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Gangrenous Cholecystitis – A Case Report

Mukul Kumar[1], Arun Agarwalla[2], Rajeev Ranjan[3]
[1,2]*Pgt-3rd year; NMCH,Sasaram, Bihar, India*
[3]*Associate professor; NMCH, Sasaram, Bihar, India*

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I. INTRODUCTION

Gangrenous cholecystitis (GC) is defined as necrosis and perforation of the gallbladder wall as a result of ischemia following progressive vascular insufficiency[1,2]. Acute cholecystitis develops in 1% to 2% of patients with asymptomatic cholelithiasis each year. Conservative treatment comprising intravenous fluid resuscitation and antibiotic therapy proves effective in 80% of patients with acute cholecystitis. As one of the severe complications of acute cholecystitis, GC develops in 2% to 20% of the cases with acute cholecystitis. Epithelial injury by increasing gallbladder wall tension owing to vascular insufficiency arising secondary to persistent obstruction of the cystic duct gives way to the development of GC, which follows a quite rapid course.5 The phospholipases released from cell membranes of damaged epithelium initiate heavy inflammatory reaction. Inflammation and ischemia of the gallbladder wall show progressive worsening as a result of deteriorating venous insufficiency with age, thereby giving rise to more necrosis and perforation. Although 73.8% of the cases in our study were aged 51 years and older, age did not influence mortality. GC is a severe complication of cholelithiasis.[3]

II. CASE REPORT

A 55 yrs, male, a resident of Eastern India came to the OPD of a Tertiary Care Centre with complaints of Pain Abdomen since 18 days, Nausea since 18 days, Itching over the whole body since last 7 days. Patient was apparently asymptomatic 18 days back when he experienced severe abdominal pain which was sudden in onset, rapidly progressive and colicky in nature. Pain was aggravated on having food and relieved by taking medication. Pain was associated with nausea . He was a known case of hypertension on regular medication since last 11 yrs, Newly diagnosed case of T2DM 1 week back and he had CVA 11yrs back. He had a habit of tobacco chewing since last 20 yrs. His BP was 90/60 mmHg, and pulse was – 64 bpm, and was having mild icterus on examination, Liver function tests were raised; Total Bilirubin – 3.0mg/dl, Direct Bilirubin – 0.6 mg/dl, Indirect Bilirubin – 2.4 mg/dl , ALP - 104 IU/L, SGOT – 32.9 IU/L, SGPT- 93.5 IU/L , the Total Leukocyte count was 16,900/mm3. Ultrasonography showed a gallbladder containing multiple calculi with sludge. The patient was managed initially with IV fluids, Antibiotics, Antiemetics and Analgesics; Elective laparoscopic cholecystectomy was planned. Intra-operatively, the entire fundus of the gallbladder was wrapped with omentum, Calot's triangle was frozen, the gallbladder wall was inflamed and distended as shown in the Fig.-1. Calot's dissection was done meticulously and gall bladder was extracted via an endobag. The whole Gall Bladder was gangrenous as shown in the Fig.-2 and was on the verge of perforation. Postoperatively the patient was kept on intravenous antibiotics and analgesics. The histopathological report of the gallbladder showed gangrenous cholecystitis. The hospital stay was uneventful and the patient was discharged on oral medication on the 4th postoperative day.

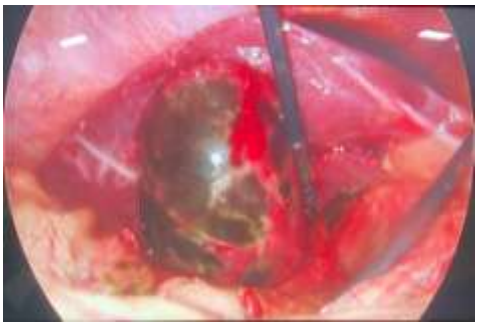

Fig 1 showing intra operative picture with distended and gangrenous Gall Bladder

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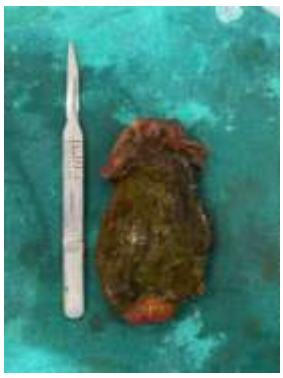

