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Aspects of the Surgical Management of Acute Biliary Pancreatitis

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ABSTRACT

This study presents a retrospective analysis of the management of 221 patients with acute biliary pancreatitis. In cases of gallstone impaction in the major duodenal papilla, endoscopic papillotomy is recommended within the first few hours; in patients with choledocholithiasis, obstructive jaundice, cholangitis, and acute biliary pancreatitis, endoscopic papillotomy combined with lithoextraction should be performed within the first 24 hours of hospital admission. Cholecystectomy—preferably performed laparoscopically—is indicated after conservative resolution of mild biliary pancreatitis, optimally within 3–7 days. When endoscopic papillotomy is uneventful, cholecystectomy may also be performed during the same hospitalization without prior discharge. In cases of acute biliary pancreatitis complicated by sterile or infected fluid collections, cholecystectomy should be deferred until complete resolution of these collections and the abatement of the systemic inflammatory response

Keyword: *acute pancreatitis; surgical management; complications of acute pancreatitis; pancreatic necrosis*.

1. INTRODUCTION:

Acute biliary pancreatitis (ABP) is a common condition, accounting for 25–30 % of all cases of acute pancreatitis, and presenting in a severe form in 15–25 % of patients [1,3,5]. Its principal etiological factors include gallstone disease, the anatomical relationship of the common bile duct and the main pancreatic duct with their embryological development as a "common channel," microlithiasis, and the impaction and migration of stones through the major duodenal papilla [2,7,9].

Currently, an active surgical approach to gallstone disease is advocated, encompassing the widespread adoption of laparoscopic cholecystectomy and endoscopic retrograde pancreatocholangiography with the option of papillotomy and stone extraction. However, outcomes of surgical management in ABP remain suboptimal, as the mortality rate in severe cases reaches 15–30 % [4,8]. Therefore, timely identification of specific laboratory parameters and findings of specialized investigations characteristic of ABP is of paramount importance. It is necessary to substantiate the choice of surgical approach—minimally invasive or "open"—based on the predominance of pathological changes in the gallbladder, bile ducts, pancreas, and parapancreatic and retroperitoneal spaces. Definitive resolution is also required regarding the necessity, efficacy, and risks of endoscopic papillotomy in the setting of complicated ABP, as well as the optimal timing of cholecystectomy after resolution of ABP of varying severity [6,10].

The aim of this study was to develop a rational surgical strategy for patients with acute biliary pancreatitis.

2. MATERIALS AND METHODS

This retrospective study analyzes the management of 221 patients with acute biliary pancreatitis treated at our clinic over a period exceeding 10 years. Patients were stratified into two groups: Group I (mild disease course): 142 patients (64.2 %); Group II (moderate to severe disease course): 79 patients (35.8 %)

Inclusion criteria were confirmed biliary etiology—defined by the presence of gallstone disease and serum amylase levels at least three times the upper limit of normal—while patients whose attacks followed alcohol consumption were excluded. Consistent with existing epidemiological data, a female predominance (1 : 1.5) was observed. Patient ages ranged from 18 to 75 years (mean ± SD, 54.5 ± 1.9 years). In the moderate-to-severe group, 84.7 % were of working age (20–50 years),

comprising 34 men (43.1 %) and 45 women (56.9 %). Laboratory evaluation included a complete blood count performed on a Sysmex XT-4000i analyzer using EDTA-anticoagulated peripheral blood, urinalysis, and a comprehensive biochemical panel (total and direct bilirubin, amylase, alkaline phosphatase, alanine aminotransferase [ALT], aspartate aminotransferase [AST], albumin, total protein, urea, creatinine, glucose, electrolytes, C-reactive protein, and procalcitonin) on 3 mL of heparinized venous blood. Hemostatic assessment comprised thromboelastography and standard coagulation profiling, and microbiological cultures were performed on sterile specimens.

3. RESULTS AND DISCUSSION

The treatment of 142 patients with mild acute pancreatitis consisted of analgesia, administration of octreotide, anti-mediator therapy with ksefocam, antibiotics in the presence of fever, antispasmodic treatment, and correction of fluid–electrolyte imbalances. Review of these patients' medical records revealed a marked difference in peak serum amylase levels between biliary and alcoholic pancreatitis. In biliary pancreatitis, amylase elevations above 800 U/L were typical; however, 34 patients with clinically mild disease demonstrated amylase levels ranging from 1,500 to 4,000 U/L. Because of the risk of developing pancreatogenic shock, these patients required transfer to the intensive care unit and received a treatment regimen comparable to that used for severe acute pancreatitis.

