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Ultrasound, CT scan and Cytopathology Correlation in Etiology of Ascites

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Abstract

Ascites, defined as the abnormal accumulation of fluid within the peritoneal cavity, is a frequent clinical condition associated with diverse etiologies. This study aimed to evaluate the causes and imaging characteristics of ascites using ultrasonography and computed tomography. Fifty patients with clinically suspected ascites were examined using both modalities and cytopathology study. Cirrhosis was the most common cause, followed by malignancy and tuberculosis. Ultrasonography effectively detected free fluid and internal echoes, while CT provided superior characterization of loculated and malignant ascites. Radiological correlation assists in identifying the underlying cause, guiding appropriate clinical management

INTRODUCTION

The term ascites is derived from the greek word “ASKOS” (bladder, belly, bag) and denotes the presence of excessive fluid in the peritoneal cavity. Many diseases are known to lead to the formation of free fluid within the peritoneal cavity. Basically the causes of ascites may be grouped into those conditions in which the pathological process does not directly affect the peritoneum and those in which the peritoneum itself is involved.

The first group includes diseases associated with sinusoidal portal hypertension (cirrhosis, acute alcoholic hepatitis, fulminant or subacute viral or toxic hepatitis, congestive heart failure, constrictive pericarditis, IVC obstruction, Budd-Chiari syndrome, hepatoveno occlusive disease) hypoalbuminaemia (Nephrotic Syndrome, protein-losing enteropathy, and malnutrition), and a variety of disorders that may cause ascites through different mechanisms, such as myxoedema, ovarian diseases (carcinoma,benign tumours, ovarian hyperstimulation syndrome),chronic ancreatitis,biliary-tract leakage (secondary to liver trauma, biliary-tract surgery, or transhepatic cholangiography), diseases affecting the lymphatic system of the splanchnic area and chronic renal failure. In the second group, ascites is formed as a consequence of primary peritoneal disease or as a result of peritoneal involvement in systemic process; tuberculous, fungal Candida albicans, Coccidioidesimmitis), parasitic and granulomatous eritonitis(sarcoidosis, Crohn’s disease, peritoneal granulomatous reaction to talc, cotton, wood fibers, starch and barium), primary or metastatic peritoneal tumours, vasculitis (systemic lupus erythematosus, Henoch-Schonlein purpura), eosinophilic gastroenteritis, and Whipples disease are the most characteristic causes of ascites in this group.

The evaluation of a patient with ascites requires that the cause of the ascites to be established. In most cases ascites appears as part of a well recognized illness such as cirrhosis, congestive heart failure, nephrosis or disseminated carcinomatosis, in these situations the physician should determine that the development of ascites is indeed a consequence of the basic underlying disease and not due to the presence

of a separate or related disease process. This distinction is necessary even when the cause of ascites seems obvious.

Diagnostic paracentesis (50-100ml) should be part of the routine evaluation of the patient, with ascites. The fluid should be examined for its gross appearance, protein content, albumin level, cell count, and differential cell count, should be determined and gram's and acid fast stains and culture should be performed. Cytologic and cell block examination may disclose anotherwise unsuspected carcinoma. Serum ascites albumin gradient (SAAG) should be calculated to determine whether the fluid has features of a transudate or an exudate. The gradient correlates directly with portal pressure, a gradient >1.1 gm/dl, high gradient ascites is characteristic of uncomplicated cirrhotic ascites and differentiates ascites due to portal hypertension > 97% of the time. Other etiologies of high gradient ascites include alcoholic hepatitis, congestive heart failure, hepatic metastasis, constrictive pericarditis and Budd chiari Syndrome. A gradient < 1.1 gm/dl (Low gradient) suggests that the ascites is not due to portal hypertension with > 97 % accuracy and mandates a search for other causes such as peritoneal carcinomatosis, tuberculous peritonitis, pancreatitis, serositis, pyogenic peritonitis, and nephrotic syndrome.

