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Cholelithiasis – A major cause of Acute Cholecystitis

Amna Khalid[1], Dr Fabiha Moeen[2] and Dr. Muhammad Awais[3]
[1]Woman Medical Officer at Pakistan Kidney and Liver institute and research center Lahore
Email: aamnazariab91@gmail.com
[2]Woman Medical Officer at Pakistan Kidney and Liver institute and research center Lahore
Email: fabiha_moeen@hotmail.com
[3]Designation : Medical Officer at Basic Health Unit 221 J/B District Chiniot, Punjab
Email: mowais299@gmail.com

Abstract

Acute cholecystitis means the inflammation of gall bladder. Gall stones is the most highly recognized cause of acute cholecsytitis which may or may not be complicated with super added bacterial infection. Gall stones are present in nearly 10% of the population[1][2]. Nearly 80% people are asymptomatic[3]. However, 1-3% develop acute cholecystitis due to these gall stones. It presents with fever, right upper quadrant pain and tenderness. This condition can easily be diagnosed with ultrasound abdomen. Occasionally HIDA scan may be needed when there is diagnostic uncertainity. Acute cholecystitis is managed conservatively to allow the inflammation to settle after which cholecsytectomy is performed however, early cholecsytectomy is also performed.

Key words: Cholecystectomy; Cholelithiasis; Cholecystitis; gall stones.

Method:

This review was prepared by searching medline for various articles using the key word " acute cholecystitis".

Anatomy of Gall Bladder and Billiary system:

It is a pear shaped organ present just under the liver having a total size of 8-12 cm and capacity of nearly 30 ml. It is anatomically divided into fundus, body and neck. The neck terminates into the infundibulum. The muscle fibers in the wall of gall bladder are arranged in cris-cross manner and inside the gallbladder there are indentations of mucosa called Crypts of Luschka. The cystic duct is 3 cm in length having a diameter of 1-3 mm . It connects the infundibulum of gall bladder with the common hepatic duct. However, this is not always the case. In certain individuals the cystic duct can open in right hepatic duct.

The common bile duct is formed by the union of common hepatic duct and cystic duct. It has total length of 8-9 cm and is anatomically divided into 4 parts supraduodenal, retroduodenal, indraduodenal and intraduodenal portions. The common bile duct opens into second part of duodenum through ampulla of vater.

Bile is produced by liver and stored in the gall bladder from where it is released into the duodenum. Bile is composed mainly of cholic acid, chenodeoxycholic acid, deoycholic acid and lithocholic acid.

Pathogenesis:

Cholesterol is solubilised in bile by aggregation with water soluble bile salts and lecithins. An adequate proportion of cholesterol and bile salts, lecithins is necessary to keep the cholesterol soluble in the bile otherwise it may precipitate out. When the bile becomes supersaturated with the cholesterol followed by hypomobility of gall bladder the nucleation of cholesterol in the bile is started.[4]

Pigment stones on the other hand are a mixture of insoluble calcium salts of unconjugated bilirubin and inorganic calcium salts. Hemolytic diseases, ileal resection and bacterial infection of billiary tract increase the risk of pigment stone formation.

Gall stones are the major cause of acute cholecystitis in nearly 90% cases. Acute cholecystitis is the inflammation of gall bladder and it is mostly caused by impaction of gall stone in the cystic duct or infuldibulum.

1. The mucosal phospholipases convert lecithins in the lumen to lysolecithins.
2. The protective layer comprised of glycoprotein is disrupted and it eposes the mucosal epithelium to the action of bile salts which act as detergents.
3. The wall of inflamed gall bladder releases prostaglandins[5] and it contributes to development of gall bladder dysmotility.
4. Distention and increased intraluminal pressure reduce the blood flow to the mucosa further aggravating the inflammation.
5. Secondary bacterial infection by pathogens in the gut lead to Acute bacterial cholecystitis.

Presentation:

Acute cholecystitis typically presents with nausea, vomiting ,severe right upper quadrant pain

radiating to right shoulder and fever. However it can also present with epigastric pain. The signs and symptoms are categorized as Local signs of inflammation and systemic signs of inflammation.

