Original article

Differentiation of malignant and benign ascites by Ultrasonography and/or CT

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Abstract:

Background: Ultrasonography is an accurate and reliable method of detecting ascites and distribution, guiding paracentesis, and monitoring the effects of therapy. Differentiation between benign and malignant ascites by ultrasound was considered as difficult or impossible; however certain ultrasonic patterns aid in distinction. Detailed characterization of ascites can be done by CT.

Objective: Detection and characterization of ascites by USG, advanced characterization by comparing with CECT Abdomen, arriving at diagnosis of cause of ascites and correlating imaging features with ascitic fluid assay.

Material and Methods: Study was conducted on Seimens Accusen Antares and ALOKA prosound Alfa 6 ufatrasound machines. The CECT Abdomen study was done on GE duo FII scanner without and with intravenous contrast. Presence of omental thickening, tethered bowel loops, extension of ascites in lesser sac, peritoneal implants, gallbladder wall thickness and existence of septa and debris in ascitic fluid was investigated.

Results: Out of 100 cases of ascites, 21 were malignant and 79 were of benign etiology. Extension into lesser sac was seen in 80 % of malignant cases. Peritoneal, omental thickening and tethered bowel sign were seen in 57 %, 80 % and 52 % of malignant cases respectively. GB wall thickening was noted in 33 % of benign cases, most of them were due to liver cirrhosis.

Conclusion: Omental thickening, peritoneal deposits, tethered bowel sign and extension into lesser sac were seen more frequently in malignant cases whereas thickening of the gall bladder wall and anechoic clear fluid was seen in benign causes of ascites.

Keywords: Benign ascites, Malignant ascites, Lesser sac, Peritoneal implants, Tethered bowel, Omental cake.

Introduction:

Ascites is an abnormal collection of fluid in the peritoneal cavity. Ascites can develop due to disease of abdominal origin or due to systemic disease. Determination of its aetiology is necessary for establishing appropriate treatment plan. Ultrasonography (USG) and Computed Tomography (CT) plays an important role in assessing amount of ascitic fluid and for ascitic sampling and draining of ascitic fluid and demonstrate associate findings. Cirrhosis contributes to more than three-quarter of cases of ascites, malignant ascites represents 10% of cases of ascites, 5% of cases are so-called ‘mixed’ ascites, since they combine several causes. USG and CT help in differentiating benign from malignant ascites.[1] Certain signs on USG and CT are helpful in predicating etiology of ascites (Benign v/s Malignant). Distribution of ascites (greater sac, greater sac and lesser sac), presence of peritoneal implants, omental cake, tethered bowel signs are extremely useful in differentiating benign from malignant ascites on imaging.[1]
Materials and Methods:
A prospective study was conducted in 100 patients of ascites diagnosed clinically and on USG and / or CT referred to a tertiary care teaching hospital from June 2012 to April 2014. USG machines used were Siemens Accusen Antares and ALOKA prosound Alfa 6. CT was conducted on GE duo FII scanner without and with intravenous contrast. Presence and distribution of ascites, existence of septa and debris in ascitic fluid, presence of omental thickening, tethered bowel loops, extension of ascites in lesser sac, peritoneal implants, gallbladder wall thickness were investigated. Ascitic fluid assay was done in all cases. Correlation of characteristics of ascitic fluid (on USG and CT) with ascitic fluid assay was done to differentiate between benign and malignant ascites. 

Statistical analysis – Fischer’s exact chi-square and Mann-Whitney U tests were carried out to evaluate the consistency of various signs on USG and CT in determining the pathologies and the pathological differences were observed between benign and malignant ascites.

