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REVIEW ARTICLE **OPEN ACCESS**

Acute Pancreatitis and Its Management

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ABSTRACT
For the past ten years, there is a significant advancement in the understanding of pathophysiology and natural history of acute pancreatitis. AP affects between 15.9 and 36.4 per 100,000 persons. AP has high mortality and morbidity rate. Etiology includes biliary tract disease, Alcohol consumption, hyper triglyceridemia, genetics, infection which includes bacterial, parasitic, viral and fungal infection, Post endoscopic retrograde cholangio pancreatography (ERCP) and 10-40% of all the cases are idiopathic or unidentifiable. The pain occurs specifically in mid epigastrium region of abdomen, it may be localized or generalized. The back pain experienced by patients can sometimes be relieved by sitting or bending forward.[1]Diagnosis is done on the basis of history collection, physical examination, laboratory investigation, CT scan, MRI, various tools to assess severity of disease which includes acute physiology and chronic health examination (APACHE) II score, Bedside index of severity in acute pancreatitis (BISAP) score, Glasgow criteria and Ranson's criteria. Management include fluid therapy, adequate nutrition, essential therapies and pain management.[2]Necrosectomy is indicated for patients with symptoms. Cholecystectomy is performed on patients with mild AP brought on by gallstones. Acute necrotic collection, walled off necrosis, pseudo aneurysm, thrombosis and abdominal compartment syndrome.
KEY WORDS: *AP- Acute Pancreatitis, CRP- C-reactive protein, APACHE- Acute physiology and chronic health examination.*

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INTRODUCTION

Over the years, there has been debate over the best way to treat acute pancreatitis, with options ranging from a cautious medicinal strategy to an aggressive surgical approach. For the past ten years, there is a significant advancement in the understanding of the pathophysiology and natural history of acute pancreatitis. Acute pancreatitis can have a mild, transitory form or a severe, necrotizing disease as its clinical course [1, 2]. The majority of acute pancreatitis episodes (80%) are mild and self-limiting, going away on their own in 3-5 days. Mild pancreatitis patients react adequately to medical care and just need intravenous fluid resuscitation and analgesics.Severe pancreatitis, on the other hand, is characterized as having pancreatitis coupled with local problems such necrosis, abscess formation, or pseudocysts, as well as organ failure. 15-20% of cases of pancreatitis might be considered severe [3].

Epidemiology

AP affects between 15.9 and 36.4 per 100,000 persons. In the near future, it is anticipated that the disease will have a greater impact on the utilization of healthcare resources are used [7]. Despite the advancements in healthcare access, imaging technologies, and therapies, AP still has a high rate of mortality and morbidity that remains significantly constant over time. The total death rate ranges from 5% to 17% for severe AP and 1.5% for mild AP.[4]There are three most important causes associated to AP which are idiopathic, alcohol-related, and gallstone/biliary-related. The majority of AP cases are caused by one of these three factors. According to estimates, between 28 and 38 percent of cases involved biliary pathology, whereas between 19- 41 percent involved alcohol [4].

Etiology

Biliary Tract Disease: According to estimates, gallstone pancreatitis accounts from 28- 38 percent of all instances of AP. Migration of gallstone leads to blockage of ducts which causes temporary impaction of the migrated stone at ampulla of duodenal, increases the pressure of duct and unregulated stimulation of the enzymes which are secreted from pancreas. The blockage may be caused by spasms, stuck calculi inside the duodenal ampulla, as well as sphincter of Oddi fibrosis [5].

Alcohol: Alcoholic pancreatitis, which is thought to account for 19 percent to 41 percent of all instances of AP, is the second most common cause of the condition. Although the link between alcohol misuse and pancreatitis is not well established, all the patients who abuse alcohol does not develop pancreatitis.[8]

Hyper Triglyceridemia: A rare cause of AP is hyper triglyceridemia-induced pancreatitis. It account for 1 percent to 4 percent of cases. The excessive breakdown of lipoproteins which is rich in triglyceride, which releases a high concentration of free fatty acid and damages the vascular endothelium and acinar cells of the pancreas, is thought to be the cause of hypertriglyceridemia-induced pancreatitis [5].
Genetic: The development of AP is associated with several mutations of genes.
Infection: AP has been linked to a number of infections which include bacterial, parasitic, viral and fungal.
Post Endoscopic Retrograde Cholangio Pancreatography (ERCP): It has been documented that 24 hours after an ERCP, there is three times increase in serum amylase level. Same thing has been reported in 1.3- 4.3 percent of ERCP procedures. Younger age, female gender, pancreatic duct opacification, cholangitis, sphincter of oddi dysfunction and duodenal perforation are the most typical risk factors for post-ERCP related AP [5].
Idiopathic: 10- 40% of all the cases are Idiopathic or unidentifiable.

SYMPTOMS

Symptoms of AP include dull gnawing pain, nearly all patients suffering from acute pancreatitis first experience severe upper abdominal discomfort. The discomfort is typically intense and ongoing. The mid-epigastrium-specific pain may be localized or generalized throughout the abdomen. The back pain experienced by about half of patients can sometimes be eased by sitting or bending forward. Other symptoms include board like abdomen, guarding, fever, rapid pulse, grey turner sign, Cullen sign, nausea and vomiting [6].

DIAGNOSIS

Inaccurate and inconsistently recognized definitions of the disease's severity and frequently occurring AP complications make it challenging to assess the disease in some cases. Acute pancreatitis is clinically diagnosed using the symptoms present in patient, a physical examination, laboratory results, and imaging findings. Practice guidelines from 2006 state that two of the following three main characteristics must be present in order to make the diagnosis: (1) pain in abdomen (2) serum amylase and lipase levels greater or approximately equal to three times the upper normal limit; and (3) Presence of AP on computed tomography (CT) scans.

