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Role of Leptospirosis Causing Infective Endocarditis - A Case Report

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ABSTRACT

BACKGROUND: *Leptospirosis is the most widespread zoonosis in the world. It can manifest inmany ways ranging from a mild febrile illness of short duration to a severe infection with jaundice and renal failure. It is caused by many strains of leptospira. Rarely other organs such as lungs, gall bladder, brain and ophthalmic fissures are involved mainly due to vacuitis[1].*

Cardiac involvement in Leptospirosis*: An analysis of data of 50 patients with serologically provenleptospirosis demonstrated that 70% of the patients had electrocardiographic abnormalities, with atrial fibrillation being the commonest major arrhythmia noted[2].*

Thirty-six percent of the patients had conduction system abnormalities and 30% had T wave changes. Another series has reported AV block in 44% of patients with leptospirosis[3]. A glycoprotein fraction of leptospiral cell wall has been incriminated in the pathogenesis of these rhythm disturbances. This protein is thought to inhibit the Na-K ATPase and may be responsible for the arrhythmia. Univariate analysis has shown that cardiac arrhythmia is more common in patients dying of leptospirosis than in the survivors. Other reported cardiac abnormalities include myocarditis and pericarditis[4]. In seven cases of fatal leptospirosis, petechial hemorrhages were found in the heart and the pericardium in all the autopsy specimens and interstitial myocarditis was found in five specimens. In another study, acute coronary arteritis was found in 70% of patients who died of leptospirosis and evidence of aortitis was present in more than half[5]. One case of leptospira endocarditis, possibly caused by serovarIcterohaemorrhagiae has been reported in literature. Infective endocarditis can also be caused by rare germs[6].

Keywords*: Leptospirosis, Infective endocarditis, Negative blood culture.*

INTRODUCTION

Case Report: A 30 year old male, a fish vendor and recreational swimmer presented with fever for 3days and altered sensorium for one day duration. His medical history was insignificant for previous systemic disease. He had no history of tuberculosis, surgical infection, dental extraction or heart disease in the past. He was an alcoholic. There was no history of promiscuity or drug abuse. On examination he was drowsy and disoriented with GCS 13/15. He was febrile with conjunctival suffusion. Examination of the other body system was essentially normal. Two days later patient developed icterus. He had mobiliform rash with few pustules over upper chest wall and right arm. There was gangrene over right middle finger. All major peripheral pulses were felt equally. There was no bruit. Subsequently patient developed gangrene of right ring finger and left big toe. There was maculopapular rash over both upper and lower limbs, palms and soles. He continued to be febrile. Patient later developed pain over left hypochondrium.

Investigations done for the Patients: Hb was 12 gms/dl, Total count 10600 cells/ mm^3, Differentialcount was 84% polymorphs, 14% lymphocytes, 2% eosinophils. ESR was 40/85 mm/hr , blood urea 122 mgs%, serum Creatinine 2.1 mgs% with normal electrolytes. LFT revealed sr. bilirubin 3 mgs%, SGOT 184 IU/L, SGPT 114 IU/L, SAP 397 IU/L. serum amylase was 88 IU/L. urine routine was normal initially. Repeat urine examination a week later showed 100 RBC/ hpf. Urine bile salts and bile pigments were positive. ECG showed sinus bradycardia. His HIV, Hepatitis B virus serology, VDRL, anti HCV were negative. CSF analysis revealed no cells with sugar 60 mgs%, protein 100 mgs%, and CSF culture showed no growth. Serial blood cultures showed no growth. Urine culture also showed no growth. Leptospira micro agglutination test was positive with 1:200 titres. The reactive serovar was australis. Leptospirae were seen in DFM. Repeat MAT after 2 weeks period showed rising titre of 1:800. Reacting serovar was australis. LeptoIg M was positive (25 u/ml). Skin biopsy revealed stratified squamous epithelia overlying collagen with collection of inflammatory cells around blood vessels - a picture suggestive of vasculitis. ANCA, ANA, anticardiolipin antibody, rheumatoid factor, serum cryoglobulins were negative. CRP was positive, serum fibrinogen was 605 mgs%. Radiological examination of chest and abdomen, CT chest and brain, ultrasonography of abdomen were normal at the time of admission. Repeat USG abdomen done after a week showed a large splenic abscess with ascites and minimal pleural effusion. Transesophageal echocardiogram showed a mass of about 10 mm attached to atrial surface of AML and freely moving vegetation. AML and PML were thickened and prolapsed. LV, RA, RV, aorta were normal. There was mild MR without pericardial effusion. A repeat TEE after ten days showed same findings with mild pericardial effusion and left pleural effusion. CT abdomen contrast showed a large splenic abscess of 11.5 x 8.5 cms with pericardial effusion and left pleural effusion and bilateral renal infarct. The above findings were suggestive of leptospirosis with infective endocarditis with metastatic complications. The patient was treated with penicillin, ceftriaxone, gentamycin and metronidazole. Supportive measures were given. Inspite of effective treatment patient died after 2 weeks due to sepsis and multi organ dysfunction.

DISCUSSION

Leptospirosis is a disease caused by pathogenic spirochetes of the genus leptospira. It's the most common zoonosis in the world. Its distribution is worldwide with greatest incidence in the tropics. Human become infected from direct contact with urine of infected animals or from exposure to soil, water or other materials contaminated with it. There is anoccupational risk including farmers, veterinarians, and commercial fish vendors. Recreational activities such as swimming, may also involve contact with organism. Our patient was a fish vendor and recreational swimmer which increased the risk of acquiring infection[7]. More than 90% of symptomatic patients have the relatively mild and usually anicteric form of leptospirosis. About 10% have severe leptospirosis or weil's disease. Severe leptospirosis causes a multisystem disorder involving hepatic and renal system. It also affects the pulmonary, hematopoietic, ocular, cardio vascular and central nervous system. Leptospirosis causing infective endocarditis though extremely rare has been documented in literatures. The incidence is less than 1%. The diagnosis usually made by clinical features suggestive of endocarditis with TEE and negative blood cultures in a serologically proven case of leptospirosis as in this case. A case of tissue proved infective endocarditis caused by leptospira species has been reported[8]. Also a case of infective endocarditis caused by Leptospiragrippotyphosa has been documented[9]. Splenic abscess is also very rare in these days. The incidence is 0.14 to 7% at autopsy cases. Splenic abscess is also associated with infective endocarditis as in this case. It occurs in 3 - 5% of cases of infective endocarditis[10,11].

CONCLUSION

Leptospirosis is an uncommon cause of infective endocarditis and splenic abscess. This case is presented for its rarity. We recommend considering leptospira as a possible etiologic agent of endocarditis.

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