Results:
In our case series, 100 cases were included. Out of 100 cases of ascites 21 cases were of malignant ascites and 79 were benign [Table 1]. Age group was from 9 – 70 years [Table 2]. 10 were males, 11 were females [Table3]. Out of 21 cases of malignant ascites, Ca ovary was seen in 8 [Figure 1 A and B], ovarian lymphoma in 1 [Figure 2], Ca stomach in 6, Ca colon in 2, perianampular carcinoma in 1, disseminated metastases in 2 and peritoneal carcinomatosis in 1 as shown in Table 4. Analysis of ascitic fluid as transudative and exudative was done after ascetic fluid tapping and its results are tabulated in Table 5. GB wall thickening was < 3 mm in all 21 cases as given in Table 6. Omental thickening / nodular deposits were noted in 17/21 cases and was not observed in 5 /21 cases. Peritoneal implants were found in 12/21 cases. Tethered bowel sign was seen in 11/21 cases. Ascites with internal echoes was noted in 4/21 cases and it was found to be clear in 17/21 cases. Concordant greater and lesser sac ascites was found in 17 out of 21 cases. Greater sac ascites was seen in 4/21 cases. Associated findings were not found in 4/21 cases, found in 17/21 cases. Pleural effusion was seen in 5/21, liver metastases in 10/21 [Figure 3], retroperitoneal lymph nodes in 4/21, obstructive jaundice in 1/21 cases [Tables 7, 8, 9]. 70 cases of benign ascites were observed. Fluid was anechoic in 53/79 cases, internal echoes were seen in 21/79 cases, hemorrhagic ascites was seen in 5/79 cases. Multiple Septations in ascetic fluid were seen in 12/79 cases. Loculated ascites was seen 1 case. Parietal peritoneal thickening was seen in 3/79 cases. Ascites in greater sac was seen in 48/79 cases, ascites in both greater sac and lesser sac was seen in 31/79 cases, of which 22 cases were pancreatitis [Figure 4], 2 cases were of duodenal perforation, 1 case of peritoneal tuberculosis, 2 cases of appendicular perforation , 1 case of cirrhosis, 1 case of CCF and 2 cases of SABP. In associated findings signs of liver cirrhosis were seen in 10, retroperitoneal lymph nodes were noted in 4 cases, mesenteric fat stranding was seen in 23 cases, mesenteric lymph nodes were seen in 10 cases as shown in table 7, 8, 9.

The sensitivity, specificity, positive and negative predictive value of significant findings (greater and lesser sac ascites, gall bladder wall thickening, peritoneal implants, omental cake and tethered bowel signs) are summarized in tables 10 and 11.

Discussion:
In our case series, 100 cases were studied. Causes were determined to be malignant in 21 cases (21%) and benign in 79 cases (79%).
Investigation of ascites usually starts with physical examination and lab test. Clinical diagnosis usually difficult with limited ascites. Ascitic fluid analysis performed following abdominal paracentesis is an easy and economical method; however it is difficult in cases of mild ascites. Differentiation between cirrhotic and malignant ascites can be done by history-taking, physical examination, blood and urine tests, abdominal ultrasound, and paracentesis.\[1, 2, 3\] Imaging alone cannot differentiate between the two. However indirect signs can aid in differentiating benign and malignant etiology. Ascites can occur due to various benign and malignant pathologies. \[4\]

In our series of malignant ascites, Ca ovary, ovarian lymphoma, Ca stomach, ca colon, periamplular carcinoma, disseminated metastases and peritoneal carcinomatosis were causative factors. Most common malignancy was Ca ovary. The causes of benign ascites were pancreatitis, bowel and gall bladder perforation, tuberculosis, SABP, liver cirrhosis, anaemia, hydroproteinemina, extrahepatic portal HTN, CCF and renal failure. Associated findings found in 17/21 cases (80.9 \%) were pleural effusion was seen in 5/21 (23.8 \%), liver metastases in 10/21 (47.6 \%), retroperitoneal lymph nodes in 4/21 (19.1 \%), obstructive jaundice in 1/21 cases (4.7 \%).

