showing a wide range in the CEA values (from 0.2 to 4400). Cytokines were quantified, and those demonstrating statistical significance between the groups are presented in Figure 1. The cytokines for NM samples were IL-1α and IL-5, and M samples were IL-10 and GM-CSF.

Interleukin-1α has been reported to have antitumor activity where IL-1α-expressing tumor cells fail to initiate tumor development or block progression in mice.[5] Interleukin-1α directly activates the antitumor activity of CD8+ T cells.[5] Consistent with these observations, our results showed the significantly higher secretion of IL-1α in NM cysts when compared with the M group. The accumulation of inflammatory stimuli, including IL-1α, can be correlated with the benign formation of a cyst but also with stalled cancer progression.[6] This cytokine facilitates interactions between malignant and immune effector cells that bear IL-1Rs promoting apoptosis of malignant cells. Furthermore, our results showed a significantly higher concentration of IL-5 in the NM group that also could be correlated to antitumor activity. Eosinophils, the main cells involved in IL-5 production, can serve as accessory cells for the attraction of tumor-specific CD8+ T cells and macrophages.[6] Interleukin-5 promotes an immunosuppressive environment by inducing apoptosis and tumor hypoxia.[2]

In addition, we also detected 2 cytokines that were secreted in significantly higher levels in the M group. Our results suggest that IL-10 and GM-CSF could assist in diagnosing or monitoring malignant transformation of mucinous cysts because their concentration was significantly higher than in the NM group. These results align with previous studies on IL-10 in several types of cancers because it polarizes T-cell responses toward the Th2 phenotype while suppressing antigen-presentation and Th1 response.[7] Furthermore, GM-CSF is produced by T cells and has been used to predict survival in patients with resectable pancreatic adenocarcinoma, as high plasma levels of this cytokine were significantly associated with shorter patients' survival after surgery.[8]

In conclusion, this study demonstrated that selective cytokines measured in pancreatic cyst fluid appear to be able to differentiate mucinous from nonmucinous cysts and may possibly be predictive of the malignant potential in mucinous pancreatic cysts. The measurement of these cytokines could represent a significant advantage over current protocols for assessment of pancreatic cysts, thus preventing unnecessary resections. Our pilot study shows a significant correlation between the cytokines identified for each cyst group, but a larger prospective trial will be needed to determine sensitivity/specificity of these markers both alone and in combination with current markers.

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OPEN

Minimally Invasive Treatment for Severe Acute Pancreatitis With Superior Mesenteric Vein and Common Bile Duct Stenosis
A Case Report and Review of the Literature

To the Editor:

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas and the annual incidence ranges from 13 to 45 per 100,000 population.[1] Complications of AP include pancreatic necrosis, fluid collection, organ failure, and so on.[2] Portal hypertension, a complication of pancreatitis, gives rise to esophagogastric varices which may lead to fatal upper gastrointestinal hemorrhage. It is commonly reported in chronic pancreatitis cases where it is mainly caused by splenic vein thrombosis or occlusion.[3,4] However, in AP cases, portal

hypertension resulted from superior mesenteric vein (SMV) stenosis is rarely concerned and there has been no report about the systemic treatment. Here, we address a case of SMV stenosis in a patient of AP with total minimally invasive treatment, including laparoscopic surgery and stent implantation.

CASE REPORT

A 62-year-old male presented with sudden onset of upper abdominal pain after high-fat diet, associated with nausea and vomiting. Physical examination was significant for epigastric tenderness. Laboratory tests showed serum calcium of 1.06 mmol/L (reference range, 2.1–2.5 mmol/L) and an elevated amylase of 2612 U/L (reference range, <100 U/L). Abdominal enhanced computed tomography (CT) revealed swollen pancreas with extensive effusion around epigastrium (Fig. 1A). The patient was accompanied with persistent respiratory failure (P_aO_2/FiO_2 was 185) and sequential organ failure assessment score was 4. The diagnosis was severe AP (SAP) with multiple organ failure.