Correction of derangements in patients with moderate and severe acute pancreatitis—characterized chiefly by transient or progressive multiple organ dysfunction—began with placement of a central venous catheter, insertion of a nasojejunal tube for intestinal decontamination and enteral feeding, and placement of an epidural catheter for anesthesia. Broad-spectrum antibiotic therapy was administered, along with prophylaxis for stress ulcers; exocrine pancreatic secretion was routinely inhibited with octreotide; anti-mediator therapy was continued; and extracorporeal detoxification was initiated when the APACHE II score exceeded 15. Over the past three years, we have added anti-mediator therapy with ksefocam to this regimen to suppress the cytokine storm. Organ-system support measures were applied as dictated by the severity of dysfunction. This comprehensive approach successfully prevented progression from moderate acute pancreatitis with transient multiple organ dysfunction to severe disease in 15 patients.

Among the 79 patients with moderate to severe disease, six exhibited impaction of a stone in the major duodenal papilla; ten showed gallbladder destruction with sterile peripancreatic fluid collections; another ten had gallbladder destruction with infected collections; twenty-three had choledocholithiasis accompanied by cholangitis and obstructive jaundice; and thirty developed infected pancreatic necrosis without signs of demarcation (Figure 1).

Patients with confirmed biliary pancreatitis complicated by impaction of a stone in the major duodenal papilla (six patients) and those with choledocholithiasis, mechanical jaundice, and cholangitis (twenty-three patients) warrant special consideration in terms of treatment organization. This cohort demands heightened vigilance because "time is a risk factor," and prolonged obstruction at the ampulla of Vater or within the extrahepatic bile ducts leads to difficult-to-manage complications such as pancreatic necrosis, suppurative cholangitis, and hepatic abscesses. Clinically, stone impaction at the papilla mimics renal colic, with patients experiencing intense, constant pain radiating to the back and exhibiting marked restlessness. Accordingly, after brief stabilization, endoscopic papillotomy—typically performed with a needle-knife electrosurgical electrode—should be carried out as early as possible. With growing endoscopist expertise at our center, this intervention can now be performed around the clock, often within 1–2 hours of admission. A similarly proactive strategy applies to patients presenting with choledocholithiasis, cholangitis, and obstructive jaundice alongside biliary pancreatitis.

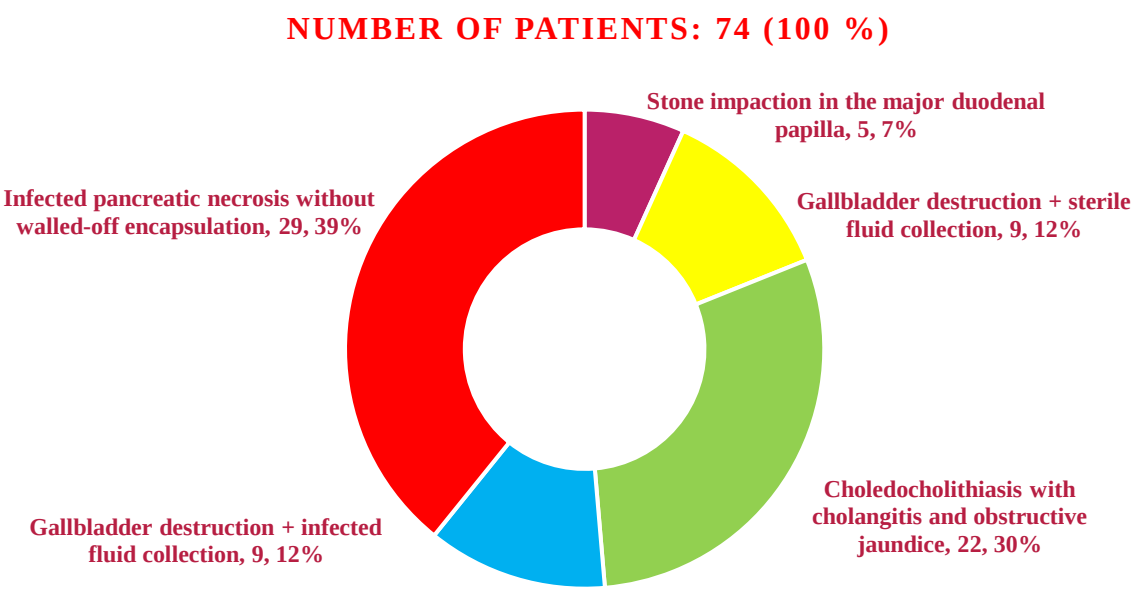

Figure 1. Distribution of patients with moderate and severe acute biliary pancreatitis.

