ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 8, Issue, 12, pp.43026-43036, December, 2016

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

ACUTE GALLSTONE PANCREATITIS

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ARTICLE INFO

Article History:
Received 29th September, 2016
Received in revised form
18th October, 2016
Accepted 05th November, 2016
Published online 30th December, 2016

Key words:

Acute,
Gallstone,
Pancreatitis.

ABSTRACT

Background: More than 220,000 patients are admitted to hospitals each year with acute pancreatitis in the United States alone. Acute pancreatitis is relatively common.
Aim: To explore the modern modalities focusing on the diagnosis and treatment of acute gallstone pancreatitis (GSP).
Methods: Systemic review of PubMed filter finds publications to support keywords of the current study.
Findings: Among the numerous causes, two factors which account for about 70 -80% of cases of acute pancreatitis are biliary tract disease and alcoholism. Abdominal pain is the major manifestation of acute pancreatitis. The pain may vary from mild and tolerable to severe, constant and incapacitating distress. Marked elevation of serum amylase levels during the first 24 hours, followed within 72 to 96 hours by a rising serum lipase level. Hyperglycemia is common, hypocalcemia may result from precipitation of calcium soaps in necrotic fat, and hypertriglyceridemia occurs in 15 to 20% of patients. Direct visualization of the enlarged inflamed pancreas by radiography is useful in the diagnosis of pancreatitis. In the majority of patients [85–90%] acute pancreatitis is self-limited and subsides spontaneously, usually within 3–7 days after treatment is started. About 5% with severe acute pancreatitis die from shock during the first week. Acute respiratory distress syndrome and acute renal failure are dangerous complications.
Conclusion: GSP is a disease with a wide spectrum of severity. Diagnosis and management have evolved in recent decades with the advent of new and improved technology. Advances in imaging techniques have limited the need for diagnostic procedures in many cases, and various treatment options are becoming more widely available.

Citation: Al Ghouthani Jamal, 2016. "Acute gallstone pancreatitis", *International Journal of Current Research,* 8, (11), 43026-43036.

INTRODUCTION

Pancreatitis is inflammation of pancreas that arise within a short period of time. Pancreatitis is exacerbated, in most cases, due to be gallstones or drinking alcohol excessively. [1] Among the other causes of pancreatitis involves intake of medications, exposure to infections, exposure to injuries, disruption in the body's metabolism or due to undergoing surgery. Around 80 % of patients who developacute pancreatitis, regardless of etiology, havemild disease. Other patients developsevere disease characterized by end organ failureand/or necrosis of the pancreatic parenchyma orperipancreatic fat [2]. Cholelithiasis is the most common cause of acute pancreatitis in the world. In most cases, gallstone pancreatitis (GSP) is a mild and self-limiting disease, and patients can proceed without complications, some cases require cholecystectomy. Severe disease occurs in about 20% of cases and is associated with significant mortality and critical management. Thorough understanding of the disease processes, diagnosis, severity, stratification and management principles is vital to the appropriate care to patients with this common disease. [3,4]

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MATERIALS AND METHODS

Cholelithiasis, or gallstones, are present in up to 10% of the general population. Risk factorsfor developing gallstones include female sex, advancing age, ethnicity and genetics, obesityand the metabolic syndrome, rapid weight loss, high fat low fiber diet, pregnancy, andcertain disease states such as cirrhosis and Crohn's disease. GSP is more common in women over 60 years, and the number of cases reported each year is increasing around the world, perhaps because of the worsening obesity. [1,5] The incidence of acute pancreatitis is estimated at 40 per 100 000, and 40% to 50% of cases are in biliary etiology. The burden of acute pancreatitis from all causes in the United States exceeds $ 2.2 billion per year, more than 300,000 hospitalizations and 20,000 deaths per year [6,8]. The

prevalence of gallstones in the US and Europe is 10% to 15%, and PSG risk factors are similar to those of the formation of gallstones: age, sex, obesity, pregnancy, genetics and family history, fasting and rapid weight loss, and biliary stasis, among others. [9,10] Although most of symptomatic gallstones commonly present as acute cholecystitis biliary colic, and the impact of the GSP development is 3% to 8%, and symptomatic gallstones carry a risk of developing GSP 0, 04% to 1.5%. [11-14]. Once gallstones are involved in acute pancreatitis, disease follows a mild course in 80% of patients, and the mortality is 1% to 3%. However, in 20% of patients' acute pancreatitis is severe, and mortality approaches 30%. [15]

