Contrast-Induced Rhabdomyolysis Occurring after ERCP in a Patient with Pancreatic Cancer: A Case Report

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ABSTRACT

Objective: We present a patient with pancreatic cancer who developed weakness, acute renal failure and significantly raised creatine kinase levels post-ERCP and who was assessed as having contrast-induced rhabdomyolysis.
Results: The patient underwent haemofiltration and ultimately succumbed to his condition.
Conclusion: Rhabdomyolysis is a potentially life-threatening condition which occurs because of damage to skeletal muscle, with release of myoglobin and electrolytes into the circulation. The mortality rate is 59% in severe cases, despite appropriate treatment.

LEARNING POINTS

- Iodine-based contrast can cause rhabdomyolysis by reducing blood flow to the muscle.
- Renal replacement therapy does not improve the mortality rate of rhabdomyolysis.
- <10% of patients present with the classic triad of myalgia, muscle weakness and tea-coloured urine; creatine kinase levels greater than 5 times the upper limit of normal are the gold standard for diagnosing rhabdomyolysis that is not related to statin use.

KEYWORDS

Iodine-based contrast, rhabdomyolysis, ERCP, pancreatic cancer, case report

INTRODUCTION

Rhabdomyolysis is a potentially life-threatening condition that results from the destruction of skeletal muscle and the subsequent release of its toxic intracellular content (myoglobin and electrolytes) into the circulation [1–5]. Serum creatine kinase (CK) levels begin to rise during the first 12 hours of rhabdomyolysis and are the gold standard for laboratory diagnosis [1]. A level greater than 5 times the upper limit of normal (ULN) diagnoses rhabdomyolysis unrelated to statin use [1, 4]. Crush syndrome is a well-known aetiology of the condition, but other established causes include immobilization, exertion, heat stroke, endocrinopathies, drugs/toxins and genetic defects [1–4]. Up to 33% of patients develop rhabdomyolysis-induced acute kidney injury (AKI) secondary to hypovolaemia, metabolic acidosis and the nephrotoxic effects of myoglobin [1, 5]. Other complications include compartment syndrome, disseminated intravascular coagulation (DIC) and electrolyte derangements (hyperphosphataemia, hyperkalaemia, hypocalcaemia) [1, 5]. Aggressive fluid management remains the cornerstone of treatment with renal replacement therapy being reserved for refractory cases [1, 3, 5]. Correction of electrolytes and the use of mannitol and sodium bicarbonate may be implemented to supplement treatment, although the verdict is still out on the latter [1–3, 5]. This case report aims to highlight contrast-induced rhabdomyolysis and the complexities involved in the diagnosis and management of the condition. This is the second case report in the literature documenting iodinated contrast as a cause of rhabdomyolysis.

CASE DESCRIPTION

A 69-year-old male presented with a 5-week history of painless jaundice, pale stools, dark urine, itching, poor appetite and a total weight loss of 12.7 kg. His vital signs were normal, he was independently mobile and his physical examination was unremarkable except for being jaundiced. His initial blood test results were normal except for deranged liver function test results, which were as follows: alanine transaminase (ALT) 228 IU/l (0–50), gamma-glutamyltransferase 256 IU/l (0–55), alkaline phosphatase 246 IU/l (30–130), albumin 26 g/l (35–50), total protein 59 g/l (60–80), bilirubin 566 µmol/l (0–21). Magnetic resonance cholangiopancreatography (MRCP) (*Fig. 1*) and computed tomography of the thorax, abdomen and pelvis (CT TAP) (*Fig. 2*) confirmed a tumour mass to the head of the pancreas with significant intra-/extrahepatic biliary dilatation and pancreatic ductal dilatation. The appearance suggested pancreatic adenocarcinoma with a radiological staging of T4N1M1 with local lymphadenopathy at the porta hepatis and patchy peritoneal disease.
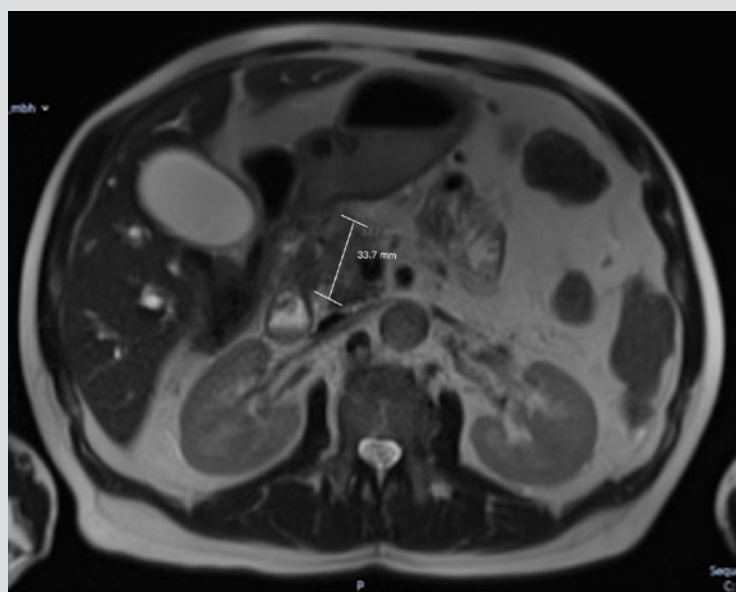

