Case report: a fatal case of severe pancreatitis associated with rhabdomyolysis

Saw Kian Cheah, Mohamed Izzad bin Isahak, Wan Rahiza Wan Mat, Shereen Suet Ping Tang, Rufinah Teo, Aliza Mohamad Yusof, Raha Abdul Rahman

Abstract

Background: Rhabdomyolysis is an uncommon presentation associated with severe acute pancreatitis (SAP). Patients usually present late and asymptomatic, thus it carries poorer clinical outcome and higher mortality rate.

Case report: We report a case of SAP in a 42-year-old man who presented with persistent epigastric pain, elevated serum amylase, and characteristics of acute pancreatitis on the contrast-enhanced computed tomography of the abdomen. Investigations revealed persistent hyperkalemia and severely elevated creatinine kinase in the presence of acute kidney injury, which led to the uncommon possibility of diagnosing severe rhabdomyolysis. He suffered complications of intra-abdominal hypertension, feeding intolerance, severe ARDS and multi-organ failure. He succumbed to death after a month despite aggressive intensive care therapies and support.

Discussion: The actual pathophysiology of rhabdomyolysis in SAP is complex. It is postulated that acute inflammation of pancreas causes ATP-dependent proton pump dysfunctions leading to skeletal muscle cells injury and therefore, rhabdomyolysis.

Conclusion: Coexistence of rhabdomyolysis with SAP have higher risk for fatality, therefore, its presence allows early triage, early aggressive resuscitation and intensive care management.

Key words: Severe acute pancreatitis, rhabdomyolysis, hyperkalemia, acute kidney injury, ATP-dependent proton pump.
Initially he was admitted to general surgical ward from the emergency department. Despite having full consciousness, he was tachypneic at 40 breaths per minute and tachycardic at 153 beats per minute with high grade temperature of 39.2 °C. However, both his conjunctivae did not show signs of anemia nor icterus. On the further examination, there were reduced breath sounds at both of his lung bases worsened by distended and guarded abdomen. His blood investigation revealed significantly elevated serum amylase level of 1061 IU/l. His Imrie score was 4 after 48 hours of admission (white blood counts 15.7x10^9/l, blood urea nitrogen 21.1 mmol/l, lactate dehydrogenase 1151 U/l, and albumin 23 g/l) indicating high risk of developing SAP. His abdominal contrast-enhanced computed tomography (CECT) on the day of admission showed heterogeneously bulky head of pancreas measuring approximately 5.6 cm x 5.3 cm x 7.4 cm with extensive peripancreatic fluid streakiness. However, there was no evidence of necrosis, calcification or peripancreatic collections. The gallbladder was distended with no calculus and the pancreatic ducts as well as biliary duct were not dilated (Figure 1).

His immediate managements were adequate parenteral analgesics, fluid resuscitations and kept fasted to rest the gastro-intestinal tract. However, he developed hypoxic respiratory failure needing endotracheal intubation and mechanical ventilation. He was then referred to the intensive care team for further management. Following admission to intensive care unit (ICU), he developed unstable haemodynamics requiring inotropics support with concurrent fluid resuscitation. On day 2 of ICU admission, he developed grade 2 intra-abdominal hypertension (IAH) as his abdomen became further distended and was tense. In subsequent days, his kidney functions deteriorated and were associated with persistent hyperkalemia. Despite fluid resuscitations and repeated cycles of the lytic cocktail regimes, his kidney functions declined further requiring renal replacement therapy (RRT). Concurrent investigation showed serum CK level elevated and peaked on day 6 of ICU admission at 105,829 U/l from the baseline of CK level of only 63 U/l on day 1 of ICU admission. With RRT, the serum CK levels gradually normalized and kidney functions improved. However, his abdomen remained distended and noted to have persistent hypocalcemia required replacement. Furthermore, he developed feeding intolerance despite initiation of prokinetics. Abdominal CECT was repeated on day twelve of ICU admission. It showed persistently, enhanced, and an enlarged pancreas but without focal areas of necrosis. However, this repeated abdominal CECT showed extensive peripancreatic fluid collections with thin enhancing wall at lesser sac measuring at 7.6 cm x 7.4 cm x 5 cm, right paracolic gutter measuring at 3.8 cm x 7.3 cm x 5.4 cm, and left anterior pararenal space measuring at 10.7 cm x 5.2 cm x 12 cm (Figure 2). As the extensive peripancreatic fluid collections contributed to the distended abdomen, a transcutaneous pigtail catheter was inserted by the interventional radiologist for decompression and drainage of the collections. Despite the drainage, the patient’s abdomen remained distended with feeding intolerance. In the following days leading up to his death, he developed nosocomial infections, severe acute respiratory distress syndrome (ARDS), and multi-organ failure (MOF).