Although the necessity and timing of papillotomy in choledocholithiasis with cholangitis and jaundice remain debated, we strongly advocate for early intervention in biliary pancreatitis accompanied by microlithiasis or choledocholithiasis, cholangitis, and mechanical jaundice. We perform endoscopic sphincterotomy and stone extraction within the first 24 hours of admission. Among our 23 patients with this constellation of findings, cholangitis and jaundice resolved and acute biliary pancreatitis regressed following ERCP with papillotomy and lithoextraction. Notably, four of these patients were transferred after 2–5 days of conservative management at other institutions where papillotomy was either deferred or technically unfeasible. No fatalities occurred in either subgroup, and none of the patients undergoing endoscopic transpapillary procedures experienced significant clinical deterioration or required transfer to intensive care. Moreover, no hemodynamic instability was observed during any ERCP or papillotomy procedures. The frequency of ERCP-related complications in our acute biliary pancreatitis cohort is detailed in Figure 2.

As shown in Figure 2, the risk of progression to pancreatic necrosis following ERCP is 4%. Importantly, no cases of serious complications with multiple organ dysfunction syndrome were observed among our patients after ERCP with endoscopic sphincterotomy. Following endobiliary intervention, patients demonstrated marked clinical improvement. In 86 % of cases, no repeat procedures were required to alleviate biliary hypertension. In these patients, intraductal pathology had been the underlying cause of impaired bile flow and pancreatitis development. After ERCP with sphincterotomy, clinical status improved and laboratory parameters normalized—serum amylase, bilirubin, transaminases, and hematologic indices returned to normal ranges, inflammatory markers subsided, and signs of systemic inflammatory response resolved. Imaging also demonstrated reduction in pancreatic size as well as normalization of bile-duct and gallbladder dimensions. Changes in laboratory and instrumental parameters before and after endoscopic intervention are presented in Table 1.

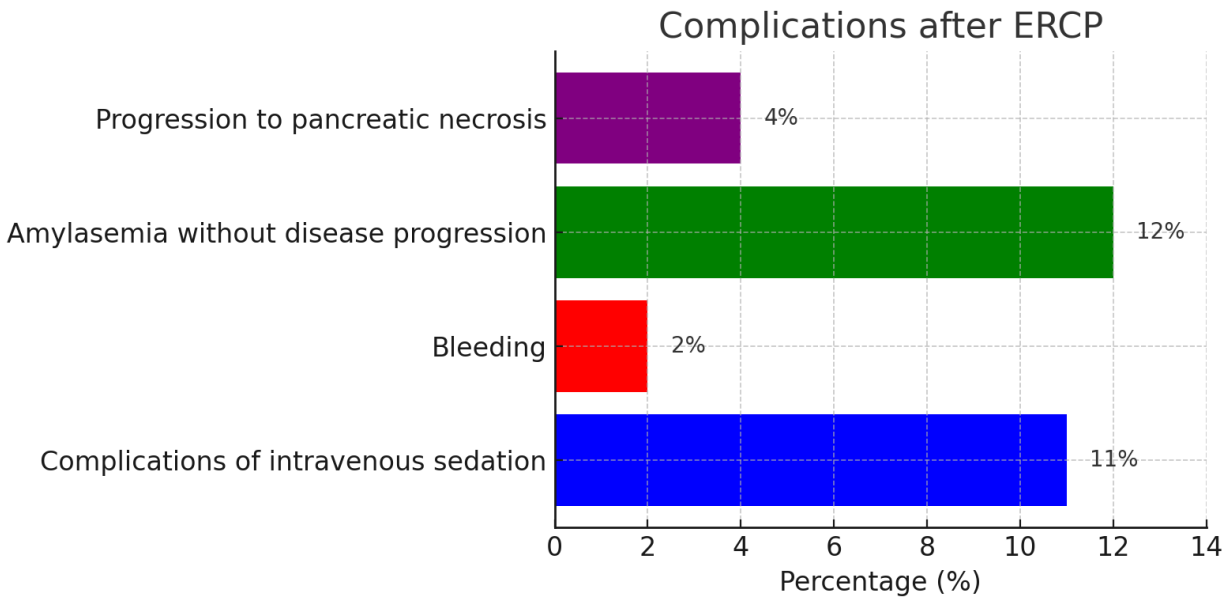

Figure 2. Frequency of complications after ERCP in patients with acute biliary pancreatitis.