Pathophysiology

Pancreatitis results from an autodigestive process. Pancreatic digestive enzymes, vasoactive materials and other toxic materials extravasate out of thepancreas into the surrounding areas, leading to a widespread chemicalirritation resulting in simple edema to severe hemorrhage and necrosis. Gallstones have been detected in the stools of up to 90% of patients with GSP, suggesting that the stones pass into the duodenum spontaneously.The composition of these stones is mainly cholesterol, bile salts, and phospholipids. [16, 17] When bile becomes saturated, overabundant cholesterol precipitates as crystals, which mixture with bilirubinate and solidify to form bile sludge, which can then aggregate to form gallstones. [18] Although the migration of gallstones in the common bile duct (CBD) is relatively common, GSP because stones with much less regularity. [19] Bernard Prince and first describes the relationship of gallstones and acute pancreatitis in 1852 and 1882, followed by Opie in 1901. [4, 20, 22] There are an impressive number of oriented basic research the complexity of this relationship and the exact mechanism by which the cause of biliary pancreatitis calculations. Risk factors include multiple small size stones below 0.5Mmin, and a large cystic duct. [23,25] Several theories have been proposed to describe how gallstones charged to the inflammatory response in acute pancreatitis, and a commonly accepted mechanism involves a transient blockage of the bile or pancreatic duct or a hit through the Pierre. Otherwise, biliary sludge can cause cholestasis or irritate the sphincter of Oddi, causing swelling and a barrier to biliopancreatic. This process causes the intracellular activation of digestive enzymes in the pancreas, but the mechanism is not well understood.Biliopancreatic reflux resulting from ductal pressure increase can contribute, but this theory has been challenged on the basis of physiological studies showing a greater secretion of pressure in the pancreatic duct in the bile duct. In addition, some researchers found that the sterile bile under physiological pressure is not harmful to the pancreas, although it has also been challenged. [26, 27]

However, the increase in intraductal pressure probably plays a role, because the extent of pancreatic lesions is related to the duration of the ampullary obstruction. [28, 29] Exocrine pancreatic sphincter, secretions, mucosal barrier, and the delay before activation of trypsinogen in the duodenum are all the protection elements of normal biliary physiology. In PGS, this homeostasis is altered and pancreatic injury is aggravated by inflammatory cytokines, which can worsen the damage and pancreatic parenchyma may induce systemic inflammatory response syndrome [SIRS] [26].

Diagnostic investigations

History and physical examination

Most patients presenting with symptoms typical of GSP complain of pancreatitis, and less can also provide a history of biliary colic. The most common complaint is the sudden onset of epigastric pain or right upper quadrant abdominal pain that is relentless, and in 50% of cases radiates to the back. [10] The symptoms are nausea and vomiting. An alcohol abuse history should arouse suspicion of alcoholic pancreatitis. [30] Examination usually shows impressive physical abdominal tenderness, and patients with pancreatitis may also have signs suggestive of an acute surgical abdomen. The immediate evaluation is necessary for patients since the presentation of the severe acute pancreatitis may mimic intestinal perforation. As with acute cholecystitis, the pain is exacerbated by eating or drinking. The Peripancreatic inflammation can lead to a generalized ileus, causing hypoactive bowel sounds and anorexia. Moderate to severe disease patients may also experience symptoms of SIRS, including fever, tachycardia and tachypnea. [8,12]

Laboratory evaluation

Laboratory analysis is essential in the initial diagnosis of acute pancreatitis. Upper abdominal pain with amylase or lipase 3 times the upper limit of normal is diagnostic of acute pancreatitis in many cases, and the addition of cholelithiasis on imaging can sufficiently identify the cause as bile ducts. Lipase is highly sensitive [> 90%] in institutions of pancreatitis, and also has an advantage over the specificity of amylase, because the lipase is produced primarily by pancreatic acinar cells, while amylase is present in saliva. Amylase level generally increases in the 2 to 12 hours after onset and to normal in 3 to 5 days, while the peaks of the lipase at 24 hours and can remain elevated for several days. [31] Of importance, the degree of elevated amylase and lipase is not correlated with the severity of the disease. [32] In spite of this limitation, amylase levels were observed in gallstones alcoholic pancreatitis compared to pancreatitis [33].A complete blood count may show leukocytosis. It was also observed that hematocrit modestly corresponds with the severity of the disease. [15] A basic metabolic panel is useful for detecting metabolic disorders, and may also demonstrate hyperglycemia slight decrease insulin secretion and increased glucagon. [7] Renal function is also important to consider in serious disease where failure is a potential sequence.In addition, patients with acute pancreatitis of any origin may present as, with hypovolemic acute renal failure corrected by adequate volume resuscitation. Bicarbonate levels are a marker of resuscitation and can correlate with the severity of the disease.The tests of liver function are also essential in the initial evaluation. Because the underlying pathology in the GSP may involve biliary obstruction, although ephemeral in most cases, patients may present with elevated bilirubin and transaminases. Transaminases are usually low that, unlike the high levels observed in viral hepatitis. However, inWhere a stone is touched they can increase markedly, but the normalization after the resolution in a day period instead of weeks. Except in a choledocholithiasis, bilirubin is usually less than 15 mg / dL, because the obstruction is usually incomplete or intermittent [34]. In 10% of cases of GSP, liver function tests [BFH] are normal. [35] Although very sensitive [48%], a level of alanine

aminotransferase more than 3 times the upper limit within 24 to 48 hours after onset is the best predictor of the GSP, with a predictive value positive 95%. [36] In addition, alkaline phosphatase and g-glutamyl transpeptidase may be high, especially if the cholestasis persists. First laboratory balance for acute pancreatitis should also include triglyceride levels and calcium for the review of hypertriglyceridemia and hypercalcemia as possible etiologic factors. In idiopathic pancreatitis, immunoglobulin G4 can help in identifying autoimmune pancreatitis.

