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(CASE REPORT)

Severe acute alcoholic pancreatitis complicated by distributive shock, abdominal compartment syndrome, and black pleural effusion

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Abstract

Severe acute pancreatitis (SAP) is a life-threatening medical emergency that can lead to multi-system organ failure and mortality. Here, we highlight a case of SAP complicated by a black pleural effusion (BPE), distributive shock, and acute abdominal compartment syndrome (ACS) requiring emergent decompressive laparotomy. BPE fluid analysis was consistent with previously reported exudative effusions from a pancreatic pseudocyst, though the computed tomography (CT) of abdomen and pelvis did not show definitive evidence of a pseudocyst. In addition, black fluid was also discovered during decompressive laparotomy for ACS. It is possible that the severely elevated intra-abdominal pressure contributed to a temporary translocation/transudation of this fluid from the abdominal cavity into the pleural space, as no re-accumulation of the BPE was observed on a follow-up CT after thoracostomy tube removal.

Keywords: Severe Acute Pancreatitis; Distributive Shock; Abdominal Compartment Syndrome; Black Pleural Effusion; Acute Respiratory Distress Syndrome; Pleural Fluid Analysis

1. Introduction

Severe acute pancreatitis (SAP) represents a complex medical condition that requires prompt diagnosis and intervention. The expected course of SAP may include inflammation, necrosis, and infection during which, potential distributive shock and progressive multisystem organ failure may develop.[1] This disease process remains challenging to acute care physicians due to the severe metabolic derangements, acute lung injury, and the possibility of an acute surgical abdomen once necrosis occurs. In our report, we present a rare case of SAP in a young, otherwise healthy male, complicated by distributive shock, acute abdominal compartment syndrome (ACS), and the development of a black pleural effusion (BPE). Despite the absence of definitive evidence of a pancreatic pseudocyst on computed tomography (CT) imaging, the patient exhibited clinical and laboratory findings consistent with pancreatic involvement, including significantly elevated serum lipase levels and pleural fluid amylase levels.

2. Case presentation

The patient is a 27-year-old, overweight male with a past medical history of gastritis, alcohol, and tobacco use, who presented to the emergency department with a complaint of abdominal pain attributed to gastritis. His vital signs were within normal limits, and physical exam was notable for a firm, diffusely tender abdomen with hypoactive bowel sounds. His serum lipase was significantly elevated to 3,042 U/L. CT of the abdomen and pelvis demonstrated moderate peripancreatic fat stranding and a small amount of peripancreatic fluid tracking into the abdomen and pelvis consistent with acute pancreatitis (Figure 1A). His initial Ranson's score was 1; therefore, he was admitted to medical floor. On hospital day three, the patient developed a sudden onset of hypoxia, hypotension, and tachycardia, requiring intensive

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care unit (ICU) admission. He was emergently intubated for acute respiratory distress syndrome (ARDS) and, initiated on renal replacement therapy for acute, anuric renal failure. He was resuscitated with intravenous crystalloids and supported with multiple vasopressors and steroids and treated with broad-spectrum antibiotics. His blood, tracheal, and urine cultures, MRSA, influenza, and COVID screening were all negative. His hepatitis panel, HIV, and fungal cultures were also negative. His Ranson's score-was 6 at 48-hour into his hospitalization. This score is associated with a possible 40% mortality.

On ICU day one, his CT of the chest showed bilateral pleural effusions, left greater than right (Figure 2A). Left thoracostomy tube was placed that returned BPE within the collecting chamber (Figure 2B). Initial pleural fluid analysis showed a markedly elevated amylase in the fluid (Table 1). On ICU day three, the patient developed ACS with a persistent bladder pressure greater than 25mmHg despite gastric tube decompression and paralysis. Repeat CT of abdomen and pelvis showed worsening SAP (Figure 1B). He was emergently taken to the operating room (OR) for decompressive laparotomy, during which 200ml of black fluid was obtained during fascial opening. Final peritoneal and pleural fluid cultures showed moderate polymorphonuclear leukocytes without organisms. On ICU day six, our patient went to the OR for abdominal closure and was extubated the following day without incident.

His thoracostomy tube was subsequently removed when the chest tube drainage was minimal, and there was no recurrent pleural effusion on CT of the chest at 48 hours (Figure 2C).

