoxication with respiratory failure that required mechanical ventilation, which is a rare complication, and indicates severity. Due to severity she was started on dialysis, even though there is controversy about the use of dialysis. In our case, we couldn't affirm there was an improve as the levels of thallium in blood, and dialysis liquid did not differ before and after it.

Table 1 (SC-37)

| Thallium levels microg/L | Arterial blood | Venous blood | Urine 24h | Dialysis liquid pre | post |
|--------------------------|---------------------------|----------------------------------|-----------|---------------------|------|
| - 09/03 | | | 28800 | | |
| - 12/03: | | 182 | | | |
| - 16/03 | | | 11400 | | |
| - 20/03 | | 37 | 4100 | | |
| - 21/03 | | 11.2 (prediálisis) 7.5 (post) | 4965 | < 1 | 2.1 |
| - 22/03 | 14.5 (pre) 11.4 (post) | 60.5 (pre) 6.2 (post) | | < 1 | < 1 |
| - 24/03 | 7.2 (pre) 6.7 (post) | 3.2 (pre) 4.6 (post) | 318 | < 1 | < 1 |
| - 25/03 | 3.3 (pre) 3.4 (post) | 1.9 (pre) 2.3 (post) | 294 | < 1 | < 1 |
| - 26/03 | 2.1 (pre) 1.9 (post) | 1.4 (pre) 1.6 (post) | 150 | | |
| - 27/03 | | 2.7 | 84 | | |
| - 28/03 | | 2.2 | 66 | | |

SC-38 DERMATITIS INDUCED BY AMLODIPINE: A RARE SIDE EFFECT

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Introduction: Amlodipine is a dihydropyridine calcium channel blocking agent which used widely in the treatment of hypertension. In this article; we report a case who has intense dermatitis at both hands that is thought to be associated with the use of amlodipine and regressed after the change of amlodipine therapy.

Case: S.S. is 47-years-old male patient, a personnel of our hospital. The patient's who was admitted to internal medicine outpatient clinic with ABP: 152/95mmHg. Essential hypertension was diagnosed and amlodipine 5mg/day started. After about three months, the patient who continues amlodipine but no other treatment, associated with some intense cracks lesions at both hands were noticed to occur. The patient was diagnosed with contact dermatitis by dermatology consultation and his treatment was started. Despite the patient has continued to the treatment about a month, his complaints of has not recovered. Dermatology consultation was repeated and was evaluated as a drug eruptions may be. Amlodipine therapy changed to candesartan 8 mg / day. The patient had continued to this treatment for about 2 weeks and a regression was observed in lesions at his hands. Candesartan treatment was changed because of side effects and Amlodipine 10 mg per day treatment was started again at the request of the patient. In the second week of the treatment; previous lesions repeats were observed and recorded. After obtaining approval from the patients, the treatment was adjusted as valsartan 160mg / day. Dose of treatment adjusted with regular visits. Approximately after 2-3 weeks the lesions completely regressed in both hands.

Conclusion: Although it is expressed at the prospectus that it may be skin rashes; extensive dermatitis in both hands, such as our case were not found at literature search. In our case, the patient was working with manual dexterity in technical services and need to use his hands in unhygienic environments at most of daily working hours. The patient whose symptoms were regressed with changing the treatment, was able to continue to antihypertensive treatment comfortably. About the use of antihypertensive; up to patient-physician relationship, knowing the side effects of the drugs used by physician is also considered to be extremely important in terms of continuity of treatment.

SC-39 HYPERIMMUNGLOBULIN M (HIM) SYNDROME WITH RHEUMATOID ARTHRITIS (RA): A RARE CASE REPORT

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Background: Although immunodeficiency and autoimmunity seem to be the two opposite sides in the spectrum of the clinical immune response, primary immunodeficiency diseases (PID) and autoimmune phenomenon may occur concomitantly in the same individual. Sometimes autoimmunity and rheumatic diseases may be the first clinical manifestation in PIDs.

Case: A 20 years old male patient referred to clinics for weakness, fatigue, morning stiffness, pain and swelling on his both knee, shoulder, wrists and hands. Previous history revealed frequent upper and lower respiratory tract infections since his childhood. On the rheumatological examination, right 2nd-3rd metacarpophalangeal (MCP), left 3rd MCP, 5th proximal interphalangeal (PIF) joints were found to be swollen and tender. Furthermore, there were also arthritides in right knee and ankle. The extension of both elbows was limited to 70°. On the laboratory investigation, erythrocyte sedimentation rate (ESR) and CRP were found to be increased (65 mm/h, and 28 mg/L, respectively). Whole blood count and blood biochemistry including renal and liver function and protein electrophoresis tests were within normal ranges. Serum immunoglobulins: IgG: 1.43 g / l (N: 8.30-18.2), IgA: 0.25 g / l (N: 0.7-4.0), IgM: 3.16 g / l (N: 0.4-2.3) were found. Auto-immune markers including ANA, anti-CCP, rheumatoid factor (RF) were all negative. T cell subtype studies were as follows: CD45: 94.7% (N: 88-100), CD4: 7.1% (N: 34-63.8) and CD8: 64.6% (N: 19-48). The presence of low Ig G and A levels and high M levels led to the diagnosis of hyperimmunglobulin M (HIM) syndrome. With respect to the articular symptoms, high frequency ultrasound examination was performed which revealed synovial hypertrophy and power Doppler activity signals in wrists and knees. MRI of the right wrist, carpal and metacarpal joint findings were consistent with rheumatoid arthritis (RA) monthly IVIG treatment was initiated for HIM syndrome and 5 mg of oral prednisolon daily and 200 mg of hydroxychloroquine daily were started for rheumatoid arthritis. At the last visit (after ~6 month) his joint symptoms were improved and ESR and CRP levels were decreased.

Conclusion: Co-occurrence of HIM and RA is quite rare, and accurate diagnosis is vital for patients. Differential diagnostic work-up especially for septic arthritis is very important. When deciding to add DMARDs to the low-dose corticosteroids should be considered cautiously and followed-up closely.



SC-40 SEVERE DIARRHEA OBSERVED AFTER ANTIHYPERTENSIVE TREATMENT. A RARE CASE REPORT

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Introduction: Hypertension is one of the leading preventable causes of death, with an incidence rate of % 26.4 effecting approximately 1,5 billion people. Telmisartan + Hydrochlorothiazide (HCT) is an antihypertensive combination used for many years. Although gastrointestinal (GI) side effects due to this combination are reported in its prospectus and drug-induced diarrhea may happen; it was not found at our literature search. Here we report a case who had severe diarrhea after the use of Telmisartan + Hydrochlorothiazide combination and recover after ceasing the drug.

Case: E.C. 44 years old man was admitted with complaints of headache, dizziness, weakness and 24-hour ambulatory monitoring and manual arterial blood pressure measurements revealed high results. He was diagnosed with stage-2 essential hypertension and Telmisartan-HCT treatment was started. A foul-smelling and juicy diarrhea 3-4 times per day, and on the following days 4-6 times per day, started 36 hours after the treatment began. He was admitted again. No pathological finding was present to explain the patient's complaints. With the antihypertensive treatment for a month, the patient's diarrhea continued. Since he has expressed that he lost 6kgs and was very exhausted in a period of about a month, his antihypertensive treatment was changed to amlodipin 10mg/day. We observed that the patient's complaint regressed in 1-2 days and blood pressure measurements turned to normal. His diarrhea was disappeared and turned to his normal weight before the treatment in about a month, after the change in medication. The patient is still under control and treatment.

Discussion: In our case, severe diarrhea after Telmisartan + HCT treatment regressed with change of treatment. Weight loss and fatigue lead to loss of labor. Even though no pathologic finding seen at abdominal USG; a reaction in an intestinal mucosal level (angioedema?) was thought to cause the current situation. While awareness and treatment of hypertension is in such a low level in the community, minimizing the side effects of drugs used to treat hypertension and physicians' knowing the possible side effects will improve patient compliance and contribute to the regulation of blood pressure.

SC-41 AN UNUSUAL CASE OF HODGKIN LYMPHOMA PRESENTING WITH SEVERE THROMBOCYTOPENIA AND IMMUNE-MEDIATED REFRACTORINESS TO PLATELET TRANSFUSION

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Case report: Autoimmune thrombocytopenia (AITP) often complicates low-grade non-HL but is rarely observed in Hodgkin lymphoma (HL) (1). We present a case of immune-mediated transfusion-refractory thrombocytopenia at initial diagnosis of HL. A 65 years old healthy man was admitted with 3 months history of fatigue, fever (with chills and night sweats), epistaxis and unintentional 30 kg weight loss over 6 months. PMH/SH were negative except for tick bite 6 months prior to admission (PTA) and a history of nephrolithiasis. Multiple hospitalizations over past 3 months –the last 1 month PTA – for similar symptoms resulted in no diagnosis. Physical examination was within normal limits except for presence of nasal packing, mild splenomegaly and diffuse petechial lesions. Laboratory studies showed moderate normocytic anemia and severe thrombocytopenia (3×10^9 cells/L). Peripheral blood smear showed mild hypochromia, anisocytosis, microcytosis and reduced thrombocytes, but no abnormal WBC morphology. Biochemistry panel and anemia workup were normal with exception of slightly elevated LDH, elevated ALP and GGT. Multiple single donor apheresis transfusions did not improve platelet (PLT) count and epistaxis continued. High dose methylprednisolone (1 mg/kg/day) started for possible ITP. Consecutive (IVIg) (1gr/kg, x two days) treatment was administered; PLT level remained unchanged. Bone marrow biopsy and CT of neck, chest and abdomen showed multiple hypodense lesions of spleen & multiple lymph nodes in the peri-aortocaval & internal iliac regions, improved in size compared to imaging 1 mo PTA. BM was mildly hypercellular with normal maturation in all lineages. Apparent increase in megakaryocytic lineage was compatible with peripheral destruction of PLTs. During workup, upon an abdominal hemorrhage; Pt was taken emergently for an exploratory laparotomy and splenectomy. Post-splenectomy, PLT level was $<10 \times 10^9$ cells/L. Refractoriness to Plt transfusions was attributed to alloimmunization, with poor platelet increase after 2 transfusions and no causes of peripheral destruction were appreciated. The PLT trend and corrected count increment (CCI) calculated from formula: $CCI = [PPI \text{ (post transfusion platelet increment)} \times BSA \text{ (m}^2)] \times 10^{11} / \text{number of platelets transfused}$ (Table). Spleen pathology established diagnosis of HL. Treatment with doxorubicin, bleomycin, vinblastine, dacarbazine (ABVD) resulted in PLT count increase to 246×10^9 cells/L within 3 days. AITP rarely complicates the course of HL. Of 1029 Pts with HL, 3 (0.29%) cases were identified at presentation (1); these were older and often had advanced disease. Two similar cases (2, 3) one of which improved with IVIG treatment (2), and a second presenting with thrombocytopenia as a manifestation of disease relapse and non

responsive to treatment (3) were published. However, immune mediated refractoriness to platelet transfusion and splenectomy has not been reported upon disease presentation. Alloimmunization is caused by Plt antigens: the human leukocyte antigen (HLA) & the human platelet antigen (HPA) systems (4). Which one is the leading cause in HL has not yet been described but ABVD combination chemotherapy seems to provide effective control of this condition as well as autoimmune thrombocytopenia.

References: 1. Dimou M, et al: *Leukemia & Lymphoma*, 2012; Early Online:1-7. 2. Dahlqvist C, et al: *Acta Clin Belg*. 2010;65:41-3. 3. Saitoh T, et al: *Clin Lymphoma Myeloma*. 2008;8:62-4.4. Tinmouth AT, et al: *Transfus Med Rev* 2006;20:294.

Table (SC-41)

| | Platelet Count | Corrected Count Increment (CCI) |
|----------------------------------|------------------------------|---------------------------------|
| Before Splenectomy | 6 x 10 ⁹ cells/L | |
| After Splenectomy | 5 x 10 ⁹ cells/L | |
| 10 minutes after Plt transfusion | 22 x 10 ⁹ cells/L | 3.825 |
| 1 hr after Plt transfusion | 6 x 10 ⁹ cells/L | 0.225 |
| 24 hrs after Plt transfusion | 2 x 10 ⁹ cells/L | |

SC-42 PARANEOPLASTIC NECROTIZING MYOPATHY IN A PATIENT WITH SIGNET-RING CELL CARCINOMA

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Introduction: Necrotizing autoimmune myopathy is an increasingly recognized autoimmune myopathy with distinct pathologic features. It is a unique immune-mediated myopathy which muscle biopsy is characterized by necrotic fibers and little or no inflammatory infiltrate.

Objective: To describe a paraneoplastic autoimmune necrotizing myopathy case.

Material and methods: Medical history and related literature review.

Clinic case: A 43-years-old woman with neither relevant medical history nor previous treatment, 20 packageyear smoker, was admitted to our Service with a four months history of progressive asthenia, hyporexia and unquantified weight loss. During this time she presented symptoms of severe progressive weakness affecting predominantly lower limbs and within the last 2 weeks, the patient was wheelchair-bounded. No skin lesions. Physical examination revealed malaise, skin and mucous pallor, limb weakness, specially involving the proximal muscles (both waists), preserved sensitivity, arreflexia of lower extremities and normal deep tendon reflexes of upper extremities. The laboratory sample showed hemoglobin 10.4 g/dL, corpuscular erythrocyte mean volume (MCV) 80fL, erythrocyte sedimentation rate 103 mm/h, c-reactive protein 7.5 mg/dL, protein 58 g/L, albumin 32 g/L, aspartate aminotransferase (AST) 81 U/L, alanine aminotransferase (ALT) 61 U/L, lactic acid dehydrogenase (LDH) 515 U/L, creatine kinase (CK) 1485 U/L, aldolase 18 U/L. Levels of tumor, autoimmunity and celiac markers were unremarkable. Syphilis, hepatitis B, C and HIV serology were also negative. The electromyogram performed to confirm myopathy showed evidence of inflammatory myopathy. Lower extremities magnetic resonance also revealed findings consistent with inflammatory myopathy that affected completely both thighs musculature. Skeletal muscle biopsy showed necrotic fibers, the absence of inflammatory infiltrate, some regenerative fibers and the absence of histocompatibility complex class I (MHC-I) antigens expression. Thoracoabdominal CT scan demonstrated thickened and ulcerated fundus gastric wall with a suggestive image of locoregional adenopathic conglomerate. Gastroscopy revealed a big ulcero-vegetative tumor in the gastric fundus infiltrating the cardia. Histopathology confirmed diagnosis with a signet-ring cell carcinoma. She was treated with pulse methylprednisolone (1g per day for three days) and afterwards prednisone was continued at 1 mg/kg per day for a month and slow tapered to the lowest effective dose for a total duration of therapy between 6-12 months. Finally, the patient underwent a total gastrectomy and partial esophagectomy (T4aN3bMx). Anti-signal recognition particle (SRP) antibodies titration was 1/160 (significant levels titers >1/160).