Fig 2 showing GB specimen post operatively with whole of the gangrenous Gall Bladder

III. DISCUSSION

GC is the last stage of gall bladder inflammation[4] and, in spite of its grave prognosis, its diagnosis can be elusive, both clinically and on laboratory investigation. The incidence of GC ranges from 2% to 29.6% in all patients with acute cholecystitis, in various surgical series, and generally occurs in older patients[5]. Many factors have been implicated in its formation. Fagan et al[6] demonstrated that nine variables were associated with GC, but Contini et al[4], showed that there is no single clinical or laboratory finding, apart from a high WCC, predictive of severe inflammation of the gallbladder.

Contini et al showed that the time of hospitalization delay plays a crucial role in the formation of GC. The time between the onset of symptoms and hospital admission was significantly longer in patients with GC. The patient's history (timely or delayed admission) and physician's attitude (general practitioner and/or surgeon) are likely to play a role in the progression towards a severe necrosis of the gallbladder wall.

Ultrasonography usually serves as the first-line imaging modality for the evaluation of patients with clinically suspected acute cholecystitis. However, CT can play an important role in evaluation of these patients if sonography is inconclusive[5]. The hallmark on sonography of GC is the presence of heterogeneous or striated thickening of the gallbladder wall, which is often irregular with projections into the lumen and pericholecystic fluid collections. The presence of intra-luminal membranes representing desquamative gallbladder mucosa is a specific finding but it is less common. The accuracy of pre-operative ultrasound in diagnosing GC remains uncertain. Twenty-eight percent of patients with GC had ultrasound reports that failed to show any evidence of acute inflammation. This was mainly due to the absence of sonographic Murphy's sign and gallbladder walls of less than 3 mm, both important radiological signs of acute inflammation of the gall bladder. The CT findings most specific for acute GC are gas in the wall or lumen, intra-luminal membranes, an irregular wall and pericholecystic abscess. GC is associated with a lack of mural enhancement, pericholecystic fluid and a greater degree of gallbladder distension and wall thickening.

There is a controversy regarding the best surgical approach to GC with some authors, such as Eldar et al[7] recommending open cholecystectomy for most men over 60 years of age who have significant co-morbidity, large bile stones and elevated bilirubin level. In contrast, Hunt and Chu[7] indicated that laparoscopic cholecystectomy can be used relatively safely and successfully in patients with gangrenous cholecystitis, reporting a success rate of 91% without increased morbidity and no mortality. Others suggested that a more reasonable approach would be an initial examination with the laparoscope, not wasting more than a few minutes to determine whether a dissection would be possible. In the hands of an experienced laparoscopic surgeon, an initial attempt at laparoscopic cholecystectomy is possible, converting to open procedure if necessary. A conversion rate higher than that for simple acute cholecystitis or symptomatic cholelithiasis is to be expected. However, when successful, laparoscopic cholecystectomy is associated with a significantly better outcome and a shorter hospital stay; the conversion rates range from 8% to 75%.

GC has a mortality rate of up to 22% and a complication rate of 16-25%. Complications associated with GC include perforation, which has been reported to occur in as many as 10% of cases of acute cholecystitis.

IV. CONCLUSION

When dealing with patients with acute cholecystitis, a high index of suspicion is essential for the early diagnosis and treatment of GC. The possibility of a patient, especially an elderly patient with acute cholecystitis, progressing to GC should always considered, even in an apparently improving patient and in spite of the absence of any firm clinical or laboratory findings. The radiological investigations may not be conclusive. There is a need for an early (if not urgent) surgical intervention in acute cholecystitis (whether laparoscopic or open surgery) in order to decrease the time elapsed from the start of symptoms to admission and treatment.

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