3. Discussion

To our knowledge, this is the first case report in an SAP patient, in whom ACS may have been complicated with BPE development. The established causes of BPE can include infection, malignancy, hemorrhage, and pancreatic pseudocyst rupture.[2] In the latter case, BPE can enter the pleural cavity via the esophageal hiatus or by a transdiaphragmatic route through a pancreaticopleural fistula or by translocation. The black color of the fluid is thought to be due to hemosiderin deposition as a result of breakdown of hemoglobin by pancreatic enzymes.[3] The diagnosis of a probable pseudocyst rupture is made by a high index of suspicion and by pleural fluid analysis with elevated amylase level confirming pancreatic origin for the effusion.[4,5] Due to his ARDS and hemodynamic instability, our patient was not able to undergo MRCP, which appears to have the highest sensitivity in pseudocyst diagnosis.[3] However, based on pleural fluid analysis (Table 1) and operative findings, were able to conclude that his elevated intra-abdominal compartment pressure appeared to facilitate in the translocation of the black abdominal fluid into his left pleural cavity. Our patient's hemodynamic parameters, ARDS, renal function, and respiratory failure slowly improved in the days following his decompressive laparotomy for ACS. His pleural effusion did not re-accumulate on chest CT. This further supported our theory that the abdominal fluid may have migrated from the intra-abdominal to the pleural cavity due to the pressure that existed within the abdomen. Relieving pressure within the abdominal cavity was paramount and life-saving. Pleural drainage appeared to further assist in his ventilatory status. Diagnostic and therapeutic fluid analysis was also helpful in guiding subsequent therapeutic decisions.

Table 1 Pleural fluid analysis shows black pleural fluid with exudative pattern and markedly elevated amylase level.

Fluid Volume	**60ml**
Fluid Color	Black
Fluid pH	9
Fluid WBC	7,267 uL
Fluid RBC	<2,000 uL
Fluid Granulocytes	70%
Fluid Lymphocytes	2%
Fluid Monocytes	28%
Fluid Glucose	171 mg/dL
Fluid Cholesterol	<50 mg/dL
Fluid Total Protein	2.9 g/dL
Fluid LDH	2791
Fluid Amylase	>1302

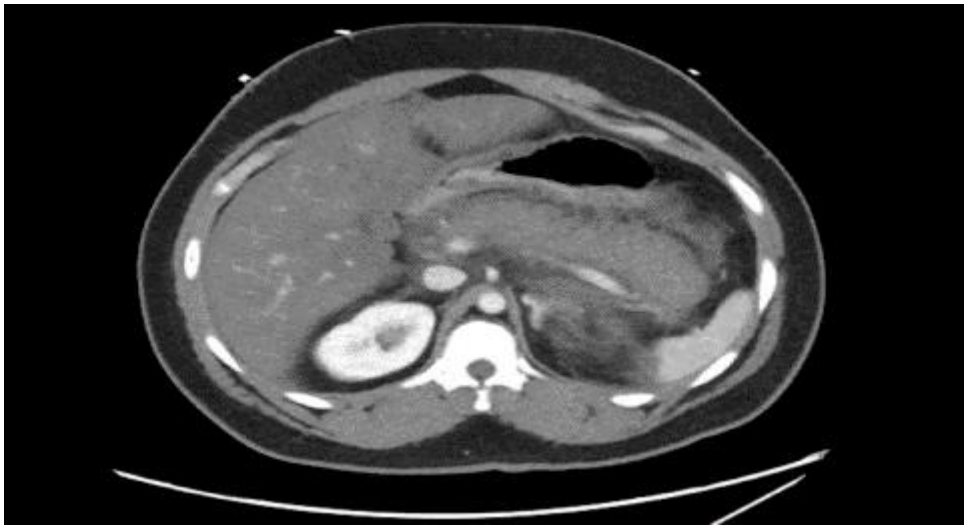

Figure 1A. CT of abdomen and pelvis on admission demonstrated extensive hypoattenuation involving most of the pancreas representing acute pancreatitis.

