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ACUTE HEMORRHAGIC PANCREATITIS PRESENTING WITH HEMODYNAMIC INSTABILITY – A CASE REPORT

General Medicine

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ABSTRACT

Acute hemorrhagic pancreatitis is a severe and life-threatening variant of acute pancreatitis characterized by pancreatic necrosis and vascular injury. We report a 45-year-old male chronic alcoholic presenting with acute epigastric pain and hemodynamic instability. Laboratory investigations revealed anemia, leukocytosis, elevated inflammatory markers, and pancreatic enzyme elevation. CECT abdomen confirmed hemorrhagic pancreatitis with necrosis. Severity assessment showed Ranson score 4, BISAP score 3, and CTSI score 8. The patient improved with intensive conservative management.

KEYWORDS

Acute Hemorrhagic Pancreatitis, Pancreatic Ascites, Hemorrhagic Ascites, CT Severity Index, Case Report

INTRODUCTION

Acute pancreatitis is an inflammatory disorder of the pancreas with a wide clinical spectrum, ranging from mild interstitial pancreatitis to severe necrotizing and hemorrhagic forms. The global incidence of acute pancreatitis has been increasing, with alcohol abuse and gallstone disease accounting for the majority of cases. While most patients experience a mild and self-limiting illness, approximately 15–20% develop severe acute pancreatitis, which is associated with significant morbidity and mortality.

Acute hemorrhagic pancreatitis represents a severe and life-threatening variant characterized by pancreatic parenchymal necrosis and enzymatic destruction of pancreatic and peripancreatic blood vessels. This vascular injury results in intrapancreatic and retroperitoneal hemorrhage, leading to hypovolemia, shock, and multiorgan dysfunction. The pathophysiology involves premature activation of pancreatic enzymes, particularly trypsin and elastase, causing autodigestion of pancreatic tissue and surrounding vasculature.

Alcohol-induced pancreatitis is associated with increased pancreatic enzyme secretion, ductal obstruction due to protein plug formation, and direct toxic effects on acinar cells. These mechanisms predispose patients to severe disease and complications such as necrosis, hemorrhage, pancreatic ascites, and systemic inflammatory response syndrome. Hemorrhagic ascites with markedly elevated ascitic fluid amylase and lipase levels strongly suggests pancreatic origin and indicates severe disease.

Early diagnosis, severity stratification using validated scoring systems such as Ranson, BISAP, and CT Severity Index (CTSI), and prompt intensive care management are crucial to improve outcomes. We report a case of acute hemorrhagic pancreatitis presenting with hemodynamic instability and hemorrhagic pancreatic ascites, highlighting the importance of early recognition, comprehensive evaluation, and aggressive supportive management.

Case Presentation

A 45-year-old male with a history of chronic alcohol consumption presented with severe epigastric pain radiating to the back, vomiting, and abdominal distension. On admission he was hypotensive and tachycardic. Abdominal examination showed marked epigastric tenderness with guarding.

Hematological parameters showed hemoglobin 7.5–8.0 g/dL, total leukocyte count 18,500–19,200/mm^3, neutrophils 85–88%, lymphocytes 8–10%, platelet count 1.6–1.8 $\times 10^5$/mm^3, and MCV 86 fL.

Renal and electrolyte parameters were serum urea 68 mg/dL, creatinine 1.8 mg/dL, sodium 130 mEq/L, potassium 3.2 mEq/L, chloride 96 mEq/L, and calcium 7.6 mg/dL.

Liver function tests showed total bilirubin 0.9 mg/dL, direct bilirubin 0.3 mg/dL, AST 46 IU/L, ALT 42 IU/L, ALP 118 IU/L, serum albumin 3.1 g/dL, and total protein 6.2 g/dL. CRP was >100 mg/L.

Serum amylase was 362 U/L and serum lipase was 302 U/L. Tumor markers were AFP 3.5 ng/mL, CEA 0.7 ng/mL, and CA 19-9 6.9 U/mL.

Ascitic fluid was hemorrhagic in color with total cell count 980 cells/mm^3, neutrophils 82%, lymphocytes 18%, protein 3.8 g/dL, ascitic fluid amylase >2000 U/L and lipase >2000 U/L.

CECT abdomen showed pancreatic necrosis with hemorrhagic peripancreatic collections confirming acute hemorrhagic pancreatitis. Ranson score was 4, BISAP score 3, and CTSI score 8.

DISCUSSION

Acute hemorrhagic pancreatitis is a severe form of acute pancreatitis associated with high morbidity and mortality. The disease results from premature activation of pancreatic enzymes within the pancreas, leading to autodigestion of pancreatic tissue and enzymatic destruction of adjacent blood vessels. Elastase-mediated vascular injury plays a central role in the development of hemorrhage, contributing to hypovolemia, shock, and systemic inflammatory response.

Alcohol is a major etiological factor in severe acute pancreatitis and contributes to disease progression through multiple mechanisms, including increased enzyme secretion, oxidative stress, and direct acinar cell injury. These factors predispose patients to pancreatic necrosis and hemorrhagic complications. In the present case, the patient's history of chronic alcohol consumption, combined with imaging findings and severity scores, supports alcohol-induced severe acute pancreatitis.

The presence of ascites in acute pancreatitis indicates severe disease. Hemorrhagic pancreatic ascites is characterized by dark blood-stained fluid with markedly elevated amylase and lipase levels, often exceeding serum values. In this case, ascitic fluid amylase and lipase levels exceeding 2000 U/L strongly supported pancreatic origin and severe pancreatic ductal disruption or necrosis. High protein content and neutrophil predominance further indicate intense inflammatory activity.

Severity assessment plays a critical role in guiding management and predicting outcomes. A Ranson score of 4, BISAP score of 3, and CTSI score of 8 in our patient indicated severe acute pancreatitis with a high risk of complications. Contrast-enhanced computed tomography

remains the imaging modality of choice for assessing pancreatic necrosis, hemorrhage, and peripancreatic collections.

Management of acute hemorrhagic pancreatitis is primarily supportive and includes aggressive intravenous fluid resuscitation, hemodynamic stabilization, pain control, nutritional support, and close monitoring for organ dysfunction. Surgical or interventional management is reserved for specific complications such as infected necrosis or uncontrolled hemorrhage. Early intensive care management in the present case resulted in gradual clinical and biochemical improvement.

CONCLUSION

Acute hemorrhagic pancreatitis is a medical emergency. Early diagnosis, severity stratification, and aggressive supportive management are essential to reduce morbidity and mortality.

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