Discussion: The idiopathic inflammatory myopathies encompass a heterogeneous group of rare disorders including polymyositis, dermatomyositis, inclusion body myositis and necrotizing autoimmune myopathy. In general, they present acutely or subacutely with marked proximal and symmetric muscle weakness.

Necrotizing autoimmune myopathy is an autoimmune mediated myopathy. Histopathologically is characterized with necrotic fibers and the absence of inflammatory infiltrate. The neoplasies most frequently associated with necrotizing autoimmune myopathy are gastrointestinal adenocarcinoma, lung, breast and prostate cancer, multiple myeloma and transitional bladder carcinoma.



Conclusion: Necrotizing autoimmune myopathy is a rare cause of idiopathic inflammatory myopathies. We present a necrotizing autoimmune myopathy associated with signet-ring cell carcinoma.

SC-43 TUBERCULOSIS: DIFFERENT TYPES OF PULMONARY INVOLVEMENT

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Introduction/ Objectives: Lungs constitute the most common site of involvement by *Mycobacterium tuberculosis* infection. The current presentation has the purpose of reflect about the pulmonary involvement by tuberculosis and its possible complications in order to review some specificities related to the diagnosis and management of this disease.

Material and methods: Revision of five clinical cases and literature review were performed. A 18 year-old caucasian female, with no prior pathologic history or evidence of trauma, referred to the Emergency Department with a right hydropneumothorax in a chest X-ray. She described asthenia, anorexia and weight loss, accompanied by productive cough and night sweating, with one month of evolution. 24 hours prior to the admission, she developed, suddenly, a right pleuritic pain associated with fever. A 52 year-old caucasian male with complaints of productive cough and pleurisy with one month of evolution. On admission he described resting dyspnoea, asthenia, anorexia and weight loss, night sweating and vespertine chills, denying fever. A 36 year-old caucasian male observed in the Emergency Department after a sudden onset of haemoptysis. He referred a two weeks prodrome of productive cough. A 54 year-old caucasian male presenting with fever, dry cough and pleuritic chest pain in the previous two weeks. He described night sweating, asthenia, anorexia and weight loss. A 51 year-old caucasian male with an insidious evolution of dyspnoea, dry cough, chest pleuritic pain and night sweating with two months of evolution. He reported the existence of asthenia, anorexia and weight loss.

Results: In the first case, a diagnostic thoracentesis was made, which demonstrated an empyema with ADA levels of 101.8 U/L (N < 40 U/L). The identification of *Mycobacterium tuberculosis* was made in sputum samples. The second and the third cases were diagnosed through the identification of *Mycobacterium tuberculosis* in sputum samples. In the fourth case, all the cultural results were negative, being the diagnosis made through the identification of *Mycobacterium tuberculosis* in a pleural biopsy. In the last case, the identification of *Mycobacterium tuberculosis* was made in samples of bronchoalveolar lavage and urine; cervical, axillary and inguinal ultrasound confirmed ganglionar enlargement. All patients were HIV negative.

Discussion/ Conclusions: Tuberculosis remains an important cause of pulmonary infection. The clinical cases presented show the wide variety of possible pulmonary manifestations of tuberculosis infection and subsequent complications, as well as the complexity of its diagnosis and therapeutic management.

SC-44 YOUNG ADULT PRESENTING WITH JOINT PAIN AND RED EYE: A CASE OF LÖFGREN'S SYNDROME

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Introduction/ Objectives: Sarcoidosis is a multisystemic granulomatous disorder characterized by the existence of noncaseating granulomas in a wide variety of tissues such as skin, lung, lymph nodes, eyes, joints, brain, kidneys and heart. Löfgren's syndrome corresponds to an acute presentation of sarcoidosis characterized by the presence of the triad: hilar adenopathy, polyarthralgia or arthritis and erythema nodosum, with or without the documentation of parenchymal infiltrates or fever. The presentation of this clinical case has the purpose of demonstrate and reflect about concepts and particularities related to the multisystemic involvement resulting from sarcoidosis.

Material and methods: Revision of one clinical case and literature review were performed. A 36 year-old caucasian male came to the Emergency Department presenting a clinic of inflammatory polyarthralgias (large joints – elbows, wrists, knees and ankles) and back pain with almost two weeks of evolution. He was previously observed by his Family Physician, who prescribed him a non-steroidal anti-inflammatory drug. On admission, he also described the existence of a redness in his left eye, denying the presence of fever. During his hospitalization he developed fever and erythematous and tender subcutaneous nodules, predominantly located on right infrapatellar region and anterolateral face of his left leg.

Results: Sacroiliac MRI demonstrated a bilateral sacroiliitis. The thoracic CT scan revealed the existence of multiple adenopathies on the medium mediastinum and diffuse accentuation of the lung reticulum – mild interstitial fibrosis. A bronchofibroscopy was made

and the bronchoalveolar lavage was compatible with a lymphocytic alveolitis (increased CD4/CD8 ratio). HLA-B27 antigen was negative.

Discussion: The presented clinical case illustrates well the multisystemic involvement resulting from sarcoidosis. Musculoskeletal disease isn't uncommon in patients with sarcoidosis. Joint manifestations are clinically relevant in patients with acute forms of this disease.

Conclusions: Musculoskeletal disease is a less common problem in patients with sarcoidosis. Joint involvement is typical in acute forms of the disease. Approximately 25 percent of the patients have an associated arthropathy. Löfgren's syndrome constitutes an acute presentation of sarcoidosis, being associated with a favourable prognosis.

SC-45 YOUNG GIRL PRESENTING WITH AN HYDROPNEUMOTHORAX: A CATASTROPHIC PRESENTATION OF A TUBERCULOSIS PRIMARY INFECTION

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Introduction/ Objectives: Primary tuberculosis refers to the development of disease following an initial exposure to *Mycobacterium tuberculosis*, being mainly a disease of childhood. Many studies have shown a growing frequency of tuberculosis primary infection in adolescents and adults. The exposition of this clinical case of severe primary tuberculosis has the purpose of reflect about concepts and particularities of this kind of clinical presentation of tuberculosis infection as well as review some specificities related with the diagnosis, management and treatment of this disease.

Material and methods: Revision of one clinical case and literature review were performed. The authors present a clinical case of an 18 year-old caucasian female, with no prior pathologic history or evidence of trauma, referred to the Emergency Department with a right hydropneumothorax in a chest X-ray. The patient described a clinical scenario, with one month of evolution, of asthenia, anorexia and weight loss, accompanied by productive cough and night sweating. 24 hours prior to the admission, she developed, suddenly, right pleuritic pain associated with fever. On admission, the patient was pale and emaciated; febrile (38.0°C), tachycardic (150 bpm), normotensive (120/70 mmHg), tachypneic (30 cpm) and with SpO2 91% (FiO2 24%). A diagnostic thoracentesis was made, which demonstrated an empyema with ADA levels of 101.8 U/L (N < 40 U/L). A thoracic tube was inserted. After the identification, in a sputum sample, of *Mycobacterium tuberculosis*, antituberculous therapy was initiated. The complementary study excluded any kind of primary or acquired immunodeficiency. The patient was discharge asymptomatic and without respiratory failure, after six weeks of treatment, being oriented to the Pneumologic Diagnostic Center.

Discussion/ Conclusion: Tuberculosis remains an important cause of pulmonary infection. Large volume hydropneumothorax constitutes a severe and rare presentation of pulmonary involvement for *Mycobacterium tuberculosis*, demanding its diagnosis a high grade of clinical suspicion.

SC-46 ATYPICAL PRESENTATION OF AN INSULINOMA

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Case report: Neuroendocrine tumours (NETs) are a wide cross section group of tumours. These tumours originate in tissues containing cells derived from the embryonic neural crest, neuroectoderm and endoderm. While NETs occur at many sites in the body, the majority are found in the gastrointestinal tract and pancreas or lungs. Although estimates vary, the annual incidence of clinically significant NETs is approximately 1-2 cases per 100000 inhabitants. They can be classified as functional or non-functional, and symptoms range from non-specific systemic symptoms due to abnormal hormonal secretions, to more localized symptoms such as those caused by obstruction. They are usually indolent (slow-growing) with a late diagnosis and a probability to be malignant; therefore having a more aggressive clinical behavior. Most insulinomas are typically hypervascular, sporadic, solitary, benign and small, measuring less than 2cm in 90% of cases. Less frequently they could have a malignant presentation (10%) and may occur as part of complex familial endocrine neoplastic syndromes, such as multiple endocrine neoplasia type 1 (MEN1), although the majority occur as non-familial (i.e. sporadic) isolated tumours. Below we report a case of an insulinoma, with both atypical diagnostic findings and histological features. The patient was a 42-year old female, who presented with multiple episodes of dizziness, tremors, anxiety,

headache and syncope during the last 14 months. These symptoms were associated with digital glycemia readings of 38-55mg/dL, but had no association with time of day or fasting status. She had never smoked cigarettes or consumed alcohol, and was on no prescribed medications. The patient reported a past history of fibromyalgia and a surgical intervention for a subarachnoid cyst at her 20s. The first outpatient blood analysis ruled out the most frequent causes of hypoglycemia. The team clinically suspected a hyperinsulinemic hypoglycemia, and the patient was admitted to hospital to perform a 72 hour fast. Six hours after initiating the fast, she presented a symptomatic hypoglycaemia, with the following biological findings: glucose 36 mg/dL, insulin 2.7 mIU/L (3-20 mIU/L), C peptide 0.9 (0-3 mg/dL), insulin/glucose index 0.075. Ketones were not present, and beta-hydroxybutyrate levels were not measured. The proven hypoglycaemia with insulin and C peptide in their lower limits of normality prompted us to repeat the fast, showing again several episodes of hypoglycaemia with similar biochemistry. A pro-insulin level could not be determined. We also performed a glucagon test after one of the episodes of hypoglycaemia, with glucose levels at baseline, 10, 20 and 30 minutes being 36, 40, 49 and 52mg/dL respectively. Puzzled by the normal insulin levels with such low glucose levels, without defining levels of hyperinsulinism, we continue investigating with a chest, abdomen and pelvic CT scan demonstrating a 7x6cm tumour at the head of the pancreas with necrotic and haemorrhagic elements compromising splenic vessels. As a result, a partial pancreatectomy was performed, including 2 involved lymph nodes, with preservation of the spleen. The histology showed small sized cells arranged in a solid and trabecular pattern surrounded by plenty of vessels. The immunohistochemistry showed a positive stain for chromogranin, synaptophysin, CD56, focal insulin areas and a 10% of Ki67. The diagnosis was pancreatic neuroendocrine tumour: insulinoma. The adenopathies showed chronic lymphadenitis without tumor lesions. Three months post-operatively, the patient had no further episodes of hypoglycaemia, with blood glucose maintained around 80mg/dL, basal insulin 2.2 mIU/L and C peptide 0.8 mg/dL. We did not find any metastasis in Octreoscan nor in PET-CT and she is still attending the outpatient clinic every 6 months. The main diagnosis approach for insulinoma remains fundamentally on the evidence of Whipple's triad during a 72 h supervised fast, with the presence of symptoms known or likely to be caused by hypoglycemia, a low plasma glucose measured at the time of the symptoms, and relief of symptoms when the glucose is raised to normal. The lack of insulin suppression in the presence of hypoglycemia leads to suspect hyperinsulinism. Hence, an insulin increase over 6mIU/L, with a low glycemia under 45 mg/dL is the best criteria to diagnose endogenous hyperinsulinism, reaching a 100% sensitivity and specificity. Also, an increase in C peptide levels over 0.6 mg/dL leads to the suspicion of insulinoma, and could have more precision than insulin levels. Insulinomas are frequently diagnosed with a medium size of 1.5cm, 24-30% are smaller than 1cm, 42% are between 1-2cm, 30% between 2-3cm, and 4% are greater than 3cm approximately. Malignant tumours incidence are less than 10%; however, a greater size has been reported to have a malignant behavior. In our case, the tumour had a big size, thus it is necessary to have a close follow up with image techniques. The immunohistochemistry showed localized positive areas for insulin, and negative for glucagon, ki 67 was 10%, which indicates a high index proliferation, therefore suggesting a non-differentiated tumour with highly aggressive potential to keep in mind through this patient follow up. Following WHO classic criteria to define an Insulinoma as malignant, it is considered: metastasis evidence, invasion degree, tumour size, mitotic rate, proliferative rate and vascular invasion; however, most authors consider the evidence of metastasis the only definitive criteria for malignancy. In any case, the rest of criteria should lead to high risk malignant insulinomas, which is why it is important to have a close follow up as in our case. A review of the literature demonstrates similar cases to ours, in which insulin levels are under the diagnosis threshold, but with a predominant proinsulin secretion, which could explain the relatively low insulin levels of our case. Insulin levels measure is influenced by the type of assay used (polyclonal or monoclonal antibodies). The use of polyclonal antibodies leads to a cross reaction with insulin by 40 to 80%, unlike the monoclonal assays that have detected minimal concentrations of insulin, that is why it has been suggested to reduce the diagnosis threshold for hyperinsulinemia when these essays are used for the diagnosis of insulinoma. In our case, we used chemiluminescence immunoassay for paramagnetic particles with monoclonal antibodies which may suggest that the inferior limit for the detection of hyperinsulinism should have been lower than usual >6mIU/L as we have referred previously. This immunoassay presented a 2.6% cross reaction with proinsulina. This aspect could explain the focal positive areas for insulin in the immunohistochemistry. Unfortunately, we could not obtain proinsulin levels and insulin levels were not indicative of endogenous hyperinsulinism, while the lack of peptide C suppression would indeed coincide with the hypoglycemia episodes that showed through all hospitalization. In conclusion, the non-elevated levels of insulin in a case of suspected hypoglycaemia induced by endogenous hyperinsulinism, required the measurement of proinsulin. Our case shows as well atypical insulinoma findings such as size, local positive areas for insulin and a high ki-67, which we will take into account for close follow up.