USG and CT are primary imaging tools to demonstrate presence of ascitic fluid distribution and associated findings. Diagnosis of ascites is not possible with physical examination when ascitic fluid level is limited. USG and CT should be used in such conditions. \[5\] Ultrasound is increasingly used in evaluating ascites. It is an accurate and reliable method of detecting ascites and distribution, guiding paracentesis, and monitoring the effects of therapy. It can distinguish transudative from inflammatory or malignant exudative fluid. USG can differentiate fluid form solid tissue. Typical sonographic appearance of ascites has been described on previous literature. \[6\] Ascitic fluid generally appears as homogeneous, anechoic surrounding and interposed between the loops of bowel and viscera in a relatively uniform manner. The bowel loops may float or sink depending on the relative amount of intraluminal gas and fluid and the density of ascitic fluid. Distribution of ascitic fluid occurs due to the effects of gravity as well as capillary attraction in the various intraperitoneal spaces. The smallest amounts of fluid collect in the flanks and superior right paracolic gutter, around the liver, and in the lowest peritoneal reflection in the pelvis. Differentiation between benign and malignant ascites by ultrasound was considered as difficult or impossible; however certain ultrasonic patterns aid in distinction.

Total ascitic fluid protein concentration of 3g\% is used as gold standard which is used in our hospital to differentiate transudative from exudative ascites. Wall thickness of anterior wall of gall bladder is taken in all patients and 3 mm wall thickness is used as upper limit of normal in distended gall bladder. Gall bladder wall thickness > 3 mm are categorised as having transudative ascites and patients with wall thickness < 3mm as exudative ascites. \[7\] Gall bladder wall thickness in ascites using high resolution USG equipment can differentiate transudative from exudative ascites radiologically. There is direct correlation between GB wall thickness and ascitic fluid total protein. High resolution USG can differentiate transudative from exudative ascites based on gall bladder (GB) wall thickness and comparing it with ascitic fluid total proteins. USG has revolutionized the work up of patients with ascites over the past two decades. It aids in therapeutic and diagnostic aspirations especially in small collections and also demonstrates factors responsible for ascites.
GB wall thickening in ascites has been interpreted differently by different authors.

According to Tsujimoto et al. thickening of GB was supposed to occur in benign ascites and not in malignant ascites. Georgiev and Mechkov used GB wall thickness as a method to differentiate cirrhotic from other ascites. Brogan et al Collia et al described GB in ascites as a sign of transudative ascites and of portal hypertension whatever be the cause. Malde et al made an attempt to differentiate transudative from exudative ascites based on echogenicity of fluid. Thickening of GB wall in ascites is due to hypoalbuminemia and passive diffusion of fluid in GB wall. In cirrhosis, venous dilatation is also proposed as a cause of GB wall thickening called as congestive cholecystopathy. Increased gallbladder wall thickness could be due to different etiologies other than gallbladder diseases such as liver diseases, hypoalbuminemia, ascites, hepatitis, congestive heart failure, kidney disease, AIDS, malignancy and sepsis. Ultrasonography has an accuracy of 93%, in determination of gallbladder wall thickness, if it is 1mm and 100% for 1.5 mm. If there is a change in plasma hydrostatic and oncotic pressure, gallbladder wall edema ensues. USG measurements of GBWT can be used to differentiate between of malignant from other causes of ascites. Convex transducer of 3.5- 5 MHz frequency was used in the right upper quadrant below costal margin. Thickness was measured by vertical beam to gallbladder wall and measured from serosa to mucosa. The patients were examined after 8 hours of fasting and only those patients were included that had a fill gallbladder. Three measurements of gallbladder wall thickness were taken at each site and the average measurement was used.