**Discussion**

Acute pancreatitis is an acute inflammation of the pancreas, characterized by the activation of pancreatic proenzymes with autodigestion, proteolysis, edema, vascular damage, interstitial hemorrhage, and fat necrosis. (1) Acute pancreatitis is a surgical emergency that can lead to local and systemic complications. According to the revised Atlanta classification (2012), SAP was defined as an acute pancreatitis with organ failures (respiratory, cardiovascular, and renal) that persist for more than 48 hours. (3) The overall incidence of SAP was about 15-20% of all cases of acute pancreatitis, yet the reported cases of SAP showed poorer clinical outcome with high mortality for up to 50%. (3,4,5).

Rhabdomyolysis is an uncommon complication seen in acute pancreatitis. The exact pathophysiology role of rhabdomyolysis in acute pancreatitis is rather complex. The possible postulated theory would be SAP induced systemic inflammatory response, persistent hyperthermia, and extensive injury of pancreas causing the excessive adenosine triphosphatase (ATP) utilization and depletion. (6) With ATP depletion, pump functions such as Na/K-ATPase and Ca^{2+}-ATPase will get affected and disrupted. These will subsequent lead to the muscle cell membrane breakdown, increased in the cell membrane permeability and get accumulated in the cytoplasm’s sodium and calcium ion concentration. The excess of the calcium ion intracellularly will further enhance the increase in the intracellular proteolytic enzymes activity causing muscle cell degradation. With the existence of the muscle cell degradation and necrosis, this leads to the severe rhabdomyolysis and large release of electrolytes such as potassium, phosphate, myoglobin,
and CK into the circulation and subsequent causing of renal tubular cell injury, obstruction, ischemia, and AKI. (7) Severe and prolonged hypocalcemia might also occur with the concurrent conditions of SAP with rhabdomyolysis. (2) In SAP, saponification of fat usually deposits in the pancreas, however, concurrent presentation of rhabdomyolysis will worsen the condition as calcium salt will further deposit in the damaged muscle cell as seen in this patient. (2,8) Although the association between rhabdomyolysis with acute pancreatitis is uncommon, when it does happen, patients usually present with acute deterioration accompanied by MOF leading to possible death. In this case report, we reported the patient initially presentation of acute pancreatitis, but rapidly developed AKI with hyperkalemia requiring RRT. Concurrently, these were accompanied by the refractory intra-abdominal hypertension (IAH), feeding intolerance, and multi-organ dysfunctions. We believed that his deterioration was largely associated with the ongoing rhabdomyolysis in SAP.

**Conclusion**
If an acute deterioration developed in an otherwise relatively stable acute pancreatitis patient, especially with early development of AKI and persistent hyperkalemia, acute rhabdomyolysis should be suspected to institute early aggressive resuscitation and intensive care management.

**Acknowledgement**
Associate Professor Dr. Faizah Mohd Zaki, Department of Radiology Universiti Kebangsaan Malaysia Medical Centre for helping in the radiograms selection.
**Figure 1.** Contrast enhanced CT abdomen of the patient at presentation in axial (a) and coronal (b)

Legend: The pancreas was swollen and homogenously enhancing (black star) with associated peripancreatic fluid and increased fat streakiness (white arrows).
Figure 2. Repeated contrast enhanced CT abdomen at day-12 of ICU admission in axial (a) and coronal (b)

Legend: There were multiple new collections with thin enhancing wall at the lesser sac and peripancreatic region, perisplenic, and right paracolic gutter (white stars). There was no dominant necrosis in the pancreas (black arrow).
References