Original Research Paper

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General Surgery

CASE REPORT OF GALL BLADDER CARCINOMA WITH COMPLEX PERFORATED GALL BLADDER

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ABSTRACT A case report of a 56 years Indian lady, a known case of cholelithiasis, presented with swelling in Right upper quadrant with complex Gall Bladder perforation – localised perforation (falciform ligament and abdominal wall – Niemeier's Type 2) and Cholecystoduodenal fistula (Niemeier's Type 3), eventually diagnosed with Stage IV adenocarcinoma gallbladder. The unique and rare presentation of the complex type of gall bladder perforation – Type 2 plus Type 3 Niemeier's classification, absence of regional lymphadenopathy or distant metastasis posed to be diagnostic and therapeutic challenge in this case.

KEYWORDS : Gall Bladder Carcinoma; Gall Bladder Perforation; Complex Gall Bladder Perforation; Modified Niemeier Classification; Adenocarcinoma; Metastasis;

INTRODUCTION

Gallbladder perforations have been reported to occur with an incidence of 1 to 4 percent of cases with cholecystitis, with type 2 as the most common presentation followed by type 3. Four types are described: Type-1 include acute perforation with generalized peritonitis, type-2 subacute with localized pericholecystic abscess, type-3 is chronic perforation with cholecysto-enteric fistula and type-4, chronic perforation with cholecysto-biliary fistula. *(Fig 1)* [1]

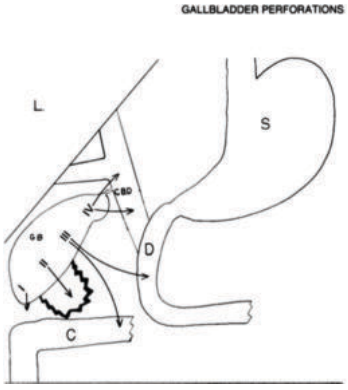


Figure 1. *Schematic representation of gallbladder perforations and modified Niemeier's classification: type 1, free perforation; type 2, perforation with abscess; type 3, perforation with cholecysto-enteric fistula; and type 4, perforation with cholecystobiliary fistula formation. C, colon, CBD, common bile duct, D, duodenum, GB, gallbladder, L, liver, and S, stomach*

Gallbladder cancers are leading malignancy of the biliary system and carry a grave prognosis with overall 5 years survival rate being less than 5%. Due to aggressive nature and nonspecific clinical presentation, 80% of patients were diagnosed at advanced stages. However, in early-stage disease, 5-year survival up to 75% is possible if appropriately managed. Gallstones and chronic cholecystitis are important risk factors in formation of gallbladder malignancies. [2]

Gallstones may cause obstruction leading to inflammation, bacterial overgrowth within gallbladder lumen and lead to eventual venous congestion, pressure necrosis and perforation of gallbladder wall.

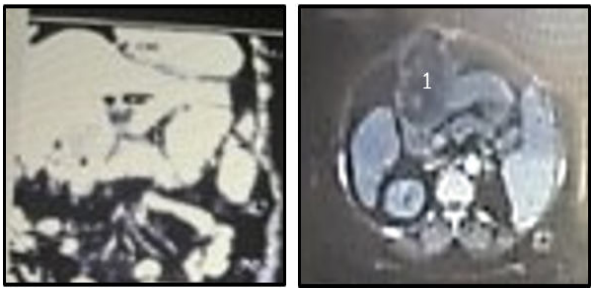
Chronic inflammation accumulates successive genomic mutations which lead to progression from pre-neoplastic dysplasia to carcinoma in situ and ultimately to invasive cancer, mostly adenocarcinoma (90%) after around 15 years of inflammation. [2]

Case Report

A 56-years hypertensive Indian lady, a known case of cholelithiasis for 8 years presented with a swelling in right upper quadrant associated with continuous dull aching pain in right upper quadrant for 3 months. Patient is a known hypertensive for 30 years and on active medication (Tab Amlodipine 5mg + Tab Atenolol 25 mg), postponed for cholecystectomy thrice in view of uncontrolled hypertension. Patient does not have any positive family or surgical history. She is a postmenopausal lady with Obstetrics history of P1L1. At presentation, she was haemodynamically stable and had a palpable mass of size 5cm x 4 cm, which was erythematous, tender, increased localised temperature, firm with regular margins and uniform consistency; intra-peritoneal in location not moving with respiration. Murphy's sign was negative. Her laboratory parameters indicated Hb – 8.3; TLC – 14,300 with P/L/E/M of 75/22/2/1 and rest of the parameters within normal limits. Her USG was suggestive of heteroechoic lesion in left lobe of liver with suspicion of rupture of Gall Bladder. CECT revealed non visualisation of Gall Bladder with multiple air pockets within GB fossa region extending to anterior abdominal wall and was suggestive of acute complicated cholecystitis with ruptured GB wall and localised abscess formation.