SC-47 EPIDURAL ABSCESS IN MAN AFTER PERFORMING A BONE MARROW BIOPSY

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Introduction: The epidural abscess is a suppurative disease that affects the central nervous system. Due to its unspecific symptoms and low prevalence, it can delay the diagnosis that can bring serious consequences and even death.

Case report: This case is about a man who is 64 year old, drinker and severe smoker, poorly controlled diabetic and followed for a monoclonal gammopathy of undetermined significance, performing 3 months before admission a bone marrow biopsy, which was complicated by a suppurative fistula for six weeks. His father and three siblings died for neoplasms. The patient shows asthenia, anorexia and weight loss of more than 10 kilograms during several weeks. Eight days before the admission the patient complained of diffuse neck pain and fever, with difficulty in walking and urinary incontinence. It is found fever of 38 degrees and neurologically highlights areflexia in lower extremities and positive Babinski sign on left foot in the physical examination. Analytically we can highlight leukocytosis with left shift, altered coagulation, elevated acute phase reactants, with high procalcitonin, and high glucose with glycated hemoglobin of 9.5. Given the suspected diagnosis of epidural abscess is requested MRI confirms it, visualizing the cervical level. Paravertebral abscesses are also observed. We are drawn to blood and urine cultures positive for methicillin-sensitive *Staphylococcus aureus*. It began with Cloxacillin antibiotic therapy for 6 weeks with clinical and radiological resolution.

Discussion: The epidural abscess has got a low incidence of 1-2 cases per 10,000 to 20,000 hospital admissions. The classic triad occurs in one third of cases with fever, spinal pain and neurological deficit. The diagnostic technique of choice is MRI with contrast, but you should find an etiologic diagnosis by blood cultures, urine, abscess,... It should be to establish empirical antibiotic therapy as soon as possible, keeping in mind that the most common organism is *Staphylococcus Aureus*. Depending on the clinical and radiological evolution, the use of surgical techniques for drainage of this should be assessed. There are three access routes: hematogenous, by contiguity or iatrogenic, with an increased incidence of the latter in recent years due to increase invasive techniques. Predisposing factors usually present are diabetes, parenteral drugs, alcoholism, HIV or immunosuppression.

SC-48 NEPHROGENIC SYSTEMIC FIBROSIS ASSOCIATED TO GADOLINIUM CONTRAST EXPOSURE

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Objectives: To identify the potential trigger agent for developing nephrogenic systemic fibrosis (NSF) in a patient, as well as to describe the clinical signs during the course of her disease.

Material and method: Retrospective review of a single case

Results: A 28-year-old caucasian female diagnosed of an autosomal recessive polycystic kidney disease and congenital hepatic fibrosis at birth, with type I diabetes mellitus since 1991. Between 1998 and 2002 she suffered repeated episodes of cholangitis, so that she underwent five times magnetic resonance (MR) imaging procedures using gadolinium-based contrast agents (Gd-CA) with gadoteric acid. In 2005 she started renal replacement therapy with hemodialysis, requiring correction of anemia with supplemental erythropoietin and red cell transfusions. In 2006 she was again exposed to Gd-CA for another MR imaging procedure due to an infectious disorder of her left breast. In 2011 she presented in our hospital with a progressive disorder characterized by hair loss, epidermal atrophy, and an erythematous papular rash involving mainly the skin of the lower extremities. She also had contractures of the joints, non-productive cough and dyspnea. Testing for autoantibodies was negative. The patient was diagnosed of NSF based on the histologic findings of both skin and muscle biopsies showing fibrosis in association with CD34-positive fibrocytes, and was admitted as candidate for liver and kidney transplantation. Finally, she developed liver abscesses with septic shock and died.

Discussion: NSF is a rare condition appearing only in patients with severe renal impairment and characterised by widespread tissue fibrosis. This patient developed a progressive, multiorgan system fibrosing disease with fatal evolution. Although the pathogenesis of this disorder remains unclear, there is evidence of a strong association between Gd-CA exposure and the triggering of this disease. Cellular elements involved in pathogenesis of NSF include bone-marrow-derived collagen-producing fibrocytes, myofibroblasts and activated macrophages.

Conclusions: Clinicians should consider the possibility that patients with severe renal failure may develop NSF after exposure to Gd-CA. We suggest avoiding these contrast media in patients with significant renal impairment unless the benefits clearly outweigh the risks.



SC-49 CASE REPORT: THE SUPERIOR MESENTERIC ARTERY SYNDROME ASSOCIATED WITH NUTCRACKER SYNDROME

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Case report: The superior mesenteric artery syndrome (SMAS) or Wilkie syndrome is a rare acquired disorder of intestinal obstruction in adults. Initial clinical manifestations are nonspecific. This process should be considered in the differential diagnosis in patients with vomiting. We speak of nutcracker syndrome when this disease is associated with compression of the left renal vein. The diagnosis of this entity is difficult, due to its low frequency and also because it produces nonspecific symptoms (back pain, hematuria...). We report a patient with SMAS, associated with asymptomatic nutcracker syndrome. A 31-year-old woman with a history of 3-month history of vomiting and loss of 13 kg of body weight. The body mass index was 15.5 kg/m². Analytical studies of blood and urine, including thyroid function, antigliadin and antiendomysium antibodies were performed, with normal results. Serology for infectious disease and the identification of fecal parasites were negative. Through an upper endoscopy and a duodenal biopsy, malabsorption and other disturbances in the intestinal wall were ruled out. According to a psychiatric study, there was no eating disorder. The radiological study showed dilatation of the duodenum due to a duodenal compression caused by narrowing of the angle between the aorta and the superior mesenteric artery. A computed tomography angiogram confirmed duodenal compression by vascular structures, and increased caliber of the left renal vein, the left gonadal vein and lumbar veins, due to the coexistence of a nutcracker syndrome. Conservative treatment was initiated with nutritional support. Due to the persistence of symptoms a duodenojejunostomy was performed. A barium study showed passage of contrast gastroduodenal through the duodenojejunostomy. At 2 months after surgery the patient continued showing weight loss. The patient didn't have any digestive symptom, but refused food intake. We conclude that we should think of this rare syndrome in patients with low body mass index, with vomiting of unknown etiology, and their possible coexistence with compression of the left renal vein without clinical nephrologic symptoms.

SC-50 H1N1 TYPE A INFLUENZA AND ACTIVE PLEUROPULMONARY TUBERCULOSIS

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Introduction: Since Type A Influenza is in a pandemic stage, when a test shows (+) results for the disease it is likely that other diagnosis are considered, especially if the findings indicate the presence of the mentioned disease. Recently a patient has been admitted to our hospital with Type A Influenza, along with H1N1 and pleural effusion. Lab tests confirmed tuberculosis in along with H1N1.

Case: A 25 year old white Hispanic woman, in a good condition, had sudden fever (40°C) and coughing along with stuffy nose, rhinorrhea and expectoration, general discomfort, headaches and myalgia. Two days later, she complains about a discomfort in the left side of the thorax resembling a pleuritic pain. Doctors decide go on with the medical exam finding a left side pleural effusion so she is hospitalized. No indication of previous contacts with affected by flu or TB. On Exam: good general state, no respiratory swelling or paleness, temperature 38.7°C; HR 108 and 95% of saturation. Hypoventilation in the left base was found. No lymphadenopathy. The rest of the exam normal. Tests included those for pneumonia, HIV and antigenuria(-). Throat culture (+) for H1N1A influenza; pregnancy test (-); sputum (-) and (+) for TB. Leukocytes 2990/mm³ (1530 neutrophil, 1070 lymphocytes/mm³), the rest of the tests normal. Blood cultures (-). Pleural fluid: yellowish, 1370 cells, 80% mononuclear; pH 7.38, proteins 4.59 gr/dl and LDH 564 U/l, ADA 69.2 U/l, microbiological and cytological tests (-). Mantoux: 15mm. X-rays showed pleural effusion with alveolar condensation in the left lobe. Patient initially treated with Oseltamivir and Levofloxacin, 5 days later, after receiving the (+) results for TB in sputum as well as the presence of an apical infiltration four other drugs were added. In 7 days, pleural effusion disappears. The patient is released with ambulatory follow-up. TB appears in the sputum culture. Ambulatory follow up is carried out with tuberculostatic for six months. At the end, X-rays shows the lung was normal and culture (-).

Discussion: The coexistence of these two diagnoses – H1N1 Influenza Type A and pulmonary tuberculosis are confirmed based on the tests and the combination of complementary tests such as PCR, X-rays, analysis of the pleural fluid with high levels of ADA and PCR and sputum culture (+) for Mycobacterium tuberculosis. At that moment it is not strange to conduct H1N1 A Influenza and TB detection tests in an independent way and in our environment, the occurrence of Influenza Type A is more frequent. However, their coexistence is rare. We have no reason to believe that a viral infection or an atypical pulmonary infection had facilitated the

development of TB in the patient; contrary to what occurs with bacterial pulmonary infections which are preceded by the above-mentioned infections. But it is known that other pulmonary infections such as TB can coexist, probably because TB opens up the way to infections, which could indirectly contribute to the morbidity and mortality rates of this disease.

SC-51 PATENT FORAMEN OVALE AND CRYPTOGENIC STROKE: DON'T BLOW THE HORN IF YOU HAVE A STROKE

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Introduction: Patent foramen ovale (PFO) is a common alteration (20%) in the general population and has been implicated in the etiology of cryptogenic stroke. There is controversy about the clinical utility of PFO closure in cryptogenic stroke patients.

Description: A 63 year old male was admitted because of an acute neurological deficit consistent with recurrent stroke. He was a wind-instrument musician. The patient had a previous right frontotemporal stroke in 2002. NMR demonstrated an old right cerebellar infarction that was previously unnoticed. Transthoracic and transesophageal echocardiography (TEE) were normal, carotid ultrasound did not show significant atherosclerotic lesions. Studies for hypercoagulable state were negative. The patient was discharged with aspirin, ACE inhibitors and statin therapy. The patient did not smoke and a regular follow up at a vascular risk clinic demonstrated optimal control of blood pressure and lipids levels. The patient was admitted again in 2006 because of a new episode of acute neurological deficit (weakness in the right leg lasting 20' with full recovery). MRI demonstrated an acute infarction in the left frontal lobe and multiple old ischemic lesions in different vascular territories highly suggestive of embolic origin. Carotid ultrasound and thrombolytic studies remained negative. 24h ECG monitoring demonstrated stable sinus rhythm. Cranial Doppler ultrasound and TEE with bubble contrast demonstrated the presence of patent foramen ovale with right-to-left shunt particularly prominent after Valsalva maneuvers. Low molecular weight heparin followed by oral anticoagulation was begun. Given the professional risk and the recurrence of ischemic events, the patient was referred for PFO closure with a GORE HELEX Septal Occluder device. The patient was maintained with acenocumarol for an additional 6 months and switched to aspirin thereafter. After 6 years of follow-up the patient remains free of new neurological symptoms.

Discussion: A recent prospective, randomized controlled trial^{1,2} suggests that PFO closure provides no clinical benefit in patients with cryptogenic stroke. However, the study included low-risk patients and used different antithrombotic therapy in patients assigned to PFO closure or medical therapy. We present a case of a patient with recurrent 'cryptogenic' strokes with a prominent right to left shunt with an additional risk as a wind-instrument musician whose clinical evolution changed dramatically after PFO closure. Carefully selected patients at high risk may benefit from PFO closure.

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SC-52 PAINFUL HEEL FAT PAD INFLAMMATION SYNDROME AS THE PRESENTING FEATURE OF TAKAYASU'S SYNDROME

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Case report: A 27 year old woman presented with a 4 weeks history of fever, subcalcaneal pain, weight loss and loss of appetite. The physical examination revealed pain over the heels, without arthritis or cutaneous lesions. The remainder of the examination was normal. Laboratory data revealed: C-reactive protein 15.68 mg/dl (normal value <0.5), ferritine 464 ng/ml (normal value <150), erythrocyte sedimentation rate 62 mm/h, hemoglobin 8.4 g/dl. Further laboratory evaluation of routine hematologic, coagulation, biochemical tests and urinalysis were otherwise normal. Tests for antinuclear antibodies, antibodies to double-stranded DNA, rheumatoid factor and antineutrophil cytoplasmic antibodies (ANCA) were negative, as well as results of test for B and C hepatitis and human immunodeficiency virus. Cultures of specimens of blood and urine were sterile. Full-body Computed Tomography, Gallium scans and technetium-99m scintigraphy were within normal ranges. A Magnetic resonance imaging (MRI) of the heel showed fat pad inflammation without plantar fasciitis, enthesophyte or bone lesions. Diclofenac and immunosuppressive agents with prednisone (0.4 mg/kg/day) and methotrexate (15 mg weekly) were prescribed without improvement, and methotrexate was discontinued. Then the patient was treated with local corticosteroid injection in the left heel also without relief. A rheumatic disease was suspected and adalimumab 40 mg every 2 weeks) was began 7months later and gradually improved her complaints. During a 12-month follow-up



period no remarkable signs were observed but did not resolved her raised acute-phase reactants and anemia and therapy with TNF-alpha blocker was stopped. On the following weeks fever and Raynaud phenomena developed and asthenia increased in severity. On examination no thoracic or abdominal bruits were found and distal pulses were normal. New testing for antinuclear antibodies, antibodies to double-stranded DNA, ANCA and complement were normal or negative. 19 months after his initial presentation, a computed tomographic angio-scan and MR angiography were performed and revealed stenosis of cranial and extracranial arteries. Angiographic features were consistent with Takayasu's arteritis and we elected to treat the patient with a return to corticosteroids and cyclophosphamide bolus (500 mg beweekly 3 months) with improvement. One year later, she is asintomatic and returning to work, without changes in the angioRM, only with 150 mg daily of azathioprine.

