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CASE PRESENTATION

Síndrome de Weil. Reporte de Caso

Weil's Syndrome. Case Report

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RESUMEN

La leptospirosis fue reconocida en sus inicios como una enfermedad ocupacional de los obreros de las alcantarillas. El Dr. Weil en 1886, describe las manifestaciones clínicas en cuatro hombres con ictericia, fiebre y hemorragias con deterioro renal. El síndrome de Weil es una forma grave de la enfermedad por leptospirosis. El objetivo del informe es describir un paciente con síndrome de Weil, sus características clínicas y el procedimiento terapéutico empleado. Paciente masculino de 25 años de edad con antecedentes de salud que asiste a servicios de Urgencias por presentar fiebre, polipnea, ictericia, mialgia y cefalea y se le confirma el diagnóstico de síndrome de Weil. Al realizarse el diagnóstico del síndrome de Weil, es necesario alcanzar un control inmediato de la enfermedad a través de un tratamiento conveniente para el logro de la supervivencia de los pacientes.

Palabras clave: síndrome de Weil, leptospirosis, ictericia,tratamiento
Descriptores: leptospirosis/diagnóstico/complicaciones; ictericia; terapéutica

ABSTRACT

Leptospirosis was initially recognized as an occupational disease of sewer workers. In 1886, Dr. Weil described the clinical manifestations in four men with jaundice, fever, and hemorrhages with renal impairment. Weil's syndrome is a severe form of leptospirosis. The aim of this report is to describe a patient with Weil's syndrome, his clinical characteristics, and the therapeutic procedure employed. A 25-year-old male patient with no prior medical history attended the Emergency Department presenting with fever, polypnea, jaundice, myalgia, and headache; the diagnosis of Weil's syndrome was confirmed. Upon diagnosing Weil's syndrome, it is necessary to achieve immediate control of the disease through appropriate treatment to ensure patient survival.

Keywords: Weil's syndrome, leptospirosis, jaundice.
Descriptors: leptospirosis/diagnosis/complications; jaundice; therapeutics

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INTRODUCTION

In 1883, leptospirosis was recognized as an occupational disease of sewer workers. In 1886, Dr. Weil described the clinical manifestations in four men with jaundice, fever, and hemorrhages with renal impairment, and in 1915 the pathogenic agent was cultured and isolated by the Japanese scientists Inada and Ido, who named it Spirochaeta icterohaemorrhagiae.[1]

Leptospirosis is a common bacterial anthropozoonotic disease in humans and animals, caused by organisms that are antigenically different but morphologically identical, belonging to the genus Leptospira.[2] There are several clinical forms; it is stated that 90 % present in a mild, self-limiting form, and 10 % progress to a severe, icterohemorrhagic form, which can be fatal.[1-3] Two phases are distinguished during the infection: a leptospiremic phase, marked by the presence of the bacteria in the blood, and an immune or leptospiruric phase, characterized by the appearance of IgM antibodies against Leptospira, mediated by the innate human immunity.

According to the WHO, the incidence of the disease can vary from 0.1 to 1 case per 100,000 inhabitants in temperate climates and from 10 to 20 cases per 100,000 inhabitants in tropical climates.[4,5]

Weil's syndrome is a severe form of leptospirosis characterized by impairment of liver and kidney functions. The most severe cases can progress directly from the acute phase with fever above 40 °C and rapid onset of liver failure, acute kidney injury, hemorrhagic pneumonitis, hemodynamic instability with cardiovascular involvement.[1,6]

Therefore, the objective of this report is to describe a patient with Weil's syndrome, his clinical characteristics, and the therapeutic procedure employed.

CASE PRESENTATION

Patient Information

A 25 year old male patient, with no prior medical history, from a rural area, working as a farmer, presented to the Emergency Department with fever, polypnea, jaundice, myalgia, and headache.

History of Present Illness

During the interview, he reported that seven days prior to admission, he had experienced a fever of 39 °C (102.2 °F), accompanied by chills, profuse vomiting, diarrhea, intense abdominal pain, myalgia, headache, and shortness of breath.

Diagnostic Evaluation

Physical Examination

-Weight: 65 kg, Height: 175 cm, Body Mass Index (BMI): 17.6 kg/m².
-Mucous membranes: dry (xx) with a jaundiced tint.
-Respiratory system: respiratory rate of 30 breaths per minute.
-Cardiovascular system: rhythmic heart sounds, central heart rate of 119 beats per minute, blood pressure of 100/62 mmHg.
-Mean arterial pressure (MAP) of 74.6 mmHg, with a capillary refill time of four seconds and distal thermal gradient at the hands and feet.
-Skin: jaundiced with hemorrhagic lesions (petechiae and ecchymosis), Figure 1.
-Urine: scant and coluric (dark urine).

