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Case Report

Severe peri-renal sepsis in established renal failure masquerading as an acute abdominal catastrophe

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Abstract

Severe worsening lactic acidosis in an elderly patient following an episode of atrial fibrillation, who is not haemodynamically compromised, usually indicates an intra-abdominal vascular catastrophe. We describe a unique case of severe peri-renal sepsis in a patient with long-standing dialysis-dependent chronic kidney disease unrelated to urolithiasis that masqueraded as an acute abdominal condition requiring emergency laparotomy and nephrectomy.

Keywords: acute abdominal catastrophe; chronic kidney disease; peri-renal sepsis

A 73-year-old lady with established renal failure of unknown cause with poor vessels for primary or secondary vascular access was admitted electively for creation of a first-stage brachiobasilic arteriovenous fistula.

In the past, she had transferred to haemodialysis from peritoneal dialysis following an episode of peritonitis which required a laparotomy. Attempts at re-initiating peritoneal dialysis had been unsuccessful, and she was dialysing via a tunnelled jugular central venous catheter. The jugular catheter had been *in situ* for 14 months.

Her co-morbidities included a previous metallic mitral valve replacement (requiring therapeutic anticoagulation), hypertension and haematuria of unknown cause. Her native kidneys had never been biopsied, and she was medically unfit for renal transplantation.

In the preoperative period, she developed diarrhoea and rigours post-dialysis, and she complained of pain around her line. Treatment was instigated for presumed catheter-associated bacteraemia. However, in spite of treatment for 5 days, she remained septic with swinging fevers and one splinter haemorrhage. A new murmur was noted along with a possible vegetation on her mitral valve on echocardiogram, and blood cultures were positive for a fully sensitive *Staphylococcus aureus*. Multiple screens for MRSA were negative.

A clinical diagnosis of infective endocarditis was reached, and an appropriate treatment was administered. Her treatment for endocarditis with antibiotics was continued for the next 4 weeks, and she appeared to respond well.

In the fifth week following this illness, she represented feeling non-specifically unwell for 1 day. Routine observations and blood investigations were normal. However, she rapidly deteriorated with a lactic acidosis. At this point, she complained of mild lower abdominal pain, but clinically, abdomen was felt to be soft with no evidence of peritonitis. Her medical condition continued to deteriorate, and she developed fast, uncontrolled atrial fibrillation. An arterial blood gas showed pH 7.13, pO2 103 mmHg, pCO2 17.3 mmHg and lactate 13.3 mmol/L.

A contrast-enhanced CT angiogram showed patent celiac and superior mesenteric artery axis and patent portal vein and features consistent with chronic pyelonephritis and a possible inflammation in the gallbladder fossa and inflammatory oedema in Morrison's pouch. She was transferred to the intensive care unit where she was further resuscitated, intubated and ventilated. She remained haemodynamically stable, but her metabolic acidosis progressively worsened, and her arterial lactic acid increased to 20 mmol/L.

In the presence of equivocal signs, an emergency laparotomy was performed to rule out an intra-abdominal source of sepsis/infarction. At laparotomy, there was sero-purulent fluid in Morrison's pouch and a macro-nodular liver with normal gallbladder. The stomach, duodenum and pancreas were normal as were the small bowel and colon. There was an extensive inflammatory change around the right kidney, and therefore, a nephrectomy was performed. The patient was transferred back to the intensive care unit and was haemofiltered for 4 h.

Thereafter, she made a speedy recovery, the arterial lactic acid returned to normal and she was discharged to the ward in 48 h.

Pathology revealed chronic pyelonephritis with sclerosis of the glomeruli, atrophy of the tubules, many of which were cystically dilated and containing polymorphs, chronic inflammation, and fibrosis of the parenchyma. There was no evidence of renal stones. The arterioles revealed a hypertensive change. In view of the findings on histopathology and at surgery, a final diagnosis of chronic pyelonephritis

with peri-renal sepsis was made. The liver biopsy showed features consistent with congestive hepatomegaly.

Discussion

Peri-renal sepsis in the setting of chronic renal failure requiring an emergency nephrectomy is rare. There is a single case report of overwhelming MRSA urosepsis in a patient with stag horn calculus who required an emergency nephrectomy [1]. Pyelonephritis accounts for 3–4 hospital admissions per 10 000 females [2], with normal renal function, and the incidence in patients with chronic pyelonephritis is not known.

In patients with chronic kidney disease who are anuric, it is difficult to establish the diagnosis of renal or peri-renal sepsis. Cross-sectional imaging may remain inconclusive early in the illness and at times take 2–3 weeks to become apparent [3]. Worsening abdominal pain with severe lactic acidosis and without peritonitis usually heralds a major intra-abdominal vascular event.

While a similar presentation has been described in the setting of stone disease and MRSA sepsis [1], to our knowledge, this is an index case where severe peri-renal sepsis presented with life-threatening metabolic acidosis in the absence of any obvious predisposing case and possibly secondary to a fully sensitive *S. aureus* infection. Metastatic sepsis from infective endocarditis [4] is a well-recognized complication, and we postulate that despite adequate antibiotic therapy, this may have been the source of sepsis in this case.

The interesting features that this case illustrates are that the absence of common conditions causing an acute abdomen, obscure renal or peri-renal sepsis should be considered as a possible source in anuric patients with chronic kidney disease, and that surgical management with a nephrectomy may prove necessary after failure of conservative management.

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