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Clinical characteristics and management of gastric outlet obstruction in acute pancreatitis

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ABSTRACT

Objective: The aim of this study was to describe the clinical characteristics and management of gastric outlet obstruction following acute pancreatitis(AP).
Background: Gastric outlet obstruction (GOO) is not uncommon in acute pancreatitis (AP) and can occur throughout the course. However, the clinical features and related treatment of GOO is rarely reported.
Methods: A retrospective review of AP patients with a diagnosis of GOO from March 2017 to June 2020 was performed. The diagnosis and management of GOO, as well as the demographic characteristics and clinical outcomes of the study patients, were collected and analyzed.
Results: Over the three years, there were 60 AP patients developed GOO, constituting an incidence of 5.7%. Thirty-three patients (55.0%, 33/60) developed GOO in the first 4 weeks and 27 patients (45.0%, 27/60) after 4 weeks from onset. Pancreatic necrosis compression (60.6%; 20/33), gastric outlet gastrointestinal edema (27.3%, 9/33) are the main causes of early-onset GOO (≤4 weeks), while wall-off necrosis (92.6%, 25/27) is the leading cause in the late phase (>4 weeks). The management of GOO incorporates both supportive and specific treatment like gastric decompression, gastric juice reinfusion, percutaneous catheter drainage, etc. The mortality of AP patients with GOO (≤4 weeks) was 21.2% and none patients who developed GOO (>4 weeks) died.
Conclusions: GOO, as a gastrointestinal complication developed in AP patients, has two peak incidences in the duration of AP and needs to be paid more attention to.

Introduction

Acute pancreatitis (AP) is an inflammatory disorder of the pancreas, characterized by edema, autodigestion, fat necrosis of pancreatic tissue consequently systemic inflammatory response [1]. Pancreas is an irregular ribbon located among dodecadactylon, stomach and transverse colon, the former two of which make up the gastric outlet position. Gastric retention is an uncommon but not rare complication of AP featured by the delayed emptying of gastric contents and can be divided into gastric outlet obstruction (GOO) and gastric hypomotility [2]. There are a couple of factors causing this complication: (1) The pancreatic necrosis with large size can compress the surrounding gastrointestinal tract [3,4]; (2) The inflammatory ooze from the pancreas in AP can infiltrate the surrounding gastrointestinal tract and causes tract edema [5]; (3) Gastric wall cholinergic nerves and smooth muscle is invased and come into dysfunction [6]; (4) High intra-abdominal pressure inhibits the motility of gastrointestinal tract [7].

GOO is featured by the obstruction of the distal stomach, duodenum and/or proximal jejunum and is a relatively rare but severe complication requiring intervention in acute pancreatitis (AP) raised in revised Atlanta classification [8,9]. The most common

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clinical symptoms of GOO patients include epigastric pain, nausea, vomiting, abdominal distension and these patients can be found a succussion splash by physical examination with the increased flow during gastric decompression [10,11]. The stenosis of gastrointestinal tracts can be caused by either by the compression of local fluid/necrotic collections or the severe gastric outlet gastrointestinal edema (GOGE) [8].

Previous studies reported several cases of GOO developed after 4 weeks from the onset of AP result from the compression of pancreatic pseudocyst (PP) or walled-off necrosis (WON) [3,12,13]. However, to our knowledge, GOO developed within the first 4 weeks of AP is rarely reported. More, there is no consensus on the management of GOO in AP. In this retrospective study, we described the clinical features, etiologies and outcomes of GOO that occurred during the entire course of AP and the management strategy in a tertiary center in China.

Method

Patient selection

This study retrospectively screened 1038 AP cases admitted to the Center of Acute Pancreatitis, Department of Critical Care Medicine from March 2017 to June 2020 Jinling Hospital, Medical School of Nanjing University. Our center is a tertiary center for severe acute pancreatitis located in eastern China. Inclusion criteria were AP patients with the complication of GOO in the whole duration of disease. All the data were extracted from a prospectively collected database with the approval of the institutional review board (No: 2020 JLAPDMC-005). Routine written informed consent was obtained for data collection, storage, and academic use of data from all patients or next of kin at admission. Additional informed consent from individuals was waived due to the retrospective and anonymous nature of the current study.

Definition

The diagnosis and local complications of AP were defined according to the 2012 revision of the Atlanta Classification [9]. Gastric outlet obstruction is defined as gastric outlet stenosis on computed tomography (CT) or upper gastrointestinal contrast study plus the clinical symptoms including mass daily gastric juice drainage which cannot be relieved by gastrokinetic agents, intolerance of entogastric nutrition with vomiting and upper abdominal distention. The pancreatic necrosis compression (PNC) (as shown in Fig. 1) and severe GOGE is diagnosed based on abdominal CT or gastroscope by at least two independent clinicians with more than 10 years of experience in treating AP from Jinling hospital. Biliary obstruction refers to blockage of the bile duct system preventing bile from flowing from the liver into the intestinal tract, which is diagnosed on the basis of the abdominal medical imaging examination (computed tomography, transabdominal ultrasound, magnetic resonance imaging and cholangiography) and laboratory test (aminotransferase, aspartate aminotransferase, bilirubin, etc.) [14].