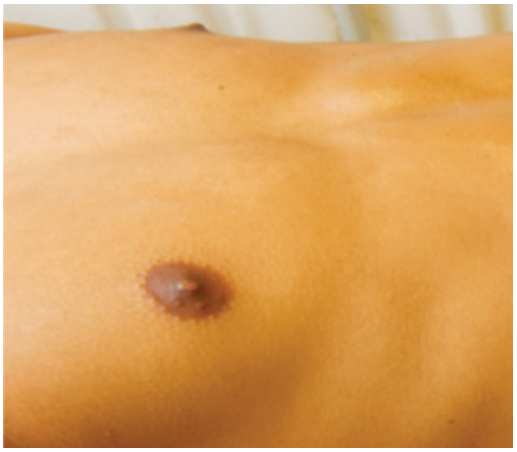

Fig. 1. Yellowish discoloration of the skin showing a jaundiced tint

Complementary Examinations

-Complete blood count: Hemoglobin: 100 g/L, Hematocrit: 0.30, Leukocytes: 12.8 x 10^9/L, Neutrophils: 0.80, Lymphocytes: 0.20.
-Peripheral blood smear: Normocytic – normochromic red blood cells.
-Coagulation profile: Platelet count: 90 x 10^9/L with macroplatelets and disaggregated platelets.
-Activated Partial Thromboplastin Time (aPTT): 60 seconds.
-Prothrombin Time (PT): Control (CT): 14 seconds, Patient PT: 24 seconds.
-Activated Partial Thromboplastin Time (aPTT): Normal value up to 35 seconds; evaluates intrinsic pathway factors (I-fibrinogen, II, V, VIII, IX, X, XI, XII, prekallikrein, and high molecular weight kininogen).
-Prothrombin Time (PT): Normal value up to

three seconds above the control; evaluates extrinsic pathway factors (I-fibrinogen, II, V, VII, X).
-Creatinine: 140 µmol/L.
-Glomerular filtration rate: 65.9 ml/min.
-Urinalysis: Turbid yellow appearance, proteins (XXX), leukocytes: 15-20 per xc, red blood cells: 1-2 per xc, abundant pyocytes and granular casts observed.
-Abdominal ultrasound: Hepatomegaly observed, free abdominal fluid, moderate bilateral pleural effusion, Figure 2.

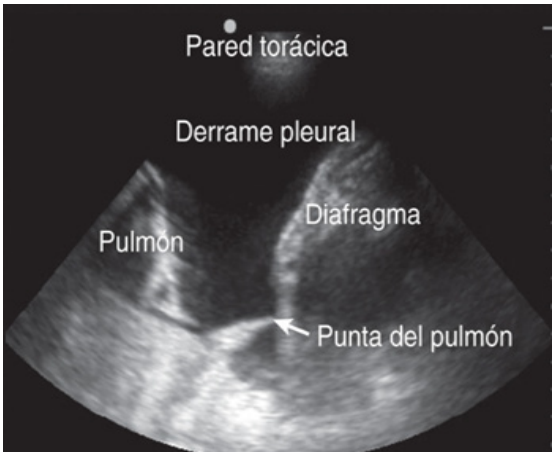

Fig. 2. Bilateral pleural effusion

-Bilirubins: Direct: 62 mmol/L, Indirect: 21 mmol/L, Total: 83 mmol/L.
-Glutamic-Oxaloacetic Transaminase (GOT/AST): 184 U/L
- Glutamic-Pyruvic Transaminase (GPT/ALT): 78 U/L
- Leptospirosis test: Positive.

The complementary examinations confirmed that the patient presented alterations in renal, hepatic, and hematological function. The leptospirosis test was positive, and together with the clinical manifestations presented, a diagnosis of Weil's syndrome was made. The patient was admitted to the Intensive Care Unit (ICU).

Therapeutic Intervention
In the ICU, hydration with crystalloids was initiated according to the patient's needs and his condition. At the same time, an antimicrobial agent was administered: crystalline sodium penicillin G, 1 million units per vial, two vials every four hours intravenously (IV) for three days, followed by one vial every six hours IV for seven days.

Additionally, one ampoule of 10 mg/ml of phytomenadione (Vitamin K1) was administered every 12 hours intramuscularly (IM) for five days, along with fresh frozen plasma, one 250 ml bag every 12 hours for two days.