Blood stained fluid with > 2.5gm / dl protein is unusual in uncomplicated cirrhosis but is consistant with tuberculous peritonitis or neoplasm. Cloudy fluid with predominance of polymorphonuclear cells > 250/ micro liter and a positive Gram's stain are characteristic of bacterial peritonitis, which requires antibiotic therapy, if most cells are lymphocytes tuberculosis should be suspected. Chylous ascites refers to a turbid milky or creamy peritoneal fluid due to presence of thoracic or intestinal lymph. Such fluid shows sudan staining fat globules microscopically and an increased triglyceride content by chemical examination. Opaque milky fluid has atriglyceride concentration > 1000 mg/dl, but a triglyceride Concentration > 200mg is sufficient for diagnosis. A turbid fluid due to leukocytes, or tumor cells may be confused with chylous fluid (pseudochylous) and it is often helpful to carry out alkalinization and ether extraction of the specimen.

Alkali tend to dissolve cellular proteins and thereby reduce turbidity, ether extraction leads to clearing if the turbidity of the fluid is due to lipid. Chylous ascites is often the result of lymphatic disruption, or obstruction from cirrhosis, tumor, trauma, tuberculosis, filariasis, or congenital abnormalities.

It may also be seen in nephrotic Syndrome.

AIMS AND OBJECTIVES

The aims and objectives of this dissertation are -

1. To study and evaluate the causes of ascites using ultrasonography, Computed tomography and cytopathology findings.
2. To correlate the imaging findings in ultrasonography, Computed Tomography and cytopathology Imaging in various causes of ascites.
3. To differentiate benign and malignant causes of ascites.
4. To make final diagnosis using ultrasonography, Computed tomography and cytopathology findings

REVIEW OF LITERATURE

DEFINITION:

Ascites is defined as the accumulation of excess fluid in the peritoneal cavity. Fluid accumulates when it enters the peritoneal cavity from the mesenteries, the peritoneum and hepatic surface at a rate greater than canbe returned to the circulation via the capillaries and lymphatics.

ANATOMY OF THE PERITONEUM

The peritoneum is a serous membrane which lines the abdominalcavity; it covers the anterior and posterior walls, the under surface of the diaphragm and the pelvic cavity, All this is the parietal peritoneum. Inplaces it leaves the posterior abdominal wall or diaphragm to form a partialor complete investment for viscera; this is the visceral peritoneum, whichforms the serous covering for many viscera.Peritoneum consists of a single layer of flattened cells, mesothelium, overlying areolar tissue which varies in both thickness and density in different places. Over expansile parts this areolar tissue is loose and cellular (e.g. transversalis fascia on the lower anterior abdominal wall) while over non-expansile parts it is often very thick (eg. Iliac fascia, psoas fascia, parietal pelvic fascia); but loose or dense, thin or thick, these variously named fasciae are part of the one continuous extra peritoneal connective tissue lying between the parietal peritoneum and the walls of theabdominaland pelvic cavities. On the posterior abdominal wall the dense psoas and iliac fasciae and the anterior layer of the lumbar fascia serve as firm bases upon which the extraperitoneal tissue can gain attachment. The posterior surfaces of retroperitoneal structures (pancreas, duodenum, ascending and descending colon) also gain a firm attachment to these fasciae,. Thus peritoneum and viscera have a firm anchorage undisturbed by the movements of contraction of the underlying muscles. Various folds or reflections of peritoneum connect viscera to the abdominal walls or to one another. Some of these are properly called folds,but others may be called a mesentery, omentum or ligament, The double foldsupporting most of the small intestine is the mesentery; the mesenteries supporting the transverse colon, sigmoid colon and appendix are the transverse mesocolon, sigmoid mesocolon and mesoappendix,. The lesser omentum connects the stomach to the liver, and the greater omentum hangsdown from the lower border of the stomach. The various ligaments associated with the liver, stomach and spleen are simply peritoneal folds andbear no relation in structure or strength to the ligaments of muscles and joints; the name as applied to peritoneum is an unfortunate one. A few of these peritoneal structures are easy to see on opening the abdomen throughthe anterior abdominal wall (e.g. the greater omentum and the mesentery), but others can only be properly appreciated when viscera are displaced or removed.