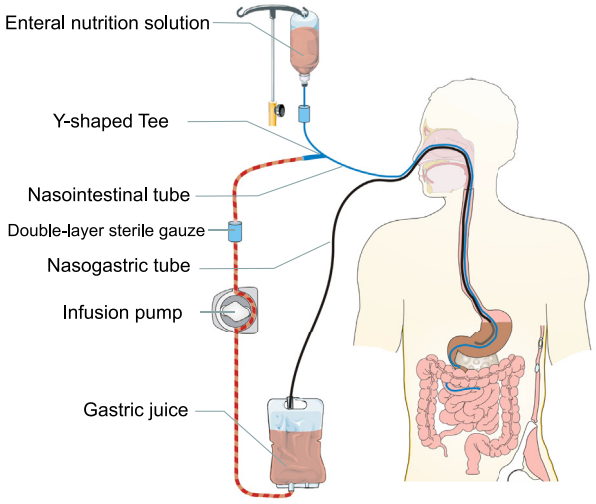

Fig. 1. Gastric outlet obstruction in acute pancreatitis and the management of reinfusing gastric juice.

Data collection

The demographic characteristics (including age, gender, body mass index, etc.), clinical features (including pancreatitis severity, organ functions, incidences of systemic and local complications, mortality, etc.), management and clinical outcomes of each patient were extracted from the database. Patients were assessed with blood routine and biochemical tests and evaluated with CECT during the first 24 h after admission. All the laboratory results including levels of lipase and amylase were obtained from the Central Laboratory of Jinling Hospital according to the standard protocols.

Management of GOO

The management of GOO includes supportive care treatment and specific management of obstruction.

Supportive care treatment: A nasogastric tube was placed for gastric decompression in these patients to relieve vomiting or abdominal distention. Proton pump inhibitors (PPIs) was administered to decrease the volume of gastric secretions. Gastric juice was drained via nasogastric tube every 4 h to observe color, character and volume. The collected gastric juice was filtered through double-layer sterile gauze and reinfused within 2 h to jejunum solution at a constant speed of approximately 100 ml/h via a three-way tube connected to nasointestinal tube (shown in Fig. 1). The PH value of the reinfused gastric juice should be above 5 to avoid affecting the intestinal environment (shown in Fig. 1).

Specific treatment: In the first 4 weeks, ANC or severe GOGE are the main causes of GOO. For the former, it was most leave unintervened until encapsulation of collection, arbitrary four weeks from onset, while for the latter, fluid restriction whenever possible were applied to improve edema.

Results

1038 patients were initially screened and eventually 60 patients were enrolled. The median age of the 60 patients was 44 years. Most patients were male (44/60, 73.3%). Most patients developed pancreatic necrosis>50% (38/60, 63.3%). Hyperlipidemic pancreatitis (29/60, 48.3%) and biliary pancreatitis (27/60, 45.0%) are the most common etiologies. Patients' demographic and clinical data are displayed in Table 1.

The abdominal CT findings of GOO patients are listed in Table 2. Based on the time interval from AP onset (13 vs 64 days), GOO patients were divided into GOO (≤4 weeks; n = 33) and GOO (>4 weeks; n = 27). The time interval from AP onset to GOO has two peaks as shown in Fig. 2. PNC (60.6%; 20/33) is the most common cause of GOO during the early phase (≤4 weeks), and WON (92.6%; 25/27) during the late phase (>4 weeks). Fig. 3 shows the CT finding of GOO caused by PNC(3a), GOGE (3b), WON (3c), and PP (3d). The

biliary obstruction was diagnosed in 26 patients (43.3%), of whom 12 patients (46.2%) developed cholecystitis and 19 patients (73.0%) received percutaneous gallbladder drainage.

Table 3 shows the clinical feature and outcomes of GOO patients(≤4 weeks). Of the 33 patients, 21 (63.6%) patients receive percutaneous catheter drainage (PCD) and the others receive conservative treatment. Moreover, 15 (45.6%) patients received endoscopic necrosectomy, and the median number of sessions was 3.5 (IQR: 1–5). Eighteen (54.5%) patients developed infected pancreatic necrosis and 8 (24.2%) patients have the gastrointestinal fistula. Organ failure includes ARDS (60.6%), AKI (51.5%), and shock (24.2%). Seven (21.2%) patients died due to uncontrolled infection and or massive abdominal bleeding.