Follow-up and Outcomes
The patient initially presented to the emergency department with fever, marked jaundice, polypnea, tachycardia, relative hypotension, oliguria, and hemorrhagic skin lesions. Complementary studies revealed:
-Hyperbilirubinemia (83 mmol/L)
-Elevated creatinine (140 µmol/L)
-Thrombocytopenia (90 x 10^9/L)
-Positive leptospira test. The diagnosis of Weil's syndrome was confirmed.

The patient was transferred to the Intensive Care Unit (ICU), where he remained for seven days. During the first three days, he continued to present with jaundice and polypnea, although with partial improvement in blood pressure.

Between the fourth and fifth days, fever and dyspnea decreased, with a progressive decline in bilirubin levels and slight improvement in renal function and platelet count. On the sixth day, the patient was more stable, with reduced jaundice and laboratory parameters showing a downward trend, which allowed his transfer to the general ward on the seventh day.

During the following four days in the ward, he evolved favorably: afebrile, with evident clinical improvement, and complementary parameters decreased, with bilirubin of 40 mmol/L*, creatinine of 118 µmol/L, and platelets of 170 x 10^9/L. He was discharged after 12 days of hospitalization, fully recovered. Due to the initial severity of the condition and the risk of late complications, he was followed up by his family physician.

Ethical Considerations
The present research respects the ethical principles established in the Declaration of Helsinki. The patient was treated according to current care protocols, and the confidentiality of his personal data was guaranteed at all times. Informed consent was requested and obtained for the use of clinical information for academic and publication purposes, ensuring that the patient's identity and rights were not violated.

DISCUSSION

Leptospirosis is a zoonosis caused by aerobic spirochetes of the genus Leptospira, of which 21 species and more than 200 serovars have been identified.[5] The risk of occurrence of these diseases is a latent problem due to the constant interaction of humans with domestic animals and livestock; furthermore, the in-

vasion of ecosystems where wild animals are found makes these diseases a challenge for public health.[7]

Anicteric leptospirosis is the most frequent and mild form of the disease, and is usually biphasic. With an incubation period of two to 20 days, it begins with the initial or septicemic phase. After an interval of one to three days of symptomatic improvement and disappearance of fever, the second or ¨immune¨ phase begins; however, in severe disease, the phases may be indistinguishable.[8]

Leptospirosis has variable clinical manifestations and can be asymptomatic; for this reason, it is frequently misdiagnosed. It is a self-limiting anicteric febrile illness in 85 to 90 % of cases and severe in 5 to 10 % of cases (Weil's syndrome). [1,3]

Weil's disease corresponds to the severe phase of the illness, also known as icterohemorrhagic fever. It is the form with the worst prognosis and can appear from the onset of the disease or in a second phase. It affects various organs, including the kidneys, liver, and lungs. The classic presentation is characterized by the triad of hemorrhage, jaundice, and acute nephropathy. Pulmonary involvement can present as alveolar hemorrhage. Weil's disease may also present with other hemorrhagic manifestations, such as gastrointestinal bleeding and epistaxis.[9]

In severe leptospirosis, from a macroscopic perspective, visceromegalies such as hepatomegaly and splenomegaly are reported in 20% of cases.[10]

It has been estimated that the annual mortality rate of leptospirosis is 0.84 deaths per 100,000 people worldwide, increasing to up to 10 % of individuals when Weil's syndrome occurs.[5] Jaundice is the first sign of severity; patients with severe jaundice have a higher probability of kidney failure, liver failure, and cardiovascular collapse.

Antimicrobial therapy constitutes the most important specific treatment and should be initiated early once the disease is diagnosed. In this case, crystalline penicillin was administered. It is understood that due to the degree of dehydration presented by the patient (caused by vomiting, diarrhea, fever, and polypnea), an appropriate hydration regimen was administered.

When vitamin K is administered intravenously, there is greater urinary excretion of its metabolites, which implies a lower capacity for tissue storage and, over time, a loss of its capacity for release into the systemic circulation. Low levels of this vitamin lead to inadequate activity of coagulation factors, increasing the risk of bleeding in various organs.[11] There are three known types of vitamin K (VK): phylloquinone (PQ), menaquinone (MN), and menadione, designated vitamins K1, K2, and K3. [12]

The use of vitamin K1 acquires great clinical relevance in this case, since its administration contributes to the synthesis of vitamin K-dependent coagulation factors (II, VII, IX, and X). This aims to reduce the risk of severe bleeding and improve patient stability.

When Weil's syndrome is diagnosed, it is necessary to achieve immediate control of the disease through appropriate treatment to ensure patient survival.

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Dra. Legna Iris Nicolarde Franco	Data curation, writing – review and editing.
Dr. Rider Brizuela Oliva	Methodology, writing – review and editing.

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