SC-53 ACUTE CEREBELLITIS AND TAKAYASU ARTERITIS CAUSALITY OR CASUALITY?

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Case report: Acute cerebellitis (AC) is common causes of acute ataxia in children but it's very infrequent in adults. It is characterised by headache, altered mental status, nystagmus and dysmetry following infection. The aetiology is unknown but it is frequently associated to infectious diseases. Takayasu arteritis (TA) is a chronic inflammatory arteritis, affecting predominantly the aorta and its main branches. It mainly affects women between 10 and 40 years. The main symptoms are weakness, fever, arthralgia. Sometimes a stroke can be the first manifestation. We report the case of a patient with a previous (AC) who later received a diagnosis of TA. The interest of this case is to set out if the relation between both inflammatory processes is accidental or causal. A 39 years old female, smoker, without cardiovascular risk or other personal or family medical history, was admitted to the hospital. She had had gastroenteritis some days before. The patient referred feeling progressively sick in the morning with cephalalgia, nausea and dizziness. Later she suffered loss of vision, strength and feeling in one half of her body. In the exploration, the patient showed horizontal nystagmus on the right, vertical nystagmus, 3^o nerve palsy, and diplopia. Finger to nose test showed pathology in the left side, tandem with lateropulsion to the left, hemiparesia and paresia of the right side of her body. Rest of the exploration was normal. Laboratory test showed leukocytosis with a sedimentation rate of 64 mm/h, the rest with thyroid hormone, ANA and serology test was negative. EKG and Chest-X-ray was normal. A lumbar puncture was normal with negative GRAM and culturing. The NMR which shows hyperintense lesions in DP, T2 and FLAIR located in both cerebellar hemispheres, although in the left hemisphere they are more extended. The patient progressed favourably with corticotherapy IV. After discharge she was revised in the neurology department where a minimum dysmetry, left dysdiadochokinesia and a grade 4/5 power in the proximal muscles of the left arm persisted. 8 months later the patient was admitted in the hospital with speech difficulty and an acute hemisensory loss of the face. On physical examination she had weakness of both arms specially the left arm with weaker pulses in the left upper limb. Blood pressure was 80/60mm Hg on the right arm and 70/40 mm Hg on the left arm. A systolic bruit could be heard in both subclavian arteries. Her higher mental functions and cranial nerves were normal. Cardiac and respiratory examinations were normal. On admission, an urgent CT revealed: Encephalomalacia areas secondary to cerebellitis. The laboratory test showed only mild leucocytosis and erythrocyte sedimentation rate (ESR) was 94. Chest X-ray was normal. The CT performed forty-eight hours later revealed Ischemic stroke in the territory supplied by the middle cerebral artery (MCA). An AngioCT was done and it showed: meaningful stenosis of the origin of the left subclavian artery and a narrowed of the right subclavian artery because of thickening of the wall. She also had meaningful stenosis of the left common carotid artery before the bifurcation in an 80% approximately. Following the criteria of the American College of Rheumatology our patient met 5 criteria of six, so she started on prednisone treatment 60mg. The patient was doing well with return in speech.

Discussion: This is a case in which two inflammatory infrequent diseases, which affect CNS of the same patient are described and we think about the possible association between them. Many associated neurologic and autoimmune diseases have been described with TA. However, until now, no associations have been described with AC. Some of the signs of TA can initially coincide with the ones of AC. Therefore we consider the possibility that the association between both diseases is underdiagnosed. On the other hand, AC generally fully improves and in our case the patient improved but a certain sign of neurologic focality persisted. It may make us think about a possible diagnosis associated. Could TA be a possible cause of AC? Currently the most frequent aetiology of AC is the idiopathic one and between the known causes, the most frequent are the infectious ones.

SC-54 CRION: AN UNKNOWN ENTITY

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Key-words: Optic neuritis; CRION; multiple sclerosis; neuromyelitis optica; aquaporin-4 (AQP4) antibodies.

Introduction: Chronic relapsing inflammatory optic neuropathy or "CRION" is a rare entity among inflammatory optic neuritis. Infectious, tumor, system vasculitis and auto-immune disorders must be eliminated by an extensive research.

Case report: A young 22 year old man without any personal or familial antecedents had a severe recurrent and bilateral retro bulbar optic neuropathy. Six attacks were registered over eighteen months with a bad visual recovery in spite of steroid treatment. Azathioprine also had been introduced after 4 attacks. The neurological examination found a relative afferent pupil defect and hypotonia in the legs. Visual evoked potential examination showed a prolongation in latency in both sides. Initial nerve image showed an enhancement of the right optic nerve. Repeated brain and medullar magnetic resonance imaging (MRI) did not display any abnormality; neither did the cerebrospinal fluid (CSF) analysis.

Discussion: Inflammatory optic neuritis can occur in several circumstances. Severe visual loss, recurrent bilateral optic neuritis, bad response to treatment, steroid dependence and negative initial research are elements which allow us to use the term CRION. An extensive investigation to find an etiology is necessary. In this case, demyelinating etiologies as multiple sclerosis and neuromyelitis optica have to be considered. Positive research of Aquaporin4 antibodies constitutes an additional tool to predict a future evolution to NMO. Nevertheless, its accomplishment had not been possible for our patient. The treatment for CRION is based on steroids and a long term immunosuppression.

Conclusions: The CRION is a subgroup among idiopathic optic neuritis. In spite of a large investigation, its etiology remains often undetermined. However, the patients are considered as a high NMO risk. So, a long multidisciplinary follow-up of these patients, repeated brain and medullar MRI, CSF analysis and AQP4 antibodies research seem to be essential to detect a possible evolution to defined demyelination pathology.

SC-55 PRIMARY NEUROBRUCELLOSIS DISCLOSED BY ATAXIA

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Key-words: Neurobrucellosis; Ataxia; Brain MRI; T2 white-matter signals.

Introduction: Brucellosis is the most frequent zoonotic infection in the world, in particular in the Mediterranean basin. It is endemic in Algeria and its notification is obligatory. Neurobrucellosis disclosure by ataxia is uncommon.

Case report: A 42-year-old man, living in a rural area of west Algeria suddenly presented acute mental confusion and high fever. The neurological examination found only ataxia and brisk tendon reflexes. He had hiccups too. All these symptoms started two days before. Erythrocyte Sedimentation Rate and C-reactive protein were increased. The brain magnetic resonance imaging (MRI) showed multiple nodular T2 white-matter signals. The cerebrospinal fluid (CSF) analysis displayed lymphocytic pleocytosis (20 elements/mm³), elevated protein content (1.38 g/l) and reduced CSF plasma/glucose ratio (0.45). Standard tube agglutination (STA) test on blood was positive at 1/160. So, brucellosis was confirmed. Treatment included doxycycline 200 mg/ day and rifampicin 1200 mg/day for 6 months. A complete resolution was achieved.

Discussion: Ataxia in neurobrucellosis has rarely been reported as a first symptom. In this case, it belongs to a primary form of neurobrucellosis. Nervous tissue may be directly affected by brucella or its toxins. Positive STA test on serum and/or CSF confirm diagnosis; but leucoencephalopathic lesions in the brain MRI as those presented by our patient can help too. Early specific treatment generally leads to a good outcome. 2 or 3 antibiotics that have a good brain diffusion and a synergic action have to be associated. The most commonly used are doxycycline, rifampicin and trimethoprim-sulfamethoxazole. Ceftriaxone can be used too. Treatment must be continued at least 2 months up to 6-12 months.

Conclusions: Brucellosis is a polymorphic infection disease. The clinical presentations are heterogeneous. Neurobrucellosis represents a rare and a serious form. It is so essential to think of neurobrucellosis when a patient presents a central nervous system symptom as ataxia, especially in an endemic area.

SC-56 CASE REPORT: MADELUNG'S DISEASE

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Introduction: Madelung's disease (MD) is characterized by painless adipose tissue mass symmetrically distributed mainly on neck, nape, trunk and proximal limbs. It affects men between 30 and 60 years of the Mediterranean area with chronic alcoholism and metabolic disorders.

Statement: 69 year old man with cervical-thoracic, abdominal and scrotum lipomatosis since he was 34 years old. He was derived to us for dyspnea of 3 years. Backgrounds: obesity (BMI 35.8 kg / m²), ex-smoker, chronic alcoholism (130 gr / OH day), cirrhosis. Elevated acid uric and triglycerides, hypothyroidism. Diagnosed at age of 45 when lipomatosis was widespread and after being intervened on several occasions of "lipomas" with recurrences. The biopsy revealed mature adipose lobules tissue. We found an obese patient, painless cervical lipomatosis occupying the entire circumference, chest, abdomen, and scrotum. Complementary proves: laboratory tests GGT 81 U / L, bilirubin 1.38 mg / dl, MCV 105.2 fL, uric acid 8.0 mg / dl. Echocardiogram, EF 72%, diastolic dysfunction type 1. CT and ultrasound of abdomen didn't identify fatty infiltration. Respiratory function tests, mild restrictive pattern.

Discussion: Because of obesity, cardiovascular risk factors and smoking history, echocardiographic and functional tests were requested, which revealed type 1 diastolic heart failure and mild restrictive lung disorder secondary to obesity. We diagnosed a multifactorial dyspnea. We decided to propose surgery but it was rejected by high risk. The pathogenesis theories relate the disease (2) with alcoholism, familial aggregation, protease inhibitors, effects on glucose and lipid metabolism, mitochondrial disorders (recently the gene mutation 8344 tRNA (lys) of mitochondrial DNA has been associated with alterations in oxidative metabolism) (3). Also been reported in patients not alcoholics (4). Up to 85% have concomitant neuropathy (4). Histologically usually is unencapsulated brown adipose tissue with normal adipocytes but smaller multivacuolated.

Conclusions: MD is an uncommon disease characterized by painless adipose tissue mass of unknown etiology. It's usually associated with alcoholism. The diagnosis is clinical, supported by imaging and histology. Stop alcohol consumption and diet, may stop or slow the progression. The gold treatment is surgery.

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SC-57 ACQUIRED HEMOPHILIA A

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Case report: A 39-year-old healthy woman was admitted to the hospital after presenting with hematuria and cutaneous hematomas. Past Medical History was significant only for a normal pregnancy 14 months earlier. Routine blood tests during the pregnancy were normal. She was not taking any medications. On presentation her physical exam showed multiple cutaneous hematomas. Significant findings on blood tests: hemoglobin 9.6g/dL, platelets 296x10⁹/L, reticulocyte count 53.5x10⁹/L, prothrombin time (PT) 13.5seconds, INR 1.1, activated partial thromboplastin time (aPTT) 75.3seconds, aPTT ratio 2.2, fibrinogen 612mg/dL, total bilirubin 0.33mg/dL, direct bilirubin 0.09mg/dL, LDH 414U/L. Autoimmunity tests, immunoglobulins, urine and serum immunoelectrophoresis were normal. Tumor markers, Bence Jones proteins and cryoglobulins were undetectable. HIV, HBV and HCV serologies were negative. She was admitted to the hospital for bleeding diathesis and an abnormal aPTT. Progressive deterioration of aPTT with increasing anemia was detected. The clinical presentation was likely secondary to aPTT prolongation, most probably due to a device present on plasma sample; factor VII, IX or XI deficit, von Willebrand Disease (vWD) or circulating anticoagulant inhibitors. The initial diagnostic suspicion was an acquired hemostatic disorder (less likely the use of heparin or vWD). The mixing test showed a persistent prolongation of the aPTT (46.1 seconds; ratio 1.32). This suggested the presence of a factor VIII inhibitor. Factor VIII levels were decreased (3%). Levels of factors VII, IX, XI and XII were normal. The diagnosis of hemorrhagic diathesis secondary to acquired factor VIII deficiency (acquired hemophilia A) was made. To control active bleeding, human recombinant factor VIIa (90mcg/kg/8hours) was given; to eliminate factor VIII inhibitor, prednisone (1mg/kg/day) was used. Gross hematuria persisted and cyclophosphamide (2mg/kg/day) was added. Cessation of bleeding was achieved after 5 days. A decrease in inhibitor titers and normalization of aPTT occurred after 2 months. The incidence of acquired factor VIII inhibitor is 1.3-1.5cases/million/year. Major identifiable causes are pregnancy or postpartum period, rheumatoid arthritis, malignancy, SLE and drug reactions. In almost one half of the patients, no underlying disorder is present. This patient had been pregnant 14 months earlier, maybe could be considered the precipitating cause of her bleeding diathesis.

SC-58 SEVERE HYPOCALCEMIA AS A PRIMARY PRESENTATION OF CELIAC DISEASE

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Introduction: Celiac disease (CD) is an immune-mediated inflammation of the small bowel caused by dietary gluten sensitivity and related proteins in genetically prone individuals. CD is a common condition which may affect all ages and that involves several organ systems. Although hypocalcaemia is a frequent manifestation in CD it is not common that severe hypocalcaemia with associated tetany announce the onset of this disease as in the present case. The gold standard of CD treatment is a strict gluten-free diet and supplementing of deficiencies [1].

Case report: A caucasian 30-year old woman, previously healthy, was admitted in the emergency room because of cramps in her extremities. Previous week she reported diarrhoea. Stools were watery and did not present blood, mucus or pus. She was afebrile. Physical examination was unremarkable. Biochemistry showed severe hypocalcaemia with calcium (Ca) levels were 6 mg/dl (N 8.6-10.2) and ionic Ca was 0.87 mg/dl (N 1.15-1.29). Other laboratory parameters and complementary explorations were normal. She declined admission as she was breastfeeding and she was discharged with oral treatment (Ca, vitamin D and magnesium). When the patient came to outpatients office she was still breastfeeding and with no diarrhoea, but persisting cramps with both Trousseau's and Chvostek's positive signs. An outpatient laboratory revealed: Ca was 5.5 mg/dl, magnesium 1.1 mg/dl (N 1.7-2.2), ionic Ca 0.87, parathormone (PTH) 79 pg/mL (N 10-65), Fe 30 µg/dl (N 37-145), folic acid 3 ng/mL (N 6-20). Other laboratory parameters were normal. D vitamin levels were: 1-25 dihydroxyvitamin D 47 pg/ml (N 18-78) and 25-hydroxyvitamin D 23 pg/ml (N 10-29). Stool culture and trypsin test were negative. Computed tomography showed loops distension in the small bowel with inner liquid, in which a round "doughnut-like" shape was visualized at hypogastrium that could be a potential intussusception. Antiendomysial and anti-transglutaminase antibodies values were 1/320 and 22.6 U/ml respectively. Due to the high levels of antibodies found an upper GI endoscopy was performed and confirms the high clinical suspicion for CD. Dual-emission X-ray absorptiometry showed osteopenia (-2.4 T-Score at L2- L4 level). The present case is diagnosis adult CD presenting with severe hypocalcaemia (with associated tetany) in the frame of a malabsorption condition and with associated iron deficiency, moderate deficiency of vitamin D, folic acid deficiency and hypomagnesaemia. The patient adheres to treatment consisting of a gluten-free diet, and oral treatment with Ca and vitamin D, magnesium, iron and folic acid. She showed improvement and osteopenia levels decreased. At present she only takes Ca and vitamin D. After one year, Ca is 9.3 mg/dl, PTH 46pg/mL, Fe 97 µg/dl, folic acid 11.1 ng/mL and the antibodies were negative.