Imaging

The goal of imaging in acute pancreatitis is 3 times: for detecting the cause of the disease [gallstones, neoplasms, anatomical variations], identify complications [fluid collections, pseudocysts, hemorrhage], and assess the severity of the disease [peripancreatic inflammation, pancreatic necrosis].

Ultrasonography

Each patient with acute pancreatitis and no other obvious cause should undergo transabdominal ultrasound to isolate gallstones as the possible cause. Ultrasound is inexpensive, sensitive, and widely available. In the last decade, technology hasdeveloped, allowing portable ultrasound devicesgreatly improved resolution. The ultrasonography parts of the pain of an invasive test and ionizing radiation computed tomography [CT]. Note ultrasound, studies are obtained by trained technicians and are dependent on the operator. In general, the presence of cholelithiasis or sludge on ultrasound [Fig. 1], in the absence of other likely causes, is sufficient evidence to diagnose GSP when combined with a typical presentation and high levels of enzymes pancreatic. Ultrasound is 95% sensitive for cholelithiasis, but GSP covering intestinal gas due to ileus can decrease sensitivity to 60% to 80% [32.37]. In the detection of choledocholithiasis, ultrasound is located 25% to 60% are susceptible [38-40]. Ultrasound is useful in detecting the expansion and intrahepatic bile ducts, which may indicate an obstruction, but is less sensitive in the context of obstruction because GSP is acute. Ultrasound may also fail to detect small stones 4 mm and small stones are a known risk factor for the GSP [41]. Despite its limitations, ultrasonography remains the standard imaging examination in the diagnosis of GSP, and in terms of imaging, it is sufficient for most patients with disease.

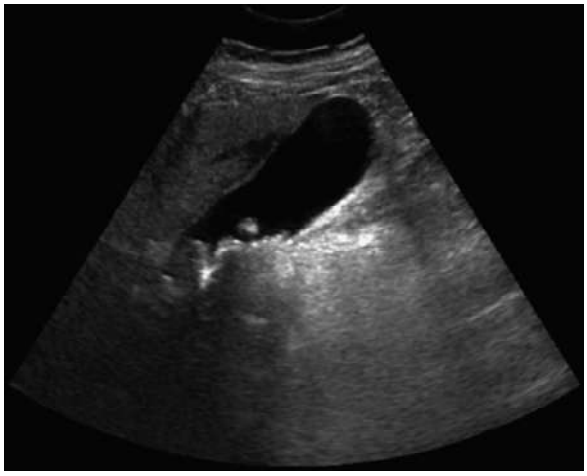

Fig.1. Typical Ultra-sonogram showing cholelithiasis and biliary sludge

Computed tomography

The use of CT in GSP is for detecting anatomical changes that correlate with complications and mortality [42].CT is often not a critical study of GSP, but provides more useful information about cases that are moderate to severe. CT is from 85% to 97% and 88% for 96% of specific common channel stones when the contrast is used [Fig. 2] [43-45]. The use of CT for the stratification of severity and direct management requires proper timing and technique. IPN is better visualized on CT 2 to 3 days after the onset of symptoms [46]. If a first CT was obtained during the diagnosis, it may need to be repeated in three days if the patient's pain is severe and persistent, and laboratory data fail to tend to normal. To optimize radiographicevaluation of pancreas, pancreatic protocol must be specified, consisting of 2 to 3 mm cross-section through the pancreas, intravenous contrast, and the two phases of pancreatic venous and imaging. Contrast orally should be avoided as it causes artifact in the duodenum which limits the study. The severity of Balthazar CT was developed to help stratify patients with acute pancreatitis.

