

A Case Report of Pancytopenia in Leptospirosis

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Abstract

Leptospirosis is a zoonotic illness that can appear with a variety of symptoms in addition to the standard ones of renal insufficiency and jaundice. In this report, we present a case of fever with the decrease in all three cellular components of blood (i.e RBC, WBC, platelets), subconjunctival hemorrhage, and hepatosplenomegaly as the presenting clinical manifestation of leptospirosis, pancytopenia reversed completely with one week of intravenous third-generation cephalosporins. Pancytopenia secondary to leptospirosis has received relatively little attention in the literature, despite the fact that thrombocytopenia, along with renal and hepatic dysfunction, has been widely described. In summation, this clinical study implies that febrile pancytopenia's differential diagnosis should include *Leptospira* infection also.

1. Introduction

Recent occurrences of leptospirosis, a serious zoonotic disease, on basically every continent serve as evidence of its apparent resurgence. Leptospirosis ranges from subclinical infection to a fulminant, lethal condition, and is brought on by pathogenic *Leptospira* species, which are spirochetes. Transmission occurs through abraded skin, cuts, and mucous membranes like conjunctival or oral mucosa exposure to flood waters or recreational freshwater activities like swimming, and windsurfing contaminated by urine from infected animals. Leptospirosis is classically biphasic. In the acute leptospiremic phase a mild form of leptospirosis presents with nonspecific symptoms like fever, headache, and myalgia. Leptospire can be cultured in urine throughout the immunological phase, and the disappearance of signs may correspond with the development of antibodies. Severe disease may be monophasic and fulminant which includes Weil's syndrome characterized by jaundice, kidney failure, and hemorrhagic manifestations. Here we present a patient with an unexplained fever with pancytopenia and hepatosplenomegaly.

2. Case History

Here we are discussing a case of 55 years old male, who presented with 10 days history of fever, severe myalgia, loss of appetite, mild reduction in weight, c/o passing red-coloured urine, mild breathlessness NYHA grade 1. History revealed that the patient accidentally fell into sewage 2 weeks back. On examination, the patient had pallor+, icterus+, subconjunctival haemorrhage (fig 1), grade 1 clubbing of fingers, and no cyanosis. On Abdominal palpation revealed an increase in size of the liver palpating 3 cm below the right costal margin, and an increase in size of the spleen 2 cm below the left costal margin(fig 2). The remaining system examinations were normal. Vitals showed a temperature of 99.8 degrees Fahrenheit, pulse was 101/min, and BP was 100/70 mmHg. On the fourth day of hospitalization, the patient developed increased Blood Urea Nitrogen and transaminitis. Urine complete analysis showed 25-27 pus cells with white granular to waxy casts.



Figure 1. Arrow Mark Showing Subconjunctival Hemorrhage

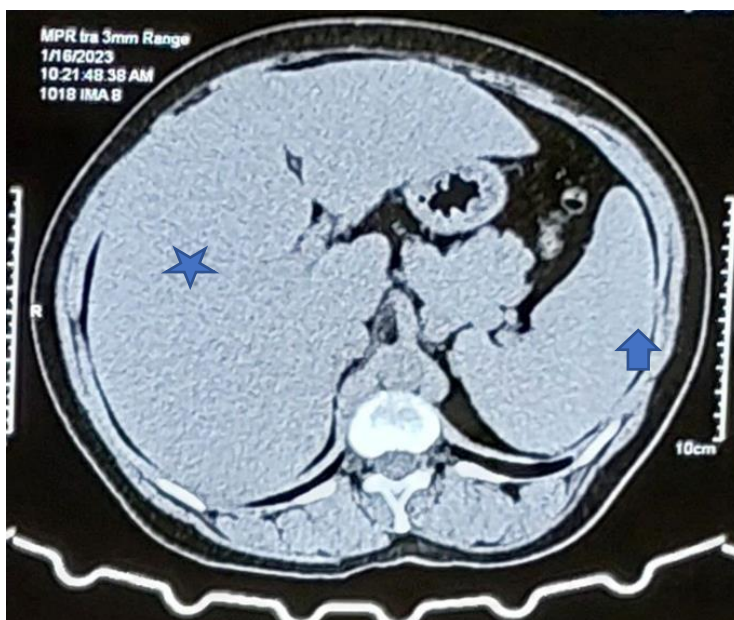


Figure 2. Star Mark Showing Hepatomegaly, Arrow Mark Showing Splenomegaly

Laboratory investigations-

Table 1 investigations at admission and during the first and second week of illness

	Hb	Total leucocyte count	Platelet count	Creatinine mg/dl	Urea mg/dl	Alanine aminotransferase u/l	Aspartate aminotransferase u/l	Bilirubin mg/dl	serology	
									IgM u/ml	IgG u/ml