CEA at the time of presentation was 123 ng/ml (0.01-4) and CA 19-9 was within normal limits.

There was a gradual increase in size of the swelling and for source control, Incision and Drainage was performed and 320 ml purulent collection was drained with cytology revealing degenerated inflammatory cells. There was persistent residual hard mass, evaluated with a repeat cytology revealing fibrofatty tissue and repeat CEA increased to 180 ng/ml. Repeat CECT of whole abdomen revealed Pneumo-bilia *(Figure 2a)*, overdistended Gall Bladder with hyperdense foci and air extending to anterior abdominal wall. *(Figure 2b)*.



(a)*1- Pneumobilia* *(b) 1- Extension to Anterior Abdominal wall*

Figure 2*. Preoperative CECT showing Pneumobilia (a-1); extension of cavity from liver, porta hepatis to anterior abdominal wall (b-1)*

Patient was optimised for forty days and explored by a Mercedes Benz incision with intraoperative findings revealing cavity with thickened wall in the region of Falciform, which was in continuity with the Gall bladder fossa. The cavitating lesion was adherent to Pylorus and Duodenum. Adjacent Liver was hard on palpation with no palpable nodules or lymph nodes. Greater Omentum was also adherent to the mass.

The cavity was opened and was filled with polypoidal projections, no separate gall bladder was identified (Cavity wall itself was most likely the ruptured gall bladder). A 2 cm X 2cm fistulous communication was found with duodenum and necrotic material with free polypoidal projections retrieved from the duodenal lumen also. *(Figure 3, 4)*.

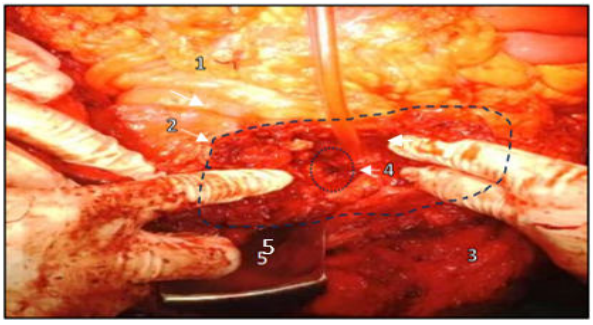


Figure 3. *Intraoperative image showing 1- Omental Adhesions; 2 - Gall Bladder Wall; 3- Duodenum; 4 – Cholecystoduodenal fistula; 5 – Liver*

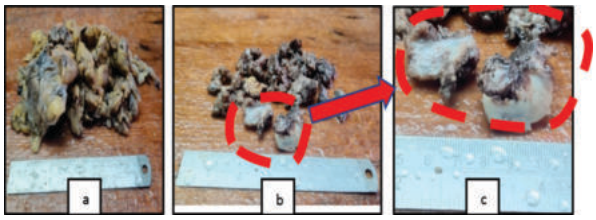
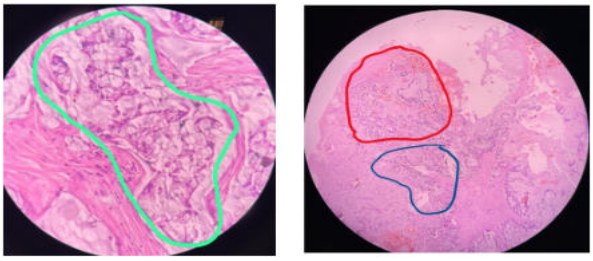


Figure 4. *(a) Gross outer surface of excised cavity wall. (b) Inner surface lining of cavity with (c) Magnified view of cavity wall section showing thick wall and polypoidal growth*

A fringe of cavity wall was left around the Duodenal Fistula opening after fulgurating the lining and the wall of cavity utilized to close the fistula.

The wall was sent for Frozen section *(Figure 5)* and Histopathological examination.

Frozen section revealed an area of adeno carcinoma, However, in view of frozen Calots, adherent Pylorus and induration at Hilum of Liver no major resection was contemplated.



(a) (b)

Figure 5. *Tumour cells in nests (a), tumour cells forming glands(red), tumour cells arranged in nests and singly scattered with fibromyxoid background, dilated blood vessels and extravasated blood (blue) confirming adenocarcinoma (b)*

On POD 10, patient developed Grade 5 SSI with bilio-purulent discharge from the wound which on conservative management, decreased in amount but minimal purulent discharge persisted. The patient was discharged with minimal discharge, antibiotics for re-evaluation at oncology center for chemoradiotherapy. However, from the site of discharge, patient developed a growth at margins of the fistula, which were everted and histopathology revealed metastatic growth. *(Figure 6)*.

Figure 6. *Metastatic growth at the fistula site*

PET CT revealed (1) heterogenous mass measuring 123*67*59 mm replacing gallbladder, infiltrating and extending beyond abdominal wall muscles up to overlying cutaneous margin with ulcer, (2) few mildly FDG avid periportal and aortocaval nodes, largest measuring 1.7*1.8 cm.

Staging – T3N2M0, Stage IVB Gall Bladder Adenocarcinoma

DISCUSSION

Underlying long standing Gall Stones, older age, polyps are high risk factors for development of malignancy[3] and were present in this case. Other risk factors are also described like adenoma, porcelain gall bladder, cholesytoenteric fistula, ulcerative colitis, anomalous biliopancreatic junction, decreased expression of 23 nm and overexpression of c-erb B2 gene products. Gall Bladder Perforation in this case was in a lady though Gall bladder perforations are more common in men[4], however, perforation in squamous cell malignancies have been reported in women in 4th-5th decade.

Atherosclerosis is a high-risk factor for Gall bladder Perforation and his patient was on antihypertensives for over 30 years.[5] Acute presentation as a liver abscess is described in literature [6] but no evidence of presentation of gall bladder perforation as abdominal wall abscess could be traced in literature searched online.

Both internal fistula and concomitant abdominal wall abscess are unique to this case. The presence of multiple small polyps filling the lumen of the gall bladder forming the wall of the cavity raised a suspicion of malignancy but inflammatory polyps in the abscess wall granulation tissue was also a possibility. Inflammation of Gall Bladder due to infection or malignancy can cause perforation[7]

The pre operative challenge of diagnostic dilemma of malignancy and inflammation persisted during surgery also. Absence of enlarged Lymph nodes in the Hepato- Duodenal Ligament and Porta Hepatis in Gall Bladder malignancy is rare, described in some cases of squamous variety of Gall Bladder malignancies[8] but raised a per operative doubt of pseudo tumour or acute on chronic inflammation.

Asymptomatic Gall Bladder perforations are reported in Literature [7] and may be missed on Ultrasonography. Superimposed inflammation in a pre-existing perforation could possibly be a reason for complex presentation of the Gall Bladder cancer with perforation, as described, are locally advanced and not amenable to resection as in this case.

Even though residual tumour is the most important factor for recurrence, R_0 resection is precluded due to local invasiveness and advanced disease at presentation in most cases primum non no cere, as should be, was the main principle in managing this case. Even with a frozen section report of malignancy, palliative procedure of primary closure of the fistula with fulgurated remnant wall of Gall Bladder was done avoiding morbidity due to dissection of frozen calots and visceral adhesion of pylorus and adherent cavity at the liver hilum. Minimalistic approach was kept in mind in view of reported poor survival outcomes of advanced Gall Bladder Malignancy (the 5–year survival of metastatic adenocarcinoma being 5%) and at the same time giving the benefit of doubt of benign or low-grade papillary malignancy as an underlying cause of disease.

In spite of non-feasibility of R_0 resection and regional recurrence, survival benefit is possible in select situations mimicking or low grade malignant conditions as in our patient who continues to live for over 10 months with normal weight and appetite.

CONCLUSION

Initial presentation as abdominal abscess, co-existence of Type 1 and Type 3 Niemeier type Gall bladder perforation, operative features of advanced malignancy with no lymph node enlargement and no distant metastasis make this case unique in clinical presentation and management and may be encountered by a surgeon as a surprise during the operative procedure.

This complex perforation with concomitant two types (any two of Type I to Type IV) could be reported as Type V extension of Modified Niemeier's classification. This is strongly proposed and must be consciously looked for in all cases of Gall Bladder Perforations.

Avoiding a radical resection in situations that mimic malignancy in the setting of a perforated gall bladder or cholecystoenteric fistula may be of benefit in terms of palliation and survival, both.

The role of Frozen section is limited in altering operative management in advanced gall bladder malignancies unless the Centre is a Specialized Centre for research and ultra radical surgical management is a possibility.

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