CASE REPORT

An infectious cause of acute kidney injury with low serum potassium

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SUMMARY

A 67-year-old man was referred to our hospital because of acute kidney injury, thrombocytopenia and hyperbilirubinemia. Despite severe kidney failure, hypokalemia was present. One infectious cause explained the whole clinical picture.

BACKGROUND

The diagnostic work up of patients with acute kidney injury (AKI) sometimes leads to unusual diagnoses of great clinical and pathophysiological interest. This case highlights the typical features of a common and often unrecognised cause of AKI.

CASE PRESENTATION

A previously healthy 67-year-old man was referred to our hospital for suspected haemolytic uraemic syndrome. He described an episode of acute diarrhoea, fever and bilateral calf myalgia during a canoe tour in France, 2 weeks before admission. The patient recovered after a short therapy with amoxicillin/clavulanic acid and metronidazole. However, 1 week later, his general condition deteriorated. Headache, vomiting, epistaxis and finally anuria led him to visit the emergency room of a regional hospital. AKI and severe thrombocytopenia were diagnosed. At admission, the patient was markedly dehydrated; scleral jaundice, conjunctival suffusions and multiple petechiae were observed.

INVESTIGATIONS

Haematological tests revealed thrombocytopenia (24 G/L), modest leucocytosis (10.3 G/L) and normal haemoglobin without schistocytes. Coagulation parameters were normal. General laboratory analyses showed markedly elevated creatinine (924 µmol/L), blood urea nitrogen (51.8 mmol/L) and an elevation of direct bilirubin (178 µmol/L) without signs of haemolysis (lactate dehydrogenase slightly elevated, normal haptoglobin, normal indirect bilirubin). Liver transaminases were modestly elevated (aspartate transaminase 115 U/L, alanine transaminase 96 U/L). Creatine kinase was 974 U/L. C reactive protein (CRP) was 226 mg/L and procalcitonin 55.3 µg/L. Surprisingly, despite the presence of severe AKI, the patient had low serum potassium of 2.6 mmol/L. Blood bicarbonate was 15.6 mmol/L. Screening immunological tests including antinuclear antibody, antineutrophil cytoplasmic antibody, HIV and hepatitis serology, as well as renal sonography were unremarkable. After adequate rehydration, the diuresis recovered.

Urinalysis revealed mild proteinuria (protein/creatinine ratio 0.031 g/mmol); urinary sediment was unremarkable.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of AKI in patients recovering from diarrhoea is broad with dehydration being the most common aetiology.¹ The proposed diagnosis of haemolytic uraemic syndrome would explain renal failure and thrombocytopenia, but this was rather unlikely in the absence of haemolysis. An interstitial nephritis after antibiotic therapy could explain the rapid loss of renal function (and elevation of liver enzymes) but not the severe thrombocytopenia. Rhabdomyolysis or hyperbilirubinemia were considered as possible causes of AKI but were not sufficient to explain the whole clinical picture. Hantavirus infection would have explained renal failure, thrombocytopenia and abdominal symptoms, while low potassium, high CRP and absence of haematuria would have been uncommon. Indeed, this patient had a complex syndrome, with a biphasic clinical presentation that culminated in AKI, a severe inflammatory state, elevated direct bilirubin and thrombocytopenia. Particularly intriguing was the low potassium level in this patient with severe renal failure, a combination suggesting renal tubular dysfunction. In consideration of the history of recreational water activity in the last weeks and of the clinical presentation, we suspected leptospirosis (Weil's disease).

TREATMENT

The patient was treated with doxycycline and required dialysis for 7 days.

OUTCOME AND FOLLOW-UP

In the following days, the patient completely recovered. A high level of IgM against *Leptospira interrogans* confirmed the diagnosis.

DISCUSSION

Leptospirosis is an often unrecognised zoonotic disease with worldwide distribution and probably a common cause of AKI in low-income countries.^{1 2} Leptospirae colonise the proximal renal tubule of carrier animals. Human infection results from exposure to infected urine, directly or via contamination of soil or water through breaches in the skin or mucous membranes. As in this described patient, in Western countries most cases occur in association with recreational activities involving immersion in water. Leptospirosis should be considered in patients with the typical history of non-

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specific prodrome followed by AKI, particularly in combination with myalgias and liver dysfunction with disproportionate hyperbilirubinemia. The pathogenesis of AKI in leptospirosis is multifactorial including hypotension, hypovolaemia, tubular damage related to hyperbilirubinemia and rhabdomyolysis and a direct nephrotoxic action of the leptospirae. Low serum potassium is typically observed in patients with leptospirosis and AKI. Previous studies revealed that leptospirae led to a dysfunction of the thick ascending limb with reduced activity of the NKCC2 transporter resulting in renal potassium wasting.^{3–6} Treatment is largely supportive: in most cases, leptospirosis resolves spontaneously, but severe cases might require intensive care support including dialysis. Weil's disease is the most severe form of this illness and leads to jaundice, renal failure and haemorrhage with reported mortality rates up to 20%. Patients presenting with poor prognostic factors such as hypotension, dyspnoea, oliguria, hyperkalaemia, thrombocytopenia, repolarisation abnormalities

on ECG and alveolar infiltrates should be treated in an intensive care unit.^{7 8} Notably, young healthy patients can also succumb in case of severe leptospirosis. The role of antibiotic therapy with doxycycline, ampicillin, amoxicillin or ceftriaxone is controversial, but most experts recommend antimicrobial treatment if leptospirosis is suspected.⁹

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Contributors PEC, RPW and SS treated the patient and wrote the manuscript. KA treated the patient.

Competing interests None declared.

Patient consent Obtained.

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Learning points

- Critically consider clinical or laboratory parameters that are not fitting in the whole clinical picture for a correct diagnosis.
- Think about infections in the differential diagnosis of acute kidney injury.
- Suspect leptospirosis in patients with acute kidney injury, myalgias and liver dysfunction with disproportionate hyperbilirubinemia.
- Hypokalemia in acute renal failure is a typical feature of leptospirosis.

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