The GB wall thickness in patients with ascites due to liver cirrhosis was 3.9±0.6 and 2.2±0.6 mm in patients with peritoneal carcinomatosis. GBWT is a helpful and simple technique to differentiate ascites due to cirrhosis and peritoneal carcinomatosis. In our series, GB wall thickening of less than 3 mm was seen in all 21 cases of malignant ascites (100 %) [Figure 5]. GBWT of less than 3 mm was seen in 53/ 79 cases of benign ascites (67%), GBWT of more than 3 mm was seen in 26/79 ( 33%) cases [Figure 6]. Amongst benign causes GBWT of more than 3 mm was found in liver cirrhosis, CCF, anemia-hypoproteinemia and renal failure [Figure 7].

The sensitivity and specificity of USG in detection of free ascitic fluid is over 90 %. Its value is limited in presence of overlying bowel gas. CT unlike USG is not limited by bowel gas. However it fails to demonstrate internal septa. USG can detect multiple, fine, complete or incomplete and mobile strands of fibrin and debris within ascites. In our study, ascites with internal echoes on USG was noted in 4/21 cases of malignant ascites (19.1 %) and it was found to be clear in 17/21 cases (80.9 %) [Figure 8]. In 79 cases of benign ascites, fluid was anechoic and clear in 53/79 cases (67 %), internal echoes were seen in 21/79 cases (26.5 %). Ascites with multiple septations were seen in 12/79 cases (15.1%).

According to our study, USG provides a simple, rapid and highly sensitive approach for detection and characteristic of ascites. Other imaging modalities like CT and MRI are rarely needed.

Ha HK et al reported that small sized peritoneal implants cannot be determined by CT scan. However they can be easily determined if they are calcified. In cases of ascites with peritoneal lesions, group of investigators reposted that USG is superior to CT (except in obese patients).

While another study group reported that CT has a prominent role. According to Vanhoenacker FM et al, USG can demonstrate diffuse hypoechoic
peritoneal thickening of 2-6 mm, irregular peritoneal nodular thickening with tiny nodules of < 5 mm, only if a considerable amount of ascites is present. CT demonstrates smooth mild peritoneal thickening and pronounced peritoneal enhancement. CT is a modality of choice for mesentery and omentum. However omentum and small intestinal mesentery involvement can be demonstrated with USG as hyperechoic mass anterior to bowel and beneath abdominal wall. USG examination was performed by a single radiologist in our series which contributed to decreased chances of technician dependent mistakes. Omental thickening / nodular deposits were observed in 17/21 cases of malignant ascites (80.9 %) and was not observed in 4 /21 cases (19.1 %) in our case series [Figure 9]. Peritoneal implants were seen in 12/21 cases (57.1 %) [Figure 10,11]. Parietal peritoneal thickening was seen in 3/79 cases of benign ascites (3.7%) [Figure 12]. They were either tuberculous peritonitis or subacute bacterial peritonitis. No peritoneal thickening was observed in other causes of benign ascites.