Conclusion: CD is a common enteropathy however it is under-diagnosed. Manifestations in the adulthood, especially in those patients presenting atypical signs, stand for the 60% of the current diagnosed cases [2]. Our case presented severe hypocalcaemia, an unusual manifestation of CD due to the malabsorption, as well as hypomagnesaemia which required a replacement treatment to readjust normal levels of Ca and magnesium.

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SC-59 A CASE OF ESOPHAGEAL INTRAMURAL PSEUDODIVERTICULOSIS (EIP)

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Objectives: EIP is a rare, benign condition caused by dilation of the deep esophageal glands, giving rise to multiple outpouchings of the wall of the esophagus. A variety of esophageal disorders have been proposed in the genesis of EIP.

Material and methods: We describe the case of a 47-year-old woman who received radiotherapy for a nasopharyngeal carcinoma 20 years ago. In addition she presented corrosive esophagitis and esophageal stenosis due to ingestion of caustic agents and esophageal dilations were necessary. She subsequently developed mechanical dysphagia and esophageal stenosis secondary to caustic agents and radiotherapy, and required repeated dilations of the proximal esophagus.

Results: Esophagogram and endoscopies were performed showing no evidence of cancer relapse or esophagitis. Outpouchings of the wall of the esophagus were demonstrated. Conservative treatment was undertaken. *Candida krusei* and methicillin-sensitive *Staphylococcus aureus* were recovered from brush cytology.

Discussion and conclusions: EIP requires no treatment in the absence of symptoms and it is directed towards treating underlying conditions and relieving symptoms. Serious complications are rare. Our patient was successfully treated with endoscopic dilation, and six months after, she remains asymptomatic.



SC-60 DO I SUFFER FROM MICROSCOPIC COLITIS (MC)?

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Objectives: MC is a disorder of unknown etiology that causes chronic diarrhea. As the colon appears normal during endoscopy, MC can only be detected on histologic examination of a biopsy specimen.

Material and methods: We report the case of a 34-year-old man who underwent hemorrhoidectomy and rectal polyp removal in his youth. He consulted for chronic non-bloody, watery and often nocturnal diarrhea, often associating diffuse abdominal pain. No dehydration was found.

Results: A total colonoscopy revealed no pathological findings. No pathological microorganisms were revealed on stool sample evaluation. There was no evidence of concomitant autoimmune disease and all laboratory tests were within normal range. A biopsy was performed, revealing histo-pathological findings consistent with MC.

Discussion and conclusions: MC may be present in patients meeting diagnostic criteria for irritable bowel syndrome (IBS). Although our patient met IBS criteria, the biopsy revealed MC, which changed our course of management. To the best of our knowledge, the amount of patients who meet IBS diagnostic criteria who in fact have MC is unknown. More studies in this direction are needed in order to assess if patients who meet IBS criteria may benefit from a biopsy to evaluate for MC.

SC-61 THROMBUS IN TRANSIT: HUNTED BY YOUNG INTERNIST PERFORMED BEDSIDE ECHOCARDIOGRAPHY

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Case report: A 88-year-old man was admitted to the internal medicine ward because of dyspnea and one year history of weight loss. Physical examination was normal except for low grade fever. A 12-lead ECG showed only sinus tachycardia and a plain anteroposterior chest radiograph was unremarkable. Blood tests showed microcytic anemia, High C-reactive protein, low serum albumin, iron deficiency and normal tumor markers. Blood cultures grew *Streptococcus bovis* and *Escherichia coli*. Bedside transthoracic echocardiography performed by the attending internist showed moderate aortic regurgitation and AN incidentally visible thrombus prolapsing across the tricuspid valve, without visible vegetation. Twelve hours later this thrombus had simply disappeared from atria in a new echocardiogram performed by the cardiologist. An immediate CT pulmonary angiography confirmed a big thrombus lodged in the main pulmonary artery with absence of filling in the segmental branch of the left basal pyramid artery. Heparin was prescribed despite of anemia, but had to be stopped because of lower gastrointestinal bleeding. Colonoscopy revealed colorectal cancer, that we can directly relate to *S. Bovis* bacteriemia, as the predisposing cause of the thromboembolic disease. Surgery was rejected based on previous Barthel index and the outcome was fatal. Acute pulmonary embolus with visible right-heart thrombi is a rare phenomenon, diagnosis of this entity has been reported almost exclusively with echocardiography, and it is associated with a bad prognosis, the estimated in-hospital mortality of thrombus-in-transit is thought to exceed 45%. This case highlights the importance of bedside transthoracic echocardiography which probably will increase the identification of cases with thrombus in-transit.

SC-62 THE LORD OF THE RINGS – A PECULIAR PRESENTATION OF MICROSCOPIC POLYANGIITIS

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Case report: Microscopic Polyangiitis (MPA) is an anti-neutrophil cytoplasmic antibody (ANCA) associated vasculitis, with small vessel necrotizing inflammation. It can affect several organs. We describe a 60 year old caucasian male, presenting in the Emergency Department (ER) with 5 months' complaints of late afternoon relapsing fever with chills. He also complained of asthenia, anorexia and a 6 kg weight loss. A week prior to admission, the patient noticed 3 erythematous skin lesions (on the left inner thigh and gluteal areas), painless and non-pruriginous. He also complained of lower limb myalgia. He remembered a similar episode one year later. Basic ambulatory diagnostic workup revealed inflammatory anemia (Hb 11 g/dL) and a C-reactive protein (CRP) of 18 mg/dL; normal renal function was documented a few months before. No relevant personal or familial history was detected. In the ER he was noted to be febrile (39°C), pale and had uremic breath. He had a 6 cm wide target-like skin lesion on the left inner

thigh, with erythematous border and scaly center, and 2 other smaller similar lesions in the gluteal areas. The ER workup showed normocytic normochromic anemia (Hb 11.8 g/dL), increased CRP (17.9 mg/dL), urea (185 mg/dL) and creatinin (9.1 mg/dL). Arterial blood gas revealed metabolic acidosis (pH 7.35, HCO₃- 18.1 mmol/L). He was started on urgent dialysis. He was admitted as an inpatient and infectious, inflammatory and neoplastic diseases were pursued. In the inflammatory group, the prime suspects were systemic lupus erythematosus and ANCA associated vasculitis. Lyme disease was also investigated because the skin lesions fulfilled criteria for epidemiologic definition of case. Further studies showed an erythrocyte sedimentation rate of 104 mm/1h, proteinuria of 835.66 mg/24h. Serologies were negative for HIV, syphilis, *Borrelia burgdorferi*, *Rickettsia conorii*, *Coxiella burnetti*, *Leptospira spp*, Huddleson test. The head, thorax, abdomen and pelvis CT showed no suspect lesions. Kidney biopsy diagnosed a pauci-immune necrotizing crescentic glomerulonephritis. Dermatology identified the lesions as erythema annulare centrifugum (the skin biopsy showed only superficial perivascular dermatitis). Anti-MPO ANCA positivity was found (165.47 U/mL); the rest of the immunology study was negative. The association of skin lesions, rapidly progressive renal failure with necrotizing crescentic glomerulonephritis and MPO-ANCA positivity, and systemic features led to a diagnosis of MPA. Glucocorticoids (GC), plasmapheresis and cyclophosphamide were prescribed. Regression of symptoms and cutaneous lesions was achieved and the patient is dialysis free. Despite therapy, 2 months after the diagnosis he presented with steppage. EMG showed a demyelinating peroneal mononeuropathy. GC therapy was stepped up. Skin lesions are frequent in MPA, prevalence ranging from 20 to 70%. So far, erythema annulare centrifugum has never been reported as a skin manifestation of MPA.

SC-63 FAST PROGRESSION CHONDROSARCOMA IN WOMAN WITH OPERATED POLINODULAR GOITER— A CASE STUDY

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Introduction: Chondrosarcoma is a rare disease. The most frequent locations are the pelvis, legs or shoulders in patients over 40 years.

History: In Nov 2011 a female patient aged 67 years came to our clinic. She complained about the development of a tumor in the last 5 weeks positioned in the lower sternum, accompanied by compressive phenomena. Patient was known previously with hypertension, diabetes mellitus, tiroidectomy. Clinical examination - prominent tumor in the lower portion of the sternum 5/3cm in size, hard, adherent to the bone and skin. The tumor was not sensitive to pressure and with no alteration of the adjacent skin.

Investigations: Soft tissue echography - encapsulated tumor 5/3cm with thick walls, central necrosis and with no Doppler signal in the periphery. On the posterior wall there was a poorly delimited tumor 1.4cm with central calcification and vascular strands. Transthoracic echocardiography - chist anterior to the right ventricle. Thoracic CT - tumor in contact with the pericardium over the right ventricle. Mixt structure, predominetly liquid with solid septum and calcifications 9/8/5cm. The presence of micro calcifications suggested a diagnosis of chondrosarcoma. Bronchoscopy excludes a primary bronchic tumor. Gastroscopy excluded a gastric tumor only a chronic gastritis.

Treatment: The thoracic surgery recommends, excision of the tumor and histopathology examination. In Dec 2011 the tumor was removed by surgery. The surgical piece was sent for histopathology and revealed a grade 3 chondrosarcoma touching the excision margins. Post surgery radiotherapy with 60GY irradiation for 30 sittings was done. At the 6 months follow up, patient's status was good and the control CT did not reveal a relapse.

Particularity: The patient's tumor developed over a short period 5 week and the radiografic and CT investigation did not reveal any bone destruction which is characteristic for the extraskeletal chondrosarcoma, although its position near the sternum might suggest a skeletal chondrosarcoma. This case was found in a patient with no risk factors for chondrosarcoma such as Olliere disease or Maffuci syndrome or metachondromatosis.



SC-64 FEMALE WITH ABDOMINAL PAIN AND FEVER DURING A POSTOPERATIVE

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Anamnesis: Female, 82 years old, admitted to hospital due to pertrochanteric fracture. On sixth day after the surgical procedure, the patient refers abdominal pain, nausea and vomiting.

Physical examination: The patient presented ill looking aspect, sweatiness, drowsiness and tachypnea. Blood pressure: 80/40; Heart rate 120 bpm; Body temperature: 101.3 °F; O₂ Saturation 82%; Auscultation cardiopulmonary: dispersed ronchi. Abdominal examination: soft, tender and dispersed painful on palpation, low intensity bowel sounds.

Diagnostic tests: A blood test highlights severe hypokalaemia, hypocalcemia and CRP elevation. The chest radiograph reveals an alveolar infiltrate in the right lung base. The patient was treated with intense hydration and Piperacillin/Tazobactam after blood cultures extraction. One day later, vancomycin was added to the therapy. An ultrasound of abdomen was reported as normal. Blood cultures were negative. The patient worsened progressively and an abdominal CT was consistent with adrenal gland hyperplasia or bleeding of the same. Cortisol levels were requested, the results were 9.54 mg/dL, ACTH 420.1 ph/mL. A Synacthen test showed cortisol levels of 9.78 and 9.84 (after 30 and 80 minutes), thus confirming the existence of primary adrenal insufficiency. The patient was treated with Hydrocortisone, being asymptomatic 48 hours after initiation.

Clinical judgement: Shock secondary to bilateral adrenal hemorrhage.

Differential diagnosis: The clinic present in our patient, abdominal pain, fever and hypotension, is often interpreted as septic shock. In the differential diagnosis of adominal pain, fever and hypotension we must consider gastrointestinal and hepatobiliary diseases, gynecological, endocrine-metabolic, hematologic and infectious disease or referred pain.