Liquid resuscitation, tracheal intubation, mechanical ventilation, and other organ function supportive treatment were performed. The organ function was gradually stable, but infectious symptom, such as fever, was present. Pancreatic necrosis with infection was considered. After the percutaneous catheter drainage for abdominal abscess, surgery was still considered to control the infection. Meanwhile, CT indicated intrahepatic bile duct expanded mildly with lower part of common bile duct (CBD) stenosis and portal vein (PV) compression. Serum total bilirubin was 156.9 μmol/L (reference range, 0–20.5 μmol/L). Percutaneous biliary drainage was performed because of obstructive jaundice. We performed a laparoscopy-assisted debridement of peripancreatic necrosis and indwelled two irrigation tubes for drainage. Infection was quickly controlled, and the patient resumed oral diet. However, bile drainage and ascites volume were large. Postoperative vascular ultrasonography indicated the left PV sagittal diameter of 0.56 cm. Postoperative CT indicated ascites increased (Fig. 1B). The coronal reconstruction of CT showed SMV stenosis (Fig. 1C). Cholangiography showed the upper segment of CBD was dilated, and the middle and lower segments were not developed (Fig. 1D). Biliary plastic stent and PV stent implantation were then executed. Intraoperative PV angiography indicated a stenosis in the SMV with collateral circulation (Fig. 1E). The PV stent and biliary plastic stent were implanted separately. Postoperative PV angiography showed smooth blood flow and cholangiography showed biliary patency (Figs. 1F-G). The patient recovered smoothly and was discharged in good condition. Computed tomography after 3 months of discharge showed a little necrosis around the pancreas without obstruction of the section from SMV to PV stent and without expansion of CBD. Low molecular weight heparin was used for anticoagulation inhouse, and