Discussion

Gastric outlet obstruction is not a rare complication of AP. GOO is not functional which is why it differed from gastroparesis. Distal stomach, duodenum and proximal jejunum make up the gastric outlet which has the physical function of the path of gastric juice, pancreatic juice and bile. The obstruction of gastric outlet may cause enteral nutrition intolerance and low protein intaking leading to the poor encapsulation of pancreatic necrosis and worse outcome. In this retrospective study, we reviewed the AP patients admitted to Jinlin hospital. We found that among 1038 patients, 60 patients (5.8%) developed GOO and the occurrence has two peaks during the course of AP corresponding to the local complication of ANC (≤4 weeks) and PP or WON (>4 weeks).

It has been reported that GOO is commonly caused by PP or WON after 4 weeks after the onset of AP [4,12]. In this study, we found that GOO associated with compressive effects of pancreatic collections within the first 4 weeks of AP was not uncommon, and severe gastric outlet gastrointestinal edema can also lead to early-onset GOO. For the late phase(beyond 4 weeks), WON is the leading cause of GOO.

The current American college of gastroenterology guidelines (2013) recommended ongoing gastric outlet obstruction is one indication for surgical intervention regardless of infected or sterile pancreatic necrosis [15]. However, IAP/APA evidence-based guidelines (2013) and WSES guidelines (2019) recommend that gastric obstruction should be intervened iwhen the necrosis collection get encapsulated, usually 4 weeks after the onset of the disease [16,17]. PCD was reported to be a minimally invasive and safe therapy for compressing GOO caused by WON and PP [4]. In AP patients complicated by GOO (≤4 weeks), the incidence of infected pancreatic necrosis and gastrointestinal fistula is much higher than reported previously [18,19]. Massive necrosis surrounding the duodenum could release enzyme-rich fluid harming gastrointestinal mobility, and facilitating the formation of edema, thrombosis, ischemia, necrosis and fistula eventually [20]. Therefore, drainage of ANC within the first 4 weeks aiming at releasing GOO should be considered in selected patients.

In our practice, we commonly apply supportive treatment including gastric decompression, PPIs, enteral nutrition support and gastric juice reinfusion rather than PCD given the risk of introducing infection. Among them, gastric juice reinfusion is not well described in the literature. A previous study had shown that gastric juice reinfusion is a simple and effective method to maintain fluid and electrolyte balance [21]. Moreover, gastric juice contains digestive enzymes which are helpful for the implementation of enteral nutrition. Futher, reinfusion of intestinal fluid to distal small bowel may diminish upper gastrointestinal secretions [22] and therefore alleviate GOO related symptoms. In our center, we reinfuse gastric juice continuously via nasojejunal tube at the rate of 100 ml/h. In view of the small number of cases in our study, more cases in multicenter studies and control studies may provide more convincing results for the optimal treatment of GOO in acute pancreatitis.

Table 1
Demographic data and baseline characteristics of the AP patients with GOO.

Parameter	Value
Total number of patients	60/1038 (5.7%)
Age, year	44 (37.25, 54.75)
Male: female ratio	44:16
BMI, kg/m2	26 (24, 27.75)
APACHE II score	10 (5,15.5)
Pancreatic necrosis,	
<30%	9 (27.3%)
30%–50%	15 (45.5%)
50%	38 (63.3%)
Cause of AP	
Biliary	27/60 (45.0%)
Hyperlipidemic	29/60 (48.3%)
Alcoholic	2/60 (3.3%)
Trauma	1/60 (1.7%)
Unknown	1/60 (1.7%)
Laboratory abnormalities	
Amylase, IU/L	131.5(39.7, 429.3)
Lipase, IU/L	791.5 (241.0, 1096.0)
Smoking history, no. (%)	19/60 (31.7%)
Drinking history, no. (%)	10/60 (16.7%)

Abbreviation: AP acute pancreatitis, GOO gastric outlet obstruction, BMI body mass index, APACHE II Acute Physiology and Chronic Health Evaluation II.

Table 2
CT finding and the number of AP patients with GOO.

Parameter	Value
GOO ≤4 weeks,	55% (33/60)
Time interval from the onset of AP to GOO, days	13 (8.5, 19.5)
PNC	60.6% (20/33)
GOGE	27.3% (9/33)
PNC + GOGE	12.1% (4/33)
GOO > 4 weeks	45% (27/60)
Time interval from the onset of AP to GOO, days	64 (32, 120)
PP	7.4 (2/27)
WON	92.6% (25/27)

Abbreviations: CT computed tomography, AP acute pancreatitis, GOO gastric outlet obstruction, PNC pancreatic necrosis compression, GOGE gastric outlet gastrointestinal edema, WON wall-off necrosis, PP pancreatic pseudocyst.