Discussion: The bilateral adrenal hemorrhage is an often fatal entity whose incidence is increasing due primarily to improved diagnostic tests specially CT. The diagnosis of acute adrenal hemorrhage should be considered in patients with a progressive deterioration of general health with unexplained pain in the abdomen, circulatory failure, fever, and hypoglycemia. A review study evaluated 431 cases of bilateral adrenal hemorrhage, we know that stress is the most common predisposing factor such as the surgery, extensive burns or trauma were responsible for 26% of cases, followed by severe infections or severe heart problems.¹ Acute adrenal insufficiency is mainly attributable to mineralocorticoid deficiency, the clinical presentation is dominated by hipotension or hypotensive shock.² To check the primary source of adrenal insufficiency should be taken blood to test basal cortisol, subsequently administered 0.25 mg intravenous Synacthen with additional extractions at 30 and 60 minutes. The stimulation is positive if cortisol levels reach over 18 mg/dL.³ Suspecting acute adrenal insufficiency should act quickly, starting with an infusion of hydrocortisone intravenously.⁴

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SC-65 A THIRTY-THREE-YEAR-OLD WOMAN WITH ANEMIA, LIVER AND KIDNEY DISEASE, HYPERCHOLESTEROLEMIA AND “STRIKING” EYES

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Case report: Athirty-three-year-old woman was referred to internal medicine presenting 850 md/dl cholesterolemia with 8 mg/dl HDL and 136 mg/dl triglycerides. The patient was asymptomatic with palpable splenomegaly of 3 cm and hepatomegaly of 1cm. She presents with yellow, rough to the touch papular lesions of less than 3 mm on both lateral sides of upper and lower extremities; “glassy” eyes with bilateral corneal opacity and yellow halo around the iris. Other laboratory data: slight normochromic normocytic anemia. ALT-415 U / L, AST 324 U / L, GGT 268 U / L, ALPK 369 U / L, BT 1.5 mg / dl. Urinalysis microalbuminuria of 19.5 mg / dL and microhematuria of 9 erythrocytes / field. Copper level, ceruloplasmin, alpha-1-antitrypsin, and AFP were normal. Serology for HAV, HBV, HCV and HIV 1 +2 negative and autoimmunity study with anti-nuclear, anti-smooth muscle, anti-mitochondriales, anti-LKM and negative anti-parietal cells. A band of 12% between albumin and alpha-1-globulins is appreciated in the proteinogram. ApoA (154 mg / dl), ApoB (145 mg / dl) and Lp (a) <6 mg / dl, are determined to be within normal values. ApoCII (118.5 mg / L) and ApoCIII (253.6 mg / L), were determined very high compared to their reference values: (ApoCII: 15-40; ApoCIII:55-105 mg / L). Liver biopsy was performed reflecting a mild microvacuolar steatosis and skin biopsy where macrophage-like cells intensely lipidized or xantomized were observed. The discordance between ApoB and total cholesterol, decreased HDLc with normal ApoA, elevated ApoC and anomalous

peak in the proteinogram, make us suspect the presence of a lipoprotein X (LPX). This is an abnormal lipoprotein rich in free cholesterol, phospholipids, and ApoC but poor in ApoB with high content in albumin (responsible for the anomalous peak obtained in proteinogram) described in: primary biliary cirrhosis and other liver cholestasis, in the deficit of LCAT and the administration of parenteral nutrition rich in phospholipids among others. The suspected presence of LpX, a HDL of 8 mg / dl and bilateral corneal disorder as well as the ruling out of liver disease as explanation for the rise of LPX, make us suspect a possible deficit of LCAT. This is an enzyme which esterifies free cholesterol with an acyl group of lecithin to form esterified cholesterol and therefore its deficit increases free cholesterol. Confirmation is requested to a second laboratory which quantifies the free cholesterol that is increasing to 89% of total cholesterol (reference value up to 40%). HDL was separated by ultracentrifugation where the total free cholesterol associated to HDL was found to be 83% (baseline up to 34%). LCAT enzyme activity, measured as cholesterol esterificadora activity, was undetectable. Sequencing of LCAT gene was done, finding a undescribed homozygous mutation Gly119Asp confirmed by restriction analysis with the enzyme MvaI.

SC-66 COLD-INDUCED URTICARIA

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Case report: Cold-induced urticaria (CU) is a syndrome that, as the name implies, develops on cold exposure. Characterized by pruritic wheals (hives) and/or angioedema, lesions can be strictly limited to the site of contact or can be generalized, with a risk of anaphylaxis. The underlying cause and pathophysiology are largely unknown. The diagnosis is principally clinical and it is confirmed by cold challenging tests (ice-cube-challenge test). Nevertheless, serologic tests are necessary to exclude secondary entities as cryoglobulinemia or infections. The authors present the case of an 18-year-old female with pruriginous erythema and papules after exposure to cold environments, especially cold air and water. Family history was found, with her grandfather having the same disorder. The ice cube test was positive and cryoglobulinemia and infections were excluded. There was a good response to treatment with antihistamine; an adrenergic drug was available in case of a life threatening situation. This case highlights an underdiagnosed disorder that can be properly treated if early recognized.

SC-67 HEMATIDROSIS: A RARE CLINICAL CASE AND REVIEW OF THE LITERATURE

Clinical case chosen for the presentation at the Young Internists' Day-Clinical Gymnasium

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Case report: Only a few cases of the rare phenomenon hematidrosis (bloody sweat) have been described in scientific literature. The underlying causes and pathological features are yet to explain but extreme psychogenic components like acute fear and persistent mental contemplation were found to be associated with this condition. We present a case of skin bleeding in a 44 year-old-man who was in acute stress because of his mother's death. Spontaneous bleeding from his intact skin especially in the forehead, face and arms was witnessed by our staff on more than 5 times. The bleeding ceased spontaneously. Bloody extravasation had identical cell components as that of peripheral blood and laboratory tests were normal. Histopathology examination was not possible because he was in panic and refused biopsy. We present a review of the literature bringing discussion the pathophysiology of this condition.

SC-68 ATYPICAL PNEUMONIA

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Introduction: A sixty-year-old woman with cough and expectoration

Personal history: Allergy to ASA and NSAIDs. Infiltrating ductal carcinoma of right breast in 2008 without evidence of disease at present. Joined in Internal Medicine 2 months ago with fever of unknown origin, without reaching certain diagnosis. Among the tests performed, we highlight: CT abdomen with hepatic simple cysts (the largest size of 11 cm which is occupying the seventh and sixth hepatic segment).



Current disease: Before joining hospital the patient was suffering from cough and abundant expectoration, dark (like chocolate) with breathlessness and pain on both sides.

Physical examination: T° 37.7, TA 170/100 and 92% SatO₂. AP: decreased breath sounds at right base.

Testing: Inflammatory anemia and leukocytosis with left shift. ESR and CRP elevated. In biochemistry it is emphasized high levels of alkaline phosphatase. Rx thorax: we notice increased density in right base probably related to condensation and possible lung pleural effusion. CT chest: image consolidation with air bronchogram at the level of the middle lobe and right lower lobe with minimal pleural effusion. Besides large fluid collection located in the liver. Liver MRI: right inferior lobe of lung cavity which communicates with cavity greater subdiaphragmatic probably intrahepatic. Amoeba and hydatid serology were negative.

Evolution: Hepatobronchial fistula was suspected and the liver MRI requested confirmed the diagnosis. She followed treatment with imipenem and metronidazole for 2 weeks. We discuss the case with general surgery, but eventually, due to the favorable evolution of the patient both clinically and in control CT, it is decided to wait and see.

SC-69 CLASSICAL HOMOCYSTINURIA AS A RARE CAUSE OF MASSIVE PULMONARY THROMBOEMBOLISM

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Case report: Homocystinurias are defined as the set of inborn errors of metabolism of methionine (essential amino acid) resulting in increased homocysteine and deposit, this being an intermediate degradation product of methionine, leading to clinical manifestations by direct toxicity to the endothelium of blood vessels, increased platelet adhesion and increased proliferation of smooth muscle cells, also will affect the synthesis of collagen and elastin tissue, so that also affect the musculoskeletal system and cutaneous phenotype very similar to Marfan's disease, ectopia lentis is characteristic. Mental retardation is present in 50% of cases cystathionine deficit. Currently the bulk of cases are due to cystathionine β -deficit synthase (CBS) that names the classical homocystinuria, whose clinical expression may be summarized by mental retardation, marfanoid habitus and thromboembolic events. Approximate incidence of neonatal screening as 1/200,000-300,000 live births, although recent studies based on mutational analysis suggest a higher frequency between 1:20,000 or greater. His inheritance is autosomal recessive. The gene encoding the enzyme protein CBS is in the long arm of chromosome 21 (21q22.3). Today we have found more than 130 mutations, 72% of whom (72%) are specific and private. The degree of enzyme activity and intensity of clinical manifestations depend on whether it is a homozygous or heterozygous with plasma total homocysteine concentrations very high between 100-250 mmol / L. We present a male patient of 17 years, a native of Ecuador, with a family history of a twin brother died at age 7 of unspecified etiology, dwarf maternal uncle, father high of 1.90 cm in height, as a personal highlight, psychotic disorder study (ANA's positive cognitive tests low score) in olanzapine requiring income one month prior, congenital dislocation bilateral crystalline diagnosed his 15 years of age and he was sent to study pediatrics by phenotype Marfanoid: also was referred for cardiological with echocardiogram showing minimal pulmonary insufficiency and tricuspid no pathological significance. Complete analytical study was requested and genetic, that the patient did not follow. The patient enters the ICU with endotracheal intubation and mechanical ventilation following respiratory arrest in street and advanced resuscitation, obtaining a first rate sinus tachycardia with cash bundle branch block morphology S1Q3T3 right, with a tendency to bradycardia and hypotension. Family tell of progressive exertional dyspnea to be done with minimal effort and fatigue since his high Psychiatry Unit last month. On physical examination highlights coma unresponsive bilateral mydriasis with no corneal reflex, habit Marfanoid preserved with anthropometry, arachnodactyly, high arched palate, large teeth and crowded. First control blood gases: pH 6.92 pCO₂ 117mmHg, P0₂ 86.1mmHg, bicarbonate 16.6, EB 19. CT scan was performed showing diffuse cerebral edema suggestive of brain damage postarrest. In the analytical study highlights Leukocytes $19.900 \times 10^9 / L$ (82% N, 5% L, 13% bands), creatinine 1.2mg/dL, calcium 7.1 mg / dL, sodium 149 mmol / L, potassium 6.1mmol / L, troponin 0.61 ng / ml and CK of 1483 U / L. Curve troponin I: 6h: 6.72 ng / ml, 12 h: 10.54 ng / ml, 18h: 10.75 ng / ml. Quick 63%, aPTT 28.8 sec, fibrinogen 507 mg / dl with other parameters within normal limits and urine toxin negative. The clinical and ECG findings suggest a pulmonary tromboembolism cause of cardiac arrest so that a CT scan of thorax showing multiple filling defects in both main pulmonary arteries and almost all segmental and subsegmental arteries of all lobes of both lungs compatible with acute massive pulmonary embolism. Since joining the patient is unfavorable from the viewpoint presented neurological BIS 2 TS 100% and peak systolic TCD with bilateral. Patient progressively worsening clinical EEG and neurological examination to confirm brain death so just be death, after 48 hours of admission. Postmortem laboratory tests are performed to confirm the suspected diagnosis from differential diagnosis between entities that present with bilateral dislocated lenses: Sd. Homocystinuria versus Marfan, being the study of normal porphyrins, featuring a free homocysteine concentration in plasma and urine high (plasma levels 194.00 mmol / L (5.00 - 12.00) with no increase in plasma methionine and urine methylmalonic acid pattern compatible with CBS deficiency. culture of fibroblasts in skin biopsy which shows defect characterized by reduced activity and affinity for the cofactor. The differential diagnosis of the case must be made clinically with Marfan syndrome, and the processes that occur with increased serum Hcy. In the case of the Marfan phenotype is similar, also with bilateral dislocation of the lens but in senior

management, in terms of cardiovascular complications: in the Marfan highlights the dissecting aneurysm and mitral regurgitation, and homocystinuria thromboembolism. However, the concentration of Hcy is normal so the differential diagnosis easy. Other diseases such as HFR or defects of cobalamin metabolism have similar biochemical patterns homocystinuria but without the characteristic phenotype may reach a diagnosis by determination of amino acids in blood and urine. The goal of treatment is to normalize serum Hcy of less than 50 mmol / L and methionine 20 to 40 mol / L. Once detected cases, these patients require analytical control every 1-3 months for checkups for early detection of complications from cardiology, neurology and ophthalmology. We also recommend conducting annual densitometry. The prognosis in untreated patients is dismal. 25% die of vascular disease before age 30. Early treatment improves the prognosis, so it is important to its early clinical suspicion.

SC-70 ACUTE DISSEMINATED ENCEPHALOMYELITIS (ADEM) AFTER REMITTING MENINGITIS

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Introduction: Acute Disseminated Encephalomyelitis (ADEM) is associated with vaccination or an antecedent respiratory infection. We experienced a rare case, ADEM after remitting meningitis.

Case: A 29-year-old healthy Japanese woman presented with a one-day history of headache, high fever, nausea and vomiting. She had closely contacted with a person with diarrhea and vomiting 3 days before admission. Vital signs were BP 92/70 mmHg, PR 107/min, RR 16/min, SpO2 100%, T 39.9 °C. Lungs were clear. No S3 nor S4. Abdomen was soft and flat without tenderness. There were stiff neck and Kernig sign but no other neurological findings. Initial laboratory data showed WBC 8400/mm³, Glu 117mg/dL. Cerebrospinal fluid (CSF) were 112 cells/ mm³ (100% mononuclear), Protein 49 mg/dL, and Glu 54 mg/dL. She was admitted as aseptic meningitis. Other differential diagnoses included septic meningitis and herpes encephalitis. Initially her fever and headache were remitted with intravenous ceftriaxone and acyclovir. However, three days after admission, she complained dysuria and urinary retention. Repeated CSF on three days after admission were 578/ mm³ (97% mononuclear, 3% polynuclear), protein 87 mg/dL, and Glu 52 mg/dL. CSF culture yielded no organism. Brain MRI on five days after admission revealed no particular findings. A week after admission, she developed tremor and gait disorder. Spinal MRI on 11 days after admission revealed no particular findings. Because she continued to have sustained fever, tremor, and urinary retention, MRI study were repeated. Spinal MRI revealed high-intensity lesion in the white matter of right lateral funiculus with C6/7 on T2 weighted image and T1 weighted image with enhanced gadolinium. We diagnosed her ADEM associated with aseptic meningitis. Her symptoms were responded well with intravenous methylprednisolone (1 g/day) followed by oral corticosteroid (30mg/day). Finally, we tapered the dose and discontinued oral corticosteroid six month after discharge. She has been free of relapse.

Discussion: There is no diagnostic criteria of adult ADEM, but the diagnosis is established when a patient has a history of recent vaccination or viral infection, acute multifocal neurological symptoms, inflammatory response of cerebrospinal fluid, and MRI findings including the white matter hyperintensities on T2 weighted image and T1 weighted image with enhanced gadolinium. We consider acute viral encephalomyelitis or multiple sclerosis as the differential diagnosis. Initial treatment includes glucocorticoid, intravenous gamma-globulin, or plasma exchange. In this case, prompt response to glucocorticoid and full recovery were characteristic. She has also no disability or relapse.

Conclusion: When we see new onset of tremor, dysuria, and urinary retention following aseptic meningitis, we should consider ADEM.

SC-71 A RARE CAUSE OF IRON-DEFICIENCY ANEMIA IN A YOUNG WOMAN

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Reason for consultation: This is a 33 year-old lady who was admitted for evaluation of a microcytic anemia.