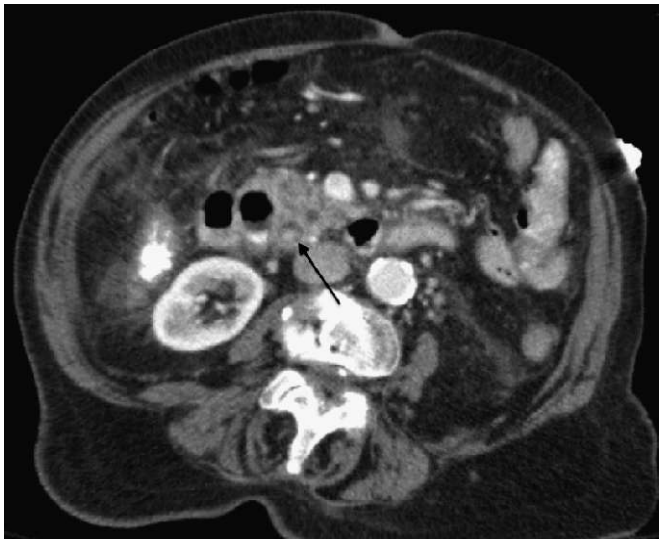

Fig.2. Computed tomography scan showing peripancreatic inflammatory changes and an obstacle to the stone [arrow] in a dilated distal common bile duct [CBD]

Magnetic Resonance Imaging

To understand the role of magnetic resonance imaging [MRI], it is important to differentiate between abdominal MRI and Magnetic Resonance Cholangiopancreatography [MRCP]. While abdominal MRI refers to a series of images of the abdomen, MRCP describes a specific protocol designed to improve fluid in the biliary system. This is a non-invasive diagnostic imaging technique that serves similar purposes to those of Endoscopic Retrograde Cholangiopancreatography [ERCP] with comparable accuracy. MRCP produces imagesthat clearly define the anatomy of the pancreatic duct and bile to delineate anatomical abnormalities such as pancreas divisum, disruption of the pancreatic duct, or filling defects that may represent tumors or gallstones. MRCP would be 85% to 90% in the sensitive detection of stones in the CBD, with 93% to 95% specificity [47,48]. One advantage of MRCP is the ability to detect stones as small as 2 mm, although this modality is still very sensitive for most smaller stones than 5 mm. MRCP is likely to confirm choledocholithiasis, and it is commonly used by clinicians to help select patients for

ERCP.Although more expensive and less available than CT, MRI excellent visualization of the bile duct, and is particularly useful in evaluating the complications of GSP. MRI can distinguish pancreatic fluid collections. Liquefied necrosis and is also useful in the diagnosis of hemorrhage of the pancreas [Fig. 3]. [10,49] Effective use of MRI is heavily based on updating technology and radiology experts that are easy to interpret the data.

Endoscopic US

Endoscopic US [EUS] is a diagnostic modality with utilization in the diagnosis of hepatobiliary abnormalities. It is done by the advancement of an endoscopic ultrasound probe skilled in the upper gastrointestinal tract. Proximity to biliary structures enables the above visualization than transabdominal ultrasound. Diagnostically, EUS is 93% to 98% sensitive and 97% to 100% for individuals' bile. [47,50] It has a negative predictive value of 93% to 100%, and may spare patients unnecessary ERCP without stones common channel. [51-53] EUS has also been used to exclude bile in pregnant women with GSP and in patients who have cons-indications of MRCP as implanted metallic devices. The safety profile of EUS is superior to diagnostic ERCP, and its use in ERCP pretherapeutic adjustment was strongly advocated. [51]

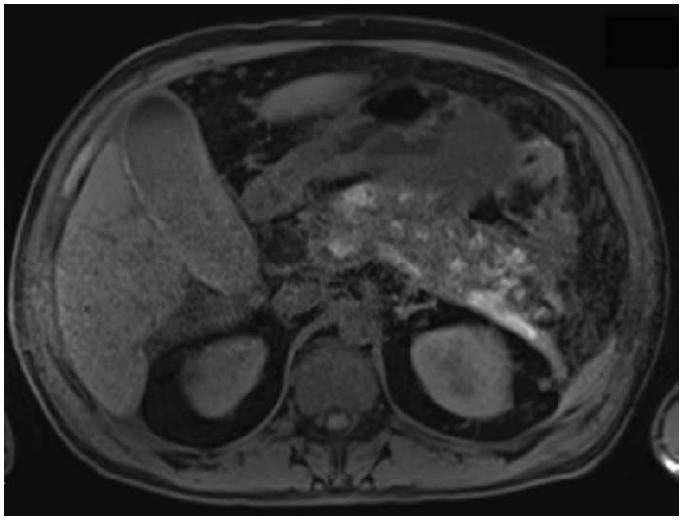

Fig. 3. Pre-contrast T1-weighted magnetic resonance-image showing a hemorrhagic pancreatitis and a wide pancreatic improvement

Management

A central principle in determining the best management plan is the prediction of disease severity. GSP is a disease with various degrees of severity, ranging from mild pancreatic resolves within 24 hours to the infected pancreatic necrosis fulminant. Pancreatitis is self-limited to a mortality rate of 1% to 3%, and describes 80% of cases. In 15% to 25% of patients with all forms of acute pancreatitis, the disease can progress to the IPN, and some of these patients will progress following pancreatic necrosis infected with a 30% mortality. [15] It is therefore essential for determining the severity of the disease at the beginning of the current hospital ensure that care and support interventions are planned while not delaying care for patients with mild disease. Several models have been developed to assist in risk stratification and predict mortality in patients with acute pancreatitis.