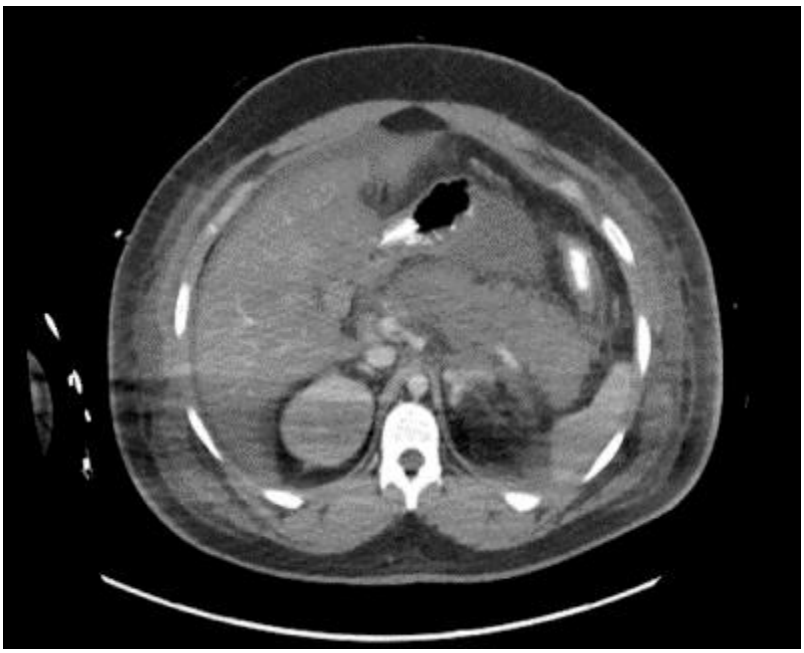

Figure 1B CT of abdomen and pelvis on ICU day 3 showed decreased enhancement of the entire pancreas with possible necrosis.

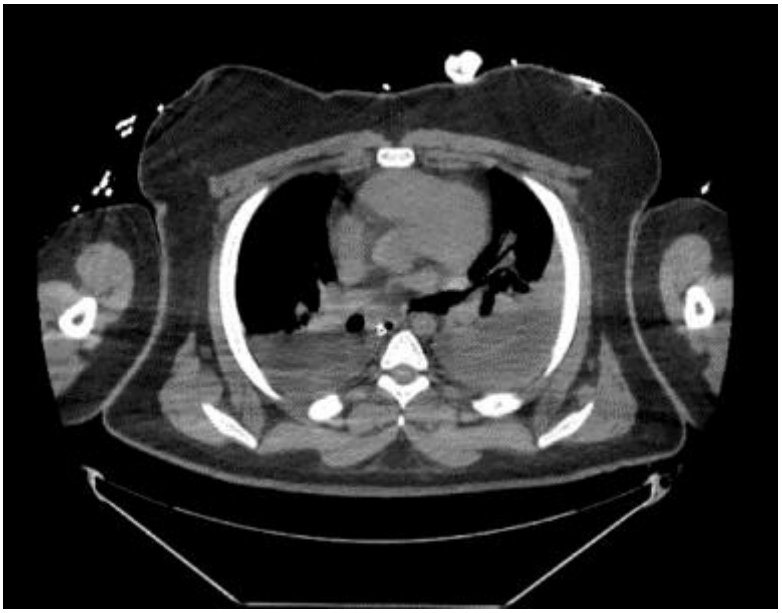

Figure 2A CT of chest on ICU day 1 demonstrated posteriorly layering bilateral pleural effusions (left greater than right) with adjacent atelectasis of bilateral lower lobes.

Figure 2B Collecting atrium was filled with exudative black pleural effusion.

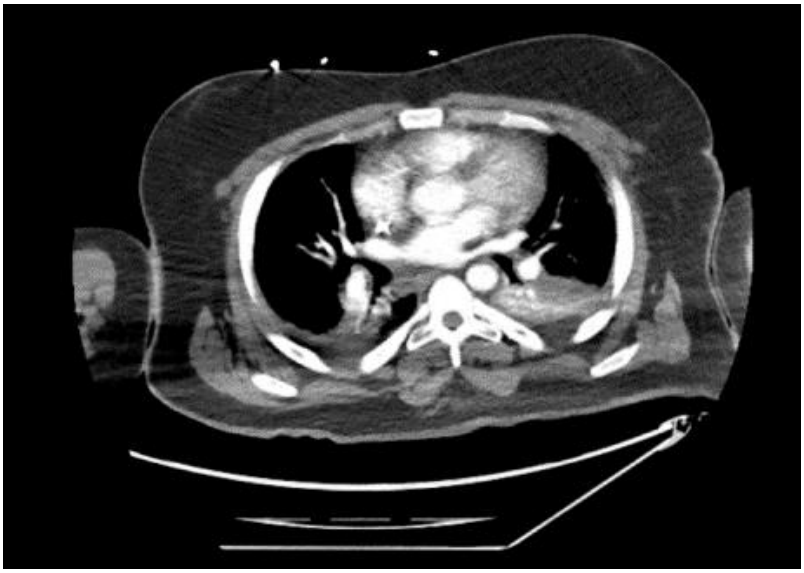

Figure 2C CT chest two days following removal of left-sided thoracostomy tube showed no re-accumulation of left pleural effusion

4. Conclusion

In conclusion, SAP has many potential life-threatening complications that require prompt recognition and treatment. When a large quantity of pleural effusion is present, intra-thoracic drainage may assist in diagnosis and therapeutically improve respiratory status. Once ACS develops, decompressive laparotomy can be life-saving as well as decreasing fluid translocation from the abdomen into the pleural space.

Compliance with ethical standards

Acknowledgments

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Disclosure of conflict of interest

The above listed authors, Drs. Nikiforov, Asllanaj, and McWhorter have no conflicts of interest to declare.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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