Figure 1. Magnetic resonance cholangiopancreatography demonstrating a mass in the head of the pancreas

Figure 2. Computed tomography of the abdomen demonstrating a mass in the head of the pancreas

The patient underwent urgent endoscopic retrograde cholangiopancreatography (ERCP) with stent placement and sphincterotomy of the common bile duct and pancreatic duct to decompress the biliary tree. The following day post-ERCP, the patient started to deteriorate clinically with generalized weakness, vomiting and anuric AKI. Although the anuria had resolved, his renal function progressively worsened for the remainder of his admission (*Table 1*), despite aggressive intravenous (IV) fluid treatment. Initially, the renal impairment was thought to be secondary to bile cast nephropathy as the patient's bilirubin levels were persistently greater than 500 µmol/l, despite an abdominal ultrasound scan post-ERCP showing decompression of the biliary system with evidence of aerobilia consistent with normal stent function. However, further investigation (*Table 1*) revealed lactic acidosis, significantly raised CK levels, hyperphosphataemia, rising potassium, hypocalcaemia and hyponatraemia. Terlipressin, calcium acetate, sodium bicarbonate and albumin were also incorporated into the patient's management, as per advice from the gastroenterologist and nephrologist, to no avail. Intensive care monitoring for possible haemofiltration also commenced at this time. The clinical impression was contrast-induced rhabdomyolysis post-ERCP with a possibility of superimposed bile cast nephropathy. Failure of conservative management prompted urgent haemofiltration on day 11 of admission, but the patient developed an upper gastrointestinal bleed immediately after the procedure and he passed away a few hours later.

DISCUSSION

This case report highlights the challenges of diagnosing and managing rhabdomyolysis in a complex patient. In the literature, iodine-based contrast is not commonly linked to rhabdomyolysis and only one case report was found highlighting this association[4]. The explanation given in that article was that iodine-based contrast media promoted skeletal muscle injury by reducing blood flow to the muscle [4]. With respect to our patient, there was a clear relationship between the use of iodinated contrast and the onset of rhabdomyolysis, as evidenced by the occurrence of renal impairment 1 day after ERCP (*Fig. 3*).
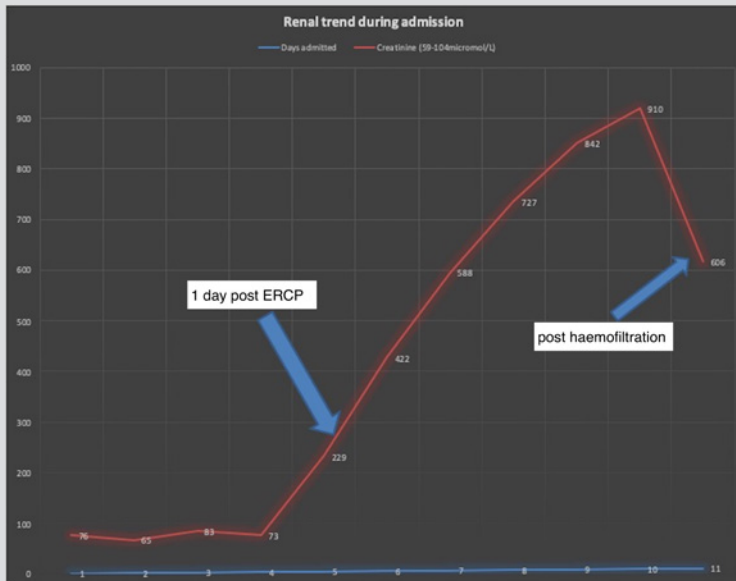
Figure 3. Graph trending the rise in creatinine levels post-endoscopic retrograde cholangiopancreatography

