Case Report

Pancytopenia a Rare Presentation of Weil’s Disease - Case Report

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ABSTRACT

Leptospirosis, a zoonotic disease, may have protean manifestations apart from the classical presentation of renal failure and jaundice. In this report, we present a case of fever with pancytopenia as the presenting manifestation of leptospirosis, which reversed completely with one week of intravenous cephalosporin. Although thrombocytopenia has been extensively reported, along with renal and hepatic involvement, pancytopenia secondary to leptospirosis has been scarcely mentioned in literature. In conclusion, this case report suggests that Leptospira infection should be included in the differential diagnosis of febrile pancytopenia.

Key words: Weil’s disease, Pancytopenia.

INTRODUCTION

Leptospirosis is a zoonotic disease caused by pathogenic spirochetes of the genus Leptospira. In 1907, Stimson described the microorganism in the renal tubules of a patient who died of the so-called yellow fever. The spirochete was first isolated in Japan by Inada and co-workers in 1915, nearly 30 years after Weil described the clinical disease in 1886. The relatively recent discovery of leptospirosis belies its long history, which was probably known much earlier in China and Japan by names such as "rice harvest jaundice" and "autumn fever." [1] This disease has been described as the most common zoonosis affecting many species of wild and domestic animals such as rodents, livestock, wild mammals, dogs and cats. Particular serovars are associated with characteristic animal hosts; e.g., L. ictero hemorrhagiae / copenhageni is the classical parasite of rats, L. grippotyphosa of voles, L. canicola of dogs, L. hardjo of cattle and L. pomona of pigs. [2]

CASE REPORT

18 year old male presented to tertiary care hospital (Kashmir, India) with 5 days history of fever with rigors, chills, myalgias and two days history of multiple episodes of vomiting. Clinical examination revealed pulse-110 beats per minute, Blood pressure -120/70mmhg, temperature-101°F and mild splenomegaly. Investigations at admission revealed pancytopenia with normal kidney and liver function tests. On third day of admission patient developed azotemia and transaminitis. Routine urine showed 20-25 pus cells with granular casts, Ultrasonography abdomen -spleen 15cm, Peripheral blood film for malarial parasite negative, dengue serology negative, low widal titres, Brucella serology negative, Hepatitis serology negative, blood culture
and urine culture sterile, Bone marrow-reactive marrow changes likely infective pathology, leptospira IgM-38.63 u/ml (negative<15u/ml) and IgG 2.13u/ml (negative <10.00u/ml). Patient was managed with injection ceftriaxone 1 gram intravenous bid for 1 week and supportive treatment. After 1 week of therapy patient became afebrile, with normalization of blood cell counts, kidney, Liver Function tests and decrease in leptospirosis titres.

DISCUSSION

Leptospirosis classically presents as a biphasic illness in humans. The first phase of the disease is commonly referred to as the septicemic phase. It is characterized by fever, headache, myalgia, conjunctival congestion, and a host of non-specific features. This phase is followed by a brief afebrile period of variable duration that, in turn, is followed by the immune phase of illness. The common organs Involved during this phase are the liver and kidneys. Both organ derangements are reversible. The severe form of leptospirosis, also known as Weil's disease, is characterized by a fulminant course with rapid onset of hepatic and renal failure and high mortality. In a retrospective report of 34 patients with leptospirosis, the common clinical features included fever (100%), headache (75%), myalgia (55%), arthralgia (45%), and vomiting (39%). Among the unusual manifestations of leptospirosis are hemorrhagic pneumonia, aseptic meningitis, myelopathy, and cerebellar dysfunction, reactive arthritis, male hypogonadism, and pancytopenia. Although thrombocytopenia has been extensively reported, along with renal and hepatic involvement, pancytopenia secondary to leptospirosis has been scarcely mentioned in literature. The pathogenesis of pancytopenia in leptospirosis has been poorly understood. Some authors postulated that this could possibly be attributed to disseminated intravascular coagulation (DIC), a toxin, or cytotoxin-mediated mechanism as a direct complication of leptospiral vasculitis or as a general phenomenon of septicemia. Pancytopenia was associated with a higher incidence of complications. From these findings, it can be postulated that the presence of pancytopenia could be an indicator of the severity of the disease. It is important for clinicians to be aware and recognize the various ways in which leptospirosis can present. Although classically occurring as an acute febrile illness with renal failure and jaundice, the other less common manifestations may predominate. The diagnosis of leptospirosis requires a high degree of clinical suspicion because the numerous manifestations of the disease can mimic other tropical infections or other nonspecific febrile illnesses, as well as noninfectious diseases such as small vessel vasculitides, systemic lupus erythematosus, or even malignancies. The initial diagnosis of leptospirosis remains a clinical one, a presumed analysis in the appropriate epidemiologic and clinical context. Routine laboratory testing is nondiagnostic but may show elevated erythrocyte sedimentation rate, peripheral leukocytosis, variable degrees of cytopenias, mildly increased aminotransferases and increased serum bilirubin and ALP. Isolation of the organism by culture of clinical specimens (blood, CSF, urine) during the first seven to 10 days of the illness is considered the gold standard of diagnosis. The majority of leptospirosis

<table>
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<tr>
<th></th>
<th>Haemoglobin gm/dl</th>
<th>Total leucocyte count</th>
<th>Platelet count</th>
<th>Creatinine mg/dl</th>
<th>Urea mg/dl</th>
<th>Alanine aminotransferase U/L</th>
<th>Aspartate aminotransferase U/L</th>
<th>Bilirubin mg/dl</th>
<th>IgM u/ml</th>
<th>IgG u/ml</th>
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<tbody>
<tr>
<td>Admission</td>
<td>11.0</td>
<td>1.6x10^3</td>
<td>16,000</td>
<td>1.4</td>
<td>34</td>
<td>36</td>
<td>40</td>
<td>1.2</td>
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</tr>
<tr>
<td>3rd day</td>
<td>10.9</td>
<td>1.8x10^3</td>
<td>24,000</td>
<td>4.0</td>
<td>65</td>
<td>168</td>
<td>108</td>
<td>3.9</td>
<td>38.6</td>
<td>2.13</td>
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<tr>
<td>10th day</td>
<td>13.6</td>
<td>4.8x10^3</td>
<td>96,000</td>
<td>1.3</td>
<td>52</td>
<td>44</td>
<td>40</td>
<td>2.6</td>
<td>15.4</td>
<td>1.9</td>
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cases are diagnosed by serologic testing of which MAT is most common. The vast majority of infections with leptospira are self-limiting, and it remains controversial if antimicrobials produce benefit in cases of mild leptospirosis without end-organ damage. The current choices of treatment for mild leptospirosis include oral doxycycline and amoxicillin. Parenteral high-dose penicillin G has long been considered the treatment of choice of fulminant leptospirosis. At present, the broad-spectrum third generation cephalosporins, cefotaxime, and ceftriaxone are also considered as acceptable agents for patients with severe leptospirosis. The use of steroids in patients with leptospirosis has not been well established. Several case reports have described the beneficial effects of glucocorticoids in severe leptospirosis with pulmonary hemorrhage, thrombocytopenia, and renal failure. In the present case, pancytopenia reversed completely with the use of intravenous cefalosporins. In conclusion, leptospirosis should be considered as a differential in febrile pancytopenia even in the absence of the usual manifestations.

REFERENCES


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