USG is accepted as gold standard for evaluating biliary system and determining GB wall thickness. GB wall thickening is due to extrinsic factors like hypoalbuminemia and portal hypertension and intrinsic factors like cholecystitis. Thus GB wall thickening is more often seen in ascites of benign etiology. A diseased mesentery is characterized by mesenteric thickening, loss of the normal mesenteric configuration and nodular lesions, consisting of micro- (<5 mm) or macro nodules (>5 mm), lymph nodes, or abscesses. Reradiating thickened mesentery causes fixing of bowel loops creating a “stellate appearance” both on the US and CT. Accumulation of ascites is more common with peritoneal carcinomatosis than in malignant peritoneal mesothelioma. Three types of tuberculous peritonitis are defined: wet ascetic type characterized by loculated fluid and thickened mesentery, dry plastic type accompanied by adhesion and enlarged lymph nodes with calcification necrosis, and fibrotic fixed type with thickened omentum. USG and CT scan findings vary according to the mentioned types. Multiple retroperitoneal and mesenteric lymph nodes with or without calcification, thickened omentum and mesentery due to granulomatous infiltration, ascitic fluid with internal echoes and moving fibrin septa, radial accumulation of intestinal loops resulting in sliced bread appearance, granulomas on liver and spleen surface, and high density ascites are the findings are usually seen in tuberculous peritonitis. However they are not specific. These can be seen in ascites of malignant origin, and a differential diagnosis is not possible. Extension of the inflammation through the peritoneum into the extraperitoneal compartment suggests tuberculosis and can be helpful in the differential diagnosis from peritoneal carcinomatosis. In ascites due to liver cirrhosis, diffuse ascites without septa and internal echoes was found. On USG liver shows diffuse coarse echotexture with fine nodular surface, splenomegaly and multiple collaterals can be seen in distal esophagus and gastric fundus, lienorenal and peripancreatic region suggestive of portal hypertension. Left lobe and caudal lobe hypertrophy, and right lobe atrophy, contributed to the diagnosis. Continuous material exchange between the vascular bed and peritoneal space. The density of simple ascites varies between 0-30HU. Ascites with a higher density occurs due to extravasation of proteins, leukocytes, blood content or contrast material into the peritoneal cavity. In malignant cases, fragile neo-vascularization,
leakage of protein-like fluid or blood from the tumor implants and secretion of substances from tumor cells that increase the vascular permeability are mentioned as the cause of the ascites staining. Cooper et al, studied the staining of ascites fluid in late images following contrast material administration and stated that, vascular-peritoneal permeability of the peritoneum was increased and staining could be enhanced. The amount of staining was independent from the type of contrast material, latency period, and clinical diagnosis of the cases. They stated that the amount of the staining is mainly determined by the amount of ascites. In cases of an excessive amount of ascites, the dilution of the contrast material leaking to the peritoneal cavity as the increased permeability will prevent enhanced staining of the ascites. Malignant ascites is rich in proteins and blood contents and hence show increased density. However, low sensitivity (68%) of this finding limits its role in differentiation of benign and malignant causes. [5]

The study criteria of characteristics of benign and malignant ascites are: ascites volume and relative distribution between the greater peritoneal cavity (GPC) and the omental bursa (OB); density of the ascites; thickness of the gallbladder wall; thickness and degree of enhancement of the parietal peritoneum and tethered-bowel sign.

The mean ascites density was measured by positioning a region of interest (ROI) on the slice with the most ascites, while maintaining a distance of at least half of the diameter of the ROI relative to the surrounding tissues. A single measurement was taken. For thickening of the gall bladder, a single measurement was taken of the maximum radial thickness in the axial plane.

Parietal peritoneal thickening and enhancement was qualitatively assessed and entered on a scale from 0 to 3, based on degree (0 = absent, 1 = moderate or visible, 2 = significant or measurable, 3 = peritoneal nodules). The analysis was performed for the parietal peritoneum with particular attention to the pouch of Douglas, the right paracolic gutter, and the right subphrenic and retrohepatic spaces which are common sites for peritoneal deposits.

The tethered-bowel sign was considered positive when there was matted bowel loops no longer floating freely in the ascites (assessed only in presence of abundant ascites in the GPC (grade 4). Mean density in ascites due to liver cirrhosis was lower, 7 +/- 3 HU and mean density in malignant ascites was higher 11 +/- 5 HU. Mean thickness of the GB wall was 3.5 mm in ascites due to liver cirrhosis and 2/5 mm in malignant ascites. [1] In our study, tethered bowel sign was noted in 11/21 cases of malignant ascites (52.3%). It was not observed in benign ascites [Figure 13,14].

Ascites in the greater sac was graded as follows - grade 1 fluid volume indicated minimal ascites layering anteriorly between the liver and anterolateral peritoneum; grade 2, fluid in Morrison’s pouch; grade 3, fluid along the paracolic gutters; grade 4, sufficient fluid to centrally displace small bowel loops.

Ascites in lesser sac was assessed on scan planes in which the stomach, pancreas, and spleen appeared at the same level.