Previous medical history: No allergies, no high blood pressure, no diabetes, no dyslipidemia. Early menopause (27 yo). Wilms' tumor (3 yo) (nephrectomy + QT + RT). Current medication: Enalapril 2.5 mg od.

Present illness: Unremarkable. Progressive asthenia. Episodes of night sweats and dyspepsia with pain in left hypochondrium.

Physical examination: Left hypochondrium pain. Rest normal.

Complementary examination: Erythrocyte 3,65mill/mcL, Haemoglobin 8g/dl, Hematocrit 25,6%, MCV 70fl, MCH 22pg, MCHC 31,3gr/dl, RDW 16,9%, Platelets 621000/mcL, seric ferrum 12mcg/dL, ferritin 5ng/ml. TSH 0.69mcU/mL.

Approach to the patient with iron-deficiency anemia: 1.- To confirm this is an iron-deficiency anemia: (Table) 2.- Causes of anemia due to iron deficiency: A.- Low iron intake. B.- Decreased iron absorption or iron transporter deficiency. Malabsorption. Gastric bypass. Defects on the iron transporter. C.- Increased needs. Pregnancy/ breast feeding. Childhood/ adolescence. D.- Blood loss. Gastrointestinal: esophageal varices, duodenal ulcer disease, NSAID, neoplasia, diverticulosis, helminthic infection. Genitourinary: menstruation, uterine myoma, neoplasia, hemoglobinuria. Respiratory: neoplasia, hemosiderosis, infections. Others: intravascular hemolysis, hemodialysis blood loss. E.- Repeated extractions: Repeated blood tests. Voluntary blood donations. Treatment (policitemia vera). F.- Others: Factitious bleeding. Genetic. 3.- Diagnostic tests for iron-deficiency anemia: A.- Hemogram (Hb, MCV, MHC, RDW, iron, transferrin, transferrin saturation, ferritin, morphologic test), fecal occult blood test. B.- Gastroscopy and colonoscopy/ barium enema. C.- Urine test, cistoscopy y pielografy. D.- Gynecological examination. E.- Intestinal angiography. F.- malabsorption tests. Evolution: the following diagnostic tests were performed: Gastroscopy: normal. Colonoscopy: normal. Gynecological examination: normal. Urine test: normal. Intestinal transit: normal. Fecal occult blood test: normal. Our patient was discharged waiting for a VCE, but she is on admission again before that due to a intestinal pseudoobstruction. Emergency surgery was done. A yeyunal mass was removed. Anatomopathologic diagnostic was LEIOMYOSARCOMA. In this case, is important to keep in mind the relationship between Wilms' tumor and small bowel leiomyosarcoma. The incidence of tumor in adults who have survived a childhood tumor is six times higher than in the general population. The overall risk was estimated to be 1.6 % at 15 years, and the highest risk was among patients who received radiation therapy and doxorubicin. Other complications resulting from radiation therapy include muscle atrophy, short stature, and scoliosis. Children who receive 10Gy or more at an age younger 1 year are at most likely to have growth impairment.

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Table (SC-71)

| Microcytic anemia | IRON-DEFICIENCY | A. CHRONIC DISEASE | THALASSEMIA |
|------------------------|-----------------|--------------------|-------------|
| MCV | ↓ | N | ↓↓ |
| MCH | ↓↓ | N | ↓ |
| RDW | ↑↑ | ↑ | N |
| Fe | ↓ | ↓ | N/↑ |
| Transferrin | ↑ | ↓ | N |
| Transferrin saturation | <10% | >10% | N/↑ |
| Ferritin | ↓ | N/↑ | N/↑ |

SC-72 ARTHRITIS, ASTHENIA, SKIN INJURIES AND EVANS SYNDROME

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Referred by: Emergency Services

Reason for referral: Skin lesions, hair loss, arthralgia, weight loss and haemolytic anaemia

Family history: Mother dead of heart attack

Previous medical history: Medical: Smoker of ten cigarettes/day. No alcohol consumption. Surgical: appendectomy, surgery of varicose veins. Allergies: no allergies. Drug intolerances: no drug intolerances. Social history: country of birth: Romania; number of years living in Spain: five; occupation: cleaner.

Current history: The patient shows in October 2011 an urticarial exanthema in arms, legs and thorax, labelled in Emergency Services as urticarial reaction possibly related to ibuprofen. In November 2011 she relates clinical manifestations as asthenia, arthralgia and anorexia; the blood test does not show abnormalities. In February 2012 she is evaluated by a Rheumatologist and starts medical treatment with prednisone. The patient does not make her better and in April 2012 goes to Emergency Services

because she continues with asthenia, anorexia and arthralgia plus 10 kilograms weight loss from October, muscular pain and hair loss. She does not refer more cutaneous lesions, not fever, nor chest pain, photosensitivity or oral ulcers. She denies cough, abdominal pain or urinary symptomatology. In the physical examination cutaneous pallor and splenomegaly are described. Among the executed tests, the blood test reveals anaemia with haemoglobin of 8.9, average corpuscular volume 85.9, mean corpuscular haemoglobin concentration 34.4, and thrombocytopenia with 98.000 platelets. The lactate dehydrogenase was 298, bilirubin 0.8. At the peripheral blood smear there were spherocytes. The Coombs test was positive. Treatment with methylprednisolone is started, 1mg/kg dose. The patient was admitted to the hospital for further studies under the suspicion of systemic lupus erythematosus with autoimmune haemolytic anaemia and thrombocytopenia.

Physical examination in our service: Blood pressure: 104/58; pulse: 95; temperature: 36.4. Facial pallor. Eupneic in decubitus. Skin: no injuries, no erythema, no signs of vasculitis. Head and neck: Mucous adequately hydrated. No oral ulcers. Two palpable bilateral nodes of one centimetre size, hard, mobile and painless. No other palpable nodes or masses. J.V.P. no elevated. Thorax: Normal chest shape. Normal respiratory movements. Cardiac auscultation: rhythmic, without murmurs. Pulmonary auscultation: normal breath sounds, without added sounds. Abdomen: appendectomy scar. Tender, no pain with palpation, palpable spleen, no hepatomegaly, no masses. Normal bowel sounds. No ascites semiology. Upper extremities: Normal. No signs of arthritis. Lower extremities: No edema, no signs of venous thrombosis, symmetric pulses. No signs of arthritis. Central nervous system: normal conscious level, preserved memory, orientated in the three spheres, preserved mood, difficulty of exploring the speech because of language barrier. Isochoric and normoreactive pupils. No nystagmus. Visual fields preserved by confrontation perimetry. Normal ocular eye movements. Rest of cranial nerves: V sensory and motor, VII, IX, X, XI y XII normal. Symmetric muscle tone and power. Symmetric sensitivity. Symmetric reflexes. No neck stiffness. Normal march.

Additional investigations: Basic biochemistry: Glucose 87 mg/dl; Creatinine 0.81 mg/dl; Sodium 135 mEq/l; Potassium 4.28 mEq/l; Totals Proteins 8.8 g/dl; Albumin 3.3 g/dl; Calcium 8.7 mg/dl; Phosphor 3.0 mg/dl; ALT 9 U/l; AST 19 U/l; Gamma-GT 117 U/l; Alkaline Phosphatase 142 U/l; LDH 236 U/l; Bilirubin 0.8 mg / dl; Cholesterol 134 mg/dl; Triglycerides 199 mg/dl; Uric Acid 6.4 mg/dl. Blood count: Erythrocytes 2.360.000; Haemoglobin 7.2 dl; Hematocrite 20.3%; MCH 30.4pg; MCV 86.1fl; MCHC 35.3 g/dl; RDW 16.3 %; Reticulocytes 13.0 x1000/mcl; Reticulocytes 0.6 %; Platelets 94000; MVP 8.0 fl; Leucocytes 5.900 (Neutrophils 4.700; Lymphocytes 700; Monocytes 400; Eosinophils 100; Basophils 0). ESR: 29 mm; C Reactive Protein 2.5 mg/dl; Rheumatoid Factor <10 IU/ml. Peripheral blood smear: Segmented 82 %; Banded forms 0 %; Metamielocytes 3 %; Mielocytes 0 %; Promielocytes 0 %; Lymphocytes. 7 %; Monocytes. 6 %; Eosinophils 0 %; Basophils 0 %; Normoblasts 3 %; Blasts 0 %. Morphology: Important erythrocytes roleaux, occasional spherocyte, proven platelets. Direct COOMBS: positive. IGG+, cd3 +. Immunology: Ab. Anti DNA-Screening MultiPlex <1; Ab. Anti Nucleus-IFI Negative; Ab. Anti Sm Negative; Ac. Anti RNP Positive; Ac. Anti RNP-A Positive; Ac. Anti Ribosomal P Positive. Anti ciclic citrulinated peptide 43.80 U/ml. ACA IgM 168, ACA IgG 80.3. The rest of antibodies were negative. Complement: C3 106.00 mg/dl; C4 13.00 mg/dl..Hypergammaglobulinemia: IgG 5150. Kappa 3980, Lambda 2720.

ECG: Sinusal rhythm. Chest X ray: normal. Abdominal Ecography: Important splenomegaly of eighteen centimetres of maximum diameter, homogeneous, without focal lesions. Nodes in the region of hepatic hilum and celiac trunk, the biggest one of two centimetres size. Homogeneous hepatomegaly. Biliary vesicle and biliary tree without alteration. Normal kidneys. Pancreas partially seen. TC Thorax-Abdomen-Pelvis: Multiple axila nodes and retropectoral bilateral of 13 mm the biggest and left supraclavicular nodes of 10 mm. Multiple small ganglions in all mediastinum territories smaller than one centimetre. Calcificated ganglions in right broncopulmonar area. No pleural or pericardic effusion. Calcificated granuloma in pulmonary right base. The rest of parenquima without alterations. Moderated difuse hepatomegaly. No focal damage. Biliary vesicle, tree, kidneys and suprarrenal glandules without alterations. Important splenomegaly of 14.5 cm, homogeneous. Bladder, uterus and ovaries without significative alterations. Minimum quantity of free liquid in Douglas that could be considered physiologic. 11 mm node in portocava region. Multiple retroperitoneal nodes mainly lateroortic inter-aortocava, of 10 mm the biggest one, and in both iliac chains. Multiple inguinal bilateral nodes, of 12 mm the biggest one.

Mycrobiology: HEPATITIS B: Ag-HBs Negative; Anti-HBc: IgG Positive 8.19; Anti-HBe positive 0.95; Anti-HBs Positive 52.17; Anti-HBs Positive 57.85. B.A.A.R in bone marrow: Negative; Lowenstein culture: negative. Leishmania serology: negative; Culture: negative. Rose Bengal test: negative.

Bone marrow puncture: Discrete diffuse reticulinic fibrosis. Nodular and interstitial infiltrate constituted by intermedium and big lymphocytes, which shows a mixed infiltrate but with many B lymphocytes (CD20 positive) activated in the immunohystoquimic. The rest of the haematopoyetic tissue appears moderately hypoplastic with elements of all the series in different madurative stages. The rest of the immunohystoquimic techniques to detect lymphoid or myeloid blasts and epithelial infiltrates are negative.

Node biopsy: Linfatic node that shows distorsionated structure because of a small and intermedium lymphocyte proliferation between a tree shaped proliferation of ephiteloid venules with many hyalin deposits and focal distorsion of the dendritic cells. This proliferation is positive for CD3, CD4, CD5 and focally for CD10. There are isolated sternbergoid cells that result positive for B marquers (CD20), LMP-1 y EBER.

Diagnosis: Angioimmunoblastic T Cell Lymphoma.



SC-73 SYSTEMIC LUPUS ERYTHEMATOSUS IN THE NORTHWEST OF SPAIN: COMPARISON WITH THE REST OF SPAIN FROM 2006 TO 2010

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Introduction: Systemic lupus erythematosus (SLE) is a chronic, relapsing, inflammatory disease with cell and tissue damage by autoantibodies and disease with a wide spectrum of clinical manifestations.

Objectives: We present a comparison between a region in the northwest of Spain (Galicia) and the rest of Spain including the main epidemiological variables of patients admitted with SLE as the primary diagnosis (PD).

Material and methods: Retrospective study from 2006 to 2010 using data from the SERGAS (Health Service of Galicia), and the rest of Spain through the MDS from the website of the Ministry of Health <http://www.msps.es>. We used ICD-9 code 710.0 for SLE and the 710 "diffuse connective tissue diseases" (excluding vasculitis), in both search. Variables such as sex, age, hospital and department of discharge were studied.

Results: There were 779 patients with a DP of SLE in Galicia and 8,086 in Spain. Regarding sex, 77.5% of cases occurred in Galician women while in the rest of the Spain was about 84%. The mean age was 37.89 (SD ± 15.47) in Galicia. Referring mortality (SLE as PD), there were 7 in Galicia (0,9%) and 96 in Spain (1%). Classifying by hospitals, the largest number of admissions was registered in A Coruña with 228 (29,3%), followed by Lugo 178 (22,8%) and Vigo 125 (16%). In Galicia the highest percentage of admissions belongs to: Rheumatology (41.5%), Nephrology (38%) and Internal Medicine (11.3%). According to the total of patients in Rheumatology, the largest number belongs to Lugo 142 (44%), in Nephrology in A Coruña 172 (58.1%) and 42 (47.7 %) in Internal Medicine from Vigo. While Internal Medicine was the first in number of admissions (30.4%), Nephrology (26.9%) and Rheumatology (26.2%), in the whole of Spain. On the other hand, admissions due to autoimmune diseases in the whole country were 14,963. Third of them, 4,880 belong to Internal Medicine, in the majority from Emergency room (57.6%), contrary to Nephrology 2,264 (32.6%) and Rheumatology 4,610 (34%) where programated admission was the primary way.

Discussion: The data analyzed in Galicia were very similar to the rest of Spain except for the percentage of admission in Internal Medicine with SLE as PD, if we compare it with other departments that also manage this disease (up to 75% less admissions than in others departments). SLE is a multisystemic disease that could benefit from a global and multidisciplinary manage. Relationship and cooperation with other specialities must be strength. As selection bias, we did not include the secondary diagnosis of SLE neither the cases followed by an ambulatory form.

