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As a different cause of kidney failure, liver failure, and fever: Leptospirosis

Abstract

Leptospirosis is the most common bacterial zoonosis globally, especially in tropical and temperate regions with heavy rainfall. Infection into humans occurs in direct contact with the urine of the sick animals' contact with the environment contaminated with urine. The clinical spectrum of leptospirosis is quite broad. It is subclinical in 90% of cases. Multiple organ failure, especially kidney, liver, and lung, can be seen in 5-10% of cases [1-2]. Weil's disease is the most severe form of leptospirosis. It progresses with liver dysfunction, acute renal failure, thrombocytopenia, and fever; If left untreated, it can be fatal at 1-5% [3]. In our case, a 60-year-old patient with fever, hyperbilirubinemia, acute renal failure, and thrombocytopenia will be discussed.

Keywords: Liver dysfunction, acute renal failure, thrombocytopenia, fever

CASE REPORT

At the age of 60, a male patient who has no previous systemic disease other than hypertension, coronary artery disease, and diabetes mellitus applied to the emergency department about one week ago with complaints of fever, nausea, vomiting, and dizziness. In the laboratory tests, requested after routine examination, leukocytosis, hyperbilirubinemia, kidney function tests, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) values detected elevated. Initial evaluation of the patient; general condition was moderate to good; tachycardia, tachypnea, fever, icterus, splenomegaly, and petechiae were examined; there were no unusual symptoms in other systemic examinations. The patient's hemogram had leukocytosis and thrombocytopenia. Also, the peripheral blood smear was compatible with the hemogram. In the biochemical examination, hyperbilirubinemia with direct dominance and creatinine values were elevated. There was metabolic acidosis with wide anion gap

in blood gas. Then, empirical ceftriaxone treatment was started. The patient was on dialysis twice due to uremic complications. After the fourth day of her clinical follow-up, the patient did not have a fever, Bun-Creatine values decreased, thrombocytopenia resolved, and ESR and CRP values decreased to normal ranges. No microorganisms were grown in the culture. The patient, who was followed up in our clinic with a prediagnosis of Weil's disease, was discharged after 14 days of ceftriaxone treatment.

DISCUSSION

Leptospirosis is a common bacterial zoonosis in the world. It is seen in farmers, sewer workers, hunters, and swimmers in streams and lakes. The route of infection to humans; is caused by spirochetes in water and soil that come into contact with the urine of infected animals and pass through the skin, mucous membranes, and conjunctiva [2,3]. Our case was also a farmer and engaged in field irrigation. Its annual incidence

varies between 0.1-1/100 000 in regions with low rainfall and 10-100/100 000 in tropical regions. The incidence is well above 100/100 000 during the epidemic after natural disasters and in risky groups [4]. In leptospirosis, leukocytosis and left shift of the oxygen dissociation curve is frequently observed as laboratory findings; thrombocytopenia is also commonly seen [5]. Some studies reported that thrombocytopenia was found in 75% of surviving patients and 83.3% of cases resulting in death [6]. Thrombocytopenia is transient and independent from disseminated intravascular coagulation. During infection, plasma 11-dehydro-thromboxane B₂ (11-DH-TXB₂) level increases. 11-DH-TXB₂ induces activation-aggregation of platelets and phagocytosis by Kupfer cells. That is an essential mechanism in the pathogenesis of thrombocytopenia [7]. Again, In some studies, the relationship between thrombocytopenia and Weil's disease has been tried to be explained by disseminated intravascular coagulation [8]. In our case, platelet values were below 50.000. Platelet values increased from the fourth day of antibiotic treatment and remained within normal ranges. The definitive diagnosis of leptospirosis is made by producing and demonstrating the agent in culture. However, a bacterial culture is not practical in routine laboratories since the growth period of the bacteria is quite long (2 weeks-6 months) and requires special media [1]. It can be seen with Dark-field microscopy in clinical samples. However, the sensitivity of Dark-field microscopy is low. In order to evaluate spirochetes in Dark-field microscopy, 104 bacteria per milliliter should be present in the sample. Other methods used for diagnosis are the Microscopic agglutination test (MAT), the Complement Fixation Test (CFT), Enzyme-Linked Immunosorbent Assay (ELISA), Radioimmunoassay (RIA), and Polymerase Chain Reaction (PCR) [5]. Generally, oral Doxycycline (200 mg/day, one week), Ampicillin (4x500-750 mg), or Amoxicillin (3x500 mg) is recommended in mild-moderate cases. In severe cases, parenteral Penicillin (4x1.5 million units) or Ceftriaxone (1x1 gr/day) is recommended [1]. We also gave our patient 2*1 gr Ceftriaxone parenteral antibiotic treatment.

CONCLUSION

In conclusion, Weil's disease should be considered in the

differential diagnosis of patients with severe thrombocytopenia, hepatic dysfunction, and renal failure who applied with the complaints of fever, muscle pain, and icterus both in our region and in our country.

Conflict of interests

The authors declare that they have no competing interests.

Financial Disclosure

All authors declare no financial support.

Informed Consent

Written consent was obtained from the patient and his parents.

REFERENCES

1. Levett PN. Leptospirosis. In: Mandell GL, Bennett JE, Dolin R, eds. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. 6th ed. Philadelphia: Elsevier Churchill Livingstone, 2005:2789-95.
2. Doudier B, Garcia S, Quennee V, et al. Prognostic factors associated with severe leptospirosis. Clin Microbiol Infect. 2006;12:299-300.
3. William AP. Leptospirosis. Goldman L, Ausiello D, Arend WP, Armitage JO, Clemmons DR, Drazen JM, Criggs RC, Larusso N. Goldman: Cecil Medicine. Saunders, An Imprint of Elsevier 24rd ed. 2011:1720-1.
4. Lau C, Smythe L, Weinstein P. Leptospirosis: an emerging disease in travellers. Travel Med Infect Dis. 2010; 8:33-9.
5. Levett PN. Leptospirosis. Clin Microbiol Rev. 2001;14:296-326.
6. Esen S, Sunbul M, Leblebicioglu H, et al. Impact of clinical and laboratory findings on prognosis in leptospirosis. Swiss Med Wkly. 2004;134:347-52.
7. Edwards CN, Nicholson GD, Hassell TA, et al. Thrombocytopenia in leptospirosis: the absence of evidence for disseminated intravascular coagulation. Am J Trop Med Hyg. 1986; 35:352-4.
8. Slofstra SH, Spek CA, Cate H. Disseminated intravascular coagulation. Hematol J. 2003;4:295-302.