Initial management

General guidelines in the management of acute pancreatitis also apply to GSP. The base of supportive care is to ensure control of pain, correct metabolic disorders, aggressive rise with intravenous fluids and prevent hypoxemia. Patients with ileus may require a nasogastric tube for decompression. Those with severe disease are best served by a multidisciplinary team in intensive care, including gastroenterologists with ERCP capabilities, hepatobiliarysurgery, comfortable withexpert intensive carephysicians, and interventional radiologists. Patients having a disease may require adequate hydration and pain control prior to cholecystectomy.

Nutrition

Patients admitted with acute pancreatitis are usually kept in the mouth from zero. However, those with mild disease have a shorter length of stay in hospital with food for immediate oral administration. [67] GSP patients are no exception, unless early feeding interferes with early cholecystectomy. Patients with severe disease have been shown to benefit from enteral nutrition within 48 hours, without exacerbation of the disease. Total Parenteral Nutrition (TPN) reserved for patients who can not tolerate enteral feeding. The gastric jejunal feeding is preferred, but food has also been shown to be safe. [68]

Antibiotics

Broad-spectrum antibiotics are indicated in the expectation of a diagnostic workup for sepsis and infected pancreatic necrosis. In sterile pancreatic necrosis, the use of prophylactic antibiotics is controversial. There are several major studies that disagreeif prophylactic antibiotics gives an advantage in pancreatic sepsis and mortality, and the recent literature does not recommend the use of antibiotics in sterile necrosis. [69-71] It has been shown that prolonged use of antibiotics broad-spectrum, increases the risk of developing a fungus infection, if appropriate use is imperative. [15]

Interventions

Unlike alcoholic pancreatitis, that management is primarily to provide supportive care, management of GSP includes several terms that are specific to the underlying cause of the disease. These measures include cholecystectomy, exploration of the CBD, ERCP with sphincterotomy and specific intervention radiology

Procedures: The objectives of these procedures are mitigating the severity of the disease to prevent the recurrence of GSP.

Cholecystectomy

The goal is cholecystectomy to prevent the recurrence of the GSP by eliminating the source of secondary gallstones. Although 1% to 2% of patients can recur even after cholecystectomy, the recurrence rate in untreated patients with GSP is up to two thirds of patients in the first 3 months of the Presentation Index. [72- 75] Recurring GSP can be more serious the initial presentation, between 4% and 50% of cases are reported as severe, and mortality and morbidity is reported in up to 10% and 40%, respectively. [13,76,77] Patient stratification as mild, moderate or severe has a profound impact on the surgical management. Historically, the

recommendation was delaying cholecystectomy for 6 to 8 weeks after acute pancreatitis attack to allow the inflammation to disappear. [78] The high rate of patients awaiting cholecystectomy lead to new guidelines. Although several studies have shown a lower morbidity and mortality in delayed operations, data from some of these studies is interpreted without regard to patient stratification. [11.78] Cholecystectomy in GSP has now been called in mild disease for several decades, but what defines the beginning of synchronization, and the challenge of patient stratification, led to a great discussion.

Published recommendations and references at the beginning of mild pancreatitis cholecystectomy range from less than 48 hours at 2 to 4 weeks of presentation. [11,58,72,79,80] Most of the surgical literature, however, during cholecystectomy even recommends admission to hospital. While many surgeons wait for the resolution of abdominal pain and standardization of pancreatic enzymes, Aboulian and others [81] found that laparoscopic cholecystectomy performed within 48 hours of admission for mild pancreatitis, regardless of the pain or laboratory values, results in a shorter length of hospital stay without compromising patient safety or unjustified difficult technical capacity of the surgeon. [82] Several other studies support the implementation of laparoscopic cholecystectomy within 48 hours of admission in mild cases, and many others advocate early cholecystectomy within the same admission to hospital. [83,84] It is a common practice for surgeons to wait for laboratory values to normalize. However, there are data demonstrating a shorter length of hospital stay without increasing morbidity when surgery is performed as laboratory values begin to trend towards normal. [85] Waiting for complete normalization of pancreatic enzymes may result in delayed care and an increase in the length of stay. But there are good reasons to recommend a cholecystectomy following idiopathic acute pancreatitis, many patients may have biliary sludge or microlithiasis undocumented. This view is supported by evidence that the biliary microlithiasis and sludge are also responsible for the pathological process mentioned in GSP. [86,87]