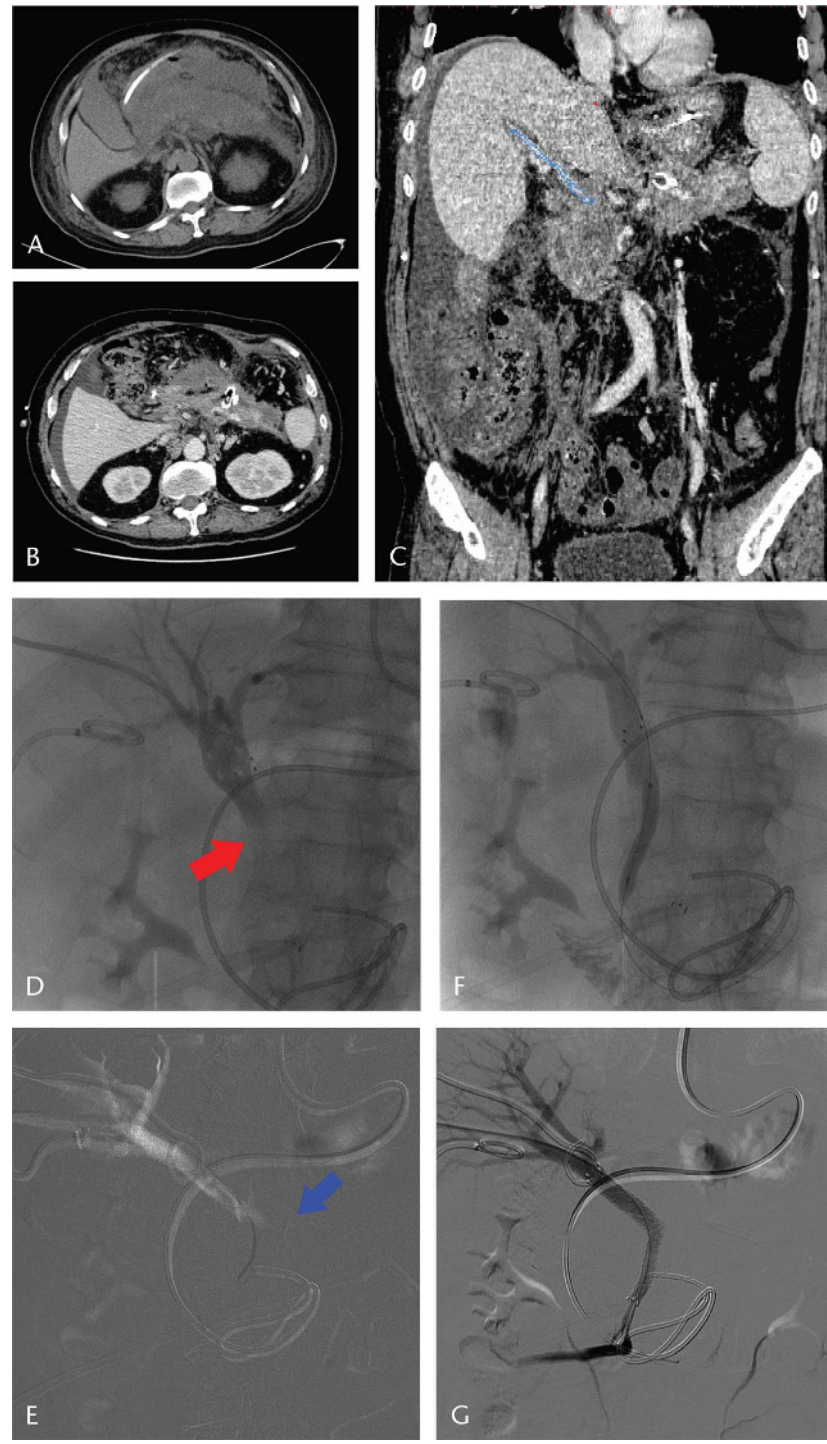

FIGURE 1. A, abdominal CT at admission. B, CT after laparoscopy-assisted debridement. C, PV trunk (blue line) and SMV stenosis in coronal reconstruction of the postoperative CT. D, Expansion and obstruction of upper segment of CBD (red arrow) in cholangiography before implantation. E, SMV stenosis (blue arrow) before implantation in angiography. F, CBD patency after implantation. G, SMV patency after implantation.

warfarin was continued for 6 months as outpatient.

DISCUSSION

There are devastating complications of AP, including pancreatic necrosis, portal hypertension, organ failure, leading to severe prognosis, even death. Extrahepatic portal venous system (PV, splenic vein, and SMV) stenosis, including thrombosis, embolism and occlusion, is more noticeable in chronic pancreatitis and rarely mentioned in AP. Only a retrospective study focusing on CT scans found the prevalence of SMV thrombosis was 14% in 100 AP patients and risk factors included formation of pseudocysts, alcoholic pancreatitis, necrotizing pancreatitis.[2] Also, another study demonstrated that 13% of 832 patients with extrahepatic portal venous system thrombosis had pancreatitis (including acute and chronic pancreatitis).[5] Moreover, there has been no standard program of prevention and treatment for AP with SMV stenosis.

In AP cases, causes of SMV stenosis include local inflammation, stasis, spasm, and mass effect from surrounding necrotic pancreas as well as direct damage of the venous wall by liberated enzymes.[4,6,7] It is more prone to happen in SAP, especially SAP with systemic inflammatory response syndrome, because of more severe inflammation, damage to the vascular endothelium, and hypovolemia.

Mortality associated with acute SMV thrombosis is extremely high, at 20% to 50%.[8] Incidence of SMV stenosis is positively correlated with the severity of pancreatitis.[2,3] Therefore, it is necessary to pay attention to SMV stenosis in AP, especially regarding prevention and clinical treatment. On one hand, anticoagulation is considered to be the first choice. However, Gonzelez et al[9] observed that almost one third of patients had recanalization, regardless of whether they received systemic anticoagulation. The patient's acquired or inherited prothrombotic states is not related to the occurrence of extrahepatic portal venous system thrombosis.[2] Whether early anticoagulation can prevent SMV stenosis in AP remains to be studied. On the other hand, debridement and drainage to remove pancreatic necrotic tissue can prevent further inflammation progression and stent implantation can help recanalization of SMV. However, there is no study on the systemic treatment about SMV stenosis in AP. In our case, we used total minimally invasive therapy, including laparoscopic-assisted debridement of pancreatic necrotic tissue and implantation of endovascular stent in SMV stenosis. The prognosis of the patient is currently good. Limited by the number of cases, systemic treatment of AP-associated SMV stenosis remains to be further studied.

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Acute Pancreatitis Associated With Myotonic Dystrophy Type I

To the Editor:

Recurrent acute pancreatitis (RAP) is clinically defined as 2 or more episodes of established acute pancreatitis, separated by at least 3 months.[1,2] Even if gallstones and alcohol are common causes of RAP, and patients with alcohol abuse and RAP are susceptible to developing chronic pancreatitis, nonalcoholic nonbiliary RAP is a difficult cluster of patients because they require a more extensive workup in order to establish the etiology.[3] Thus, we believe it would be of interest to report the case of patients with RAP associated with Steinert disease. A 47-year-old man, neither an alcohol drinker nor a smoker, with a normal body mass index was admitted to our unit in January 2012 with persistent abdominal pain associated with nausea and vomiting. He had had a cholecystectomy for symptomatic gallstones in 2002 and a right hemicolectomy for bowel obstruction due to Meckel diverticulum in 2010. Five years before admission, he had had a diagnosis of myotonic dystrophy type I, also known as Steinert disease, associated with an expansion of 220 to 600 copies of a CTG triplet repeat in the 3′ noncoding region of *DMPK*, the gene encoding the DM protein kinase, on chromosome 19.[4] He had also experienced 2 bouts of acute pancreatitis of unknown origin and of mild clinical course, one on September 2010 and the other one on July 2011. In January 2012, he was admitted in our department for another attack of acute pancreatitis. At admission, blood tests were carried out. He had normal hematocrit, C-reactive protein, and renal function and a slight elevation of transaminases (aspartate aminotransferase 47 IU/L [reference