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Case Report

Acute renal failure caused by prolonged djembé drumming

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Background

Acute renal failure (ARF), like pneumonia, can be divided into hospital- and community-based acquisition types. Hospital-acquired renal failure occurs at an incidence rate of 2–5%. The mortality rate of hospital-acquired renal failure, specifically in intensive care units, is high despite a vast array of treatment modalities [1]. In hospital-acquired renal failure, the presence of comorbidities has directed the challenge towards finding proper treatment rather than finding the cause. Community-acquired renal failure has an overall incidence of 1% of hospital admissions. After excluding pre- and post-renal causes, this type of ARF presents more of a diagnostic puzzle although treatment is straightforward [2]. In both hospital- and community-acquired renal failure there is place for prevention. In the hospital setting, prevention of radiocontrast nephropathy is not difficult but prevention of renal failure during multi-organ failure is still elusive. In a community, it is more difficult to find strategies for prevention of ARF because of its numerous and more exotic causes [3].

In the following case history of community-acquired renal failure, we will describe the completion of a diagnostic puzzle and suggest preventive measures for similar cases.

Case report

A 37-year-old Caucasian man presented at the emergency room with ARF. His medical history showed recurrent episodes of epigastric pain, nausea and biliary vomiting followed by passing of dark urine. Because the current episode was of longer duration, the patient visited his general practitioner, who measured a serum creatinine of 434 μmol/L (a previous value was 99 μmol/L). At presentation at our hospital the next day, physical examination was unremarkable. Laboratory results showed a serum creatinine level of 332 μmol/L, urea of 30.9 mmol/L, haemoglobin 8.1 mmol/L, reticulocyte count 0.4%, leucocytes 7.1 $\times$ 10^9/L, platelets 226 $\times$ 10^9/L, potassium 5.9 mmol/L, aspartate aminotransferase (AST) 62 U/L, alanine aminotransferase (ALT) 75 U/L, lactate dehydrogenase (LDH) 517 U/L, total bilirubin 9 μmol/L and indirect bilirubin 2 μmol/L. A dipstick urine specimen was positive for haemoglobin, and microscopic examination of the urine sediment showed 0–4 erythrocytes/hpf. The abdominal ultrasound showed a right kidney of 5 cm diameter and a slightly enlarged left kidney. As the renal impairment had already regressed, a conservative treatment was adopted. Within 2 weeks, serum creatinine returned to 100 μmol/L. The combination of passing dark urine and renal failure suggested haem-pigment-induced acute tubular necrosis (ATN) [4]. A renewed medical history revealed that he was a professional drum player and that previous percussion sessions with his full hands on a West-African drum (the djembé) had resulted in similar episodes. Six days before the present episode, he had continuously played for 8 h for the first time. This sequence of drumming followed by the passing of dark urine and subsequent renal failure led us to a presumptive diagnosis of mechanically induced haemolysis. To support our diagnosis, we asked the patient to play continuously for 3 h and for 4 h. Haptoglobin, which was measured to demonstrate active haemolysis, decreased to 0.19 and 0.08 g/L, respectively. After drumming 4 h, LDH had increased to 329 U/L. The serum creatinine levels did not significantly increase.

To exclude intracorpuscular pathology of the red blood cells (RBC), which may have rendered them more susceptible to mechanical destruction, we examined the spectrin content (99%) and performed the acid glycerol lyses test (>1800 s) to demonstrate congenital spherocytosis, which were normal. The presence of glycophosphatidylinositol-linked proteins CD24, 55, 59 on all granulocytes and the presence of CD14 on monocytes excluded paroxysmal nocturnal haemoglobinuria (PNH). Antibodies directed against RBC membrane components may also cause susceptibility to destruction. A direct Coombs test was negative.

Discussion

Haem-pigment-induced ATN typically presents with pigmented granular casts in the urine, a red-brown colour of

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the urine and a rise in the creatinine level. The causes of this ATN are multi-factorial and include obstruction with intratubular haem-pigment casts, tubular cell injury by free chelatable iron of the haem centre, volume depletion and renal ischaemia. Haem-pigment-induced ATN is divided into myoglobinuria due to rhabdomyolysis and haemoglobinuria due to intravascular haemolysis. In our case, the decrease in haptoglobin, an expression of the binding of free haemoglobin to haptoglobin, excluded myoglobin as a source of the haem-pigment.

The aetiology of haemolysis, in turn, can be divided into intracorpuscular and extracorpuscular causes. To differentiate between these, it is necessary to exclude intracorpuscular RBC pathology. Intracorpuscular defects can affect either the haemoglobin molecule, the RBC membrane or the metabolic machinery within the cell. Most of these defects are hereditary (e.g. thalassaemia, congenital spherocytosis, pyruvate kinase deficiency or glucose-6-phosphate dehydrogenase deficiency), but some are acquired (e.g. PNH). Because our patient was not known to have enzyme deficiency, we performed an acid glycerol lysis test and counted the RBC spectrin to exclude spherocytosis. The presence of glycophosphatidylinositol-linked proteins excluded PNH.

Extracorpuscular defects can be caused by autoimmune haemolytic anaemia, hypersplenism and other exceptional causes. A direct Coombs test eliminated autoimmunity against RBC membranes. The major cause of extracorpuscular RBC destruction, however, is mechanical trauma. This can be found following implantation of mechanical heart valve [5], physical torture [6], left ventricular assist device use [7] and strenuous exercise [8]. Marching by soldiers is the classic example of strenuous exercise. Nevertheless, other causes have been described in the literature and include playing the drums (bongo [9], conga [10]). Our present case provides a further example with the West-African djembé. This and other percussion instruments must therefore be considered in the differential diagnosis of haemoglobinuria. Although some patients may be more vulnerable to mechanical RBC destruction due to underlying abnormalities, such as spherocytosis, the present case illustrates that destruction can also occur in those with normal RBCs. In such cases, prevention can be achieved by limited and controlled exposure to the source of trauma.

Conflict of interest statement. None declared.

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