Local signs of inflammation include Right upper quadrant pain or tenderness and positive murphys' sign. A hand placed at right upper quadrant patient and asking the patient to take breath. The arrest of breathing due to the placement of hand in right upper quadrant is termed as positive Murphys' sign.

Systemic signs of infection include fever which may be associated with rigors and chills, raised white cell count, ESR and/or CRP. Bilirubin may be raised in due to inflammation and edema of billiary tree. However marked elevation of bilirubin more than 60 mmol/L is should make the clinicians diagnosis of choledocolithiasis.

One of the important differential of acute cholecystitis is Billiary colic. In billiary colic, there is transient and colicky pain in the right upper quadrant preferably after meals but without murphys' sign and fever. Ascending cholangitis , infection of common bile duct presents with fever, right upper quadrant pain and jaundice known as charcots' triad.

Investigations and Diagnosis:

Investigations for a suspected case of acute cholecystitis include those required for detecting extent of systemic involvement, ruling out alternative diagnosis and definitive imaging.

Complete blood count , ESR and CRP are usually routinely ordered. CBC may show a raised leukocyte count and specifically neutrophilia. Serum electrolytes may be deranged due to continuous vomiting which might be associated with acute cholecystitis. Serum bilirubin might help in ruling out choledocolithiasis and ascending cholangitis.

The gold standard investigation in acute cholecystitis is Ultrasound abdomen[6]. It is inexpensive, readily available, and quick to perform. It has more than 95% sensitivity for detecting gall stones. In case of acute cholecystitis, ultrasound may show presence of pericholecystic fluid, thickness of gall bladder wall and overall increase in size of gall bladder and any impacted gall stone in the gall bladder neck.

If diagnosis remains in doubt after transabdominal ultrasound then Biliary scintigraphy (hydroxyiminodiacetic acid (HIDA) scan) is performed.Technetium99-m labeled derivatives of iminodiacetic acid are taken up by liver and excreted into gall bladder. In normal individuals , gallbladder is seen within 30 minutes in 90% of individuals whereas it is visualized in in one hour in almost all. Non visualization of gall bladder is diagnostic for acute cholecystitis.

Management:

Most of the patients respond very well to medical management[8]. This includes resting the gall bladder by making the patient nill per oral (NPO), giving adequate fluids and analgesia. NSAIDs such as indomethacin can reverse the inflammation of gall bladder and and contractile ability[7]. There is always a risk of superimposed infection in patients of acute cholecystitis hence empirical antibiotics are started in acute cholecystitis. Antibiotic choices include 2nd and 3rd generation cephalosporins and metronidazole.

This reduces the inflammation of gall bladder and any impacted stone in neck is pushed back into the gall bladder lumen and cystic duct empties. Only 20% patients of acute cholecystitis need immediate surgery. Conservative treatment should be abandoned if patients symptoms worsen despite the resuscitation, detection of gas in billiary tree, peritonitis, intestinal obstruction and gangrenous gall bladder. In such cases emergency cholecystectomy should be performed.

The treatment of choice for acute cholecystitis is Cholecystectomy however there is debate regarding timing of cholecystectomy[9]. Early cholecystectomy means cholecystectomy within 2-3 days of illness in the same admission. However, Interval cholecystectomy is done 6-10 weeks after the acute episode has settled. This lets the inflammation to settle completely and surgery can be performed conveniently.

Complications of Acute Cholecystitis:

1. Multiple risk factors have been identified in the development of gangrenous gall bladder. Presence of heart disease was the most frequently associated risk factor with the gangrenous cholecystitis. A study conducted by Akin Onder showed 24.3 percent patients having gangrenous cholecystitis were having cardiovascular disease[10]. In addition prolonged delay prior to hospital admission , low white cell count , pericholcystic fluid were other factors contributing to gangrenous cholecystitis.
2. Acute inflamed gall bladder can form billiary enteric fistula.Most commonly at the duodenum but also at the colon. A study conducted in the New york Hosital- Connell medical center showed that 77% of billiary enteric fistulas were

cholecystoduodenal whereas 15% were cholecystocolic[11].

3. Gall stone ileus is a sequlae of cholecystoenteric fistula. Gall stones can obstruct the small intestine and presents as acute intestinal obstruction. A significant mortality risk of 15-20 is associated with it[12]

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