As our data demonstrate, endoscopic interventions in patients with acute biliary pancreatitis are highly effective, yielding statistically significant improvements in both laboratory and imaging parameters. To address intraductal pathology in this cohort, endoscopic correction is the preferred approach. Of the 23 patients in this subgroup, 15 underwent surgery within 2–3 weeks after resolution of pancreatitis, jaundice, and cholangitis: 12 had laparoscopic cholecystectomy (with conversion to open cholecystectomy in two cases), and two required open cholecystectomy three days after endoscopic papillotomy due to development of acute cholecystitis. Five additional patients were operated on 4–7 days post-papillotomy owing to acute cholecystitis accompanied by stone migration into the bile ducts; they underwent open cholecystectomy, choledocholithotomy, and T-tube drainage of the bile ducts. In ten patients with gallbladder destruction and sterile peripancreatic fluid collections, ultrasound-guided drainage of both the gallbladder and the collections was performed, followed by cholecystectomy. In another ten patients who developed walled-off infected collections, either percutaneous catheter drainage or open intervention was carried out, with cholecystectomy deferred until complications had resolved. Sterile or infected pancreatic fluid collections generally mandate ultrasound-guided drainage: percutaneous catheter drainage is indicated for sterile collections larger than 10 cm or multiple collections, and for infected collections that are well-demarcated, solitary, and contain no more than 100 mL of pus. Collections that do not meet these criteria often require conversion to open surgery.

Table 1. Changes in laboratory and imaging parameters in patients with acute biliary pancreatitis before and after endoscopic intervention.

Indicator	**Before ERCP with EST**	**Next day after ERCP with EST**	**p-value**
Number of SIRS criteria	1.4±0.3	0.9±0.2	< 0.001
SOFA score in MODS patients	3.8±0.5	2.2±0.5	0.002
Common bile duct diameter (mm)	11.8±0.6	7.5±0.4	< 0.001
Gallbladder length (mm)	92.6±3.1	67.1±1.5	< 0.001

Pancreatic head diameter (mm)	35.2±2.1	26.9±1.2	< 0.001

Correction of the disturbances seen in moderate and severe acute pancreatitis—marked by transient or progressive multiple organ dysfunction—began with placement of a central venous catheter, insertion of a nasojejunal tube for intestinal decontamination and enteral nutrition, and placement of an epidural catheter for analgesia. Patients received broad-spectrum antibiotics, stress-ulcer prophylaxis, mandatory inhibition of exocrine pancreatic secretion with octreotide, anti-mediator therapy, and extracorporeal detoxification when the APACHE II score exceeded 15.

We observed 30 patients with infected pancreatic necrosis without signs of encapsulation. In addition to the above measures, organ-system support was provided as needed. This regimen successfully prevented progression from moderate pancreatitis with transient organ dysfunction to severe disease in 14 patients. In 23 of these 30 patients—whose necrotic collections extended into the parapancreatic, left, right, or both retroperitoneal spaces and the greater omentum—an open abdominal approach was selected, and cholecystectomy, choledocholithotomy, and T-tube drainage were performed. In the remaining seven cases, extensive inflammatory infiltration precluded cholecystectomy and bile-duct surgery; instead, cholecystolithotomy and cholecystostomy were carried out. Given the involvement of the parapancreatic and retroperitoneal spaces and the greater omentum, conservative or minimally invasive interventions are unlikely to succeed. Accordingly, we recommend the following clinic-developed principles for managing such patients: use of bilateral subcostal or unilateral (left or right) subcostal incisions based on necrosis location; wide opening of the omental bursa with mobilization of the right and/or left colonic flexures; exposure of the pararenal spaces; direct access to the pancreas; maximal preservation of viable pancreatic tissue; evacuation of all purulent collections and removal only of freely mobile necrotic debris (avoiding forced necrosectomy and limiting indications for pancreatic resection); packing of the omental bursa and retroperitoneum without reliance on indwelling drains; provisional wound closure with sutures; and avoidance of on-demand relaparotomy in favor of planned lavage every 48–72 hours. Notably, the duration of illness prior to admission in this group ranged from 7–12 days up to 2–3 weeks. Implementation of this strategy yielded encouraging results in this particularly high-risk cohort, with seven fatalities among 30 patients (23.3 %).

4. CONCLUSIONS

1. The choice of an appropriate management strategy for acute biliary pancreatitis hinges on the patient's clinical severity, laboratory findings, the presence of organic changes in the biliary tract and pancreas, and any involvement of the retroperitoneal space in the inflammatory process.
2. Endoscopic papillotomy reliably relieves obstruction of the biliary and pancreatic ducts in acute biliary pancreatitis and abolishes ductal hypertension—the primary driver of suppurative cholangitis and necrotizing pancreatitis.
3. Cholecystectomy—preferably performed laparoscopically—is best undertaken after conservative resolution of mild biliary pancreatitis, optimally within 3–7 days. When endoscopic papillotomy has been completed without complications, cholecystectomy may be performed during the same hospitalization. In cases complicated by sterile or infected fluid collections, cholecystectomy should be postponed until these collections have fully resolved and the systemic inflammatory response has abated.

The diagnostic and therapeutic algorithm we propose for biliary pancreatitis, together with evidence-based indications for different surgical procedures, facilitates an individualized surgical approach. In our series, mortality among patients with severe pancreatic necrosis was 23.3 %.

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