PERITONEAL CAVITY: GREATER AND LESSER SACS

The serous-coated organs fill the abdominal cavity so that visceral surfaces are in contact with one another or with the parietal peritoneum. The space between them is only potential, not actual, and it contains only a few millilitres of tissue fluid which lubricates adjacent surfaces so they can slide over one another, this is the general peritoneal cavity, body cavity or coelom, and is opened up when incisions that include parietal peritoneum aremade through the abdominal wall. Another name for it is the greater sac. The lesser sac properly called the omental bursa, is a diverticulum of the peritoneal cavity behind the stomach. It exists because of the way the liver, stomach and spleen change their positions and shapes during development, and its purpose is to provide a slippery surface for the necessary mobility of the posterior surface of the stomach. It opens into thegreater sac through a slit-like aperture in front of the inferior vena cava, the epiploic foramen. Theoretically the cavity of the lesser sac should extend down between the layers of the greater omentum but because of the fusion of layers it rarelyextends much below the stomach, the lesser omentum and stomach form theanterior wall of the sac to the left the sac extends to the hilum of the spleen where the peritoneum forms the lienorenal and gastrosplenic ligaments, while at its right edge is the epiploic foramen, also described below with thelesser omentum. The sloping roof of the sac is the peritoneum covering thecaudate lobe of the liver and this is continuous with the peritoneum of the posterior wall which overlies part of the diaphragm, pancreas, left kidney andsuprarenal gland. The lowest

part of the posterior wall is the transversemesocolon, attached to the lowest part of the pancreas Many of these posterior wall features can only be properly understood when this region of the posterior abdominal wall and its associated structures have beenconsidered, but some further details should be noted now.A finger introduced through the epiploic foramen cannot explore the whole of the lesser sac, but some features are palpable. Behind the posteriorwall to the left of the inferior vena cava is the aorta, here giving off thecoeliac trunk, two of whose branches may be felt. The common hepaticartery curves down to the right behind the peritoneum and then turns upbehind the first inch of the duodenum to enter the lesser omentum. It raisesthe pancreaticoduodenal fold, which can be felt to the left of the fold thefinger tip passes steeply downwards behind the pylorus, as if over a step.The left gastric artery runs up towards the oesophageal opening to enter thelesser omentum on its way up it raises the palpable pancreaticogastric fold.These two folds together produce a slight hourglass constriction of the sacbeyond which the cavity becomes extensive, but the examining finger cannot reach its limits.

PHYSIOLOGY OF PERITONEUM

Peritoneum is a complex serous membrane which lines the abdominal wall and is reflected over the viscera within the abdomen. The parietal and visceral layers are developed respectively, from the somatopleural andsplanchnopleural layers of the lateral plate mesoderm.The total area of the peritoneal surface in the adult is between 1.5 and2m 2 approximately equal to the toal body surface area. The blood flow tothe peritoneum is 50-70 ml/mt.The normal peritoneum consists of a single layer of flattenedmesothelial cells, microvilli protrude from the free mesothelial surface whichis lubricated by a small column of serous fluid, In women of reproductiveage the amount of fluid varies, being greatest during the luteal phase, Ifthe ovaries are inactive the volume is 4ml only .

CLASSIFICATION AND CAUSES OF ASCITES

Ascites causes may be classified in two ways. Basically the causes of ascites may be grouped into those conditions in which the pathological process does not directly affect the peritoneum and those in which the peritoneum itself is involved. Otherwise ascites can also be classified according to the high gradient and low gradient concept. By the first methodascites can be classified as follows.