This was further substantiated by the rise of serum CK to 32 times the ULN. Regarding the patient's non-specific symptoms, Torres et al. observed that <10% of patients have the classic triad of myalgia, weakness and tea-coloured urine [5]. Additionally, his dark urine was multifactorial in cause and was most likely a combination of bilirubinuria and myoglobinuria. The results of the urinalysis were not typical for rhabdomyolysis, and so, the presence of myoglobinuria remained inconclusive as urine myoglobin detection is no longer a routine investigation for the confirmation of muscle injury. Furthermore, a study carried out by Alhadi et al. concluded that the presence of haem on a urine dipstick with absent red blood cells on microscopy is an insensitive test for rhabdomyolysis and the absence of this finding does not exclude the condition [6]. The sequelae of metabolic acidosis and electrolyte derangements were also in keeping with a rhabdomyolytic picture. However, the patient failed to respond to aggressive fluid therapy, which is the hallmark of management, and progressed to renal failure. There is no clear evidence in the literature to support the use of mannitol or sodium bicarbonate in the treatment of rhabdomyolysis [1–3,5]. Mannitol was contraindicated as the patient was initially anuric [1] and sodium bicarbonate did not help to resolve the metabolic acidosis. Renal replacement therapy was indicated as the renal function, metabolic acidosis and electrolyte derangements failed to improve with initial therapy. A review article by Chavez et al. noted that, despite the improvements in myoglobin and electrolyte concentrations in patients treated with haemofiltration, the mortality rates of up to 59% with severe forms of rhabdomyolysis remained unchanged [1, 2]. This was the case for our patient who developed an upper gastrointestinal bleed after haemofiltration, which was a likely combination of DIC and hepatic impairment that culminated in an unfavourable outcome.

CONCLUSION

In a patient with non-specific symptoms, an uncommon aetiology and a complex medical history, the diagnosis of rhabdomyolysis may be obscured. In a case such as this, one has to have a high index of suspicion to arrive at the diagnosis. However, even with early diagnosis and treatment, the prognosis may remain poor.

Days admitted	2	4*	5	8	10	11*
Sodium (133–146 mmol/l)	132	130	127	115	117	126
Potassium (3.5–5.3 mmol/l)	3.6	3.5	3.5		5.2	5
Phosphate (0.8–1.5 mmol/l)	0.72		1.57		2.63	1.71
Calcium (2.20–2.60 mmol/l)	2.35		2.17		2.01	2.02
Urea (2.5–7.8 mmol/l)		4.8	12.9	28.8		42.5
Creatinine (59–104 µmol/l)	65	73	229	727	910	606
eGFR (>60 ml/min)	>60	>60	25	7	5	8
Creatine kinase (40–320 IU/l)					10,498	
Lactate dehydrogenase (208–378 IU/l)					1,678	
pH (7.35–7.45)				7.284	7.284	7.331
pO2 (11.1–14.4 kPa)				11.7	13.7	15.8
pCO2 (4.30–6.40 kPa)				3.72	3.29	1.97
Bicarbonate (22–29 mmol/l)				13.2	11.7	7.8
Anion gap				16.1	17.2	18.7
Lactate (0.6–1.4 mmol/l)				1.0	1.6	7.8
ALT (0–50 IU/l)		206		283	295	234
ALP (30–130 IU/l)		226		208	196	151
GGT (0–55 IU/l)		164		137	131	100
Bilirubin (0–21 µmol/l)		586		592	550	453
Albumin (35–50 g/l)		25		27	32	25
INR					1.6	2.1
PTT						1.27
Amylase (22–80 IU/l)			25			
Urine dipstick					3+ blood + protein 3+ bilirubin	
Urine microscopy					3+ RBC + WBC	

Table 1. The relevant results of investigations carried out during admission

eGFR: Estimated glomerular filtration rate; pH: power of hydrogen; pO2-: partial pressure of oxygen; pCO2: partial pressure of carbon dioxide; ALT: alanine transaminase; ALP: alkaline phosphatase; GGT: gamma-glutamyltransferase; INR: international normalized ratio; PTT: partial thromboplastin time; RBC: red blood cells; WBC: white blood cells.

4* – Day of ERCP   11* – Day of haemofiltration

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