Physical examination

An inflammatory disorder of the pancreas, pancreatitis can affect nearby and far-off extra-pancreatic tissues. The results are related to the severity of disease. On palpation patients having less severe disease are present with tenderness in abdomen whereas patients with severe disease are present with extreme abdominal pain and absence of bowel sounds.

Laboratory Test

Serum amylase and/or lipase levels greater or approximately equal to three times the upper normal limit. Trypsinogen activation peptide, which is also included in AP, is released from trypsinogen to form active trypsin. Both the serum and urine can be used to measure this. These tests are not frequently utilized in clinical practice since they are not easily accessible. As part of their initial laboratory work-up, every patient with AP should be tested for complete blood count, liver function test, basic metabolic panel test, coagulation profile, C-reactive protein, and total albumin. Patients with hypoxia should have an arterial blood gas test.

Radiological Investigation

The best imaging modalities for displaying pancreatic pathology are contrast-enhanced CT scan and magnetic resonance imaging of whole abdomen. Although individuals with moderate AP are not typically advised to have these tests. The appearance of localized or widespread amplification of the pancreas is the hallmark sign of AP.

Assessment of Severity

Various tools are developed to assess the severity of disease.

Acute physiology and chronic health examination (APACHE) II SCORE

The APACHE II score was initially created for individuals admitted in intensive care unit and makes use of 12 variables which provide a score that is utilized at the time of admission, 24 hours after admission, and 48 hours after admission. It has the benefit as it allows for appropriate changes and interventions as the score is computed throughout the patient's stay. In order to stratify a patient's risks, each of the variables is converted into weight by using the original score [10].

Bedside Index of Severity in Acute Pancreatitis (BISAP) Score

It is a mortality-based tool used by physicians to utilize it within the first 24 hours of admission, this score was created in 2008. Five factors are included in the score system: Blood urea nitrogen> 25 mg/dL, impairment of mental status, age more than 60 years, or the occurrence of pleural effusion. Death rate was found to be more than 20% in the group at the highest risk (score of 5) and less than 1% in the individuals at lowest risk (score of 0).

Glasgow Criteria

Eight of the factors included in the Ranson's Criteria are part of this scale. This scale has been applied to AP caused by gallstones.

Ranson's Criteria

It is the first standards for determining the seriousness of AP was Ranson's criteria. The score is based on 11 characteristics, out of which 5 are assessed upon admission and 6 of which are assessed 48 hours later. This scoring system has a drawback that patients detail should be taken properly and at appropriate time and its result are not obtained for 48 hours following admission [14].

Management

Adequate nutrition, essential therapies, vigorous intravenous hydration, and pain management are the fundamentals of AP management. The most recent and available AP treatment options are mentioned here.

Fluid Therapy

After Six and 24 hours of admission, the need for vigorous fluid resuscitation should be assessed, and the rate of fluid administration should be modified in reaction to variations in mean arterial pressure, urinary output, changes in Blood Urea Nitrogen, and respiratory condition. The first treatment of AP is mainly supportive, focusing on optimizing electrolyte balance, replacing lost fluids, providing enough calories, and preventing or recognizing and treating any local or systemic consequences. Fluid selection for AP patients has generated a lot of discussion in recent times. One randomized trial revealed that Ringer's Lactate reduces the rate of systemic inflammation as compared to Normal Saline, supporting previous recommendations that Ringer's Lactate is used as the fluid resuscitation therapy in patients with AP [15].

Pain Management

Treatment of the extreme pain in acute pancreatitis requires effective opiate analgesic. For effective pain management, patient-controlled analgesia is frequently helpful.

Nutrition

As opposed to the traditional nil per oral techniques was utilized in the past, now the concept of nutrition in AP has changed to early beginning of nutritional supplementation. In patients with mild AP, early oral feedings (within 24 hours) are started [15]. These recommendations did not specify a particular diet type, but it is better to start early feedings as it serves to safeguard the gut-mucosal barrier and decreases the migration of bacteria, which minimize the chance of more adverse results [10].

Antibiotics

Antimicrobial use in acute pancreatitis has been controversial, much like the discussion around nutrition in this condition. Gram-positive bacteria, anaerobes, and fungi also have been identified using cultures of infected pancreatic necrosis, along with gram-negative aerobic bacteria and anaerobes. In cases of acute pancreatitis, penicillin's, amino glycosides, first-generation cephalosporin's, and tetracycline's remain ineffective [10]. Infected pancreatic necrosis can be effectively treated using antibiotics which are efficient to treat Gram-negative bacteria, such as imipenem, piperacillin, clindamycin, quinolones, and flagyl. Although usage of imipenem dramatically decreased the frequency of infected pancreatic necrosis, carbapenems are linked with a considerable reduction in mortality when compared to other intravenous antibiotics [11].

Surgical Management

Cholelithiasis and infected necrosis that has persisted for at least 4 weeks after receiving antibiotics and necrosectomy in patients with symptoms are the indications for surgery [12]. Cholecystectomy should be performed on all person with mild AP brought on by gallstones. The presence of pseudocysts, necrosis of the pancreas, or extra pancreatic tissue in asymptomatic patients does not necessitate surgical intervention.[14] According to current recommendations; stable patients should wait four weeks before having a necrosectomy. In individuals with severe AP, it has been demonstrated that delaying surgery is related with a death rate reduction from 39 percent to 12 percent. In contrast to surgery, minimally invasive techniques like endoscopic necrosectomy are still advised for symptomatic patients with infected necrosis [13].

Complications

There are various complication of AP which includes Acute Necrotic Collection, walled off necrosis, thrombosis, pseudo aneurysm and abdominal compartment syndrome.[9]

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