Abdominal Bleeding Complicating Necrotizing Pancreatitis and Successfully Treated by Intravascular Embolization

Rachelle Abboud¹
Fady Nader²
Charbel Nahkoul¹
Chahid Farah¹
Lourdes Khalifeh³
Boutros Youssef³
Tony El Murr⁴

¹Department of Internal Medicine diseases, Faculty of Medical Sciences-LU, Beirut, Lebanon
²Department of Surgery, Faculty of Medical Sciences-LU, Beirut, Lebanon
³Division of Internal Medicine Diseases, Head of Medicine Department at MEIH-UH affiliated with the Faculty of Medical Sciences-LU, Bsalim, Lebanon

Abstract
Severe intra-abdominal bleeding is a relatively rare complication of acute necrotizing pancreatitis that urges adequate management to save the patient's life. It results mainly from direct vascular injury leading to pseudo aneurysm formation and/or arterial rupture within the pancreas vasculature. Other types of vascular complications in acute pancreatitis may result from stress-induced peptic ulcer diseases or gastro-esophageal varices that bleed into the gastro-intestinal tract. In this article, we report the case of a 53-year-old male patient with acute necrotizing pancreatitis complicated by severe life-threatening intra-abdominal bleeding pseudo aneurysm and successfully treated by intravascular embolization. This report, with the following literature review of similar cases, is to highlight the importance and effectiveness of a prompt interventional therapy in treating severe vascular complications in necrotizing pancreatitis.

Key words
Pancreatitis; Pseudoaneurysm; Abdominal Bleeding; Intravascular Embolization

Case Report
A 53-year-old male obese (BMI = 39 kg/m²) patient presented to our university hospital for severe unbearable epigastric pain associated with vomiting and abdominal distension. In his previous medical history, we noted recurrent alcoholic pancreatitis since three years, moderate hepatic steatosis, essential hypertension, hypercholesterolemia, and severe alcohol consumption (5 liters of whisky 40% alcohol by volume (ABV) per day, which means 40%*5000ml/1000 = 200 units per day) since more than 10 years. He is non-smoker and has no known allergies. The only medication he is taking was a beta blocker and a statin.

On admission, his blood pressure (BP) was 140/85 mmHg, body temperature of 38°C, respiratory rate 22, heart rate 95 bpm, and oxygen saturation 94% on room air. His physical examination revealed a well-nourished, normally-colored skin and conjunctiva, conscious diaphoretic patient in moderate distress. He has diffuse abdominal distension and tenderness and normal cardio-pulmonary auscultation. He has no lower limb edema and no palpable peripheral lymph nodes. Blood tests showed serum amylase of 650 IU/L, lipase 860 IU/L, high peripheral white blood cells (WBC = 12.5 G/liter; 70% neutrophils and 25% lymphocytes), hemoglobin of 16.6 g/dL, platelet count of 362000/µL, and serum creatinine of 1.39 mg/dL. Axial contrast-enhanced CT image showed an ill-defined hypo attenuating region in the head and body of the pancreas, along with ill-defined heterogeneous peri pancreatic fluid, and increased fat attenuation (Figure IA, IB and IC). His physical examination revealed a well-nourished, normally-colored skin and conjunctiva, conscious but diaphoretic patient in moderate distress. He has diffuse abdominal distension and tenderness and normal cardio-pulmonary auscultation. He has no lower limb edema and no palpable peripheral lymph nodes. Blood tests showed serum amylase of 650 IU/L, lipase 860 IU/L, high peripheral white blood cells (WBC = 12.5 G/liter; 70% neutrophils and 25% lymphocytes), hemoglobin of 16.6 g/dL, platelet count of 362000/µL, and serum creatinine of 1.39 mg/dL. Axial contrast-enhanced CT image showed an ill-defined hypo attenuating region in the head and body of the pancreas, along with ill-defined heterogeneous peri pancreatic fluid, and increased fat attenuation (Figure IA, IB and IC). He was diagnosed as having acute necrotizing pancreatitis (Balthazar E) and started on IV hydration, antiemetic, proton pump inhibitor, and pain killers.