Grade 1- minimal fluid causing up to 0.5 cm separation of the contrast filled stomach and the retropenitoneal fat lying anterior to the pancreas; grade 2, separation of the pancreas and stomach by 0.5-1 cm; grade 3, separation by 1 -2 cm; grade 4, separation by greater than 2 cm. [15] USG can locate fluid within lesser sac unless gas filled stomach obscures the view.

Abdominal CT is the investigation of choice to diagnose and predict aetiology of lesser sac
pathology. Ascites within the lesser sac is not a typical manifestation of generalized peritoneal ascites, and its presence should direct a search for pathology in neighbouring organs and for peritoneal malignancy.

Relative distribution of fluid in greater and lesser sac varies according to aetiology. This suggests that the foramen of Winslow impedes free communication between these two potential spaces, which is contrary to what is suggested by most standard textbooks of anatomy.

Three patterns of ascites were observed.\[15\]

1) More Fluid in Greater Sac than Lesser Sac: Congestive cardiac failure, alcoholic cirrhosis, greater sac abscesses, and those on peritoneal dialysis. Ascitic fluid assay demonstrated, in general, transudative ascites with negative cytologic examination.

2) More Fluid in Lesser Sac than Greater Sac seen in patients with disease processes occurring in organs directly bordering the lesser sac (e.g., pancreatitis, penetrating gastric ulcer with abscess formation).

3) Same Amount of Fluid in Both Sacs- seen in Ca stomach, Ca ovary, Ca colon. Ascitic fluid assay in these patients demonstrated an exudative, occasionally bloody ascites that generally had positive cytology.

The accumulation and distribution of intraperitoneal fluid is influenced by a number of factors: volume, specific gravity, colloidal content, rapidity of formation, site of origin, intraperitoneal pressure gradients, patient position and gravity, adhesions, abdominal wall pliability, mesenteric partitions, ligamentous attachments, and peritoneal compartments.

Volume of ascitic fluid does not influence fluid communication between the greater and lesser sacs.

In patients with massive greater sac fluid due to cirrhosis, congestive heart failure, or dialysis, little if any fluid could be identified in the lesser sac. In patients with large lesser sac ascites, little fluid was seen in the greater sac. Gravitational manoeuvres do not seem to change the relative distribution of fluid in these two spaces. However, few exceptions are noted.

This could be explained as

a) Seeding of tumor cells in the lesser sac produces fluid within this compartment.

b) Subdiaphragmatic lymphatics are responsible for 80% of fluid absorption from the peritoneal cavity, which eventually drains into the venous circulation through the right lymph duct and the left thoracic lymph duct. The subdiaphragmatic location of the lesser sac with very effective lymphatic drainage in that region may be speculated to explain absence of fluid within it.\[15\]

In our study, concordant greater and lesser sac ascites was found in 17 out of 21 cases of malignant ascites (80.9%). Greater sac ascites was seen in 4/21 cases of malignant ascites (19.1%). Ascites in greater sac was seen in 48/79 cases of benign ascites (60.7%), ascites in both greater sac and lesser sac was seen in 31/79 cases of benign ascites (39.2%) [Figure 15,16].

**Conclusion:**

USG and CT can predict etiology of ascities (benign Vs malignant) in a non-invasive way which is useful in treatment plan. Distribution and characterization of ascites can be done. Omental thickening, peritoneal deposits, tethered bowel sign and extension into lesser sac were seen more frequently in malignant cases whereas thickening of the gall bladder wall and anechoic clear fluid was seen in benign causes of ascites.
### Table 1 - Causes of ascites:

<table>
<thead>
<tr>
<th>Causes</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign</td>
<td>79</td>
<td>79 %</td>
</tr>
<tr>
<td>Malignant</td>
<td>21</td>
<td>21 %</td>
</tr>
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</table>