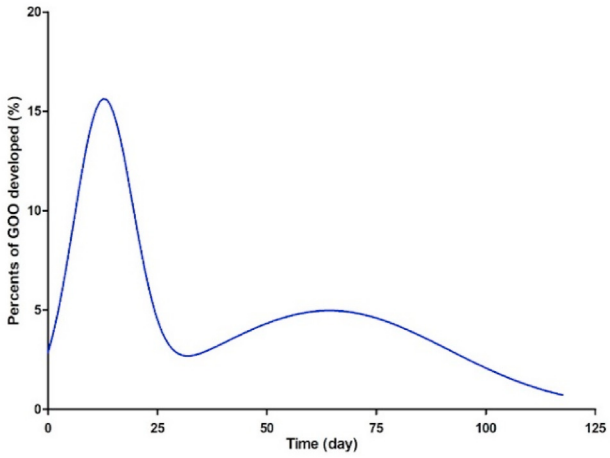

Fig. 2. The percentage of gastric outlet obstruction developed in the duration of acute pancreatitis.

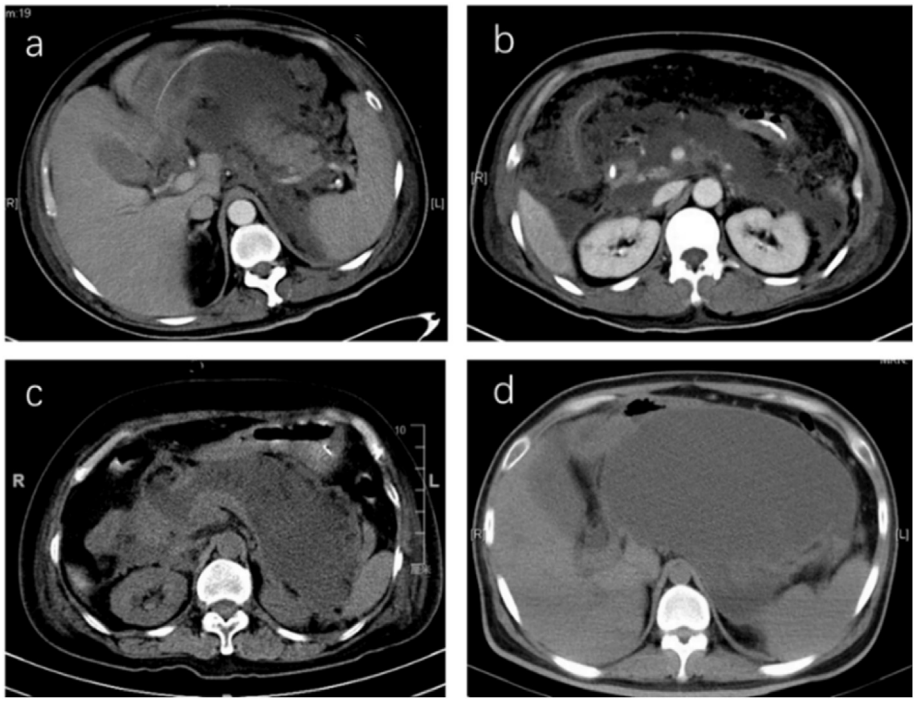

Fig. 3. Abdominal CT findings of GOO patients caused by PNC(3a), GOGE (3b), WON (3c), and PP (3d)
Abbreviation: CT Computed Tomography, GOO gastric outlet obstruction, PNC pancreatic necrosis compression, GOGE gastric outlet gastrointestinal edema, WON wall-off necrosis, PP pancreatic pseudocyst.

Table 3
The clinical outcomes of the AP patients developing GOO (≤4 weeks).

Parameter	GOO (≤4 weeks) (n = 33)
Time interval from the onset of AP to GOO [days]	13 (8.5, 19.5)
PCD	21 (63.6%)
Infected pancreatic necrosis	18 (54.5%)
Bleeding	10 (30.3%)
Gastrointestinal fistula	8 (24.2%)
Intra-abdominal hypertension	15 (45.5%)
Biliary obstruction	26 (43.3%)
Organ failure	
ARDS,	20 (60.6%)
AKI	17 (51.5%)
Shock	8 (24.2%)
Hospital stay [days]	44 (21, 72.5)
ICU stay [days]	34 (14.5, 53.5)
Open necrosectomy	5 (15.2%)
Mortality	7 (21.2%)

Abbreviations: AP acute pancreatitis, GOO gastric outlet obstruction, PCD percutaneous catheter drainage, ARDS acute respiratory distress syndrome, AKI, acute kidney injury, ICU intensive care unit.

Conclusion

GOO, as a gastrointestinal complication developed in AP patients, has two peak incidences during the course AP. Treatment of GOO should consider both supportive treatment and etiological treatment in different phases of the disease.

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Declaration of competing interest

No conflict of interest was declared by the authors.

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