Conclusion: Our results showed that there were differences in admissions of SLE as a primary diagnosis between the Galician Internal Medicine departments and also compared to the rest of Spain.

SC-74 DAPSONE IN CUTANEOUS LUPUS TREATMENT

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Case report: Systemic Lupus Erythematosus (SLE) diagnosis is often complex due to its variety of presentation forms. This variability also presents itself in therapeutics, and, in some cases, it's a true challenge, which can lead to the use of a less conventional treatment in first line therapeutic failure/intolerance cases. The authors present a 79 years old male patient case report with SLE diagnosed at the age of 75, having the following classifying criteria: photosensitivity, discoid lupus, positive ANA and Anti-DNA. Initially he had been treated with chloroquine, ceased due to retinopathy, and was then medicated with low dosage corticotherapy. He's being followed in Auto-Immune Consult since August 2009. At the first observation he presented no cutaneous lesions, and complained of xerostomy and xerophthalmia. In October of the same year, he presented scalp, dorsal (single lesion) and limbs' lesions, compatible with discoid lupus, beginning topic tacrolimus with successful result. At this time he presented positive Anti-SSA and SSB, chronic inflammatory process in salivary glands' scintigraphy and positive Schirmer's test. Secondary Sjogren' Syndrome was assumed. Because of the cutaneous lesions' worsening azathioprine was added, with lesion improvement, but ceased after due to hepatotoxicity. With its suspension marked lesion exacerbation occurred, extending to all dorsal and anterior thorax regions, and erythematosus cutaneous lesions in all anterior fingers' face, as well as detachment of the most superficial plans. Due to previous toxicity to chloroquine and azathioprine, it was chosen to begin dapsona with clear clinical improvement, sustained until now. Dapsona's use in SLE has various outcomes, existing only case reports and series in literature. It's the front line treatment in bullous skin lupus lesions. In other forms of lesions it has appeared as second choice drug to antimalarials' intolerance or refractory disease, sometimes with excellent outcomes such as in the case presented here. Its use must be individually considered.

SC-75 DORSAL PAIN: MULTIPLE MYELOMA WITH NORMAL PROTEIN ELECTROPHORESIS

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Introduction: The clonal proliferation of plasmocytes may have a varied presentation, with plasmocytoma and multiple myeloma (MM) as the most relevant clinical entities. The detection of the M protein and the identification of plasmocyte proliferation remain the cornerstone for the diagnosis of plasmocyte dyscrasias. The M protein usually results of the production of both heavy and light chains (LC), however in about 15% of MM cases the plasmocytes only produce LC, identifiable in the urine as Bence Jones protein or in the serum as free light chains (FLC).

Aim: We report a case of LC MM whose diagnosis was only possible through the detection of FLC and take it as the starting point for the discussion of the growing role of FLC in the approach of plasmocyte dyscrasias.

Case report: An 82 year-old male was admitted for dorsal pain that radiated to the ribs, with no other complaints or findings in the physical examination, including neurologic abnormalities. A fracture with collapse of the 8th dorsal vertebra was identified in conventional x-ray examination and confirmed by computed tomography and magnetic resonance imaging. These additionally showed osteolytic lesions in the femur and vertebral bodies of the 7th and 8th thoracic and 4th and 5th lumbar vertebrae with protrusion compressing the spinal cord. The laboratory investigation showed no anaemia, renal failure, calcium changes, elevated lactate dehydrogenase, sedimentation rate or beta-2 microglobulin. The serum protein electrophoresis was normal, with a slight Kappa band in seric immunofixation and 9% plasmocytosis in the bone marrow examination.. The urinary immunofixation revealed Bence Jones protein (Kappa LC) with no albumin detectable. The serum FLC assay yielded a Kappa concentration of 1610mg/L (reference interval 3.3-19.4mg/L) and a Kappa/Lambda ratio of 97.6. The diagnosis of Kappa Light Chain Myeloma was therefore established. Two months after chemotherapy (dexamethasone plus prednisone and cyclophosphamide) and directed radiotherapy there was a 74% reduction in Kappa FLC (415 mg/L) and 89% in the Kappa/Lambda ratio (11.4).

Discussion: This case illustrates a plasmocyte dyscrasia with atypical presentation where, despite multiple peripheral bone involvement, the serum electrophoresis and immunofixation were normal and the bone marrow examination did not fulfil criteria for MM. The diagnosis was established through the evaluation and characterization of the serum FLC and urinary immunofixation. We stress the usefulness of this test in the approach to plasmocyte dyscrasias, that ranges from Monoclonal Gammopathy of Unknown Significance (MGUS) to MM. Studies show that in MGUS it helps to clearly identify the group of patients (40% of all patients) with lower risk of progression to MM (only 5% over 20 years). In MM, its use allows the reclassification of as much as 70% of non-secretory MM in oligosecretory or LC MM, which therapeutic approach and prognosis is distinct from the latter. The prognostic value of the FLC assay is reinforced by the fact that higher levels are associated with a higher 3-year mortality. Recent guidelines for the use of this test specify 4 major indications: i) in the diagnostic evaluation of MM or related disorders; ii) for prognostic value in all plasmocyte dyscrasias; iii) for monitorization of oligosecretory plasmocyte dyscrasias; iv) for the documentation of stringent complete response of MM treatment.

SC-76 RIGHT SINUS OF VALSALVA ANEURYSM AS CAUSE OF ACUTE AORTIC REGURGITATION AND ACUTE CORONARY SYNDROME

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Case report: Right sinus Valsalva aneurysm is a rare congenital anomaly. They are usually asymptomatic until symptoms develop due to compression of adjacent structures or, rupture occurs with subsequent intracardiac shunting and acute heart failure insues. We present a 20 year old woman, who went to the emergency department with sudden onset of intense dyspnea and chest pain. Past medical history was relevant for congenital cardiac problem of unknown etiology. On admission she had severe hypoxemia and was in shock. She was placed on ventilator support and vasopressors were started. Chest X-ray showed bilateral interstitial infiltrates compatible with pulmonary oedema. Electrocardiogram showed ST segment depression in DII, aVF and v4-v6 leads; blood work was remarkable for elevated blood natriuretic peptide and cardiac enzymes. Transesophageal echocardiogram showed an aneurysm in the right sinus of Valsalva of the aortic root with an important fistula being ascertained to the right ventricle's ejection tract. Severe intermittent aortic regurgitation was documented probably due to a "suction" effect. The patient was transferred to a cardio-thoracic surgery centre where the documented fistula was closed. Remaining hospital stay was uneventful and she was discharged home asymptomatic. Follow-up exams revealed definitive resolution of her aortic regurgitation, preserved systolic ejection fraction and



no contractility abnormalities. Congenital sinus of Valsalva aneurysm is caused by separation of the vessel media from the annulus fibrosis of the aortic valve. It has a prevalence of 0.1– 1.5% in Western populations with an estimated 3– 4:1 male predominance. Sinuses of Valsalva aneurysms are asymptomatic until rupture occurs, often in early adulthood. Prompt surgical correction (open-heart surgery or in selected cases percutaneous closure) is mandatory with subsequent good prognosis (88% in some published series). Patients with concomitant aortic insufficiency fare less well, with only 40% of patients having complete resolution of symptoms. This clinical report illustrates well the importance of prompt referral of these cases for adequate management.

SC-77 VENTRICULAR FIBRILLATION: AN EXTREME CASE OF PRIMARY HYPERALDOSTERONISM

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Case report: Primary hyperaldosteronism is estimated to affect 11% of hypertensive patients referred to speciality consults or clinics for resistant hypertension. They usually present with elevated blood pressure, and hypernatremia and hypokalemia in blood work. Failure to diagnose and treat leads to structural vascular, cardiac and renal lesions which translate clinically in cardiovascular events such as stroke, arrhythmias or acute myocardial infarction. We present a 79 year old man which was brought to the emergency department because of altered mental status. His past medical history was relevant for long dating arterial hypertension which was difficult to control with ACE inhibitor, diuretics and beta-blocker, prostate cancer which was treated with radio and hormone therapy. He was in his usual health status until 5 days before admission when he complained of vomits and diarrhea which persisted until the day of admission. On admission he had altered mental status, with an Glasgow coma scale of 8; blood pressure was 180/96; EKG showed bigeminism with runs of ventricular tachycardia; arterial blood gases showed metabolic alkalosis; blood work was remarkable for a slight hypernatremia of 147 and a serious hypokalemia of 1.6 mEq/L; CT and MRI scan of the head showed no meaningful alterations. Despite aggressive supplementation with magnesium sulphate and potassium chloride (120 mEq in 3 hours) serum potassium was unchanged. Cardiac arrest in ventricular fibrillation ensued which was reverted after 1 cycle of advanced life support. He was admitted in the intensive care unit. First 24 hours with marked electrical instability inspite of adequate therapy. Blood work showed a low renin and elevated levels of aldosterone. 24 urine sample showed elevated levels of potassium; CT scan of the abdomen showed a 13 mm nodule in the left suprarenal gland. Potassium achieved normal levels after several days of supplementation and use of ACE inhibitor, angiotensin receptor blocker and spironolactone. He was discharged with minor cognitive changes, normalized blood pressure and serum potassium levels and was oriented to a speciality consult. This clinical case shows the importance of prompt diagnosis and adequate treatment of secondary hypertension, namely primary hyperaldosteronism in order to avoid its deleterious effects.

SC-78 MAN OF 42 YEARS OLD WITH LATE POSTOPERATIVE FEVER AND EXANTHEMA

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Case report: A 42 years old man with no known drug allergies was admitted to the Internal Medicine department with a generalized, diffuse, centrifugal, maculopapular, erythematous, petechial, pruritic rash starting in chest and including palms and soles. He associated malaise, 39,9°C of fever, facial edema, enanthem, enlarged tonsils and an injected oropharynx without cervical lymphadenopathy. He was hospitalized a month earlier in the orthopedic service for a right fractured patella and patellar tendon, which was complicated after surgery with wound infection. He was initially treated with cefazolin and gentamicin for three days, and cefepime with vancomycin subsequently for 20 days with good clinical outcome. He was discharged with rifampicin and levofloxacin, taking them for two days, and suspended because of the rash. From the second day of hospitalization, he showed elevated liver enzymes to a peak of 662 ALT, AST 307, GGT 660 and AP 464 with normal bilirubin and eosinophilia to a peak of 1500. He developed proteinuria of 300 mg/L without effects on renal function and interstitial infiltrates consistent with pneumonitis in right lung base. The leukocyte count, CRP, fibrinogen, procalcitonin, and the other complementary examinations were normal or negative. We performed a biopsy of skin lesions that showed perivascular and interstitial infiltrate in the papillary and reticular dermis consisting of lymphocytes, some neutrophils and abundant eosinophils, confirming the diagnosis of DRESS syndrome (Drug Rash with Eosinophilia and Systemic Symptoms). Treatment was started with 60mg/day prednisone, presenting progressive resolution of the signs and symptoms, proteinuria, pulmonary infiltrates, and laboratory abnormalities in a period of 10 days. Only acetaminophen and dexchlorpheniramine were administered as symptomatic treatments. DRESS syndrome has been associated with multiple drugs, especially anticonvulsants

such as carbamazepine, Phenobarbital, phenytoin and also with other drugs such as minocycline, allopurinol, dapsone, abacavir and nevirapine. In recent years there have been case reports associated with antibiotics, especially vancomycin, cephalosporins and tuberculostatic drugs. In our review we found no literature related to gentamicin and levofloxacin. We can not exclude the etiologic relationship of any antibiotics, because all of them were administrated within the time-related onset of this syndrome (2-6 weeks).

SC-79 DISSEMINATED NOCARDIOSIS

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Introduction: Nocardiosis is an uncommon gram+ infection caused by *Nocardia sp*), that most often occurs in immunocompromised. *Nocardia spp* causes localized (lung, skin or CNS) or disseminated disease, because of its ability to disseminate to any organ.

Case clinic: A 62 years old male, with complicated pneumoconiosis and idiopathic thrombocytopenic purpura, treated with oral prednisone. The patient is admitted by presenting cough, respiratory secretions, dyspnea and fever. He also presents dorsolumbar pain that doesn't yield with tramadol and progressive abdominal distension with constipation. At the physical examination the patient features fever, tachycardia, dyspnea, roncus in both hemithorax, relaxed abdomen with decrease of peristalsis and pain to the mobilization at dorsolumbar spine. In the biochemistry highlights the presence of leukocytosis and elevation of PCR, VSG and LDH. Chest radiograph and thoracic-CT show a diffuse rough-nodular pattern. Dorsolumbar radiograph shows degenerative changes. Abdominal radiograph and abdomen-CT show an important colonic dilation. With the diagnosis of respiratory infection, Ogilvie's syndrome 2º to opioids and Degenerative dorsolumbar pain, was initiated treatment with amoxicillin-clavulanate, and analgesia, yet the described symptoms and fever persist. 48 hours of income, the patient presents loss of strength and sensitivity in both lower limbs along loss of control of sphincters, detecting paraplegia of lower limbs with sensitive level below intermammary line. The spine-RMN provides an epidural collection (C7-L1) with compression of spinal cord cordon. The patient was submitted to urgent surgical intervention for the decompression of the spinal cord cordon, obtaining an epidural empiema that was cultivated with growth of *Nocardia cyriacigerogica*. We complete the study with brain-CT that shows the existence of frontal intracranial abscess and bronchoscopy, confirming the presence of *nocardia* in cultivation of BAL. The patient was diagnosed for disseminated nocardiosis and treatment with intravenous 3G cephalosporine, amikacin and TMP-SMX was administered (6 weeks) with improvement of symptoms and radiological findings. Oral treatment with TMP-SMX has been maintained indefinitely.

Discussion: The major risk factor for nocardial infection is the immunosupresion. Pulmonary disease is primary site of nocardial infection (two-thirds of cases) and approximately 50% of all disseminate to sites outside the lung, most commonly the brain. The hallmark of CNS nocardiosis is formation of a parenchymal abscess. Brain imaging should be performed in all patients with primary pulmonary nocardiosis. A definitive diagnosis requires the isolation of the organism from a clinical specimen. Antibiotics that are typically effective against *nocardia spp* include TMP-SMX, amikacin, imipenem and 3G cephalosporins.

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