are known such as endocardial fibroelastosis, dilated cardiomyopathy, and valvular insufficiency. The early clinical diagnosis in utero is essential to be specified due to myocardial tissue damages can be reversible. In the prevention and in the confirmed cardiac involvement the first line therapies are chloroquine, dexamethasone and intravenous immunoglobulins, and also a regular foetal echocardiography is of essential importance.

Objectives: Main objective of this report is description of successful treatment of an anti-SS-A antibody exposed foetus with cardiac manifestation.

Methods: Case report of a 25-year-old pregnant woman and her baby. The mother was diagnosed with Sjogren's syndrome in 2013. In previous case history there were two late foetal deaths at the 23rd and 33rd gestational age in 2016 and 2017, respectively as a consequence of foetal bradycardia. During her 2nd pregnancy the mother received chloroquine and azathioprine. At present she was admitted to our Institute in October 2019 at 23rd weeks of gestation without any compliant and any abnormality of pregnancy. Foetal development was normal. Mother received azathioprine and chloroquine from the beginning of pregnancy and dexamethasone from the 16th weeks of gestation. Foetal echocardiography was performed at the 16th gestational week, and every week thereafter. Reflective areas, reflecting oedema and inflammation, appeared at 24th gestational week, localised to the left atrium.

Results: The case was referred, and the combo therapy was completed with 1 mg/maternal kg intravenous immune globulin, dexamethasone dose was increased to 4 mg for a week, then decreased to 2 mg. Intratect was given every 2nd week. Progression was stopped as according to control foetal echocardiography after the 2nd infusion. After 4th IVIG the involved area of myocarditis decreased significantly, localised to anterior wall of left atrium and the atrial primum septum. However, at 32nd g. week pericardial fluid was visualised in maximum 9 mm width without signs of pericardial tamponade.

At the end of last December, the baby was born at the 35th gestational week with 50 cm and 2570 g and no signs of any congenital anomaly. Pericardial fluid was 4 mm maximum. Her development is normal.

Conclusion: Neonatal lupus with various cardiac manifestations may develop in anti-SS-A antibody exposed babies. Therefore, these pregnancies require stringent gynaecologic and cardiologic controls. Although congenital heart block is the most frequent complication developing between the 18-28th gestational weeks, other manifestations also may occur. High dose intravenous immune globulin therapy can be effective even after failure to combined traditional immune suppressant therapy.

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THU0596

ABNORMAL RIGHT VENTRICLE RESERVE ON EXERCISE PREDICTS PULMONARY HYPERTENSION IN MIXED CONNECTIVE TISSUE DISEASE: A CASE REPORT

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Background: Pulmonary hypertension is one of the most common complications in patients with mixed connective tissue disease (MCTD). Patients are usually at the late stage and have irreversible right heart dysfunction when diagnosed as pulmonary hypertension with the rest echocardiography. Early detection of right heart dysfunction before pulmonary hypertension is essential to ensure that patients receive timely and appropriate treatment for this progressive disease. We aimed to use exercise stress echocardiography to detect early right heart dysfunction in patients with CTD and without pulmonary hypertension.

Objectives: To present a clinical case of MCTD with normal right ventricular (RV) function at resting but presenting RV dysfunction on exercise, who developed pulmonary hypertension after one-year follow-up.

Methods: Case report. The patient was subject to the treadmill exercise stress echocardiography. The autoantibodies including anti-nRNP/Sm, anti-Ro-52, and antinuclear antibody (ANA) were detected. The patient was followed-up to one year.

Results: A 31-year-old female patient was admitted to our department in 2018, with a history of MCTD for five years. Autoantibodies testing revealed that the patient was positive for anti-nRNP/Sm (+++), anti-Ro-52 (+++), and ANA (1:3200). Echocardiography revealed no obvious cardiac dysfunction. However, the velocity of tricuspid valve regurgitation was 3.0m/s following treadmill exercise stress. The patient was followed-up to one year. Then, she developed occult pulmonary hypertension with the velocity of tricuspid valve regurgitation of 3.3m/s following treadmill exercise stress. Accordingly, MTX and prednisone were switched to MTX, prednisone, hydroxychloroquine (HCQ) and beraprost.