Thus, for purposes of this review, all acute pancreatitis caused by gallstones, microlithiasis and biliary sludge are called GSP. Despite the evidence in support of cholecystectomy, compliance is low and many patients are sent for interval cholecystectomy. In the Western world, the rate index for cholecystectomy surgery is appropriate between 10% and 60%. [88-94] Factors associated with patients who are not subject to early cholecystectomy in the US include age, black race, admission to a non-surgical services, comorbidity, and the absence of a surgical consultation. [94] Access to medical care in some populations may play a role. [95] Surveys of surgeons who do not exercise early cholecystectomy cite reasons such as the occupation of operating rooms, budget concerns, lack resources, and care for a difficult dissection. [90,96] In contrast to these concerns, the feasibility of cholecystectomy during admission and the Index, the most in two weeks, has been studied and found to be neutral from the standpoint of cost and practical. [97] In addition, surgeons involved in a related study reportedthat the dissection to be more difficult in late, rather than early, laparoscopic cholecystectomy. [98] Of the note is a study with atypical results of a busy public hospital. Clarke and others [99] reported that running cholecystectomies index put excessive strain on hospital resources, and length of stay was actually higher in hospitalized patients awaiting index cholecystectomy in patients directed towards the discharge and admission for elective surgery. Morbidity was similar in both groups, but 6.5% of patients in the interval laparoscopic cholecystectomy group had unplanned readmissions for benign recurrent pancreatitis.Severe GSP is associated with significant morbidity and mortality significantly higher, and the disease process is like that the surgical management follows a more conservative course. Much of the morbidity and mortality in early studies that warnedagainst early cholecystectomy is attributable to patients with severe forms of GSP. In one of these studies, Ranson [14] excluded patients with mild pancreatic edema and considered patients who underwent operation with pancreatic inflammation in addition to the fat necrosis or pancreas, hemorrhage and reported a high mortality rate. Other studies have reported similar results where high rates of morbidity and mortality in patients undergoing early cholecystectomy were attributable to patients with moderate and severe disease. [100] Once a patient is stratified as moderate or severe GSP, the initial management is to benefit from support services and management of complications. Follow-up care includes cholecystectomy, interval delayed at least three weeks after the resolution, if clinical circumstances allow. Early cholecystectomy is against-indicated in moderate and severe of the GSP, and is associated with increased infectious complications and sepsis. [101]

Nealon and his colleagues [103] reported that among the patients moderate to severe GSP undergoing early cholecystectomy, regardless of the CT-proven peripancreatic fluid collections, 63% required reoperation and 44% had postoperative complications. Most reoperations were for the definitive management of pseudocysts, and infectious complications were probably a result of pseudocysts which were sterile but were infected at the time of early cholecystectomy. The researchers therefore recommend delaying jusqu'cholécystectomie.It is possible to manage all of the gallbladder and pseudocysts simultaneously. Under the exclusion, almost all researchers advocating early cholecystectomy patients with mild disease specify, and warn against using too soon on moderate or severe GSP. Pseudocysts occur in acute pancreatitis due to the disruption of pancreatic duct and pancreatic excretory extravasated liquid. A collection of fluid may or may not communicate with the pancreatic duct, and finally forms a fibrous wall around the collection [Fig. 4]. Pseudocysts can be properly diagnosed with CT or MRI contrast. A general rule is to wait for six weeks before intervening to allow the pseudocyst wall to mature. [103 104] Exceptions to this rule are cases of symptomatic infection or pseudocysts, when intervention may be indicated. However, there is no universally accepted guidelines for post-GSP pseudocyst management, and a comprehensive analysis of pseudocyst management is beyond the scope of this. Generally, management is conservative, because many of these pseudocysts resolve spontaneously, especially if there are no permeability between the pseudocyst and the pancreatic duct. Because patients with GSP peripancreatic fluid collections or pseudocysts are often referred to cholecystectomy, appropriate monitoring interval is essential.Management interventional pseudocysts with walls most often gastric or maturity includes proximal enteric drainage or open endoscopic techniques. Several studies have demonstrated the resolution rate comparable both technical pseudocyst, and it favors the endoscopic method in single pseudocysts accessible from gastric or duodenal ring lumens. [105-110] Internal surgical drainage may be indicated. In complex pseudocysts and those

that are not easily accessible by endoscopy. Use of Sue can improve viewing and make it inaccessible, manageable pseudocysts with endoscopic drainage. [105,111] It has a role of percutaneous drainage in infected pseudocysts and symptomatic cases and endoscopic surgery where options are limited. However, this technique may create a fistula, pancreaticocutaneous which may persist for an extended period. [108, 112]

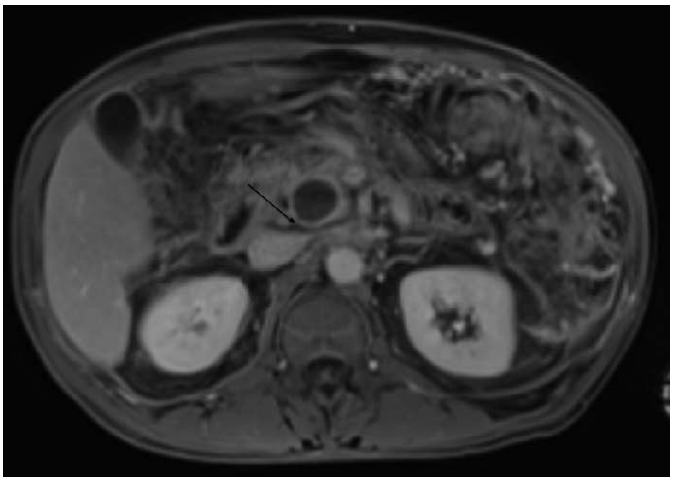

Fig. 4. Although circumscribed in the head of pancreas pseudocyst[arrow] to the mechanical origin The obstruction of the CBD

