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Research Article

ATYPICAL RENAL PRESENTATION IN SEVERE LEPTOSPIROSIS

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A B S T R A C T

Rarely, leptospirosis may cause an autoimmune condition known as Weil disease. It occurs after the bacteria have been cleared from the circulation during a period of bacteremia, but antibody-mediated mechanisms continue to have an effect on the patient's internal organs. Symptoms often manifest as acute kidney damage and high-grade hyperbilirubinemia, weeks after exposure. In severe cases of chronic Weil disease, treatment with antibiotics, plasmapheresis, hemodialysis, and glucocorticosteroid therapy may be required to speed the patient's recovery.

INTRODUCTION

Rarely, leptospirosis may cause an autoimmune condition known as Weil disease. It occurs after the bacteria have been cleared from the circulation during a period of bacteremia, but antibody-mediated mechanisms continue to have an effect on the patient's internal organs. Symptoms often manifest as acute kidney damage and high-grade hyperbilirubinemia, weeks after exposure.

CASE REPORT

A 57-year-old man was hospitalised with acute renal failure and hyperbilirubinemia but had no previous medical history. He was hospitalised after a two-week bout with fever and chills. After the early signs subsided, muscular soreness and a reduced ability to exercise set in. Urinary output decreased and jaundice became noticeable shortly after. After three days, he finally had to check into the hospital.

The results of the first blood work showed elevated serum levels of creatinine (6 mg/dl) and urea (180 mg/dl), as well as elevated serum inflammatory markers (C-reactive protein levels >60mg/l) and total bilirubin (>18 mg/dl, mostly conjugated). All of the other indications of liver damage were also within normal limits; these included alanine- and glutamate-pyruvate transaminases, prothrombin time, and alkaline phosphatase in the blood. Albumin levels were lower than normal (22 g/l) and total serum protein levels were lower (41 g/l). Tests for nephrotic-range proteinuria and erythrocyturia were positive. Platelet count (28,200/ml) and haemoglobin (Hgb) level (9.2 g/dl) were both lower than normal in a full blood count. The results of the hepatitis B virus and hepatitis C virus serological testing came back negative.

Initial treatment included both cephalosporin (cefotaxime) and continuous renal replacement therapy.

Vital signs were normal, there was jaundice, there was generalised swelling, and there were symptoms of thrombocytopenic purpura. No evidence of respiratory or circulatory failure, or isolated neurological symptoms, were seen. A urine analysis revealed extreme proteinuria (475 mg/dl) and erythrocyturia (all visible urine was red and the number of leukocytes in the urine ranged from 2 to 5). 7.5 mg/dl was the creatinine concentration. Creatine phosphokinase (CPK) was 31 U/l (normal), and potassium was 5.0 mmol/l in the blood.

Persistent jaundice and oliguria necessitating renal replacement medication highlighted the possibility of a Leptospira infection, as did the patient's proximity to a rice field and a lake, as revealed by a study of his medical history. Both a polymerase chain response investigation of Leptospira microorganism presence and a minuscule agglutination test were performed on gathered blood and urine tests. After seeing the severe encephalopathy, it was decided to have a plasmapheresis using albumin. Two sessions later, the patient’s condition had improved dramatically. After that time, the patient had solely hemodialysis therapy, and his urine production began to rise sharply. However, the microscopic agglutination test showed a highly positive result with an antibody titer of 1:3300, despite the fact that the polymerase chain reaction test for Leptospira in both urine and blood had previously shown negative results.

The patient's physical state was steadily improving, and the majority of the symptoms that had been present upon admission had vanished. Renal biopsy was performed because of the persistent renal insufficiency. Staining with Grocott's

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methenamine silver revealed cellular inclusions in the tubular lumen of the kidney biopsy samples that resembled whole Leptospira.

The results of a kidney biopsy. Inclusions (arrows) that look like Leptospira were discovered by Grocott's methenamine silver staining in the lumen of renal tubules (original magnification, 100).

After the biopsy, the patient received methylprednisolone infusions (250 mg/d) intravenously for 3 days before switching to oral prednisone (20 mg/d). This caused the patient's urine output to rise to as high as 6,000 ml per day, and the patient's blood creatinine and bilirubin levels to gradually decrease. Dialysis was stopped after 1 week and bilirubin and haematological markers reverted to normal. There was no proteinuria or any other aberrant findings on urinalysis, and the serum creatinine level dropped to 1.3 mg/dl after an additional three weeks of prednisone treatment. Inflammatory markers and serum bilirubin levels have returned to normal. The patient was taking 20 milligrammes of prednisone at the time of discharge, with a 5 milligramme decrease scheduled to occur each week. The patient presented with no symptoms and normal physical exam results four weeks later.

DISCUSSION

There is a significant fatality rate associated with Weil illness, one of the most severe forms of leptospirosis. Weil disease often causes acute renal failure with symptoms including decreased urination, low potassium levels, inability to concentrate urine, proteinuria, and blood in the urine. Interstitial injury causes renal involvement more often than not, although glomeruli are spared. The patient presented with proteinuria and anasarca in the nephrotic range, and light microscopy of a kidney biopsy material showed typical interstitial inflammation but no glomerular involvement. Regular urinalysis results were obtained over the course of therapy. Nephrotic-range proteinuria, however, has a strong candidate in podocytopathy (e.g., minimum change disease).
The standardisation of care for Weil illness is lacking. Kidney function may often improve after a few days of therapy with antibiotics and dialysis. Our case differed in that the duration of early symptoms (fever, weariness, myalgia) lasted more than 3 weeks, and these symptoms persisted despite the use of antibiotics.

The patient's health started to improve when they were given corticosteroids, maintained on antibiotics, plasmapheresis, and a hemodialysis schedule. Two rounds of plasmapheresis did wonders for his mental health. One theory for what causes encephalopathy is that antibodies cause harm to the nervous system, and that patients who undergo plasmapheresis would see rapid neurological improvement but no change in their kidney function.

However, polyuria and a reduced blood creatinine level were seen after intravenous methylprednisolone infusions. As the jaundice cleared up, renal function also improved.

Positive findings on a microscopic agglutination test and a negative polymerase chain reaction test for Leptospira indicated an active infection of Leptospirosis. Polymerase chain reaction testing on both samples turned up negative, proposing that the industrious side effects were made by the invulnerable reaction contamination rather than the bacterium itself. Leptospira are only detectable in the bloodstream for 7–14 days after infection and in the urine for about a month.

CONCLUSION

In severe cases of chronic Weil disease, treatment with antibiotics, plasmapheresis, hemodialysis, and glucocorticosteroid therapy may be required to speed the patient's recovery.

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