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THU0593

LIBMAN SACKS ENDOCARDITIS COMPLICATED WITH CEREBRAL EMBOLISM REVEALING SYSTEMIC LUPUS ERYTHEMATOSUS: A CASE REPORT

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Background: Libman Sacks endocarditis (LS) is an uncommon sterile endocarditis mostly associated with malignancies. It can also complicate the evolution of systemic lupus erythematosus (SLE) in 1 out of 10 patients after many years of evolution of the disease.

Objectives: To raise awareness of the possible rare complications of LS in SLE

Methods: We report a case of a complicated Libman Sacks endocarditis revealing the diagnosis of SLE.

Results: A 53 years old female patient with history of hypertension was admitted to the rheumatology department for polyarthritis since 2 months with fatigue and dyspnea. On physical examination, she had no fever nor cutaneous lesions, her pulse rate was accelerated to 112 per minute and there was no heart murmur. Musculoskeletal examination revealed the presence of arthritis of little joints of the hands and bilateral arthritis of the knees. Biological results showed an accelerated sedimentation rate of 49 mm the first hour, elevated c-reactive protein of 79 mg/l, anemia and lymphopenia. Renal and hepatic functions were both normal. Transthoracic cardiac echocardiography showed no valvulopathy but a 5 mm sessile vegetation on the mitral valve. To rule out infectious endocarditis, blood cultures came out negative, serological tests for brucella, parvovirus B19, coxiella brunettii, mononucleosis, were also negative. Culture of knee joint fluid for common bacteria and polymerase chain reaction tests for tuberculosis were also negative. The patient presented rapidly with agitation, cerebellar ataxia, and unilateral paresis. CT-scan showed the presence of ischemic lesions in the lenticular nucleus and the semiovale centers. Lumbar puncture showed no abnormalities. She was prescribed large spectrum antibiotics and anti coagulants but with persistent worsening of her neurological state. She was then transferred to the intensive care unit. Anti-nuclear antibodies, came out by then positive 1:3200 with an homogenous fluorescence and with positive anti nucleosome anti bodies. The diagnosis of LS endocarditis associated with SLE complicated with brain embolisms was confirmed with delay.

Conclusion: LS endocarditis is not common but when it presents it is often associated with high morbidity and mortality. Health care professionals should consider LS diagnosis in patients with underlying SLE and should be aware of the risk of embolisation. Tests to rule out infectious disease may delay initiation of appropriate treatment leading to severe prognosis. The treatment of LS endocarditis is still not well codified.

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THU0594

A CASE OF TAKAYASU'S ARTERITIS IN A PATIENT WITH TUBERCULOUS LYMPHADENITIS

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Background: Takayasu's arteritis (TA) is a large vessel vasculitis that principally affects the aorta and its main branches. The incidence has been reported at between 1.2 – 2.3 cases per million per year, more commonly in the Asian population. The age of onset is typically between tenth and fourth decade; between 80 and 90 percent of the cases are female.

The relationship between Mycobacterium Tuberculosis (mTB) and TA has long been considered; both demonstrate chronic inflammatory changes on histological examination and some granuloma formation in arterial walls. There is increasing evidence implicating mTB in the pathogenesis of TA through molecular mimicry between the mycobacterium heat shock protein -65 (mHSP-65) and the human homologue HSP -60 (hHSP-60). However, no definitive link between the two diseases has been explained.

Objectives: Case presentation.

Results: A 23-year-old lady was referred to our outpatient rheumatology clinic with a twelve-month history of persistently enlarged cervical lymph nodes on the left side for which she had received six months of anti-Tuberculosis medication. She had been referred to the respiratory physicians who had diagnosed presumed Tuberculous Lymphadenitis, with caseating granulomas demonstrated on biopsy, positive acid-fast bacilli smear but a negative culture. The patient had been initiated six months of anti-Tuberculosis medication; however, her lymphadenopathy showed no improvement. More recently she described a five-month history of weakness, paraesthesia and claudication symptoms in her left upper limb with episodes of dizziness and blurred vision, episodes occurring 2-3 times per day and lasting between a few minutes to a few hours.

Her examination at this presentation revealed an unrecordable blood pressure in the left upper limb and 104/67 mmHg in the right. There was significant tender lymphadenopathy of the left cervical lymph nodes and diminished pulses in the left upper limb. Right sided pulses were normal. The rest of her examination was normal.

Investigations at presentation revealed elevated inflammatory markers with C-reactive protein (CRP) of 116 mg/dL and erythrocyte sedimentation rate (ESR) of 128 mm/h. Complete blood count (CBC) found her to be anaemic with a haemoglobin of 100 g/L, with a mean cell volume of 71.3 fl, and have elevated platelet count of 649 x 10⁹/L. Recent computerized tomography scan with contrast of the thorax demonstrated features consistent with Takayasu Arteritis. Marked left subclavian stenosis was found on magnetic resonance imaging. High dose prednisolone at 60 mg once daily along with Azathioprine 2 mg/kg/day was started with a follow up appointment in two weeks.

Conclusion: There is increasing evidence implicating mTB in the development of TA and a few cases recognising this link have been reported. We report a case of TA in a patient recently diagnosed and treated for Tuberculous lymphadenitis who then developed symptoms of TA. There should be a low threshold for suspecting a diagnosis of Takayasu's arteritis in patients previously or actively infected with Mycobacterium Tuberculosis. Further research exploring the relationship between mTB and TA is required.

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SUCCESSFUL COMBINED IMMUNE-MODULATING THERAPY OF IN UTERO MYOCARDITIS AS THE MANIFESTATION OF NEONATAL LUPUS IN A RO/SS-A ANTIBODY EXPOSED NEWBORN

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Background: Congenital heart block (CHB) occurs in 2-5% of pregnancies due to maternal Ro/SSA and/or anti-La/SSB antibodies, and has a recurrence rate of 12-20% in the following pregnancy. The highest risk for CHB of the foetus is in the gestational age between 16- and 28-weeks. Without immunotherapy, this autoantibody mediated disorder comes with substantial mortality with a rate of 15-30%. Ro/SSA and/or anti-La/SSB antibodies attack the atrioventricular node with the subsequent inflammation and fibrosis. In addition, other cardiac manifestations