Conclusion: This study showed that treadmill exercise echocardiography could detect right heart dysfunction early before diagnosed as pulmonary hypertension with rest echocardiography in patients with MCTD in its early stage.

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THU0597

CORNEAL MELT - DON'T ALWAYS BLAME RHEUMATOID ARTHRITIS

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Background: Corneal melt is a rare inflammatory disease of the peripheral cornea; it may lead to perforation of the globe and visual failure. Corneal melt can be a manifestation of systemic vasculitis in patients with RA and other conditions, such as cancer. Without early and aggressive treatment it may be associated with a poor visual outcome and a high mortality. It has been reported in patients with stable RA.

Objectives: A case report in a patient with long standing but well controlled Rheumatoid Arthritis (RA) and metastatic disease.

Methods: A 75 year old male with a background of sero positive Rheumatoid Arthritis for more than 10 years presented to the Eye Casualty with a two week history of a painful left red eye. His other medical history was significant for Stage IIB poorly differentiated cancer of the left lower lobe. Left lower lobectomy with a patch of diaphragm resected. Intratumoural lymphovascular invasion noted. He completed Adjuvant Carboplatin/Vinorelbine chemotherapy September, 2017. He had DVT proximal left leg 22nd of September, 2017. Follow up CT in 2018 demonstrated a right renal upper pole lesion for which he was awaiting biopsy with metastatic lung disease vs primary renal carcinoma. His RA was well controlled on Methotrexate 10mg weekly. He had been treated by the ophthalmology team for left marginal Keratitis for the prior 2 months with steroid eye drops without significant improvement. On presentation to ED, he described sharp eye pain, waking him from the sleep, associated with watery discharge and photophobia. Examination showed corneal melt in left eye involving 25% of inferior portion of the cornea and spastic entropion with injecting eye lashes. He had no active joints and there were no other signs of vasculitis. CRP was 4.1. He had a negative ANA and ANCA; viral swabs were negative. He was admitted under the medical team. Intravenous Methyl Prednisolone was started. The patient felt better after 5 days of Methyl Prednisolone. Left temporary tarsorrhaphy was done by Ophthalmology. Cyclophosphamide was initiated after discussion with Oncologist pending the result of the renal biopsy. Patient was discharged after 5 days of admission in the hospital

Results: The renal biopsy was positive for metastatic Squamous cell carcinoma of lung. Cyclophosphamide was withdrawn and he was started on Carboplatin/Gemcitabine. The corneal melt improved with complete resolution of his visual symptoms.

Conclusion: In this case, although the history of RA was felt by the ophthalmology team to be the most likely association with the corneal melt, we would argue the oncological diagnoses were likely the driving force behind the presentation.

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THU0598

A CASE REPORT ON A RARE PRESENTATION OF GOUT INVOLVING THE PATELLAR TENDON

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Background: Gout is an inflammatory arthropathy associated with long-standing hyperuricemia. The first metatarsophalangeal joint is the most commonly involved joint, although gout is often polyarticular. Involvement of tendons has been described, but is rare. We report a case of gout involving the inferior portion of the patellar tendon.

Objectives: A Case report to highlight a rare presentation of gout.

Methods: A 50 year old male presented to the Rheumatology emergency clinic with severe right knee pain, unable to weight bear. He had been recently diagnosed with Sero negative Inflammatory Arthritis and treated with Methotrexate for 4 months. He had presented one week previously to the general medical team on call with similar but less severe pain. He was discharged on steroids, which were of no significant benefit. The pain progressively worsened to the extent that he was not able to bear weight on the right knee. He denied pain or swelling of any other joint.

On examination of his right knee, it was extremely tender, slightly erythematous with increased local temperature. There was no arthritis in any other joint and there were no tophi. Joint aspiration was attempted but there was no fluid.

He was admitted with query Septic arthritis and started on intravenous antibiotics. Uric acid level was 583u/l. US of the knee showed small fluid in the pre patellar bursa. Orthopaedic team was involved and he was taken to the operation theatre. Knee joint was aspirated which was negative for crystals and there was no growth on cultures.

He ultimately had an MRI of his right knee which showed significant soft tissue oedema and abnormality in the patellar tendon.