### Table 2 - Age wise distribution (n=100)

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Benign (79)</th>
<th>Malignant (21)</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>0-10</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>11-20</td>
<td>5</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>21-30</td>
<td>14</td>
<td>0</td>
<td>14</td>
</tr>
<tr>
<td>31-40</td>
<td>23</td>
<td>1</td>
<td>24</td>
</tr>
<tr>
<td>41-50</td>
<td>26</td>
<td>6</td>
<td>32</td>
</tr>
<tr>
<td>51-60</td>
<td>9</td>
<td>8</td>
<td>17</td>
</tr>
<tr>
<td>61-70</td>
<td>2</td>
<td>5</td>
<td>7</td>
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### Table 3 - Sex wise distribution

<table>
<thead>
<tr>
<th>Sex</th>
<th>Benign (n=79)</th>
<th>Malignant (n=21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>55</td>
<td>10</td>
</tr>
<tr>
<td>Females</td>
<td>24</td>
<td>11</td>
</tr>
</tbody>
</table>

### Specific findings:

### Table 4 - Pathological distribution of cases (n = 100)

<table>
<thead>
<tr>
<th>BENIGN ASCITES CAUSES</th>
<th>N=79</th>
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</thead>
<tbody>
<tr>
<td>Pancreatitis</td>
<td>25</td>
</tr>
<tr>
<td>Perforation duodenal</td>
<td>2</td>
</tr>
<tr>
<td>Perforation appendicular</td>
<td>2</td>
</tr>
<tr>
<td>SABP</td>
<td>2</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>21</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>10</td>
</tr>
<tr>
<td>Anaemia, hypoproteinemia</td>
<td>6</td>
</tr>
<tr>
<td>Extrahepatic portal HTN</td>
<td>2</td>
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</tbody>
</table>
### Table 5- Etiologies in Cases of Confirmed Ascites

<table>
<thead>
<tr>
<th>Etiologies</th>
<th>No.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Transudative</strong></td>
<td></td>
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<tr>
<td>Cirrhosis</td>
<td>10</td>
</tr>
<tr>
<td>CCF</td>
<td>3</td>
</tr>
<tr>
<td>Renal Failure</td>
<td>4</td>
</tr>
<tr>
<td>Anemia with hypoproteinemia</td>
<td>6</td>
</tr>
<tr>
<td>Extrahepatic portal HTN</td>
<td>2</td>
</tr>
<tr>
<td><strong>Exudative</strong></td>
<td></td>
</tr>
<tr>
<td>Malignancy</td>
<td>21</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>21</td>
</tr>
<tr>
<td>SABP</td>
<td>2</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>25</td>
</tr>
<tr>
<td>Appendicular abscess</td>
<td>1</td>
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<tr>
<td>Bowel perforation</td>
<td>5</td>
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### Table 6- GB wall thickening

<table>
<thead>
<tr>
<th>GB Wall Thickening</th>
<th>Benign</th>
<th>Malignant</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3 mm</td>
<td>53</td>
<td>21</td>
</tr>
<tr>
<td>&gt; 3 mm</td>
<td>26</td>
<td>0</td>
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</table>

### Table 7- Distribution of Ultrasonic Findings among Transudative and Exudative Ascites

<table>
<thead>
<tr>
<th>Type of ascites</th>
<th>Transudative</th>
<th>Exudative</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infective / inflammatory</td>
<td>Neoplastic</td>
</tr>
<tr>
<td>Clear ascites</td>
<td>53</td>
<td></td>
</tr>
<tr>
<td>Internal echoes</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Septations</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Peritoneal thickening</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Omental cake</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Peritoneal implants</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Tethered bowel sign</td>
<td></td>
<td>11</td>
</tr>
</tbody>
</table>