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AMISSIO N DAY	9.0	3900	9000	1.5	37	41	37	1.3	
4 th day	10. 8	6007	19000	3.9	66	179	109	3.8	37.9 2.10
11 th day	12. 1	10,000	80000	1.2	51	40	41	2.7	15.6 2.0

Other laboratory findings- Blood and urine culture tests showed no growth of the organism, erythrocyte sedimentation rate was high, its 134mm/h. serum iron low (26 mu/dl), serum TIBC low(121mg/dl), and serum ferritin were 401ng/ml. Hemophagocytic lymphohistiocytosis was ruled out as a peripheral blood smear showed a microcytic hypochromic picture and the fasting lipid profile was in normal limits. Viral serology of HIV, VIRAL Hepatitis A, B, C, and E markers are negative. Thyphidot IgM and IgG, CMV IgM and IgG, Malaria MP, MF, QBC, and Dengue serology were negative. Leptospira MAT was positive, IgM 40.01 (negative is <15u/ml)

The patient was managed with an injection of ceftriaxone 1gm iv twice a day for 7 days, and 4 units RDP and 1 unit SDP were transfused, and adequate fluid resuscitations are given. After 1 week Leptospira titers begins to reduce, fever got settled, Liver and kidney tests begins to normalize, and platelet count started improving. At 4th week of hospitalization, hepatosplenomegaly disappeared . . He was very well on his control after one and four months of his discharge

3. Discussion

The persistence of leptospire in the kidneys among several domestic and wild reservoir hosts accounts for their prevalence in nature and the danger they pose to people. The leptospiral life cycle comprises shedding in the urination, persistence in the environment, recruitment of a new host, and persistence in the ambient environment in addition to extramedullary propagation to the renal via the glomerulus or peritubular capillaries brush edge of the proximal convoluted tubular epithelium is colonized by leptospire after they have acquired access to the renal tubular lumen. From this point on, urine spilling can continue for a very long time without significantly harming the host of the reservoir. Because of this, the

leptospiral infection of the reservoir host may be viewed as a commensal relationship. Rats in particular are the main source of leptospirosis in rodents. ^[1,2] In humans, leptospirosis typically manifests as a biphasic sickness. The septicemic phase of the illness is the name given to its initial stage. Fever, head pain, muscle aches, conjunctival congestion, and a number of other generic symptoms are its defining characteristics. The short, variable-duration afebrile phase that follows this phase is then followed by the illness' immunological phase. The liver and kidneys are the primary systems involved in this phase. Both organ abnormalities can be reversed. Weil's disease, also known as the aggressive form of leptospirosis, is characterized by a fulminant course with a rapid onset of liver and renal failure and a high rate of mortality^[3]. Pancytopenia secondary to leptospirosis has received minimal attention in the literature, despite the fact that low platelet count, along with renal and hepatic dysfunction, has been widely described. Uncertainty surrounds the pathophysiology of pancytopenia in leptospirosis. Some researchers proposed that this might be explained by a mechanism involving a toxin, cytotoxin, or disseminated intravascular coagulation as a result of leptospiral vasculitis or as a general occurrence of bacteremia. ^[4] The incidence of complications was increased in people with pancytopenia. From these results, it can be hypothesized that the occurrence of pancytopenia may be a sign of the disease's severity. Physicians must be aware of and adept in identifying the varied manifestations of leptospirosis. The other, less common signs may predominate despite the fact that acute febrile sickness with renal failure and jaundice is the traditional presentation. The various symptoms of the illness might imitate other tropical infections, other indeterminate febrile illnesses, as well as noninfectious conditions such as small vessel vasculitides, SLE, IgA vasculitis or even carcinomas, making the diagnosis of leptospirosis highly clinically suspicious. Leptospirosis's first diagnosis is still clinical, based on

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a presumptive examination in the proper epidemiologic and clinical setting.

4. Conclusion

The most accurate way to diagnose an infection is to isolate the organism by cultivating clinical specimens (blood, CSF, and urine) within the first seven to ten days of the illness. The majority of leptospirosis cases are identified with serologic testing, the most frequent of which is MAT [5]. It is still debatable whether antimicrobials are beneficial in situations of moderate leptospirosis with no end-organ damage because the great majority of leptospira infections are self-limiting. Amoxicillin and oral doxycycline are the two options for the treatment of mild leptospirosis currently available. Injection of high-dose IV penicillin G has long been considered the management of choice of fulminant leptospirosis. Currently, patients with severe leptospirosis are also thought to benefit from using broad-spectrum third-generation cephalosporins including cefotaxime, cefoperazone, ceftazidime, and ceftriaxone. Leptospirosis patients have not been extensively studied when it comes to the usage of steroids. In extreme leptospirosis with bronchial bleeding, low platelets, and renal failure, the therapeutic effects of corticosteroids have been recorded in a number of case reports. Cephalosporins

used intravenously in this patient completely reversed pancytopenia. In conclusion, even in cases of febrile pancytopenia without the typical symptoms, leptospirosis should be considered as a differential diagnosis. [6]

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