In high risk patients, it may be necessary to use other means for decompressing the biliary system. Elderly, comorbidity, and / or critically ill patients can be considered unsuitable candidates for surgery or ERCP, yet still need emergent management of obstruction of the common bile duct stones causing septicemia or aggravate acute pancreatitis. In these patients, interventional radiologists can perform percutaneous Cholecystostomy, often through the use of ultrasound and fluoroscopy. This procedure uses the Seldinger technique, and is a minimally invasive method of decompression of the biliary system. However, patients who require this degree of interventional minimalism tend to have a poor prognosis, and the 30-day mortality was reported to be as high as 15.4% in patients undergoing percutaneous cholecystostomy for acute cholecystitis. [113]

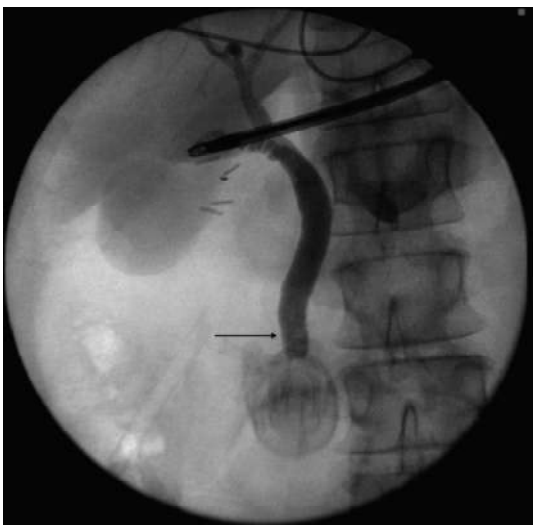

Fig.5. Demonstration of lucency irregular intraoperative cholangiogram in the CBD[Arrow], which represents a stone

Intraoperative cholangiography

The role of intraoperative cholangiography [IOC] in GSP is controversial, and its use varies considerably between surgeons. [114] Some surgeons regularly practice IOC, while others do so only when there is a strong suspicion of a stone in the common channel [Fig. 5]. Although the IOC is reported to be 94% sensitive and 98% specific for gallstones, a study found that air bubbles in ducts can mimic stone in appearance and routine use may be associated a false positive rate significant. [115] Many surgeons perform a CIO if indirect evidence of choledocholithiasis, such as an obstructive pattern on BFH or a relatively large common channel ultrasound. The diagnosis of the GSP should be considered as poor indirect evidence of a stone canal, as most stones causing acute pancreatitis pass into the duodenum spontaneously. However, the diagnosis of the GSP is associated with an increase of the use of the IOC. [114] Johnson and Walsh [116] demonstrated that patients undergoing GSP during a cholecystectomy IOC were more likely to ERCP or postoperative exploration of the CBD during surgery, but without influencing the result of pancreatitis. This is borne out in the literature, as a recent systematic review of the use of the IOC did not identify sufficient evidence to show benefit. [117]

Laparoscopic exploration of the common bile

Gallstones passing through the common channel are the agents involved in the GSP, and although most pass into the duodenum spontaneously without incident, 7% to 28% of the time the stones may remain in the common channel. [21 118-121]Given sufficient evidence for bile mild pancreatitis coexisting, it is safe and effective to remove the stone at the time of laparoscopic cholecystectomy CBD exploration [LCBDE]. [122]Hit rocks causing cholangitis or aggravating GSP serious are usually removed emergently ERCP, discussed in the next section. LCBDE has been used for more than 2 decades and the technology available to effectively deliver safe and the procedure has evolved considerably, helping to make it as efficient as ERCP in a few hands. [123-125] However, due to risks related to the handling of the CBD and the exceptional level of skill required, most surgeons do not perform this procedure. LCBDE is most commonly performed by surgeons with other conditions or laparoscopic training. The transcystic method is the preferred approach among most surgeons who perform LCBDE, and is most suitable for small stones in a small common channel. Choledochotomy is reported to be better for larger, more stones in a dilated common channel. Although technically more difficult by LCDBE choledochotomy may be a more definitive approach. [116, 123, 126] The stone hit game LCBDE has been reported in several studies to be equivalent to ERCP, it decreased morbidity, cost, and shortenedlength of stay. [127-131] When LCBDE is not an option and patients are either diagnosed or suspected of having led stones, ERCP is favored in most cases, more open exploration of the CBD.