### Table 8- Distribution of ascites on USG and / or CT

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of cases</th>
<th>GS ascites</th>
<th>GS + LS ascites</th>
<th>LS ascites</th>
<th>Paracentesis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Transudative</td>
<td>Exudative</td>
<td>CYT positive for Malignant cells</td>
<td></td>
</tr>
<tr>
<td>Benign</td>
<td>79</td>
<td>48</td>
<td>31</td>
<td>0</td>
<td>25 54 0</td>
</tr>
<tr>
<td>Malignant</td>
<td>21</td>
<td>4</td>
<td>17</td>
<td>0</td>
<td>0 21 21</td>
</tr>
</tbody>
</table>

### Table 9- Pathological distribution with USG and CT

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Benign (79)</th>
<th>Malignant (21)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>USG(Done in 79)</td>
<td>CT (Done in 61)</td>
</tr>
<tr>
<td>Ascites in GS</td>
<td>48</td>
<td>46</td>
</tr>
<tr>
<td>Ascites in GS+ LS</td>
<td>31</td>
<td>26</td>
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<tr>
<td>Ascites in LS</td>
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<tr>
<td>Septations</td>
<td>12</td>
<td>4</td>
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<tr>
<td>Peritoneal thickening</td>
<td>3</td>
<td>5</td>
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<tr>
<td>GB Wall Thickening</td>
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<td>13</td>
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<tr>
<td>Matted bowel loops</td>
<td>15</td>
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<tr>
<td>Peritoneal enhancement</td>
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<td>7</td>
</tr>
<tr>
<td>Observations and signs on USG</td>
<td>Sensitivity(%)</td>
<td>Specificity(%)</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>----------------</td>
<td>----------------</td>
</tr>
<tr>
<td>1. Greater and lesser sac ascites</td>
<td>80.95</td>
<td>60.76</td>
</tr>
<tr>
<td>2. Gall bladder wall thickening</td>
<td>-</td>
<td>67.09</td>
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<tr>
<td>3. Peritoneal implants</td>
<td>47.62</td>
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<td>4. Omental cake</td>
<td>61.90</td>
<td>96.20</td>
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<td>5. Tethered bowel sign</td>
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</table>

Table 10: Percentages of USG findings showing significant difference between malignant and benign ascites causes.

<table>
<thead>
<tr>
<th>Observations and signs on CT</th>
<th>Sensitivity(%)</th>
<th>Specificity(%)</th>
<th>Positive predictive value (PPV)(%)</th>
<th>Negative predictive value (NPV)(%)</th>
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<td>80.95</td>
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<td>3. Peritoneal implants</td>
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<td>4. Omental cake</td>
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<td>5. Tethered bowel sign</td>
<td>52.38</td>
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<td>100</td>
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</table>
Image 1A: Carcinoma left ovary with malignant ascites

Image 1B: Case of carcinoma ovary with malignant Ascites

Image 2: A case of bilateral ovarian lymphoma

Image 3: malignant Ascites with multiple hepatic metastases.

Image 4: Pancreatitis with ascites in lesser sac and a pseudocyst in left anterior pararenal space

Image 5: Malignant Ascites in greater and lesser sac with normal gall bladder wall thickness.

Image 6: Benign Ascites showing mild gall bladder wall thickening (3.5 mm)
Image 7: Benign ascites with gall bladder wall thickening (3.5mm) with no extension in lesser sac.

Image 8: Ascites with multiple internal echoes.

Image 9: Omental cake just beneath anterior abdominal wall

Image 10: Ascites with peritoneal metastatic deposits showing vascularity on colour Doppler

Image 11: Malignant ascites with peritoneal deposits in right paracolic gutter.

Image 12: Tuberculous ascites with mild peritoneal thickening and enhancement with soft tissue infiltrates in omentum.

Image 13: Negative tethered-bowel sign- Freely floating bowel loops coming in contact with anterior parietal peritoneum.
Image 14: Positive tethered-bowel sign: bowel loops not freely floating in ascites not coming in contact with anterior parietal peritoneum due to fixation.

Image 15: Ascites in greater sac with no extension in lesser sac on USG.

Image 16: Malignant Ascites with extension in lesser sac on USG.

References:


