"ULTRASOUND STUDY OF GALL BLADDER DISEASES"

By

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DOCTOR OF MEDICINE

In

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Under the guidance of

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ABSTRACT

BACKGROUND & OBJECTIVES

Diseases of the gall bladder include wide spectrum of pathologies. Conditions like cholelithiasis, acute cholecystitis, membranous cholecystitis, present with right upper quadrant pain other conditions like gall bladder polyp are asymptomatic. In many cases of carcinoma gall bladder the symptoms manifests very late, when conservative and curative treatment is not possible. In such conditions the inclusion of ultrasound abdomen in routine investigation protocol will help in early diagnosis and initiation of therapy.

SOURCE OF DATA:

The source of data for this study is patients referred to the DEPARTMENTOFRADIOLOGY AND IMAGING at Shri B.M. Patil Medical College Hospital and Research center, Bijapur for transabdominal ultrasound.

AIMS AND OBJECTIVES

- To describe sonographic findings in spectrum of gall bladder diseases.
- To discuss the advantages and limitations of ultrasound in diagnosis and differentiationof gall bladder diseases.
- To correlate clinical diagnosis with ultrasound diagnosis.
- Comparison with CT/MRI where ever possible.

METHODS AND MATERIAL

Patients with gall bladder diseases usually presents with pain in the right hypochondriac region which may be radiating to the epigastrium or around the lower ribs or to the back. Evaluation of the gallbladder by ultrasound was performed in sagittal and transverse planes with low frequencyand high frequency sector probes.To avoid missing gall bladder pathologies special maneuver such as subcostal oblique view with the left edge of the transducer more cephalad than the right edge is used.An attempt to elicit sonological Murphy's signwill be made in order to detect cholecystitis.

RESULTS

- In a series of 202 cases, majority of the gall bladder diseases includes gall stones. Among non calculus diseases inflammatory conditions, acute cholecystitiswas commonest, others include chronic cholecystitis, membranous cholecystitis, polyp, perforation, adenomyomatosis.
- In our study neoplasms were rare compared to inflammatory conditions of which malignant were more common than benign.

CONCLUSION

The ultrasound diagnosis in gall bladder diseases had favourable&impressive results against clinical diagnosis.

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INTRODUCTION

Diseases of the gallbladder include wide spectrum rangingfrom gallstones, sludge, polyps, gallbladder mucocele, porcelain gallbladder, inflammatory conditions and neoplasms, both benign and malignant.

These conditions are seen almost everyday in radiology practice, early identification of which helps in bringing down the morbidity and mortality associated with them.

The radiological evaluation of gallbladder diseases has undergone an ocean of change with the advent of ultrasound in 1978.

Oral cholecystography which was gold standard in yesteryears is no more an option due to better appreciation of gallbladder diseases by ultrasound.

Ultrasound techniques on the other hand have found greater acceptance by every faculty overwhelmingly throughout the world with its high degree of sensitivity and specificity for nearly entire spectrum of gallbladder diseases.

Several studies using ultrasound have led to the revisions in clinical approaches to the diagnosis and treatment of gallbladder diseases and hence this study of gallbladder diseases by sonography appears appropriate.

NEED FOR THE STUDY

Gallbladder pathologies constitute about 8-9 cases per week referred for ultrasound to our department.

Patients with gallbladder diseases usually presents with pain in the right hypochondriac region which may be radiating to the epigastrium or around the lower ribs or to the back.

Certain conditions like carcinoma gallbladder present with vague manifestations like weight loss and other conditions like gallbladder polyp are asymptomatic.

It has also been noted that in many cases of carcinoma gallbladder, the symptoms manifests very late, when conservative and curative treatment is not possible.

It is in such conditions that the inclusion of ultrasound abdomen in routine investigation protocol will help in early diagnosis and initiation of therapy[1].

Ultrasound is easily available, reliable, has no harmful ionizing radiations and less expensive than other modalities.

Ultrasound has the highest sensitivity and specificity for evaluating patients with suspected biliary pathologies[2].The most important advantage of ultrasound over other imaging techniques in the investigation of acute cholecystitis is the ability to assess sonographic Murphy's sign, which is a reliable indicator of acute cholecystitis with a sensitivity of 92%[3]. An increased gallbladder wall thickness of > 3.5 mm has been found to be a reliable and independent predictor of acute cholecystitis[4].

Transabdominal ultrasound is frequently the first imagingtechnique employed for patients presenting withbiliary-type symptoms as it is more accurate thanCT for diagnosing acute biliary disease[5]. Imaging isusually performed following a 4-hour fast, allowing thegallbladder to fill and reducing obscuring upper abdominal gas.Ultrasound allows dynamic assessment and by moving the patient helps differentiatestones,sludgeandpolyps.

Dopplerultrasoundallows assessment ofvascularity, while focal gallbladdertenderness can be determined using probe pressure.

Hence this study is undertaken to evaluate gallbladder pathologies by ultrasonography in diagnosis of clinically suspected cases with regard to diagnostic performance characteristics, technical success, safety and cost effectiveness.

OBJECTIVES OF THE STUDY

The objectives of the dissertation titled "ULTRASOUND STUDY OF GALLBLADDER DISEASES"is as follows

1. To describe sonographic findings in spectrum of gallbladder diseases.

2. To discuss the advantages and limitations of ultrasound in diagnosis and differentiationof gallbladder diseases.

3. To correlate clinical diagnosis with ultrasound diagnosis.

4. Comparison with CT/MRI where ever possible.

MATERIAL AND METHODS

Source of data:

The source of data for this study is patients referred to the DEPARTMENTOFRADIOLOGY AND IMAGING at ShriB.M. PatilMedical College Hospital and Research center, Bijapur for transabdominal ultrasound with right upper quadrant pain.

Period of Study:November 2015 - April 2017

Study Design:Cross-sectional study

Inclusion criteria:

- Patients presenting with history of abdominal pain, tenderness in right upper quadrant, clinical features of obstructive jaundice.
- Clinically diagnosed / suspected gallbladder diseases.
- Patients with previous history of gallstones.
- Known case of pancreatitis.

Exclusion criteria:

- Abdominal pain other than in right upper quadrant.
- All cases of acute abdomen like those due to peptic ulcer, hollow viscus perforation, intestinal obstruction.
- All post-operative patients of cholecystectomy.

METHODS OF COLLECTION OF DATA

All clinically suspected patients of gallbladder diseasereferred to the Department of Radiodiagnosis, Shri B.M.Patil Medical College Hospital and Research Center fortransabdominal ultrasound were included in the study.

Evaluation of the gallbladderwill be performed in sagittal and transverse planes.Scanning will be performed with two probes one with low frequency (8 to 5 MHz) and one with high frequency (12 to 3 MHz).

To avoid missing gallbladder pathologies special maneuver viz, use of subcostal oblique view with the left edge of the transducer more cephalad than the right edge.

An attempt to elicit sonological Murphy's sign (maximal abdominal tenderness from pressure of the ultrasound probe over the visualized gallbladder)will be made, in order to detect cholecystitis.

Ingestion of food, particularly fatty food, stimulates the gallbladder to contract. The contracted gallbladder appears thick walled and may obscure luminal or wall abnormalities. Therefore the examination of the gallbladder should be performed after a minimum of 4 hours of fasting.

The machines which will be used in the study are SIEMENS ACUSON X700 and PHILIPS HD11-XE.

SAMPLE SIZE

Based on the incidence of patients for gallbladder diseases (0.83%)[6],at 99% confidence level and +/-2 margin of error, the sample size calculated is 202.

$$n = \frac{Z^2 \sim XpX(100 - p)}{d^2}$$

Here,Z_α = Z is value at α level

p =incidence rate

d = margin of error

STATISTICAL ANALYSIS:

Data will be analyzed using,

Diagrams

Mean ± SD

chi square test/fisher exact test

REVIEW OF LITERATURE

EMBRYOLOGY

The gallbladder derives as an outpouching from embryonic biliary tree.

The proximal part of the pouch gives rise to cystic duct, and the distal portion forms the gallbladder.

Within the cystic duct, small mucosal folds called the spiral valves of Heister; these are identified on sonography.

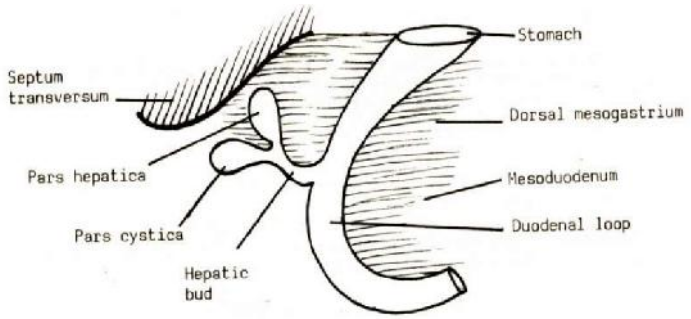


FIG 1

During its initial development, the gallbladder lies in an intrahepatic position, but as it migrates to the surface of the liver, it acquires a peritoneal covering (part of liver capsule) over 50% to 70% of its surface. The remainder of the gallbladder surface is covered with adventitial tissue that merges with connective tissue in contiguity with the liver [6]

ANATOMY

Gallbladder is a pear-shaped organ lying in the inferior border of the liver, between right and left lobes. Middle hepatic vein, interlobar fissure lies in the same

anatomic plane separating the two hepatic lobes, extends from the right portal vein origin to the gallbladder fossa and may be used to find it.

This fissure has been seen in up to 70% of hepatic ultrasound studies[7] and used as a landmark for the gallbladder fossa.

The gallbladder is divided into three parts, the fundus, body, and neck; the fundus liesas anterior and inferior segment. At the region of the gallbladder neck, there may be an infundibulum, called Hartmann's pouch, which is site for impaction of gallstones.

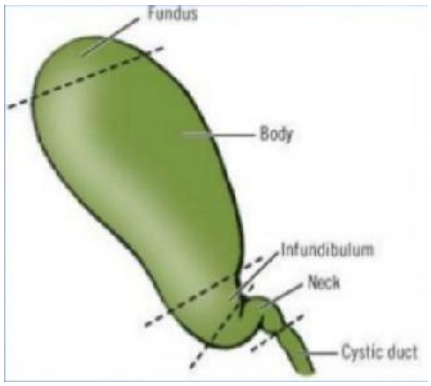


FIG 2

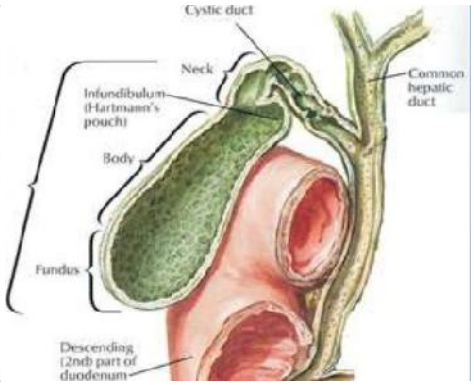


FIG 3

Primary blood supply to gallbladderis by the cystic artery, which is a branch of the right hepatic artery, and the cystic veins, which drain into the portal vein.

The gallbladder function as reservoir for the storage and concentration of bile, which is secreted by the liver, composed mainly of bile salts, bile pigments and small amounts of cholesterol, lecithin, fatty acid and mucin.

The gallbladder can store upto 50 ml of bile and can increase the concentration of bile by five to ten times.

Release of bile from the gallbladder is stimulated by the presence of fatty food, in the alimentary tract. Duodenum secretes cholecystokinin, which stimulates contraction of the gallbladder andreflexive contractions of wall of gallbladder both in conjunction with relaxation of the neck of the gallbladder, force bile out into the duodenum[8].

ANOMALIES AND ANATOMIC VARIATIONS

Gallbladder agenesis

It is caused by developmental failure of the caudal division of the primitive hepatic diverticulum or vacuolization failure after the solid phase of embryonic development. Atresia or hypoplasia of the gallbladder represents aborted development of the organ [9-10].

On ultrasound, a linear echogenic area is seen in gallbladder fossa due to dense fibrous tissue.

On ultrasound,gallbladder was not visualized, but strong echoes with acoustic shadowing were seen[11].

Duplication of the gallbladder

It is caused by incomplete revacuolization of the primitive gallbladder, resulting in a persistent longitudinal septum that divides the gallbladder lengthwise. Another mechanism is the development of two cystic budsseparately.

To establish the diagnosis, two separate gallbladder cavities, each with its own cystic duct must be present. These two cystic ducts may separately enter common duct or form a Y configuration before a common entrance [11].

On ultrasound, two cystic structures will be seen in region of gallbladder fossa which are connected to a common cystic duct or two separate cystic ducts that lead to a common hepatic duct [12].

In 2002, Lisa L et al, Sonographic Detection of Gallbladder Duplication;

On ultrasound 2 fluid-containing structures adjacent to the fetal liver were seen. These were saccular in morphology and were situated in the same plane. A small rim of tissue was noted between them. Duplicated gallbladder was diagnosed [13.]

Phrygiancap

Deformity is characterized by a fold or septum of the gallbladder between the body and fundus. Two variations can be seen. In the retroserosal type, the peritoneum smoothly invests the gallbladder, and the mucosal fold that projects into the lumen may not be visible externally. In the serosal type the peritoneum follows the bend in the fundus, reflects on itself as the fundus overlies the body[14].

Easily visualized **on ultrasound**as asymptomatic folding of the gallbladder fundus over body[15].

Multiseptate Gallbladder

Characterized by multiple septations of different sizes internally and a faintly bosselated surface externally [16-19].

These septations cause stasis of bile and gallstone formation.

On ultrasound studies, multiple communicating septa and locules are seen bridging the gallbladder lumen [20].

On ultrasound distended gallbladder with a thickened wall. There was also a transverse septum which divided the gallbladder into two cavities. No calculi were seen and the common bile duct was within normal limits [21].

Diverticula

Gallbladder diverticula are rare and most of the times clinically asymptomatic. No site of predilection in the gallbladder and are usually single and are of varyingsizes.

Congenital diverticula are true diverticula and contain all the layersof wall, on the contrarypseudodiverticula of adenomyomatosis, have no smooth muscle component in their walls[22].

On ultrasound, local outpouching of the entire gallbladder wall with a narrow neck is seen [23].

In 2009, SelimDoganay et al, True Diverticulum of the Gallbladder;**Abdominal Ultrasound** depicted a local outpouching of the entire gallbladder wall with a narrow neck, diagnostic of true diverticulum; its diameter was approximately 25 mm, and a little biliary sludge in the lumen of the gallbladder was also present. The wall of thegallbladder was mildly thickened (4 mm). No evidence of gallstone. MRCP confirmed, true diverticulum with dilatation of the intrahepatic biliary ducts[24].

Wandering Gallbladder

When the gallbladder has an unusually long mesentery, it can "float " or “wander” [25-29].

The gallbladder may "disappear" into the pelvis or may be seen in front of the spine or to the left of the abdomen. Rarely, it can be seen herniating through the foramen of Winslow into the lesser sac.

Ectopic Gallbladder

The gallbladder can be seen in a various anomalous positions; Intrahepatic,suprahepatic, retrohepatic, supradiaphragmatic, and retroperitoneal, can also be seen in the transverse mesocolon, falciform ligament, and anterior abdominal wall.

In patients with cirrhosis, small or absent right lobes, or chronic obstructive pulmonary disease, the gallbladder together with the colon is interposed between the diaphragm and the liver[30].

Left-sided gallbladder location is seen in situs inversustotalis.Gallbladder in the left upper quadrant without situs inversus is even more rare. Intrahepatic gallbladdersarelocated in subcapsular region in the anterior inferior right lobe of the liver. This leads to problem for scintigraphy, as an intrahepatic gallbladder can cause a focal defect; sonography ishelpful in these cases [31-32].

On ultrasoundneck of the gallbladder is seen anterior to the right branch of the portal vein helps in identification of gallbladder[33].

For confirmation the patients were givenfood with high fat. Decrease in the size of the cystic area was noted after repeating the ultrasound examination after two hours of intake of fatty meal indicating the contraction of ectopic gallbladder.

PATHOLOGIES

Acute cholecystitis

In 2014, Hamish Hwang et al, in the article Does acute cholecystitis can be diagnosed accurately byultrasonography? Improving diagnostic accuracy based on a study at a regional hospital, in 107 patients, found that ultrasonography had 100% sensitivity, 18% specificity, 81% positive predictive value (PPV) and 100% negative predictive value (NPV) for cholelithiasis.

In case of acute cholecystitis, sensitivity was 54%, specificity of 81%, 85% PPV and 47% NPV. For chronic cholecystitis the sensitivity was found to be 100% with ultrasonography[34].

In 2011, Aldo Benjamim Rodrigues Barbosaet al, in Gallbladder wall thickening at ultrasonography: how to interpret it?cholecystitis considered responsible for the wall thickening found in only eight patients. The rest had hepatitis, alcoholic liver disease with hypoproteinemia, heart failure, renal disease, and multiple myeloma; however, all lacked clinical evidence of biliary disease. Diagnosis of cholecystitis only on the basis of wall thickening alone should be cautious[35].

In 2010,Oliveira GAet al, Transient reticular gallbladder wall thickening in severe dengue fever: a reliable sign of plasma leakage;Flavivirusisresponsibleforacute infection,**Dengue fever (DF)**. Mild symptoms were seen in most of the patients;only few progressed to a severe form characterized by hypovolemic shock and hemorrhagic phenomena. Plasma leakage was characteristically observed in severe form of DF.

Ultrasound triad of plasma leakage in DF include wall thickening, ascites and pleural effusion.

Abdominal ultrasound examination was done in37 children with severe DF on first day of admission at the time of discharge andon 7th day after the first examination if the child was still hospitalized.

Of the 37 children, 33 (89.2%) presented gallbladderwall thickening, 29 (78.4%) ascites and 26 (70.3%) pleural effusion. By the time of second examination, most of the findings had resolved. Of the 33 gallbladder wall thickenings, reticular pattern of presentation was characteristic of plasma leakage in patients with severe DF in29 (87.9%).

Gallbladder wall thickening, pleural effusion and ascites werereactive findings in DF. They concluded that acalculous cholecystitis should not be diagnosed on the basis of typical reticular pattern ofgallbladder wall thickeningbut, they can be used to diagnose and follow up of patients with severe DF[36].

In 2007,van Breda Vriesman AC et al, studied on diffuse gallbladder wall thickening;When there is clinicalsuspicion of acute cholecystitis ultrasound is generally the preferred initial imaging technique. The sensitivity of ultrasoundranges from 80% to 100% and specificity is 60% to 100%. Imaging findings may include gallstones, gallbladder wall thickening (of more than 3-5 mm), pericholecystic fluid, and positive sonographic Murphy sign. Gallstone within the gallbladder neck or cystic duct, distended gallbladder and echogenic sludgeare other less-specific findings.

Gallbladder wall thickening with positive sonographic Murphy’s sign and cholelithiasis increased diagnostic accuracy of acute cholecystitis.

There was increase in PPV for acute cholecystitis by 4% when combination of gallstones and positive sonographic Murphy's sign to 92% from 88% with gallstones alone[37].

In 2006, Kane MA et al,Ultrasound findings in acute viral hepatitis: 177 acute viralhepatitis patients which were clinical diagnosedwere studied from Jun 2004 - 2006. 32patients were excluded, 145 patients which were serologically confirmed withuncomplicated acute viral hepatitis were studied.

Sonography was done after overnightfasting in supine positiononall patients.

USG was repeated after 3-4 weeks.Collapsed gallbladder with increased wall thickness was most common findingsseen in more than 50% of all types of hepatitis. These findings were seen in all patients with HAV hepatitis and in 84% cases of Hepatitis E. 4 to 18 mmwerewall thicknessrange. Liverenzymes were elevated proportionally as the gallbladder wall thickness increased. 51%of Hepatitis E patients showed pericholecystic oedema. Associated sludge was seen inless than 50%. High transaminase levels accounts for more gallbladderultrasound findings.However, these ultrasound findings were temporarily seen and in mostof the patients,findings disappeared within 3 weeks of the first ultrasoundexamination.

They concluded thatultrasound findings were seen in more than 80% of hepatitis patients with enteric route of transmission.As a result when biochemical tests are not availableultrasound can be used to diagnose acute hepatitis[38].

In 1995, M.L. Kulkarni et al, studied on Acute Acalculous Cholecystitis in Typhoid Fever, they conducted in a study of 50 patients with typhoid fever examined by Sonography.

Out of 50 patients, 30 were culture positive and remaining had characteristic signs and symptomswithWidal test positive ('O' titre>l:160).All patients were subjected to abdominal sonography in fasting state.4 (8%) patients were diagnosed of acalculous cholecystitis. 3 out of 4 children presented during the first week of illness.

The Ultrasound criterias for acute acalculous cholecystitis include a thickened gallbladder wall (more than 3 mm), sonographic Murphy's sign, a round shape, pericholecystic collection and the absence of gallstones.Sonography was repeated at weekly intervals, showed normal appearance after 3 weeks to 3 months. Rarely cholecystectomy was required in cases of acalculous cholecystitis with associated suppuration, ischemia or septic complications. They concluded acalculous cholecystitis, frequent complication was detected frequently in typhoid fever [39].

Gangrenous cholecystitis

Gallbladderis mainly supplied by cystic artery. In case of embolism, arteriosclerosis is the usual abnormality which occurs as a result of occlusion/stenosis of cystic artery leading to compromised viability of the gallbladder. Vascular insufficiency is the main cause of emphysematous cholecystitis, with associateddiabetes mellitus, predominance in males, high frequency of gangrene, and occurrence in older patients.Vascular compromise of the cystic artery results in ischemia ofthe gallbladder and facilitates the growth of gas-forming organisms(eg, **Clostridium or Escherichia coli**) and bacterial proliferation in the devitalized tissue with low oxygen saturation.

Among anaerobic organisms,**Bacteroidesfragilis** were most frequently isolated. High rate of isolation of anaerobic bacteria from bile in patients with

gangrenous cholecystitis (72%).Bactericidal bile is rendered alkaline, facilitating infection of the bile.

In 2015, Teena Dhira et al,Old man gallbladder syndrome: Gangrenous cholecystitis in unsuspected patients;Gangrenous cholecystitis (GC) is a rare but fulminant complication of acute cholecystitis. The pathophysiology of ischemia and necrosis of the gallbladderoccurssecondary to distendedgallbladder with increased tension and pressure on the gallbladder wall.

Acute cholecystitis complicating togangrenous cholecystitisis a common surgical condition.Increased postoperative complications, morbidity and mortality were seen in gangrenous cholecystitis. Predictive factors for gangrenous cholecystitisinclude age more than 45, male predominance, WBC count more than 13,000/mm^3 and **negative sonographic Murphy's sign.**

The incidence of gangrenous cholecystitiswas 2% to 30% in patients with acute cholecystitis and has been seen commonly in elderly patients.The risk of gangrenous cholecystitiswas also increased in patients with a history of diabetes.

Ultrasound findings predictive of GC include increased wall thickness along with elevated WBC. Positive predictive finding of GC include findings of discontinuous and/ or irregular mucosal enhancement pattern, and it was found that the lack of mural enhancement was statistically significant correlating to gangrenous cholecystitisalong with gallbladder distension and wall thickening of >4.0–4.5 mm.Other findings that help predict gangrenous cholecystitisinclude perforation of gallbladder, pericholecystic stranding and a **sonographicMurphy's sign negative.**

The reasoning for a negative Murphy's sign, due to denervation of the gallbladder, decreasing the suspicion for a more pathological process. Due to the transmural necrosis of the gallbladder wall, the afferent nerves die and the inflammation spreads to the parietal peritoneum, leading to generalized abdominal pain.

They concluded that gangrenous cholecystitisis a fulminant form of acute cholecystitis with associated increased morbidity, mortality and worse postoperative outcomes. Risk factors for GC including age > 45 years, male gender, WBC > 13,000/mm^3, ultrasound and CT findings of irregular gallbladders with lack of mural enhancement and were later found to have GC. Though the finding of GC does not change the surgical management of disease, knowing that a patient is at increased risk of GC should prompt earlier surgical intervention to prevent some of the known complications from this disease[39].

In 1991, Teefey SA et al,Gangrenous cholecystitis: new observations on ultrasound; ultrasound findings suggestive of gangrenous change include floating intraluminal membranes, echogenic foci withshadowing consistent with gas within the gallbladder wall/lumen, frank defect of the gallbladder wall and abscess formationin pericholecystic area. Characteristicdiagnosticsign of gangrenous cholecystitis is striations ofgallbladder wall, or presence of alternating hyperechoic and hypoechoic mural linear areas seen in up to 40% of patients[40].

Emphysematous cholecystitis

In 2002,Konno K et al, Emphysematous cholecystitis: sonographic findings were assessed**;** Acute cholecystitis rarely complicates with emphysematous cholecystitis (EC) early diagnosis is necessary to prevent management delay.

Comparison was done with clinical and ultrasound features of 11 surgically proven cases of EC (with minimal amounts of gas in three cases and large amounts in eight cases).

In cases with minimal amounts of gas, sonography showed a hyperechoic line with a distinct ring-down artifact or a "powder snow-like" speckled posterior shadowing, with increased amounts of gas, a wide spiculated echogenic band with a powder snow-like speckled posterior shadowing. Gallbladder wallwas not visualized in all cases due to presence of gas. Differentiation of gas localized to the gallbladder wall and gas extending to the surrounding hepatic tissue was not possible by sonography. Gas was seen throughout the intrahepatic bile ducts in 2 diabetic cases [41,42].

Perforation of gallbladder

In 2006, Derici Het al[43], Diagnosis and treatment of gallbladderperforation; gallbladder perforation is an important complication of gangrenous cholecystitis. Gall-bladder perforation is caused by transmuralnecrosis in a case of acute cholecystitis.

2–11% of cases of acute uncomplicated cholecystitis gradually progress to perforation, with a reported mortality rate of up to 60%. After perforation, patients got significantly reduced from pain. Perforationis classified into three types. Type I perforation involves spillage of gallbladder intraluminal contents into the peritoneal cavity, type II perforation is subacuteprocess with an adjacent abscess formation. Type III perforation is a chronic processwith the formation of a cholecysto-enteric fistula[44]. Fundus of gallbladder is the most common site of perforation**. Gallbladder wall shows a focal defect on ultrasound**, CT or MRI. **An extraluminal gallstone is**

a characteristic imaging finding that indicates perforation. Features of perforation are nonspecific andincludepericholecystic fluid, luminal collapseof gallbladder and abscess in pericholecystic area[45].

Xanthogranulomatous cholecystitis

In 2015,Hideki Suzukiet al[46], studied on Xanthogranulomatous cholecystitis (XGC): Difficultto differentiate from carcinoma of gallbladder;XGC is a rare form of chronic cholecystitis, seen in 1.3% to 5.2% of resected gallbladder specimens.

From Apr 2000 to Dec 2013, in 6 patients of XGC extended surgical resection was done.16 patients were proved withcarcinoma gallbladder, according to extended surgical resection. Only XGC cases were chosen for study having indistinct borders with the liver in most of the situations it is difficult to distinguish these patients from advanced carcinoma gallbladder. Comparison withultrasound findings, clinical feature and computed tomography findings between XGC and advanced carcinoma gallbladder. Clinical features likeage, gender, symptoms and tumor markerswere retrospectively assessed.

The CT findings were used to compare two conditions, to detect the co-existing gallstones, pattern of gallbladder wall thickening (focal/diffuse), the presence of a hypodense intramural nodule and continuation of the mucosal line.

Preoperative evaluation was carried out with ultrasound, CT, MRI and FDG-PET.

Ultrasoundfeatures of Xanthogranulomatous cholecystitis include grossgallbladder wall thickening with oval hypoechoic nodules[47]. Kim et al[48]

suggested the combinationof diffuse wall thickening and ultrasoundfindings and nodular wall thickening are highly suggestive of Xanthogranulomatous cholecystitis.

67% of patients presented with abdominal pain with Xanthogranulomatous cholecystitis, however, there were nomajor differences in clinical symptoms, including fever, between the two groups.

83% of patients had cholelithiasis with XGC, whencomparedto carcinoma gallbladder it was33%. Hypodense intramural nodule was seen in 3 patients with Xanthogranulomatous cholecystitis (3/6, 50%), but in only 1 patient with carcinoma gallbladder(1/16, 6%). The gallbladder wall thickness, continuous mucosal line, and dilatation of bile duct were not significantly different between Xanthogranulomatous cholecystitis and carcinoma gallbladder.

The macroscopic features of Xanthogranulomatous cholecystitis include abnormal gallbladder wall thickening with irregular soft to-firm, yellow-brown various sized nodules within wall with cholecystitis. Complications include gallbladder perforation, abscess formation, cholecystoduodenal fistula and spread of inflammation to adjacent liver and transverse colon[49]. Involvement of adjacent organs suggestsaggressive nature of Xanthogranulomatous cholecystitis, as does advanced carcinoma gallbladder. Thus it is importantto differentiate Xanthogranulomatous cholecystitis from advanced carcinoma gallbladder preoperatively to avoid unnecessary surgery.

The clinical features of Xanthogranulomatous cholecystitis are of acute or chronic cholecystitis. The primary symptoms include pain in right hypochondriac region (93.9%) radiating to shoulder and back pain (42.4%), fever (24.2%), nausea (33.3%) and vomiting (24.2%). Patients with Xanthogranulomatous cholecystitis

more commonly presents withpain abdomen jaundice and fever in comparison withcarcinoma gallbladder[50]. Excluding features like weight loss orfeatures of ascites or metastases in advanced carcinoma gallbladder, clinically it is difficult to differentiate between these two conditions.

The mechanism behind Xanthogranulomatous cholecystitis is initially biliary obstruction with acute or chronic cholecystitis and increasing intraluminal pressure, followed by a granulomatous reaction.

Obstruction to normal flow of bile causesgranulation reaction that leads to the formation of intramural nodules as a result of extravasation into the gallbladder wallthrough a small ulceration in the mucosa with involvement of the Rokitansky-Aschoff sinuses[51].

The radiological features of Xanthogranulomatous cholecystitis andcarcinoma of gallbladder,likegallbladder wall thickening andinvolvement of neighboring organs are nearly similar. However, Uchiyama et al[52]stated that continuous enhancement of mucosal line is characteristic for Xanthogranulomatous cholecystitis. Cholelithiasis adds more to the diagnosis ofXanthogranulomatous cholecystitis. In their study,cholelithiasis was seen more commonly with Xanthogranulomatous cholecystitis in comparison tocarcinoma gallbladder.

They concluded that pseudotumoralXanthogranulomatous cholecystitis has led to difficulty in terms of surgery. Even with advanced imaging techniques it is difficult to differentiate between Xanthogranulomatous cholecystitis and malignant lesions of gallbladder. Macroscopically also it was difficult to differentiatebetween Xanthogranulomatous cholecystitis from carcinoma gallbladder when Xanthogranulomatous cholecystitis is seen with irregular growth and involving

adjacent organs. Hence carcinoma radical resection including liver gallbladder is justified asXanthogranulomatouscholecystitis and malignancy cannot be completely excluded.

In 2000, Parra JA et al, Xanthogranulomatous cholecystitis: studied on clinical, ultrasound, and CT findings in 26 patients; rareinflammatory disorder characterized by abnormal nodules within gallbladder wall[53].Rokitansky–Aschoff sinuses become occluded and ruptureresulting in formation of intra mural nodules. Bile is then forced into the gallbladder wall causingan inflammatory reaction, which is comprised of histiocytes, multinucleatedgiant cells and fibroblasts. Superadded is infection is most frequently seen in elderlypatients.

Gallstones and thickened GBwall are frequent features onsonography and CT, in patients with Xanthogranulomatous cholecystitis. Wall thickening may be segmental or diffuse. Pericholecysticinflammatory reactions are seen.

Intramural hypoechoic on ultrasound orhypoattenuatingnodules on CTor bands are diagnostic of XGC[54].

Cholelithiasis

Bile is synthesized in liver transported to gallbladder for storage and is concentrated, gets emptied into duodenum which helps in breakdown of fats.Not all cases are symptomatic. However,cholecystitis results due to obstructed stone within the bile duct or gallbladder.

There are three types of gallstones

Cholesterol (10%)

More than 50% cholesterol contents; form with excessive concentration of bile, nucleation and growth of calculus

Mixed (80%)

20-50% cholesterol is causative agent as seen with cholesterol calculi

Pigment stones (10%)

Contains less than 20% cholesterol; has high bilirubin component and occur when there is concentrationof unconjugated bilirubin

Pigment stones are divided into,

Black pigment stones: due to hemolytic anemias, liver cirrhosis, intestinal malabsorption (ex; Crohn disease)

Brown pigment stones: bacterial&parasitic inclusions(ex;Clonorchissinensis) andstasis of bile.

In 2014, Yen-Chun Chen et al[55], studied on The Prevalence and Risk Factors for gallstone disease in Taiwanese Vegetarians;The of load gallstone disease (GSD) and its problems, including cholecystitis, pancreatitis and cholangitis, are major health problems globally. Many sufferers withgallstone disease are asymptomatic and around 20% become symptomatic after 10 years of record. Ultrasoundis first modalityfor diagnosing gallstone disease[56].

It was observed in 425 males and 1296 females vegetarians who were showing willingness for the study.

The diagnosis of gallstone disease was confirmed by ultrasound.In their study, there was no sex predeliction and there was weak association of increasing age with gallstone disease. Incidence of gallstone disease in male and female vegetarians is similar in this study.

Body mass index is weakly associated with gallstone disease in females. Gender is accountedas a risk factor for gallstone disease. Few Asian studies have provedslightly higher incidenceof gallstone disease in women but not as high as in Western populations. Pregnancy also plays significantrole ingallstone disease. After delivery few cases showed disappearance of sludge and gallstones[57].In this study, increased total bilirubin levels were considerably associated with gallstone disease in male vegetarians in comparison to females. They proposed that the increased probability for gallstone disease in veg population vary with gender. Increasing age is a main and collective risk factor for gallstone disease. Increased total bilirubin levels and body mass index also appeared to be risk factors in male and femalescorrespondingly[58].

In 2012,Laura M. Stinton et al, in analysis of gallbladderpathologies in defined population: gallstones and carcinoma**,**affirmed as common pathologiesand reveal as cholelithiasis and carcinoma gallbladder.Ultrasound beingnoninvasive and non ionizing radiation, is gold standard todetermine precisely the occurrence of cholelithiasis and can precisely distinguish the occurrence of cholelithiasisin a given asymptomatic group of subjects.

Today ultrasoundexamination is widely in use to study GB pathologies at any point in contrast to clinical or necropsy based evidence previously. Hazardous triggers likesevere obesity, losingweight quickly, inactive way of life and mainlynutritional factorswhich can be modifiedshould berecognized andoffera chance to avoidgallstones. Some of the causatives responsible for cholelithiasisare concernedwith carcinoma gallbladderpathogenesis[59].

Gallbladder polyp

In 2015,Vincent MMellnick et al, studied on Polyp like Lesions of the GB: Disease variety with Pathologic Correspondence, Gallbladder polyps are defined as sessile projections of the gallbladder wall into the lumen[60]. They are typically incidentally found at ultrasound. Unlike gallstones, gallbladder polyps are not significantly associated with female sex, obesity or multiparity.

Gallbladder polyps are quite frequent, incidence is 3%–7% at abdominal ultrasound and 2%–12% in cholecystectomy specimens. A wide array of pseudotumors, as well as benign and malignant tumors, may manifest as gallbladder polyps. By far, most gallbladder polyps are benign. Since malignant lesions can be resectable for cure, early detection is of crucial importance.

Ultrasound findings used to stratify gallbladder polyps into three groups: those that need no further follow-up, those that require follow-up, and those that should be excised (i.e, cholecystectomy).

Ultrasound protocols should include multiplanar gray-scale images, as well as color and spectral Doppler images of detected lesions. Lesions should be imaged in more than one position (e.g. supine and left decubitus) to avoid mistaking mobile

sludge balls for polypoid lesions. It is important to note the size and shape (e.g. pedunculated or sessile) of a polypoid lesion and the presence of gallstones, which increase the likelihood, that the polyp is a neoplastic lesion. Other findings include gallbladder wall thickening adjacent to the polypoid lesion, multiple polyps, biliary strictures and hepatic masses. The presence of twinkling artifact may help diagnose adenomyomatosis.

The cholesterol polyp is by far the most common polypoid lesion found in the gallbladder, accounting for 60%–70% of lesions in some studies. It predominantly occurs in middle-aged women. Cholesterol polyps are typically multiple and need not be associated with gallstones. No risk of carcinogenesis from cholesterol polyps. Ultrasound, there sizes are not of worrisome, round, smoothly contoured, intraluminal lesions that are attached to the wall. The stalk is rarely seen, an appearance that gives rise to the “ball on the wall” sign. Cholesterol polyps are usually echogenic with no acoustic shadowing; however, particularly when multiple cholesterol polyps are confluent and/or larger than 1 cm, they cannot be definitively differentiated from other benign or malignant lesions at imaging.

In 2011, Michael T. Corwin et al,Incidentally Detected Gallbladder Polyps: Is Follow-up Necessary?studied on 346 patients, GB polyps are incidentally detected in around 4 to7% wheregallbladder ultrasound was done.

Main apprehension was progression to malignancy like adenocarcinoma, since the lesions were recognized as neoplastic. Risk of malignancy is more in polyps of 1 cm or more diameter,single polyp,sessile polyps, polyps with adjoining increased wall thickness and also with increasing age.

When two or more polyps were recognizedand increase in size of 0.2 cm or more on follow-up study it was of significance. They were distinguishedas stable, resolved, increased/reduced in size on the basis of the highestlengthmeasurement.

They studied on 346 patients with mean age of 52 yrs (20–93 years). 156 were males and 190 females 45% & 55% respectively. 216 patientsshowed single polyp and were multiple in 130 patients. Range was 1 to 1.8 cmwith a mean size of 0.5 cm+/- 2.4.30 patients showed associated gallstonesi.e,9%. On 149 patients 43% ultrasoundfollow up study was done. In 90 patients i.e, 60% polyp size was unchanged, in eight patientsi.e5% size was reduced, in only 1 patient<1% increased dimension was seen and resolved in 50 patients i.e,34%. 42 patients (12%) undergone with resection of gallbladder, of which 13 i.e 31% had polyps, 24 (57%) with cholelithiasis and no polyps, and five (12%) with neither a cholelithiasis nor polyp.None wererecognized with carcinoma gallbladder out of 346 subjects. Mean polyp size was 0.5cm(1–1.8 cm). Between 0.1–0.6 cm no neoplasticity was identified, 1 polyp between 0.7–0.9 cm was neoplastic and 2 neoplastic polyps were found at 1 cm or more.

They concluded that, even further evaluation or follow up is also not necessary in cases where polyp size is <0.6 cm and risk of carcinogenesis is exceptionally low[61].

In 2002, D Chattopadhyay et al, in result of gallbladder polypoidal lesions detected by abdominal sonography: 9 yr study conducted andassessment of all patients who underwent sonographywho were referred to gastrointestinal surgeon at district health centre.They included all subjects with polyp like lesions in gallbladder. Out of 651,23 pts were recognisedby sonography to have a polyp like lesions prior to surgery. Post surgical resection microscopic study revealed 12 cases with

cholelithiasis, 7 cases as polyps of cholesterol, 3 adenocarcinomas within polyps and one normal gallbladder.

Ultrasound is 92% specificfor recognizing polyp like lesions. In general ultrasound has 66 % sensitivity,is 100% specific in suspicion of carcinomaprior to surgery.All the true polyps were malignant.Ultrasound showed 100% sensitivity and 87 % specificity with a PPV of 50% in the diagnosis of malignancy in polyp like lesions, when size of polyp is taken 1 cm as cut-off. Most of the patients withpolyp like lesions shown to haveassociated cholelithiasis. When polyps of more than 1 cm are encountered on ultrasound additional studies like endoscopic ultrasound/CT/MR weresuggested[62].

Adenomyomatosis

In 2006, Alexis R. Boscak et al, studied on Adenomyomatosis of the Gallbladder: is a benign hyperplastic cholecystosis, frequently identified, in at least 5% of surgical GB specimens, also called as adenomyomatous hyperplasia of the GB. There is no definite ethnic/sex based increased occurrence[63].

Gallbladder wall has four layers: mucosa, lamina propria, muscularispropria,serosa; there is no muscularis mucosa/submucosa. Mechanism of development of adenomyomatosis is overgrowth of mucosa, muscularispropria layers. It has been named as “strawberry gallbladder” due to gross appearance which is the result ofCholesterolosis,due to accumulation of triglycerides and cholesterol esters in lamina propria. Bile gets accumulated inRokitansky-Aschoffsinuses, which are small outpouchings occurring within wall lined by mucosa, and as a resultcholesterol gets increasingly collected in the gallbladder lumen.

Adenomyomatosis is inconsistent and degree of involvement, site and varying sonographic forms such asdiffuse, segmental, and focal are encountered.

Focal form is more frequently seen as hemispherical to rounded thickened GB wall, commonly at the fundus. Generalized form shows extensiveinvolvement ofgallbladder.Segmental/annular formseen asincomplete circumferentiallyinvolving thewall causing stenosis of lumen within the body of gallbladder, attributing to hourglass formation.**Ruling out of malignancy** is challenging in annular and localized cases;actually, localizedform look asdistinct mass, called adenomyoma. Metabolic categorization withfluorine 18 FDG PETis helpful add-on in difficult cases.

They concluded that adenomyomatous hyperplasia is fairly frequent benigngallbladder pathology with characteristic gross and microscopiccharactersrelatingto comparatively**precise characteristics at ultrasound**. Cholecystectomy is indicated in problematic cases[64].

In 2006 J. CarvajalBalagueraet al,diffuse adenomyomatosisof the gallbladder: uncommon entity with strenuous exercise to diagnose prior to surgery; Itisuncommonentity of the gallbladder with features of thickening of muscular layer of gallbladder wall due to epithelial proliferation resulting inpouch formationwithin gallbladder wall also known as Rokitansky-Aschoff sinuses. Many subjects are clinically silent hence the condition is detected secondarily due to sonographydone for other symptoms.

Adenomyomatous hyperplasia is an uncommon condition of unknown etiologywhich growsslowly. It can involve any site within gallbladder, frequently seen at fundus and rarely seen in the biliary tree. Since patients are clinically silent,

detection is not easy. In cases of clinically active lesions they mimic cholelithiasis. Sonography and CT are helpful in reaching the diagnosis.

Out of 11, 9 cases were seen in the fundus of gallbladder, 1 in cystic duct, 1 in distal CBD, rarely it is seen in right and left hepatic ducts, CHD & hepatopancreatic duct.Further it can also be seen instomach, small intestine, Meckel's diverticulum, sigmoid colon, recti muscles, uterus, uterine supports, ovary, abdominal cavity and also relatedwith Gardener syndrome.

Thickened gallbladder wall of more than 5 mm, herniation of mucosa into muscular layer forming pouches named as Rokitansky-Aschoff sinuses, distended lumen and sluggish growth are the characteristic features. Its patho-physiology is analogous to colon diverticulosis[65]. Histopathologically, it is characterized by a rapid growth of flat muscular fibres and epithelial adenomatous cells. Based on their site of involvement and varied ultrasound appearances, 3 forms have been described: Focal 48%, diffuse 26% and segmental form 26%[66]. Mixed forms are also recognized[67].

Conditions like in 89% gallstones&chronic cholecystitis, in 22% choledocholithiasisand in 22%with previoushistory of pancreatitis of biliary origin, were commonly associated (81%) with adenomyomatous hyperplasia.Hence, they inferredpersistent inflammation of the biliary mucosa is recognized as etiological factor[68]. Rarely it is seenrelated to GB adenocarcinoma or leiomyosarcoma[69-72], to congenital defects of the biliary tree or to duodenal diverticula[73].Nabatameet al[74] stated that annular form of adenomatous hyperplasia is associated with increased risk of carcinoma GB (6.6%) with increasing age (15.6%). These tumours have also been called as adenomyosis, hamartomas or hyperplastic adenomyomatosis[75].

GB adenomyomatosisaffects men and women between 40 and 70 years in similar proportion. Peak incidence is around 50 years. Occasionally it is also seen in pediatric population[76,77].

Diagnosis prior to surgery is complex as there is no clinical suspicionand is seen in several forms in function of the localization. Qiao et al[78]accounted that only in seven out of 42 cases it was diagnosedaccurately prior to surgery. Clinically patients may be silent (60%)[100]or present with vague symptoms. These symptoms can be: indigestion, right hypochondriacpain, PUO, acute/chronic cholecystitis[79]. When it involvesbiliary tree, it can cause symptoms of extrahepatic cholestasis, hemobilia, cholangitis or pancreatitis. Specifically an adenomyoma of the cystic ductpresents with colicky pain and gallbladder hydrops[80].

Patients with generalized adenomyomatous hyperplasia,**ultrasound feature** thickened wall with tiny outpouchesindicating the Rokitansky-Aschoff sinuses. Annular type manifestsanalogous to cholecystitis.

In tiny and plane lesions, ultrasound was unable todistinguish between benign and malignant lesions[81].

They concluded that adenomyomatous hyperplasia is infrequent and patients are clinically silent and when presents with symptoms,they are similar to cholecystopathy. Since there is no suspicion, diagnosis prior to surgery is worrisomeand is found incidentally most of the timeson ultrasound. Surgical excision is widely accepted managementbecause of indefinite evolution of this entity.

In 1983 Raghavendra BN et al,Sonography of adenomyomatosis of the gallbladder: radiologic-pathologic correlation;Adenomyomatosis seen in 9% of post

surgical specimens and accounts for about 25% of polyp like lesions of GB[104]. The focal form of adenomyomatosis may be seen as a fundalpolypoid lesion **at ultrasound**. The segmental form typically affects the body, demonstrates concentric circumferential wall thickening and may give rise to an hourglass configuration of the gallbladder.

Imaging findings of adenomyomatosis parallels its histologic features: intramural diverticula thatmay be filled with inspissated bile and appearas multiple small cystic spaces that are anechoic **at ultrasound**. When the intramural diverticula containsludge, stones or papillary projections, they appearechogenic with multiple acoustic interfacesat ultrasound, creating twinkling or comet-tail artifacts[82]. The cystic spaces may be visible at CT,which can help differentiate between fundal,adenomyomatosisand gallbladder carcinoma[83].

They concluded that adenomyomatous hyperplasia of the GB should be suspected when (a) generalized or annular GB wall thickening and (b) outpouchings within GB wall are seen as anechoic or hyperechoicareas with or without associated acoustic shadows or reverberation artifacts.

Carcinoma gallbladder

In 2008, M Barbhuiya, T Singh, S Gupta, B Shrivastav, P Tiwari, in Incidence of gallbladder cancer in rural and semi-urban population of north central India.

They collected information on the medical diagnosis and demographics of all the 464 different categories of gallbladder disease patients who were admitted for treatment during the above period. Out of 464 patients, 365 had GBC with Gallstone (abbreviated as GSC onwards), 15 had Gallstone (abbreviated as GS), 36 with

Gallbladder Cancer without stone (abbreviated as GC), 30 with Chronic Cholecystitis (abbreviated as CC) and 18 having Gallbladder Cancer with Cholecystitis (abbreviated as GCC). They excluded the data of patients who left the hospital after check- up at outpatient door for personal problems and studied all the comparative data available from the hospital record for those who got admitted and properly diagnosed. The cases were confirmed on the basis of clinical investigations, like X-ray, ultrasound, cytological examination (FNAC), histopathological examination and blood biochemistry reports. About 80% of the diagnosis is based on the ultrasound, chest X-ray and cytological tests (FNAC) and of remaining 20%, after post surgical histopathology. Sample t- test was carried out for the average values of the parameters in five different categories of gallbladder disease (collectively abbreviated as GBD). A total of 419 gallbladder cancer (abbreviated as GBC) with or without gallstones/ cholecystitis were included in the present study. The statistical analysis was performed using Graph Pad Prisma problem in the diagnosis and treatment.

Gallbladder Cancer was the fourth most common cancer constitutingabout 11% of patients admitted at the hospital [84].

In 2007, Randi Get al, Gallbladder carcinoma:found that cholelithiasis is an important risk factor for the development of gallbladder cancer. Up to 95% of gallbladder cancers are associated with gallstones. They conducted cohort study, the relative risk of developing gallbladder cancer in patients with gallstone disease was 8.3 compared to the general population. Case control studies also confirmed the association between cholelithiasis and gallbladder cancer with relative risks ranging widely from 2.3 to 34.4 between different studies. There also appears to be an association between gallstone size and the risk of developing gallbladder cancer.

Patients with gallstones larger than 3 cm have an approximately 10-fold higher risk of developing gallbladder cancer.

Elevated body mass index and multiparity are alsocorrelated with an increased risk of developing gallbladdercancer. Cohort study including over two million people and 1715 cases of gallbladder cancer showeda relative risk of developing gallbladder cancer of 2.53 for women aged 20-44 years patients with a body mass index greater than 30.

Salmonella infection is an important causative factor in the pathogenesis of gallbladder cancer. Themost compelling evidence was from a cohort study based on a typhoid outbreak where 507 cases of typhoid or paratyphoid werereported.

Gallbladder cancer have three distinct appearances on ultrasound: (1) a mass replacing the gallbladderor invading the gallbladder bed, (2) an intraluminalgallbladder growth or polyp, or (3) asymmetric gallbladderwall thickening. In cases of locally advanced disease, USGhas a sensitivity of 85% and an overall accuracy of 80% indiagnosing gallbladder cancer. However, in earlier lesions, especially where the tumor or cancerous polyp is flator sessile and is associated with cholelithiasis, ultrasoundexamination can fail to detect the lesion. In a series of71 patients with early gallbladder cancer, ultrasound hada sensitivity of 53% for sessile tumors. Color Doppler ultrasound may assist in diagnosis as detection of higher flowwithin a lesion has been reported to correlate with malignancy. Besides ascertaining the diagnosis of cancer, ultrasound is also useful in staging the disease by defining theextent of biliary tree involvement as well as confirming thepresence of hepatic arterial or portal venous invasion[85].

In 2007 Alessandro Furlan et al, Gallbladder Carcinoma Update: Multimodality Imaging Evaluation, Staging, and Treatment Options.

In cases of suspected gallbladder disease, sonography is often the first imaging technique because of its relatively low cost and widespread availability.

Gallbladder carcinoma appear as a mass completely occupying or replacing the gallbladder lumen, focal or diffuse asymmetric gallbladder wall thickening, or an intraluminal polypoid lesion[86].

Mass Occupying or Replacing the Gallbladder Lumen, this pattern is seen in 40–65% of patients with gallbladder carcinoma at initial detection. On ultrasound the presence of a large gallbladder mass that nearly fills or replaces the lumen, often directlyinvading the surrounding liver parenchyma, is highly suggestive of gallbladder carcinoma. On ultrasound heterogeneous, predominantly hypoechoic tumor fills much or all of the gallbladder lumen. Anechoic foci of trapped bile or necrotic tumor may be present, as well as echogenic shadowing foci from gallstones, porcelain gallbladder, or tumor calcifications.

The initial detection of gallbladder carcinoma as a polypoid lesion occurs in 15–25% of cases. Malignant lesions are usually larger than 1 cm in diameter and may have a thickened implantation base. The differential diagnosis of a polypoid gallbladder lesion includes adenomatous or hyperplastic cholesterol polyps as well as uncommon tumors such as carcinoid or metastases such as melanoma. At ultrasound, if movement of a polypoid mass occurs with a change of the patient’s position, then a pseudotumor of biliary sludge or clot can be diagnosed.

It is imperative to closely scrutinize the gallbladder, particularly in patients who are at increased risk of developing gallbladder carcinoma, for subtle morphologic abnormalities that indicate cancer. Recognition of the characteristic imaging appearances of primary gallbladder carcinoma and understanding its pathways of spread and staging criteria help optimize patient triage to appropriate treatment regimens.

In 2001 Angela D. Levyet al,Gallbladder Carcinoma: Radiologic-Pathologic Correlation,Primary carcinoma of the gallbladder is an uncommon, aggressive malignancy that affects women more frequently than men. Older age groups are most often affected, and coexisting gallstones are present in the vast majority of cases. The symptoms at presentation are vague and are most often related to adjacent organ invasion.

Ultrasound reveals a mass replacing the normal gallbladder, diffuse or focal thickening of the gallbladder wall, or a polypoid mass within the gallbladder lumen. Adjacent organ invasion, most commonly involving the liver, is typically present at diagnosis, causing biliary obstruction. Periportal and peripancreatic lymphadenopathy, hematogenous metastases and peritoneal metastases are also seen.

Carcinomas that completely replace the gallbladder have irregular margins and heterogeneous echotexture at ultrasound. Heterogeneous echotexture reflects varying degrees of tumor necrosis. Echogenic foci and acoustic shadowing associated with the tumor may be related to coexisting gallstones, gallbladder wall calcification or tumoral calcification. Direct extension to the liver and biliary tree is a common associated finding with large, advanced carcinomas. In these cases, the tumor is inseparable from the adjacent liver.

Wall thickening is the most diagnostically challenging of the three patterns because it mimics the appearance of more common acute and chronic inflammatory conditions of the gallbladder. Subtle areas of wall thickening may reflect early carcinomas. However, they are difficult to detect, since they cause only mild elevation of the mucosa when viewed on ultrasound. Pronounced wall thickening (i.e.,1.0 cm or more) demonstrated by ultrasound, with associated mural irregularity or marked asymmetry should raise concerns for malignancy or complicated cholecystitis.

Knowledge of the varied appearances of gallbladder carcinoma at ultrasound is important so that the diagnosis can be considered preoperatively[87-89].

In 2000, Manoj Pandey et al carcinoma of gallbladder: Role of sonography in diagnosing and staging,made an attempt to define the sonographic characteristics of gallbladder cancer. They retrospectively analyzed the sonographic findings in 203 cases of gallbladder cancer &confirmed by cytology or histopathology.

A mass in the gallbladder and gallbladder wall thickening (> 12 mm) were cardinal sonographic findings of carcinoma,their results have proved that ultrasound is highly accurate for detecting mass lesions, gallstones, liver infiltration, metastasis and ascites.

They concluded that sonography was found to be a good diagnostic tool for carcinoma of the gallbladder; however, its sensitivity was poor for staging nodal spread of the disease[90].

OBSERVATIONS AND RESULTS

TABLE 1: DISTRIBUTION OF CASES ACCORDING TO FINAL DIAGNOSIS

FINAL DIAGNOSIS	N	%
ACUTE CHOLECYSTITIS	25	12.4
ADENOMYOMATOSIS	1	0.5
CALCULOUS CHOLECYSTITIS	13	6.4
CALCULOUS CHOLECYSTITIS WITH ACUTE PANCREATITIS	1	0.5
CHOLECYSTITIS WITH ADENOMYOMATOSIS	1	0.5
CHOLELITHIASIS	110	54.5
CHOLELITHIASIS AND CHRONIC PANCREATITIS	1	0.5
CHOLELITHIASIS WITH ADENOMYOMATOSIS	1	0.5
CHOLELITHIASIS WITH SLUDGE	16	7.9
CHRONIC CHOLECYSTITIS	3	1.5
GB MALIGNANCY	2	1
GB PERFORATION	1	0.5
GB POLYPS	20	10
GB SLUDGE	5	2.5
MEMBRANOUS CHOLECYSTITIS	1	0.5
PANCREATITIS WITH ACUTE CHOLECYSTITIS	1	0.5
TOTAL	202	100

FIGURE 4: DISTRIBUTION OF CASES ACCORDING TO AGE

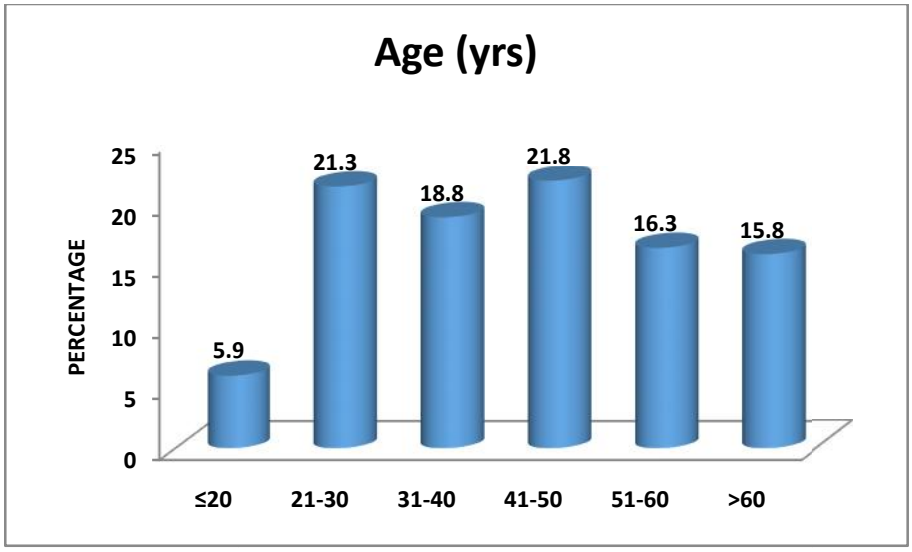


FIGURE 5: DISTRIBUTION OF CASES ACCORDING TO SEX

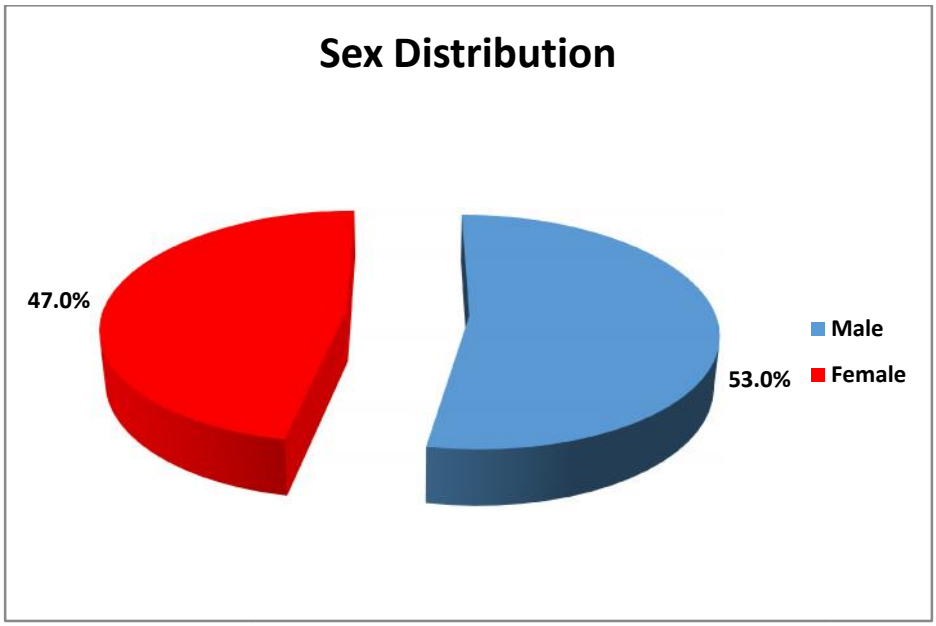


TABLE 2: INCIDENCE OF CHOLELITHIASIS WITH AGE AND SEX

Sl. no	Age	Male	Female	Total
1	≤20	2	0	2
2	21-30	10	15	25
3	31-40	11	13	24
4	41-50	13	10	23
5	51-60	13	5	18
6	>60	7	11	18
Total		56	54(49%)	110

In present study, cholelithiasis was found in110cases, there were 56 males and 54 females. The maximum incidence was in 2nd and 3rdecades

FIGURE 6: DISTRIBUTION OF CASES ACCORDING TO SIZE OF CALCULUS

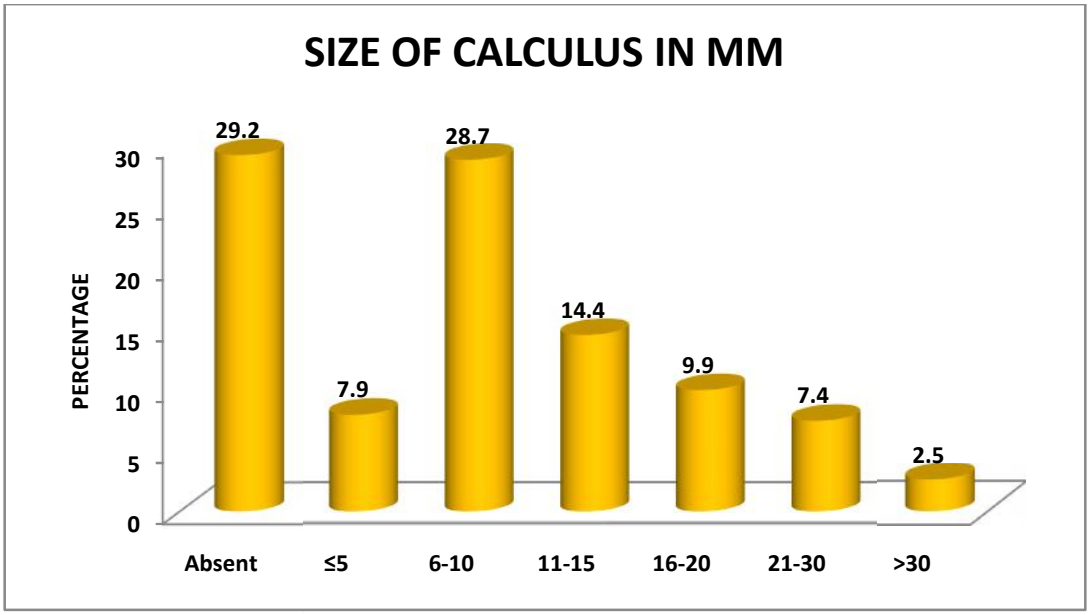


FIGURE 7: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX AMONG ACUTE CHOLECYSTITIS CASES

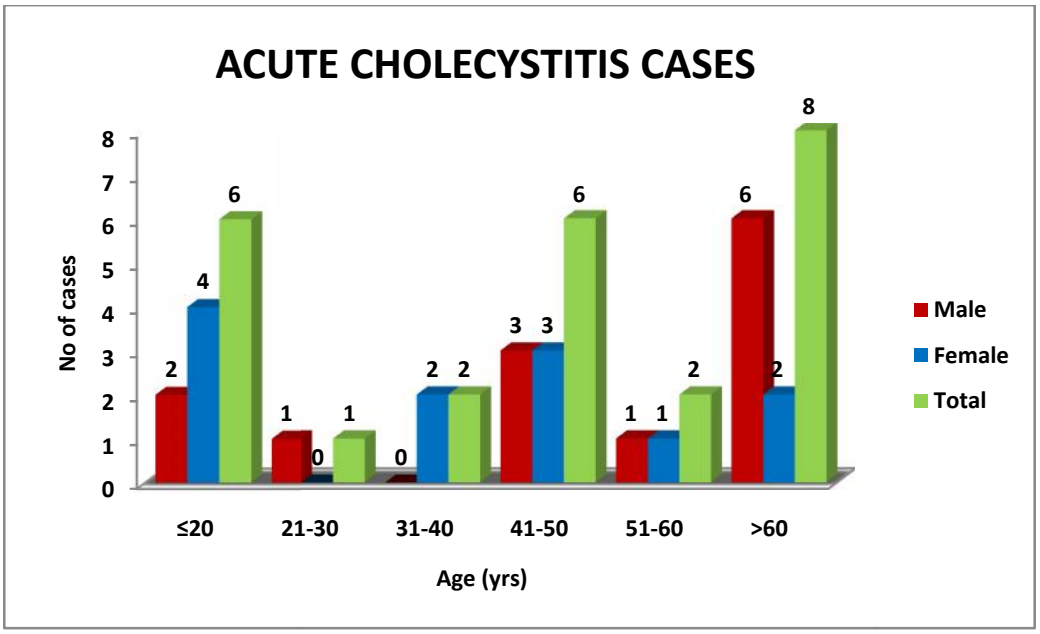


TABLE 3: DISTRIBUTION OF CASES ACCORDING TO GB WALL THICKNESS

GB WALL THICKNESS IN MM	N	%
4-5	22	10.9
6-8	20	9.9
9-10	3	1.5
Total	202	100

TABLE 4: MURPHY'S SIGN AND PERICHOLECYSTIC FLUID AMONG ACUTE CHOLECYSTITIS CASES

	Total	ACUTE CHOLECYSTITIS	% out of total
MURPHY'S SIGN	36	19	52.8
PERICHOLECYSTIC FLUID	26	16	61.5

Murphy's sign and pericholecystic fluid were found to be reliable indicators in cases of acute cholecystitis.

TABLE 5: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX AMONG CHOLELITHIASIS WITH SLUDGE CASES

Age (yrs)	Male		Female		Total		p value
	N	%	N	%	N	%	
≤20	0	0.0	1	9.1	1	6.3	0.581
21-30	0	0.0	3	27.3	3	18.8	
31-40	0	0.0	1	9.1	1	6.3	
41-50	3	60.0	2	18.2	5	31.3	
51-60	1	20.0	3	27.3	4	25.0	
>60	1	20.0	1	9.1	2	12.5	
Total	5	100.0	11	100.0	16	100.0	

16 cases of cholelithiasis were associated with sludge

TABLE 6: DISTRIBUTION OF CASES ACCORDING TO AGE AND SEX AMONG GB POLYPS CASES

Age (yrs)	Male		Female		Total		p value
	N	%	N	%	N	%	
≤20	2	14.3	0	0.0	2	10.0	
21-30	5	35.7	1	16.7	6	30.0	
31-40	3	21.4	4	66.7	7	35.0	
41-50	2	14.3	1	16.7	3	15.0	0.416
51-60	2	14.3	0	0.0	2	10.0	
>60	0	0.0	0	0.0	0	0.0	
Total	14	100.0	6	100.0	20	100.0	

TABLE 7: DISTRIBUTION OF CASES ACCORDING TO NO. OF POLYPS

NO. OF POLYPS	N	%
1	15	7.4
2	0	0
3	4	2
4	0	0
5	1	0.5
Total	202	100

INCIDENCE OF GALLSTONES IN OTHER CONDITIONS

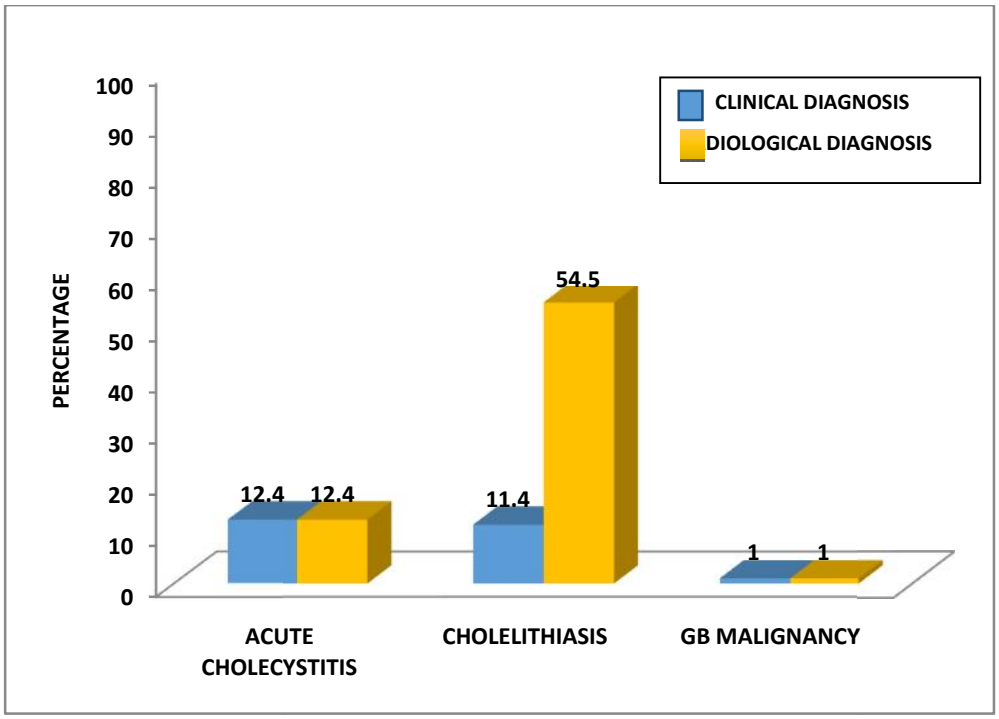
Following conditions, each of 1 case showed evidence of gallstones which include,adenomyomatosis,acute pancreatitiswith cholecystitis and chronic pancreatitis.

INCIDENCE OF OTHER GALLBLADDER DISEASES

Adenomyomatosis -3

Membranous cholecystitis – 1

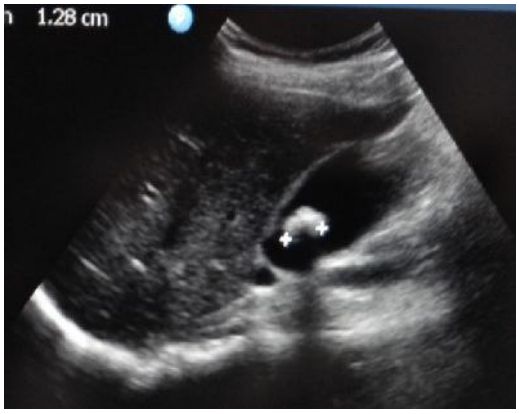
FIGURE 8: COMPARISON OF PARAMETERS BETWEEN CLINICAL AND RADIOLOGICAL DIAGNOSIS



IMAGES

Case 1

CHOLELITHIASIS



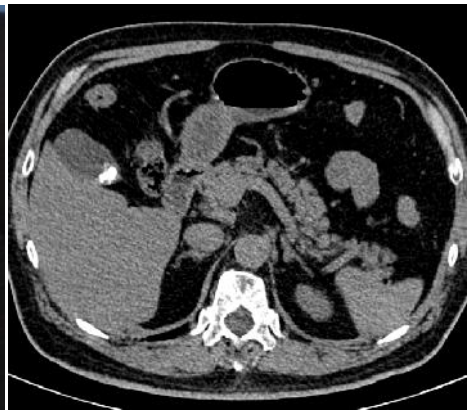


FIG 9 **FIG 10**

USG image showing well defined hyperechoic focus with posterior acoustic shadowing, corresponding CT shows hyperattenuating gall stone at the neck of gall bladder

Case 2

GALLBLADDER SLUDGE

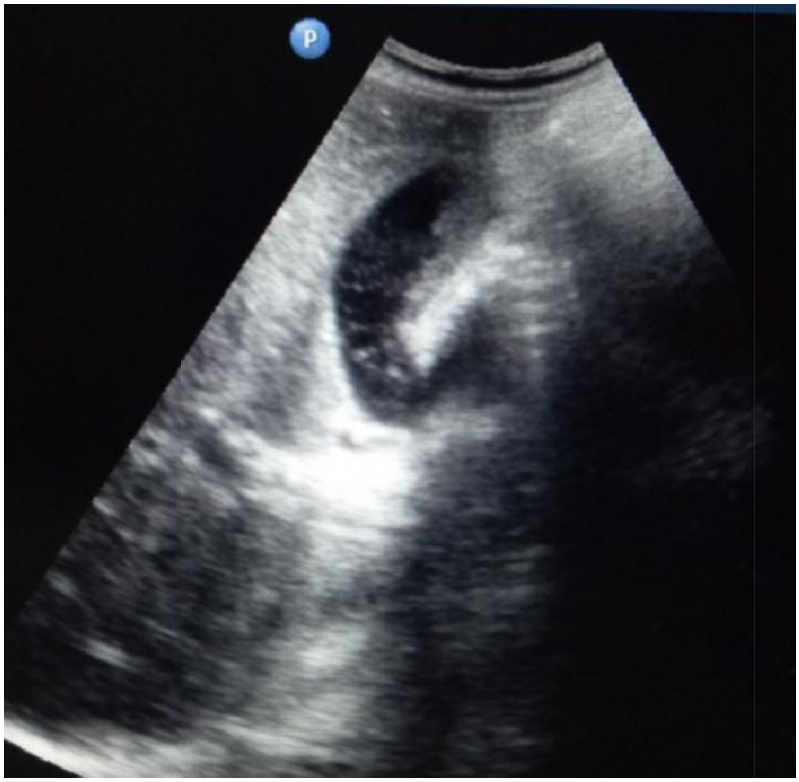


FIG 11

USG image showing hyperechoic content at the dependent part of the body and internal echoes indicative of sludge

Case 3

ACUTE CHOLECYSTITIS

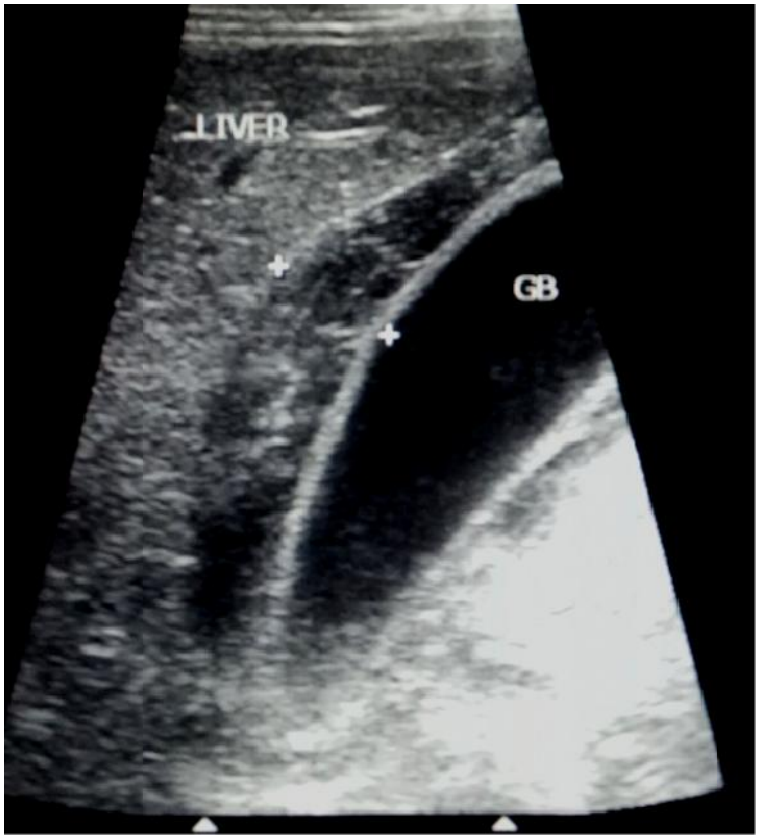


FIG 12

USG image showing thickening of gall bladder wall with mural edema and mild pericholecystic fluid

Case 4

CALCULUS CHOLECYSTITIS

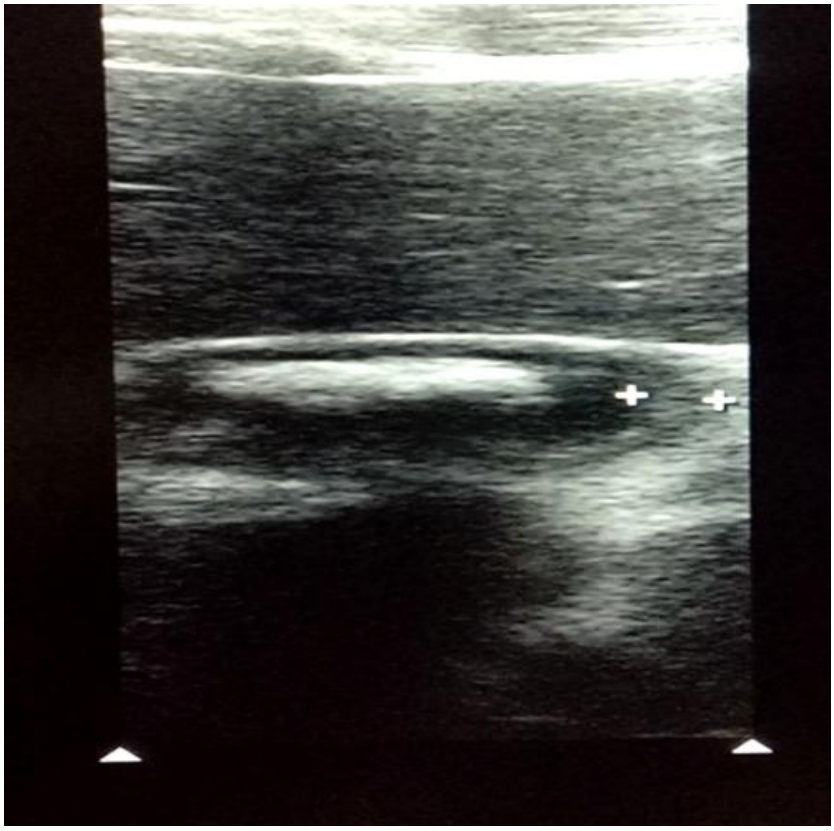


FIG 13

USG image showing gall bladder wall thickening with echogenic focus within lumen

Case 5

MULTIPLE GALLBLADDER POLYPS

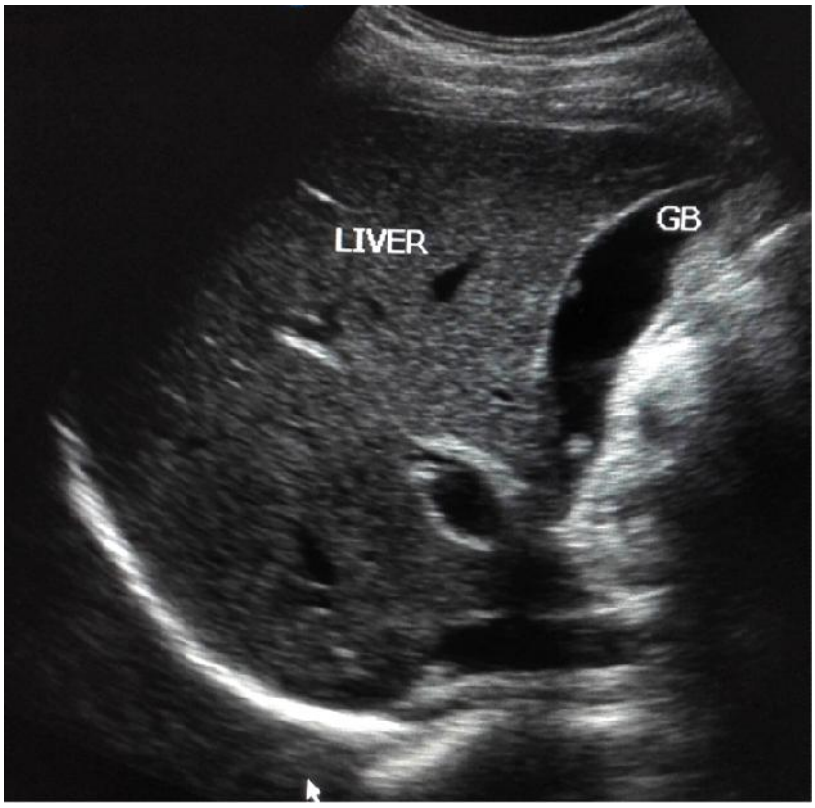


FIG 14

USG image showing multiple well defined echogenic foci adherent to wall

Case 6

ADENOMYOMATOSIS

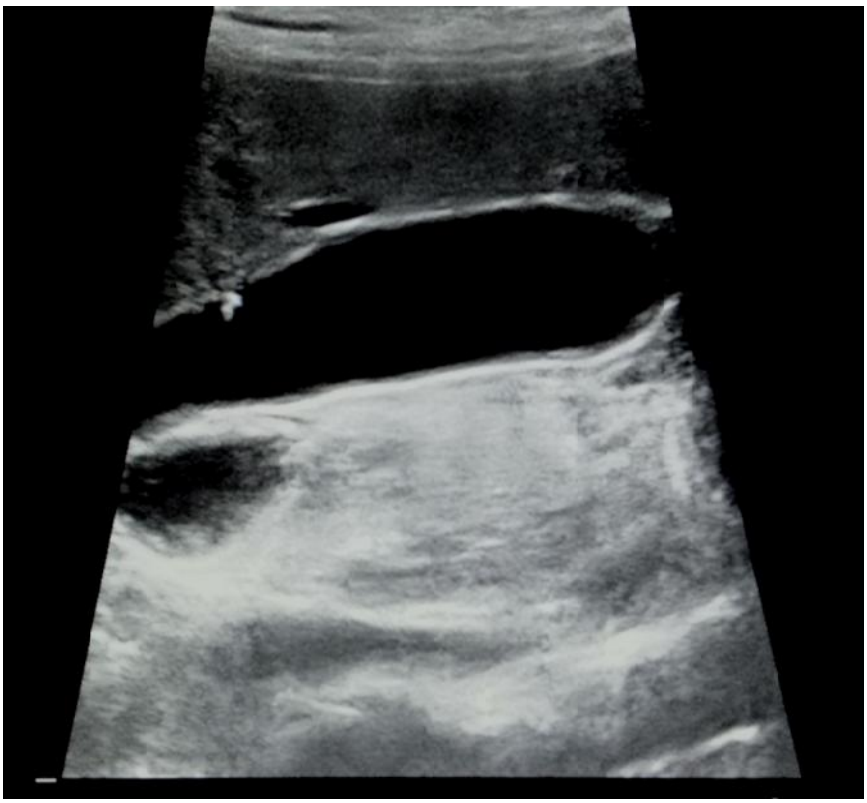


FIG 15

USG image showing well defined hyperechoic focus with comet tail artefact

Case 7

MEMBRANOUS CHOLECYSTITIS

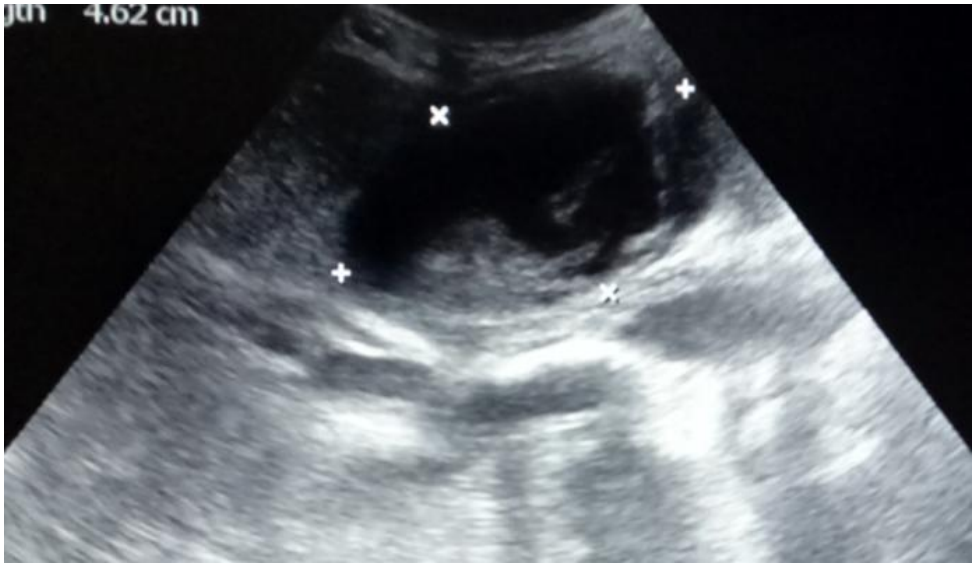


FIG 16

USG image showing well detached membranes within lumen and asymmetrical wall thickness

Case 8

CARCINOMA GALLBLADDER WITH HEPATIC INVASION

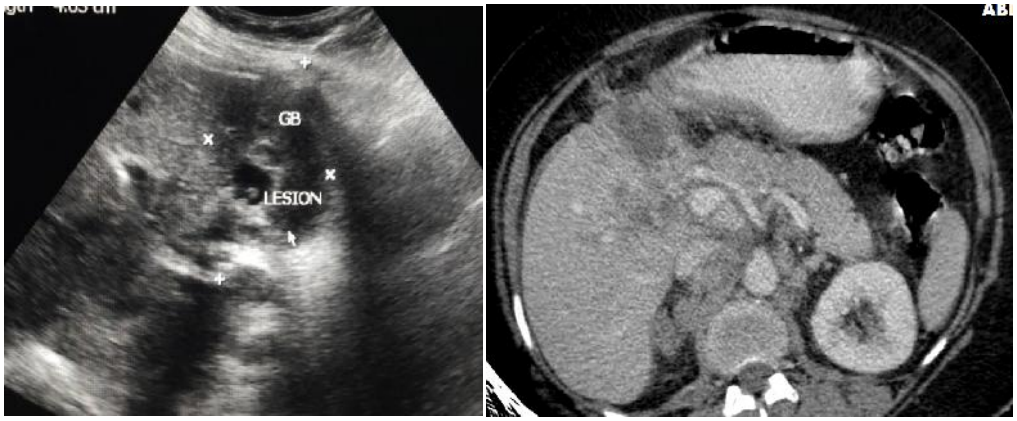


FIG 17FIG 18

USG image showing ill defined heteroechoic irregular mass lesion in gall bladder fossa completely obscuring gall bladder, corresponding CT shows heterogeneously hypodense lesion in the GB fossa with hepatic invasion

Case 9

GALLBLADDER MALIGNANCY

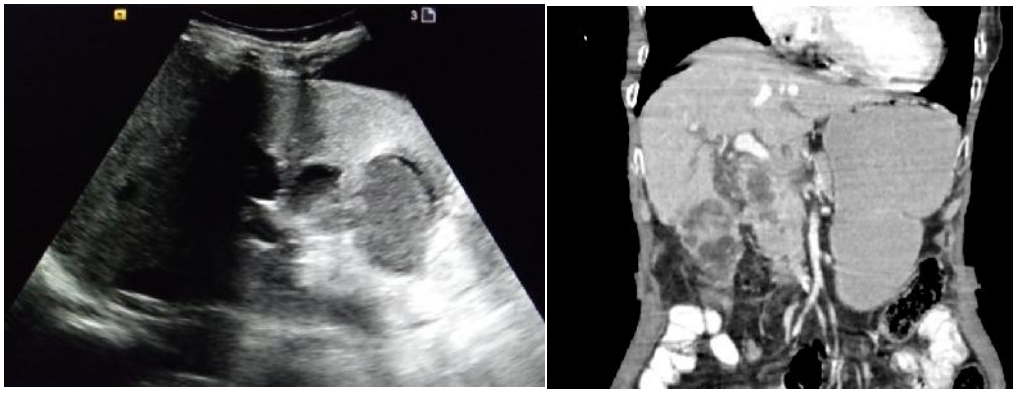


FIG 19FIG 20

USG image showing ill defined mass lesion nearly obscuring the lumen of gall bladder, corresponding CT shows heterogeneously hypodense lesion in the gall bladder

DISCUSSION

Gallbladder diseases are very common entities with diverse features of presentation at clinical, radiological settings, with variable morbidity and mortality with altered biliary function.

In our study, majority of them involvegallstones (54.5%), followed by inflammatory conditions and neoplasms.

Among inflammatory conditions, acute cholecystitis was common.

Others include membranous cholecystitis, polyps, adenomyomatosis and carcinoma.

Benign neoplasms are very rare, compared to malignant ones but are uncommon to inflammatory conditions.

Gallbladder diseases were seen in all age groups, with peak incidence at second and fifth decades.

Incidence of gallbladder diseases with age and sex

In our study there were 107 males and 95 females, and maximum incidence was seen in 2^{nd} and 5^{th} decades.

Clare Bayram et al. Gallbladder disease; has described that prevalence of GBD increases with age, and is more common in women than men[91].

Both the studies showed similar age distribution however, there was male predominance noted in our study. This variation may be due to changing life style and limited period of study.

Incidence of gallstones with age and sex

In present study, cholelithiasis was found in110 cases, there were 56 males and 54 females. The maximum incidence was in 2nd and 3rddecades.

Hardeep Singh Gill et al. epidemiology of gallstone disease; in a study of 50 cases, statedGallstones occurred in relatively younger patients, fifty percent of whom were between 11-40 years while 24% were between 41-50 years and 4% were between 11-20 years, prevalence increased with age in both sexes reaching a maximum in the third decade in men and fourth decade in women[92].

Incidence of gallstones in our study was 54% with detection accuracy of 100%.

Significance of size of gallstones

In our study, among 110 cases of cholelithiasis, 58 cases (28.7%) were between 6-10 mm, 29 cases were 11-15 mm and 20 cases in range of 16-20 mm.

In a similar study done by Sultan Alshoabi[93] on the gallstones and their characteristics like site, size, prevalence by ultrasonography, observed 184 patients with gallstones,11-20 mm in 54 cases (29.51%),followed by less than 5mm in 38 cases (20.77%), 20-30 mm in 24 cases (13.11%) and so on.

Results were similar in both the studies.

Association of cholelithiasis with sludge:

In our study, 16 cases of cholelithiasis were associated with sludge of which 11 were females and 5 were males, most common in the 4th decade and in females.

In a similar study by Sauerland S. stated, children under the age of 16 years rarely develop gallstones. In adults, prevalence steadily increases;female gender is an important risk factor for biliary lithiasis[94].

Incidence of acute cholecystitis with age and sex:

Among 25 cases, 13 were males and 12 females, after 2nd decade incidence increased with age.

In a similar study done by, Eskelinen M et al. Acute cholecystitis cases account for 3%–10% of all patients with abdominal pain among 1333 patients[95].

Telfer S et al. The percentage of acute cholecystitis cases in patients under 50 years old with abdominal pain (n = 6317) was low, at 6.3%, whereas that in patients aged 50 years and over (n = 2406) was high, at 20.9%[96].

Both the studies showed similar results of increased prevalence with age group.

GB wall thickness in acute cholecystitis:

In our study, wall thickness of more than 3 mm is taken as cut-off value for acute cholecystitis, 22 cases were between 4-5 mm, 20cases in range of 6-8 mm, 3 cases of 9-10 mm.

In a similar study, Deitch and Engel J M. Thickening of the gallbladder wall is the most reliable criterion with reported specificity of 90% using 3.0 mm and 98.5% at a 3.5 mm wall thickness, whereas sensitivity was 100% at 3.0 mm but only 80% at 3.5 mm, they recommended acceptance of gallbladder wall thickness of 3.5 mm or

greater as definitive evidence of acute cholecystitis, whereas 3.0 mm is suggestive but not conclusive evidence[97].

Murphy's sign and pericholecystic fluid in acute cholecystitis:

In acute cholecystitis along with wall thickness of >3mm,sonographic Murphy's sign and pericholecystic fluid were found to be more reliable indicators.

In a similar study done by VriesmanAC et al,In 2007,Accuracy in diagnosing acute cholecystitis increased when using a combination of findings includingcholelithiasis, gallbladder wall thickening and a positive sonographic Murphy's sign[36].

Incidence of calculus cholecystitis with age and sex:

In our study, there were 13 cases with 10 males and 3 females, 6 patients were seen between 41-60 yrs in males and all 3 females are in the age group of 21-30 yrs.

In a similar study done by R.Jai Vinod Kumar et al, studied among 50 cases, 33 were females and 17 were males and maximum incidence was seen in 3rd and 4th decades[98].

In contrast to the above study male predominance was seen in our study and increased incidence was also seen in 5th decade.

Incidence of GB polyps with age and sex

In present study, there were 20 cases of gallbladder polyps with 14 males and 6 females, increased age of incidence in 21 – 40 yrs.

In a similar study W Kratzer et al. studied among1027 patients, 128 cases were GB polyps and sex incidence was fairly equal of about 6.1 % each, average age of presentation was 42 yrs[99].

Significance of size of GB polyps

In our study among 20 cases of GB polyps, 9 cases were between 3-4 mm, 3cases each in 1-2 mm and 5-6 mm, remaining 5 cases were of 10 mm or greater.

Polyps of more than 10 mm were considered to be with increased risk of malignancy in older age group (>60 yrs) and were followed up with subsequent scans for increase in size.

In a similar study by Michael T. Corwin et al. studied on 346 patients with mean age 52 yrs (20–93 years) and found that risk of malignancy is more in polyps of 1 cm or morediameter,single polyp,sessile polyps, polyps with adjoining increased wall thickness and also with increasing age.When two or more polypswere recognized, and increase in size of 0.2 cm or more on follow-up study, was of significance. They were distinguishedas stable, resolved, increased/reduced in size on the basis of the highest lengthmeasurement[61].

Comparision between clinical and radiological diagnosis in cholelithiasis, acute cholecystitis, GB malignancy

In our study, cholelithiasis was detected without fail on ultrasound in 54% of cases which was far superior to clinical suspicion which was only 11.4 %, acute cholecystitis was detected both clinically and radiologically with equal rates of detection (12.4 %), and among 2 cases of malignancy,intraluminal mass replacing the gallbladder with intrahepatic biliary dilatation, adjacent hepatic invasion seen in one

case on both ultrasound and CT. In second case clinically it was suspected as hepatoma.

In 2007, Randi G et al, Mass Occupying or replacing the Gallbladder lumen, was seen in 40–65% of patients with gallbladder carcinoma atinitial detection. On ultrasound the presence of a large gallbladder mass that replaces the lumen, directly invading the surrounding liver parenchyma is highly suggestive of gallbladder carcinoma.

In comparison, ultrasound reliably recognized both the malignant cases[85].

In 3 cases of focal adenomyomatosis, comet tail reverberation artefact was seen and is diagnostic feature.

In 2006 Boscak AR et al, Echogenic intramural foci from which emanate V-shaped comet tail reverberation artefactsrepresenting the unique acoustic signature of cholesterol crystals within the lumen of Rokitansky-Aschoff sinuses, are highly specific for adenomyomatosis[63].

CONCLUSION

Gallbladder diseases are very common and it is appropriate to accentuate that, understanding of these conditions is essential. In present study of 202 patients, an attempt was made to correlate clinical & ultrasound features of gallbladder diseases and to ascertain the sensitivity of ultrasound in evaluation of gallbladder diseases.

The ultrasound diagnosis in gallbladder diseases had favourable&impressive results against clinical diagnosis.

Ultrasound could detect gallstones and acute cholecystitis unfailingly; other conditions such as calculous cholecystitis, cholelithiasis with sludge, adenomyomatosis, polyps, membranous cholecystitis, carcinoma were also diagnosed.

Only in cases of chronic cholecystitis ultrasound had limited diagnostic value since wall thickness was not appreciable in all the cases and hence it was tricky to recognize without prior clinical history.

In case of malignancy, ultrasound was inferior to CT in staging and also in detecting lymph nodes.

The use of ultrasound is rapid taking only few minutes, non-invasive, no ionizing radiation, easy to perform and interpret, cost effective with good repeatability and reliability, the features which help to score over other imaging modalities in emergency situations.

SUMMARY

Gallbladder diseases include gamut of conditions, and are seen almost everyday in radiology practice, early detection of which helps in bringing down the morbidity and mortality associated with them.

Patients with gallbladder diseases usually presents with pain in the right hypochondriac region.Certain conditions like carcinoma gallbladder present with vague manifestations, cases of gallbladder polyps are asymptomatic.

In such conditions, the inclusion of ultrasound abdomen in routine investigation protocol will help in early diagnosis and initiation of therapy.

Ultrasound has the highest sensitivity and specificity for evaluating patients with suspected biliary pathologies.

Abdominal ultrasound is the first imagingtechnique employed for patients presenting withbiliary-type symptoms as it is more accurate thanCT for diagnosing acute biliary diseases.

The overall good sensitivity, specificity and accuracy of ultrasound in diagnosis of gallbladder diseases has led to the proposition that ultrasound is the diagnostic technique of choice in evaluation of gallbladder diseases.

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ANNEXURES

ANNEXURE I

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR – 586103
INSTITUTIONAL ETHICAL COMMITTEE No/Sec/2015 20/11/15

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 17-11-2015 *at* 03 pm *scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.*

Title "Ultrasound Study of Gall bladder diseases"

Name of P.G. Student : Dr. Holebasu
Dept of Radiology

Name of Guide/Co-investigator : Dr. B.R. Dhamangaonkar
Professor

DR.TEJASWINI VALLABHA
CHAIRMAN

CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:
1)Copy of Synopsis/Research Project
2)Copy of informed consent form.
3)Any other relevant documents.

ANNEXURE II

CASE PROFORMA

Name: Age: Sex: MRD No:

Chief complaints:

History of present illness:

1. Pain abdomen
2. Vomiting
3. Fever

PAST HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

CLINICAL DIAGNOSIS:

ANNEXURE – III

INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, BIJAPUR – 586103, KARNATAKA

TITLE OF THE PROJECT **:** ULTRASOUND STUDY OF GALLBLADDER DISEASES

PRINCIPAL INVESTEGATOR : DR.HOLEBASU

DEPARTMENT OF RADIODIOLOGY

Email:holebasu@yahoo.co.in

PG GUIDE : DR.B.R.DHAMANGAONKAR$_{MDRD}$

PROFESSOR

DEPT. OF RADIOLOGY

BLDE UNIVERSITY'S,

SHRI B. M. PATIL MEDICAL COLLEGE

VIJAYAPURA.

PURPOSE OF RESEARCH:

I have been informed that this is being done todescribe the role of ultrasound in evaluationof gallbladder diseases.

I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I/my ward have been explained that, I/my ward will be subjected to ultrasound scan of abdomen to describe various diseases regarding the gallbladder.

RISKS AND DISCOMFORTS:

I/my ward understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I/my ward understand that my participation in this study will describe the role of ultrasound in the study of gallbladder diseases.

CONFIDENTIALITY:

I/my ward understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigators research file

and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may askmore questions about the study at any time**.** **Dr.Holebasu** is availableto answer my questions or concerns. I/my ward understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. And that a copy of this consent form will be given to me for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I/my ward understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I/my ward also understand that Dr.Holebasuwill terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: Dr. B.R.Dhamangaonkar Dr. Holebasu

(Guide) (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I/my ward confirm that Dr.Holebasu has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I/my ward have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

KEY TO MASTER CHART

ACUTE CHOLECYSTITIS	-	AC
CHRONIC LIVER DISEASE	–	CLD
ACUTE GASTRITIS	–	AG
ACUTE PEPTIC DISEASE	-	APD
OBSTRUCTIVE JAUNDICE	-	OJ
PANCREATITIS	-	P
CHOLELITHIASIS	–	CL
CARCINOMA GB	–	CA GB
ALCHOLIC LIVER DISEASE	–	ALD
GALLBLADDER	–	GB
SLUDGE	–	SL
CALCULOUS CHOLECYSTITIS	-	CC

SL NO.	NAME	AGE/SEX	DATE	OP/IP NO.	CLINICAL DIAGNOSIS	SONOGRAPHIC MURPHY'S SIGN	SIZE OF CALCULUS IN MM	SL	GB WALL THICKNESS IN MM	PERICHOLECYSTIC FLUID	FOCAL WALL THICKENING WITH VASCULARITY ON COLOR DOPPLER	NO. AND SIZE OF POLYP IN MM		DETACHED MEMBRANES	ADENOMYOMATOSIS	PERFORATION	CT FINDINGS	FINAL DIAGNOSIS
1	DEEKSHA	6/F	2.11.2015	401523	AC	Y	A	A	5	P	A	A		A	A	A		AC
2	NIRMALA	12/F	2.11.2015	35713	HEPATITIS	N	A	P	A	A	A	A		A	A	A		GB SL
3	KALLAPPA	54/M	4.11.2015	404385	CLD	N	8	A	A	A	A	A		A	A	A		CL
4	LALITA	30/F	10.11.2015	412487	AG	N	14	P	A	A	A	A		A	A	A		CL WITH SL
5	GURABAI	52/F	10.11.2015	412506	APD	N	6	A	A	A	A	A		A	A	A		CL
6	SHAILA	22/F	12.11.2015	36723	AG	N	A	P	A	A	A	A		A	A	A		GB SL
7	HANAMANTAPPA	63/M	13.11.2015	414802	CLD	N	A	A	8	P	A	A		A	A	A		AC
8	DEEPA	21/F	16.11.2015	418338	AC	Y	7	A	6	P	A	A		A	A	A		CC
9	GOURAMMA	66/F	19.11.2015	422691	AG	N	18	A	A	A	A	A		A	A	A		CL
10	BASAVARAJ	63/M	24.11.2015	38043	APD	N	10	P	A	A	A	A		A	A	A		CL WITH SL
11	MEGHA	6/F	26.11.2015	38308	OJ	Y	A	A	5	P	A	A		A	A	A		AC
12	RAMAGOND	45/M	28.11.2015	434801	CL	N	17	A	A	A	A	A		A	A	A		CL
13	ZAMEER	50/M	1.12.2015	439173	P	N	A	A	5	A	A	A		A	A	A		AC
14	KASIMSAB	50/M	3.12.2015	441943	CLD	N	9	A	A	A	A	A		A	A	A		CL
15	GEETA	36/F	4.12.2015	39181	AG	N	15	A	A	A	A	A		A	A	A		CL
16	PRAKASH	60/M	4.12.2015	442065	APD	N	A	A	A	A	A	1	3	A	A	A		GB POLYP
17	SULOCHANA	40/F	8.12.2015	447109	AC	Y	A	A	6	P	A	A		A	A	A		AC
18	KEERTI	45/F	9.12.2015	449602	HEPATITIS	N	A	A	A	A	A	3	3	A	A	A		GB POLYPS
19	MEENAXI	42/F	16.12.2015	457573	P	N	A	A	5	P	A	A		A	A	A		AC
20	TUBSUM	65/F	17.12.2015	406100	PUO	N	12	A	A	A	A	A		A	A	A		CL
21	BAYAKKA	43/F	18.12.2015	460481	AC	Y	A	A	10	P	A	A		A	A	A		AC
22	SHIVAKUMAR	44/M	21.12.2015	465162	APD	N	16	P	A	A	A	A		A	A	A		CL WITH SL
23	SHANTABAI	69/F	22.12.2015	41175	PUO	N	9	A	A	A	A	A		A	A	A		CL
24	MANJULA	46/F	26.12.2015	469979	CL		14	A	A	A	A	A		A	A	A		CL
25	MALLIKARJUN	32/M	5.1.2016	4837	AG	N	6	A	A	A	A	A		A	A	A	A	CL
26	GURU	28/M	8.1.2016	715	AG	N	7	A	A	A	A	A		A	A	A	A	CL
27	KRISHNAPPA	70/M	9.1.2016	861	AC	Y	A	A	6.6	A	A	A		A	A	A	A	AC
28	PARAGOND	55/M	10.1.2016	12105	AC	Y	9	A	4.8	P	A	A		A	A	A	A	CC
29	BHUVANESHWARI	45/F	12.1.2016	1106	HEPATITIS	Y	A	A	6	A	A	A		A	A	A	A	AC
30	MAHADEVI	52/F	19.1.2016	23439	APD	N	A	P	A	A	A	A		A	A	A	GB SL	GB SL
31	MALLAPPA	58/M	19.1.2016	1937	OJ	N	A	P	4	A	Y	A		A	A	A	AC	AC
32	SAHEBI	40/F	20.1.2016	25580	CL	N	30	A	A	A	A	A		A	A	A	A	CL
33	SAVITRI	30/F	21.1.2016	25541	AC	Y	9	A	7	P	A	A		A	A	A	A	CC

34	SUMITRA	48/F	26.1.2016	2457	APD	N	32	P	A	A	A	A		A	A	A	A	CL
35	HANAMAWWA	28/F	26.1.2016	2686	AG	N	17	A	A	A	A	A		A	A	A	A	CL
36	LALBI	28/F	30.1.2016	3248	CL	N	22	A	A	A	A	A		A	A	A	A	CL
37	SHANMUKH	32/M	01.02.2016	41462	AG	N	9	A	A	A	A	A		A	A	A		CL
38	SHARADA	35/F	05.02.2016	3925	AC	Y	25	A	A	A	A	A		A	A	A		CL
39	KASHIBAI P	43/F	10.02.2016	4549	APD	N	26	A	A	A	A	A		A	A	A		CL
40	KALLAPPA	65/M	27.02.2016	6162	AC	Y	A	A	5	P	A	A		A	A	A		AC
41	SATTU	70/M	02.03.2016	6993	HEPATITIS	Y	A	A	4.5	P	A	A		A	A	A		AC
42	SHANKAR JAMAKHANDI	28/M	02.03.2016	104113	CLD	N	14	A	4	A	A	A		A	A	A	CALCULUS IS NOTED IN THE NECK OF GB. WITH WALL THICKENING	CC
43	LALITHABAI	60/F	03.03.2016	82878	AG	N	6	A	A	A	A	A		A	A	A		CL
44	VIJAYLAXMI	34/F	05.03.2016	84884	HEPATITIS	N	10	P	A	A	A	A		A	A	A	CALCULUS IN NECK OF GB WITH SL	CL WITH SL
45	SHIREPPA	80/M	08.03.2016	5917	APD	N	24	A	A	A	A	A		A	A	A		CL
46	BHAVASING	43/M	09.03.2016	91144	AC	Y	4	A	12	P	A	A		A	A	A	GB WALL THICKENING WITH MULTIPLE CALCULI IN THE NECK AND PERICHOLECYSTIC FAT STRANDING IS SEEN.	CC
47	REVANASIDDA	30/M	13.03.2016	95103	AC	N	A	A	A	A	A	A		A	P	A		ADENOMYOMATOSIS
48	BORAMMA	30/F	17.03.2016	98925	CL	N	6	A	A	A	A	A		A	A	A		CL
49	NEELAMMA	40/F	21.03.2016	104069	APD	N	A	A	A	A	A	1	8	A	A	A		GB POLYP
50	BALAWWA	80/F	26.03.2016	111472	AG	N	13	A	A	A	A	A		A	A	A		CL
51	RESHMA	30/F	04.04.2016	11017	P	N	16	A	A	A	A	A		A	A	A		CL
52	MAHADEVI	24/F	05.04.2016	11201	OJ	Y	7	A	7.5	A	A	A		A	A	A		CC
53	BHARATI	34/F	05.04.2016	123519	AG	N	A	A	A	A	A	3	5	A	A	A		GB POLYPS
54	SHOBHA	45/F	12.04.2016	11920	AC	Y	15	P	A	A	A	A		A	A	A		CL WITH SL
55	SUNITA	29/F	15.04.2016	135803	AG	N	21	A	A	A	A	A		A	A	A		CL
56	VIJAYA	13/M	15.04.2016	12620	HEPATITIS	Y	A	A	7	P	A	A		A	A	A		AC
57	PARVATI	30/F	17.04.2016	138214	OJ	N	18	P	A	A	A	A		A	A	A		CL WITH SL
58	SHARADABAI	50/F	21.04.2016	143586	CL	N	21	A	A	A	A	A		A	A	A		CL
59	SHANTABAI	35/F	01.05.2016	154861	APD	N	23	A	A	A	A	A		A	A	A		CL
60	YALLAWWA	30/F	04.05.2016	15924	P	N	7	A	A	A	A	A		A	A	A		CL

61	RAJAKSAB	40/M	04.05.2016	14743	P	N	9	A	A	A	A	A		A	A	A	GB CALCULUS NON-ENHANCING LESION (NECROSIS) IN THE BODY OF PANCREAS, PERIPANCREATIC FAT STRANDING AND MULTIPLE CALCIFIC FOCI IN HEAD OF THE PANCREAS.	CL AND CHRONIC P
62	MANGALU RATHOD	75/M	05.05.2016	15005	HEPATITIS	N	10	A	A	A	A	A		A	A	A		CL
63	SATAWWA	70/F	06.05.2016	14941	PUO	N	20	A	A	A	A	A		A	A	A		CL
64	BHIMABAI	70/F	14.05.2016	15960	P	N	28	P	A	A	A	A		A	A	A		CL WITH SL
65	SANGEETA	30/F	16.05.2016	173952	APD	N	7.5	A	A	A	A	A		A	A	A		CL
66	SAROJINI	51/F	17.05.2016	16236	HEPATOMA	N	A	A	A	A	P	A		A	A	A	IRREGULARLY THICKENED GB WALL WITH INVASION INTO LIVER	GB MALIGNANCY
67	BHIMBAI	70/F	18.05.2016	15960	AC	Y	A	A	4	P	A	A		A	A	A	GB IS OVER DISTENDENDED WITH THICKENED WALL AND PERICHOLECYSTITIC FAT STRANDING.	AC
68	ROOPA	65/F	21.05.2016	180641	AC	Y	A	A	8	A	A	A		A	A	A		AC
69	SHRISHAIL	42/M	21.05.2016	16906	AC	N	A	A	13	A	A	A		A	A	A		AC
70	MALLAPPA	60/M	22.05.2016	181319	P	N	18	A	4.5	A	A	A		A	A	A		CC
71	REVANASIDDA	25/M	23.05.2016	182813	P	Y	10	A	4	A	A	A		A	A	A	GB IS DISTENDED WITH CALCULUS IN NECK AND WALL THICKENING WITH ENHANCEMENT	CC
72	RAJSHEKAR	23/M	23.05.2016	182262	AG	N	11	A	A	A	A	A		A	A	A		CL
73	UMA R JADHAV	46/M	24.05.2016	17142	P	Y	15	A	5	A	A	A		A	A	A	MULTIPLE CALCULI WITH ENHANCEMENT AND THICKENING OF GB WALL. BULKY ENCHANCING PANCREAS WITH PERIPANCREATIC FLUID COLLECTION	CC WITH ACUTE P
74	AMBIKA	35/F	26.05.2016	188933	HEPATITIS	N	6	A	A	A	A	A		A	A	A		CL
75	SIDDAPPA	68/M	26.05.2016	17406	P	Y	A	A	6	P	A	A		A	A	A		AC
76	TIMANNAGOUDA	49/M	28.05.2016	188500	APD	N	10	A	A	A	A	A		A	A	A		CL
77	RAJ KUMAR	43/M	28.05.2016	17731	CL	N	9	A	A	A	A	A		A	A	A		CL

78	MALLAPPA	49/M	02.06.2016	194253	OJ	N	10	A	A	A	A	A		A	A	A		CL
79	RAJSHEKAR	23/M	05.06.2016	18460	APD	N	11	A	A	A	A	A		A	A	A		CL
80	SWATHI	14/F	05.06.2016	18636	AC	Y	A	A	10	P	A	A		A	A	A		AC
81	IRAYYA	80/M	05.06.2016	18632	PUO	N	A	P	A	A	A	A		A	A	A		GB SL
82	SHIVAPPA	59/M	07.06.2016	18722	AG	N	7	A	A	A	A	A		A	A	A		CL
83	M S CHAVAN	46/F	08.06.2016	202491	P	N	11	A	A	A	A	A		A	A	A		CL
84	MAHADEV	46/M	09.06.2016	228798	CA GB	N	4	A	A	A	P	A		A	A	A	PEDUNCULATED IRREGULAR MINIMALLY ENHANCING LESION IN THE BODY OF GB	GB MALIGNANCY
85	NEELAWWA	40/F	10.06.2016	204738	APD	N	8	A	A	A	A	A		A	A	A		CL
86	CHANDRAMAPPA	62/M	12.06.2016	26785	CHOLECYSTITIS	N	A	P	6.7	A	A	A		A	A	A		CHRONIC CHOLECYSTITIS
87	SHIVAKUMAR	42/M	16.06.2016	210843	P	N	12	P	A	A	A	A		A	A	A		CL WITH SL
88	GEETA	55/F	18.06.2016	19010	AC	Y	A	A	9	P	A	A		A	A	A		AC
89	TIMANGOUDA PATIL	49/M	20.06.2016	216777	P	N	30	A	A	A	A	A		A	A	A		CL
90	CHANDRAKANTH	57/M	21.06.2016	20432	APD	Y	A	A	4	A	A	A		A	A	A		CHRONIC CHOLECYSTITIS
91	MADIWALAMMA PUJARI	57/F	24.06.2016	222839	CL	N	7	A	A	A	A	A		A	A	A		CL
92	MALLAPPA S H	70/M	28.06.2016	20887	PERITONITIS	N	A	A	A	A	A	A		A	A	3 MM	GB IS OVER DISTENDED WITH A DEFECT AND COLLECTION COMMUNICATING INTO THE PERITONEAL CAVITY	GB PERFORATION
93	ABDUL KHADER	65/M	30.06.2016	21640	ALD	N	3	A	A	A	A	A		A	A	A	GB SHOWS MULTIPLE CALCULI	CL
94	SHRISHAIL	56/M	04.07.2016	23408	AG	N	14	A	A	A	A	A		A	A	A		CL
95	MALEWWA	65/F	05.07.2016	21173	PUO	N	34	A	A	A	A	A		A	A	A		CL
96	AMBUJA	59/F	06.07.2016	236476	CL	N	7	P	A	A	A	A		A	A	A		CL WITH SL
97	PARASHURAM	24/M	07.07.2016	25665	P	N	13	A	A	A	A	A		A	A	A		CL
98	AMBIKA	45/F	17.07.2016	23219	AG	N	6.5	A	A	A	A	A		A	A	A	CL	CL
99	NAGAMMA PATIL	25/F	19.07.2016	252348	AG	N	16	A	A	A	A	A		A	A	A		CL
100	MEENAKSHI N HIREMATH	35/F	19.07.2016	252339	APD	N	A	A	A	A	A	1	3.6	A	A	A		GB POLYP
101	BASAVARAJ	48/M	20.07.2016	252325	CL	N	10	A	A	A	A	A		A	A	A		CL
102	GOURAMMA TORAVI	58/F	22.07.2016	256461	AG	N	10	A	A	A	A	A		A	A	A		CL
103	INDUMATI	44/F	28.07.2016	262265	P	N	23	A	A	A	A	A		A	A	A		CL
104	SS PUJAR	58/M	02.08.2016	268380	AC	Y	15	P	5	P	A	A		A	A	A		CC
105	ANIL A KAMBLE	29/M	02.08.2016	269398	APD	N	A	A	A	A	A	1	1.2	A	A	A		GB POLYP
106	DEVENDRA	33/M	03.08.2016	271348	AG	N	A	A	A	A	A	1	10	A	A	A		GB POLYP
107	SHIVAPUTRAWWA	40/F	09.08.2016	279460	P	N	2.2	A	A	A	A	A		A	A	A		CL

108	GIRIJA	22/F	08.08.2016	25462	AG	N	3	A	A	A	A	A		A	A	A	TINY CALCULUS IN GB WITH NO OBVIOUS PERICYSTIC FAT STRANDING OR FLUID.	CL
109	PRAKASH	13/M	08.08.2016	27738 4	AC	Y	A	A	6	A	A	A		A	A	A		AC
110	SURESH Y GUGYAD	35/,M	11.08.2016	26498	P	Y	8	A	7	P	A	A		A	A	A		CC
111	GIRISH	31/M	14.08.2016	28630 8	AC	y	8	A	5.2	A	A	A		A	A	A	FEW CALCULI THE NECK OF GB WITH PERICHOLECYSTIC FAT STRAND INCREASED WALL THICKNES.	CC
112	ANAND	26/M	19.08.2016	29162 6	AG	N	23	A	A	A	A	A		A	A	A		CL
113	VEERUPAKSHAMMA P	80/F	19.08.2016	29246 4	CL	N	10	A	A	A	A	A		A	A	A		CL
114	CHANDRU	18/M	21.08.2016	29491 4	PERITONITIS	N	5	A	A	A	A	A		A	A	A	FEW CALCULI IN GB	CL
115	MEERABAI	80/F	22.08.2016	29522 5	PUO	N	20	A	A	A	A	A		A	A	A		CL
116	SHAKUNTALA	37/F	23.08.2016	29755 3	AC	Y	A	A	5	P	A	A		A	A	A	GB WALL IS THICKENED WITH ENHANCEMENT SURROUNDING PERICHOLECYSTIC FLUID COLLECTION	AC
117	MALLANVI MULLA	45/F	02.09.2016	31032 7	HEPATITIS	N	23	P	A	A	A	A		A	A	A		CL WITH SL
118	SAROJA	58/F	02.09.2016	28708 7	CL	N	8	P	A	A	A	A		A	A	A		CL WITH SL
119	TUKARAM	45/M	06.09.2016	31399 2	P	N	A	P	5.4	A	A	A		A	A	A		CHRONIC CHOLECYSTITIS
120	PRABHU	54/M	06.09.2016	31351 9	APD	N	13	A	A	A	A	A		A	A	A		CL
121	ABDULRAJAK	24/M	07.09.2016	31585 7	HEPATITIS	N	A	A	A	A	A	1	8	A	A	A		GB POLYP
122	MAHADEVI	38/F	09.09.2016	31795 2	APD	N	18	A	A	A	A	A		A	A	A		CL
123	AS YENDIGIRI	42/M	10.09.2016	30904 8	PUO	N	A	A	A	A	A	1	6	A	A	A		GB POLYP
124	FATIMA	22/F	14.09.2016	32459 0	AG	N	12	A	A	A	A	A		A	A	A		CL
125	SHASHIKALA	58/F	22.09.2016	33508 0	AC	Y	A	A	6	P	A	A		A	P	A		CHOLECYSTITIS WITH ADENOMYOMATOSIS
126	SWETHA	26/F	23.09.2016	33572 6	AG	N	15	A	A	A	A	A		A	A	A		CL
127	LAXMIBAI	65/F	27.09.2016	34010 9	AG	N	26	A	A	A	A	A		A	A	A		CL
128	SIDDANNA	20/M	03.10.2016	34717 6	AG	N	A	A	A	A	A	1	8	A	A	A		GB POLYP
129	ISHWAR	50/M	17.10.2016	36241 9	CHOLECYSTITIS	N	7	A	11	A	A	A		A	A	A		CC
130	SOUMYA	20/F	17.10.2016	36271 1	HEPATITIS	N	7	P	A	A	A	A		A	A	A		CL WITH SL
131	MAHADEV	27/M	19.10.2016	36538 5	APD	N	10	A	A	A	A	A		A	A	A		CL

132	AMBUJA	55/F	24.10.2016	371183	CL	N	6	P	A	A	A	A		A	A	A		CL WITH SL
133	ARUNA	36/F	24.10.2016	371343	CL	N	5	A	A	A	A	A		A	A	A		CL
134	KAVITA	24/F	26.10.2016	374186	HEPATITIS	N	A	A	A	A	A	3	4	A	A	A		GB POLYPS
135	TANABAI	90/F	29.10.2016	36022	AC	Y	A	A	5	P	A	A		P	A	A		MEMBRANOUS CHOLECYSTITIS
136	VEENA R B	27/F	02.11.2016	36009	HEPATITIS	N	10	A	A	A	A	A		A	A	A	MULTIPLE TINY CALCULI IN GB	CL
137	SHANMUKH	35/M	02.11.2016	381237	APD	N	8	A	A	A	A	A		A	A	A		CL
138	BANUBAI	46/F	03.11.2016	381981	APD	N	17	A	A	A	A	A		A	A	A		CL
139	MARUTI	19/M	04.11.2016	382921	PUO	N	A	A	A	A	A	5	7	A	A	A		GB POLYPS
140	AMIT	27/M	05.11.2016	385164	HEPATITIS	Y	A	A	9	A	A	A		A	A	A		AC
141	AMBIKA	36/F	14.11.2016	395299	AG	N	9	A	A	A	A	A		A	A	A		CL
142	MAHADEVAPPA	65/M	14.11.2016	395948	ALD	N	7.5	A	A	A	A	A		A	A	A		CL
143	ABDULBEE M	50/F	17.11.2016	400041	CL	N	8	A	A	A	A	A		A	A	A		CL
144	GEETANJALI	39/F	21.11.2016	404470	AG	N	9	A	A	A	A	A		A	A	A		CL
145	HAJARAT	27/M	22.11.2016	405658	P	N	8	A	A	A	A	A		A	A	A		CL
146	CHANDRASHEKHAR	65/M	23.11.2016	406935	ALD	N	9	A	A	A	A	A		A	A	A		CL
147	N M HIREMATH	67/M	05.12.2016	421534	P	N	9	A	A	A	A	A		A	A	A		CL
148	PADMAWWA	30/F	07.12.2016	424680	AG	N	A	P	A	A	A	A		A	A	A		GB SL
149	PRASANNA	20/M	09.12.2016	426516	AG	N	10	A	A	A	A	A		A	A	A		CL
150	JAYAPPA	26/M	10.12.2016	427340	HEPATITIS	N	9	A	A	A	A	A		A	A	A		CL
151	MUKHESH J	37/M	12.12.2016	429992	P	N	14	A	A	A	A	A		A	A	A		CL
152	TARAWWA	28/F	12.12.2016	431118	APD	N	17	A	A	A	A	A		A	A	A		CL
153	DANAPPA	47/M	15.12.2016	432878	P	N	16	A	A	A	A	A		A	A	A		CL
154	NAJEERAHAMMAD	48/M	18.12.2016	437242	ALD	Y	A	A	5	A	A	A		A	A	A		AC
155	AKSHATA	18/F	18.12.2016	437285	DENGUE	N	A	A	9	P	A	A		A	A	A		AC
156	MALLIKARJUN MANDRUP	36/M	20.12.2016	439763	PUO	N	16	P	A	A	A	A		A	A	A	GB CALCULI AND SL	CL
157	CHANDRAYYA	39/M	28.12.2016	449216	HEPATITIS	N	5	A	A	A	A	A		A	A	A		CL
158	LATIF AHMED	56/M	29.12.2016	450647	PUO	N	6	P	A	A	A	A		A	A	A		CL WITH SL
159	S C KEMBAVI	55/M	30.12.2016	451404	ALD	N	5	P	A	A	A	A		A	A	A		CL
160	ARJUN YADRAWAKAR	46/M	02.01.2017	689	P	N	A	A	A	A	A	1	5	A	A	A		GB POLYP
161	J B DARGA	59/M	04.01.2017	3416	AG	N	23	A	A	A	A	A		A	A	A		CL
162	H M HOTAGI	43/M	05.01.2017	4754	APD	N	5	A	A	A	A	A		A	A	A		CL
163	BHEEMASHYAN	38 M	05.01.2017	4746	ALD	N	17	A	A	A	A	A		A	A	A		CL
164	SUJATA M C	37/M	11.01.2017	11945	PUO	N	7	A	A	A	A	A		A	A	A		CL
165	BHIMASHANKAR	50/M	18.01.2017	20234	CL	N	11	A	A	A	A	A		A	A	A		CL
166	BASAPPA	55/M	19.01.2017	22211	AG	N	14	A	A	A	A	A		A	A	A		CL
167	DIVYA	38/F	24.01.2017	2626	HEPATITIS	N	12	A	A	A	A	A		A	A	A		CL

168	B K BIRADAR	58/M	27.01.2017	31124	ALD	N	15	A	A	A	A	A		A	A	A		CL
169	B S KOLAKAR	57/M	27.01.2017	31108	ALD	N	38	A	A	A	A	A		A	A	A		CL
170	M D MANDRUP	50/M	28.01.2017	32350	CL	N	5	A	A	A	A	A		A	A	A		CL
171	A S BAJANTRI	54/M	31.01.2017	35991	AG	N	20	A	A	A	A	A		A	A	A		CL
172	H H BILAGI	28/M	01.02.2017	37280	P	N	10	A	A	A	A	A		A	A	A		CL
173	SUNANDA	42/F	01.02.2017	43830	CL	N	10	A	A	A	A	A		A	A	A		CL
174	SUNANDA	58/F	05.02.2017	44058	APD	N	46	A	A	A	A	A		A	A	A		CL
175	BJ HALLIMIANI	55/M	07.02.2017	45117	APD	N	21	A	A	A	A	A		A	A	A		CL
176	DB PATIL	26/F	13.02.2017	52394	HEPATITIS	N	8	A	A	A	A	A		A	A	A		CL
177	MM UJJANI	48/M	14.02.2017	53793	ALD	N	55	A	A	A	A	A		A	A	A		CL
178	BASAMMA	68/F	20.02.2017	62372	AG	N	19	A	A	A	A	A		A	A	A		CL
179	HANUMAPPA	65/M	20.02.2017	62410	AC	Y	A	A	8	P	A	A		A	A	A		AC
180	DEVARAJ	40/M	20.02.2017	62917	CL	N	10	A	A	A	A	A		A	A	A		CL
181	SAHAN	33/F	20.02.2017	53623	P	N	A	A	A	A	A	1	3	A	A	A		GB POLYP
182	DEEPAK	53/M	21.02.2017	63622	AG	N	11		A	A	A	A		A	A	A		CL
183	JS MOGALI	32/M	22.02.2017	64267	P	N	13	A	A	A	A	A		A	A	A		CL
184	VS BASARIGIDAD	36/F	22.02.2017	64270	CL	N	11		A	A	A	A		A	A	A		CL
185	MAHADEV	24/M	23.02.2017	65583	AG	N	A	A	A	A	A	1	3	A	A	A		GB POLYP
186	ASHOK	35/M	24.02.2017	67337	HEPATITIS	N	A	A	A	A	A	1	3	A	A	A		GB POLYP
187	RAMJAN	30/M	25.02.2017	68307	APD	N	A	A	A	A	A	3	3	A	A	A		GB POLYPS
188	MALLIKARJUN	52/M	28.02.2017	71906	PUO	N	A	A	A	A	A	1	2	A	A	A		GB POLYP
189	SUSHILABAI	70/F	05.03.2017	7004	PUO	N	17	P	A	A	A	A		A	A	A		CL
190	B S HONNUTAGI	50/M	06.03.2017	79797	APD	N	5	A	A	A	A	A		A	A	A		CL
191	CHINAPPA	40/M	06.03.2017	79923	CL	N	7		A	A	A	A		A	A	A		CL
192	VITOBA	70/M	08.03.2017	81393	PUO	N	3		A	A	A	A		A	A	A		CL
193	SS JOGIN	27/M	08.03.2017	81820	AG	N	A	A	A	A	A	1	2	A	A	A		GB POLYP
194	NAGAPPA	50/M	10.03.2017	84476	AC	Y	19	P	5	P	A	A		A	A	A		CC
195	MAHADEVI	47/F	14.03.2017	89875	AG	N	4	A	A	A	A	A		A	P	A		CL WITH ADENOMYOMATOSIS
196	BHARATI	23/F	15.03.2017	90493	AG	N	5	A	A	A	A	A		A	A	A		CL
197	N DAVEEDU	54/M	19.03.2017	99490	CL	N	12	A	A	A	A	A		A	A	A		CL
198	RAMESH	26/M	20.03.2017	97064	HEPATITIS	N	11	A	A	A	A	A		A	A	A		CL
199	ASHOK K	34/M	22.03.2017	98954	AG	N	A	A	A	A	A	1	3	A	A	A		GB POLYP
200	RUKMA NAIK	40/F	23.03.2017	101141	P	Y	A	A	4	P	A	A		A	A	A	ACUTE OEDEMATOUS P WITH AC	P WITH AC
201	SAVITA	30/F	27.03.2017	85771	HEPATITIS	N	7	P	A	A	A	A		A	A	A		CL WITH SL
202	BASAVARAJ	46/M	30.03.2017	108653	CL	N	11	P	A	A	A	A		A	A	A		CL WITH SL