MRI of Rt knee showing marked soft tissue edema (arrow)

MRI of Rt knee showing marked soft tissue edema (arrow)

Targeted US of the knee done by a consultant rheumatologist showed significant changes in the inferior portion of the patellar tendon consistent with infiltration. The external tendon boundaries were normal. It was thought to be likely that these represented gouty tophi.

USG of the rt knee showing infiltrates in the inferior portion of patellar tendon with increased Doppler flow

He went on to have a patellar tendon biopsy which has shown changes consistent with gout.

He was started on Colchicine 0.5mg bd leaving the hospital and Allopurinol was started two weeks later.

Results: He was seen in the clinic as follow up with complete resolution of his pain. Methotrexate was stopped. He has reported no flares and Allopurinol is being titrated up to achieve serum uric acid concentration <300u/l.

Conclusion: The most important finding in this case report is that interstitial gouty tophi of the patellar tendon, can cause debilitating symptoms and can prove difficult to diagnose and treat.

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THU0599

PULMONARY ARTERY ANEURYSM, BUDD CHIARI SYNDROME, INTRACARDIAC AND INFERIOR VENA CAVA THROMBOSES: AN UNUSUAL CASE OF BEHÇET'S DISEASE

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Background: Behçet's disease (BD) or "Silk Road" disease is a rare multi-systemic inflammatory disease of unknown etiology. Vascular involvement manifested as thrombosis, arterial aneurysm, and occlusion can carry a high mortality risk. BD can be a diagnostic conundrum with its broad array of clinical presentations.

Objectives: Identifying vasculo-Behçet's disease and its management.

Methods: A 25-year-old man born in Malaysia and known for cirrhosis due to idiopathic Budd Chiari syndrome presented to the emergency room with a transient ischemic attack. An inferior vena cava (IVC) occlusive thrombus and a patent foramen ovale (PFO) were discovered. Thrombolysis, angioplasty, PFO closure, and a transjugular intrahepatic portosystemic shunt (TIPS) procedure were performed. The following year, the patient experienced numerous IVC and TIPS-associated thromboses as well as a right atrial thrombus attached to his PFO closure device, all of which were refractory to anticoagulation. A few months later, the patient suffered from an acute right anterior cerebral artery stroke, with no etiology uncovered at the time. It was later determined that the patient had experienced years of recurrent oral and genital aphthae, thereby prompting a strong clinical suspicion of BD. Six months later, after only one appointment at the rheumatology clinic during which he was prescribed colchicine, the patient presented to the hospital with hemoptysis. A computed tomography (CT) pulmonary angiogram revealed a right lower lobar pulmonary arterial aneurysm with a peripheral thrombus, a right bronchial artery dilatation, and pulmonary emboli. The patient declined anticoagulation and was sent home. Two months later, he returned to the hospital, this time with hematemesis. A repeat CT pulmonary angiogram was performed and showed an increasing pulmonary emboli burden and an enlarging aneurysm. A thrombophilia workup was negative.

Results: A diagnosis of BD with pulmonary aneurysms was made and treatment was initiated with methylprednisolone pulses and monthly intravenous cyclophosphamide as recommended by the European League Against Rheumatism. A month later, there was radiological evidence of significant improvement in the burden of pulmonary emboli, an interval decrease in the aneurysm's diameter, and resolution of the right atrial thrombus.

Conclusion: BD with vascular involvement or vasculo-Behçet's disease can affect small, medium, and large vessels of both the venous and arterial vasculatures and is thought to originate from vessel wall inflammation. Thrombi in vasculo-Behçet's disease are typically quite adherent to the vessel walls and tend not to embolize. In this case, pulmonary arterial thrombosis burden was significantly decreased after immunosuppression alone, favoring a diagnosis of in situ thrombosis rather than thromboembolism. Moreover, pulmonary artery aneurysm, Budd-Chiari syndrome, and vena cava thrombosis, which are quite uncommon and carry the highest mortality risk in vasculo-Behçet's, were all present in this case. Early recognition can be life-saving as immunosuppression is the first-line therapy rather than anticoagulation, which carries a significant risk of pulmonary hemorrhage in the presence of a pulmonary artery aneurysm.