After one week at hospital, he started having progressive icterus with increase in cholestatic liver enzymes levels, caused by common bile duct (CBD) compression from severe edema in the head of pancreas as was shown clearly on Magnetic Resonance Cholangiopancreatography (MRCP) (Figures IIA, IIB, and IIC). The next day at midnight, the patient had developed suddenly a hypotension associated to very severe abdominal pain refractory even to opioid; blood tests revealed significant drop in hemoglobin (from 14.6 to 8 g/dL) and additional increase in liver enzymes levels. The patient was transferred to intensive care unit for stabilization and four units (2000ml) of whole blood was given within 24 hours. Abdominal angioscan revealed rupture of pancreatico-duodenal pseudo

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aneurysm with huge intraperitoneal hemattoma. Urgent angiography confirmed the rupture and served to intravascular embolization through the pancreatico-duodenal artery. This was followed by biliary and hematoma drainage (Figures IIA, IIB, and IIC). Three days later, and while the hemoglobin level was still stable, the patient started to have progressive left leg edema, erythematic and calf pain associated to moderate hypoxemia. Ultrasound Doppler of lower extremities showed Deep Vein Thrombosis (DVT) of the left common femoral vein but thoracic angioscan did not reveal pulmonary embolism. Knowing the high risk of re-bleeding in this patient, an Inferior Vena Cava (IVC) filter was inserted to avoid anticoagulation therapy on the short run and prevent further complications. One week later, the patient became clinically better; his hemoglobin remained stable and the inflammatory markers as well as the liver function tests decreased significantly. The abdominal drains were removed and the patient was discharged home to start the low molecular weight heparin for the DVT with close follow up at home.

Discussion

Acute pancreatitis can be mild, moderately severe or severe according to the absence, transient < 48h or persistent > 48h organ failure. It is one of the most gastrointestinal (GI) causes of hospitalization in the United States [1].

Vascular complications of pancreatitis occur in up to 14% of all pancreatitis cases and are more common in chronic than acute pancreatitis. This complication can result in life-threatening bleeding, from direct vascular injury or indirect causes. Direct vascular injury is infrequent but the most feared one and is caused by local inflammatory insult and enzymes activation, leading to erosion of the elastic vessel wall causing vessel rupture or pseudo aneurysm formation with subsequent risk of rupture [2,3]. This vessel rupture may occur either into a pseudo cyst, pancreatic duct, peritoneal cavity or the GI tract. The indirect causes of bleeding are more prevalent and include stress gastritis, peptic ulcer disease, gastro esophageal varices and Mallory weiss syndrome. Patient with pancreatitis and intra-abdominal hemorrhage presents usually with abdominal pain, drop in hemoglobin, blood into external drain or hemorrhagic shock [2,4]. In front of these findings, immediate diagnosis should be made by noninvasive abdominal imaging such as conventional CT scan and/or angioscan or vascular mapping using invasive angiography if available. Vascular complications develop mainly in severe and necrotizing pancreatitis especially in the presence of sepsis, multiorgan failure, fluid collections like abscess, pseudo cyst and walled of necrosis and in case of previous pancreatic surgery such as necrosectomy or Whipple procedure [5,6]. The most common involved vessel is the splenic artery followed by gastro duodenal and pancreaticoduodenal artery [24,6]. Mortality in pancreatitis
with bleeding is three times than without bleeding [6]. Treatment of pancreatitis-associated vascular complications can be surgical or endovascular. Surgery consists either of bleeding vessel ligation or pancreatectomy. It is mandatory in unstable patients or when angiography failed or is unavailable. Instead, interventional radiology when possible is becoming widely preferable; it encounters less invasiveness, higher success rate and lower mortality in such critically ill patients. It is based either on direct stenting through endovascular approach or embolization using coil, N-butyl cyanoacrylate or gel foam [2,3,4,6]. In his retrospective analysis about thirty seven patients that underwent endovascular intervention to treat pancreatitis-related hemorrhage, Kim et al. revealed clearly a success rate of 92% and most of the complications were due to pseudoaneurysms (78%) and treated mainly by transcatheter embolization (95%) [3]. In his retrospective study of thirty-seven patients that underwent endovascular intervention to treat pancreatitis-related hemorrhage, Kim et al. revealed clearly a success rate of 92% and most of the complications were due to pseudoaneurysms (78%) and treated mainly by transcatheter embolization (95%) [3]. Recurrent bleeding after coil embolization is relatively low and was estimated to be around 14% in the retrospective study of Philip et al. [5].

In our case, the pancreatitis was severe, necrotizing and with pancreatic collections, complicated by formation of a pseudoaneurysm in the pancreaticoduodenal artery bed that subsequently ruptured into the peritoneal cavity. Hemostasis was successfully achieved by embolization using a coil through interventional radiology.

Conclusion

In pancreatitis, major vascular complications remains rare but should be diagnosed early and treated as an emergency by a multidisciplinary approach due to their potentially lethal consequences. Interventional radiology using stenting or embolization is still the gold standard in the diagnosis and can be used as first line therapy because it is effective, safe, and minimally-invasive especially in those with multiple co morbidities.

References