Endoscopic Retrograde Cholangiopancreatography

ERCP refers to the contrast imaging of the bile and pancreatic vesicle cholangiopancreatography[conduits] using a side to the endoscope display and fluoroscopy. When the papilla of Vater is available this way and CBD cannulated with a guide wire, the endoscopist can then perform an endoscopic balloon dilation or sphincterotomy biliary Sphincter followed by

extraction of stones using a balloon or basket for stones that do not pass spontaneously [Fig. 6]. Especially for large stones, there are devices available for performing intraluminal lithotripsy to help in extraction. For purposes of diagnosis, the sensitivity of ERCP for choledocholithiasis is 90% to 97%, with 95% to 100% specificity. [132] This diagnostic performance is similar to that of MRCP, which is non-invasive. The ERCP success rate in stone extraction is around 95%. ERCP has been available for over 30 years, and has largely replaced surgical exploration of the CBD in the isolated cases of bile. The role of ERCP in GSPwas discussed lengthily, and it is widely accepted that in mild cases of GSP without evidence of biliary obstruction or cholestasis there is no utility of ERCP for diagnostic or therapeutic purposes. [133,135] Most patients with high concentrations of pancreatic enzymes transiently without an increase in bilirubin can go forward in early cholecystectomy without preoperative or postoperative ERCP. Although previous reports called ERCP within 24 hours for all applicants with GSP, this strategy has been tremendously contested. [136] It is now widely accepted evidence that ERCP in patients with GSP, but without cholestasis or cholangitis, confers no advantage in terms of complications or mortality. [137,138]In patients with GSP and evidence of choledocholithiasis, including increased liver function, elevated bilirubin persistent, persistent pain, or viewing on MRCP, performing ERCP within 72 hours reduces the rate of sepsis, mortality and complications, including pancreatic necrosis. [134 139-142] In all patients with GSP, other common stones evidence leads justifying intervention includes gastric aspirate without bile and increasing the number of bilirubin level. Performing ERCP within 48 hours in these patients can reduce morbidity. [143] Acute cholangitis may complicate GSP in up to 10% of cases, and early ERCP is indicated to decompress the biliary system in these patients. [58]ERCP plays a role in soft GSP in patients who are unfit or unwilling to undergo surgery.

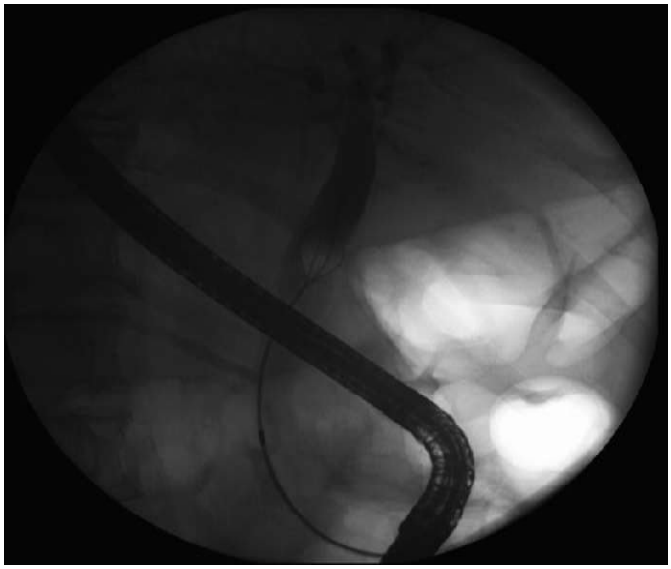

Fig. 6. fluoroscopic image of retrograde cholangiopancreatography for The balloon extraction a large stone

Although it is well-established that patients with GSP have a high recurrence rate without cholecystectomy, ERCP with sphincterotomy is a protection against recurrence of acute pancreatitis. However, since the bladder is left in place, the rate of acute cholecystitis and biliary colic remain high. [2,72,144,145] ERCP is indicated in patients with GSP who can not undergo cholecystectomy, or have a prolonged delay before cholecystectomy. The complications of ERCP. Pancreatitis, bleeding, perforation, cholangitis, and stenosis of the sphincter of Oddi [127, 146] Although increased risk of cholangiocarcinoma after ERCP has been discussed in the literature, there is currently insufficient evidence to support this concern.

Conclusion

GSP is a disease with a wide spectrum of severity. Diagnosis and management have evolved in the recent decades with the emergence of new and improved technology. Advancements in imaging techniques have limited the need for diagnostic procedures in many cases, and various treatment options are becoming more widely available. The paradigm continues to shift towards an operation earlier in mild cases, with more judicious interventions in severe disease. Risk stratification is essential to provide the best possible care for all patients, and clinical judgment is paramount in selecting the most relevant species and invasive diagnostic procedures at the most appropriate moments.

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