CAUSES OF ASCITES:

(I) ASCITES NOT ASSOCIATED WITH PERITONEAL DISEASE:

- **Intra-hepatic sinusoidal portal hypertension**
  - Cirrhosis
  - Acute alcoholic hepatitis
  - Fulminant hepatitis (toxic or viral)
  - Sub-acute hepatitis (toxic or viral)
  - Hepatic veno-occlusive disease
  - Massive liver metastasis
- **Extra-hepatic sinusoidal portal hypertension**
  - Congestive heart failure
  - Constrictive pericarditis
  - Inferior vena-caval obstruction
  - Hepatic vein obstruction (budd-chiari syndrome)

- **Hypoalbuminemia**
  - Nephrotic syndrome
  - Protein losing enteropathy, Malnutrition
- **Miscellaneous disorders**
  - Myxoedema
- **Ovarian disease**
  - Carcinoma
  - Benign tumours
  - Ovarian hyperstimulation syndrome
- **Pancreatic ascites**
- **Bile ascites**
- **Chylous ascites**
- **Nephrogenic ascites**
- **Acquired immunodeficiency syndrome**

(II) ASCITES DUE TO PRIMARY PERITONEAL DISEASE:

- **Malignant ascites:**
  - Primary peritoneal mesothelioma
  - Secondary peritoneal carcinomatosis
- **Granulomatous peritonitis:**
  - Tuberculous peritonitis
  - Chlamydia trachomatis peritonitis
  - Fungal and parasitic peritonitis (Candidaalbicans, Histoplasmacapsulatum, Coccidioidesimmitis,
- Cryptococcus neoformans, Schistosomamansoni, Strongyloidesstercoralis, Entamoebahistolytica)
  - Sarcoidosis
  - Starch granulomatous peritonitis
  - Barium peritonitis
  - Vasculitis (SLE, Henoch schonlein purpura)
- **Miscellaneous peritoneal disease:**
  - Eosinophilic gastro-enteritis
  - Whipple's disease, Endometriosis

CLASSIFICATION OF ASCITES BASED ON HIGH GRADIENT AND LOW GRADIENT CONCEPT

- **HIGH GRADIENT ASCITES**
- **Hypoalbuminaemia**
  - Nephrotic syndrome
  - Protein loosing enteropathy
- **Venous Hypertension**
- **Poor return of blood to Right side of heart**
- Eg. - Congestive Heart Failure
  - Tricuspid regurgitation
  - Constrictive pericarditis
- **Blockage of Hepatic veins and / or Venacava**

- Budd Chiari Syndrome
- Veno-occlusive disease

- **Portal vein obstruction**
- **Diffuse Hepatic disease with portal hypertension.**

- All forms of cirrhosis

- **Infiltrative process of Liver**

- Tumours,
- Lymphoma.
- Granulomatous diseases

II. LOW GRADIENT ASCITES

- **Inflammatory diseases of the peritoneum**
- Eg. - Ruptured viscus with or without
- an Intraabdominal sepsis

- Tuberculosis
- Bacterial peritonitis
- Pancreatitis
- Bile peritonitis

- **Malignancy:**

- Metastasis to liver or peritoneum
- Hepatocellular carcinoma
- Cholangiocarcinoma
- Primary mesothelioma

- **CHYLOUS ASCITES:**

- Trauma to Thoracic duct
- Filariasis
- Mediastinal tumors

PATHOGENESIS:

BODY FLUID DISTRIBUTION:

Of the total fluid in the human body two-thirds reside inside the cell (i.e intracellular fluid) and one-third resides outside the cell (i. extra cellular fluid). The patient with generalized oedema has an excess of ECF. The ECF resides in two locations: in the vascular compartment (plasma fluid) and between the cells of the body, but outside the vascular compartment (interstitial fluid). In the vascular compartment approximately 85% of the fluid resides on the venous side of the circulation and 15% on the arterial side.

STARLING'S LAW:

It states that the rate of fluid movement across a capillary wall is proportional to the hydraulic permeability of the capillary, the trans capillary hydrostatic pressure difference, and the trans capillary oncotic pressure difference.

PATHOGENESIS OF ASCITES IN CIRRHOSIS OF LIVER:

To explain the initiating event three theories have been proposed.

The Under filling theory:

This suggests that the primary abnormality is the inappropriate sequestration of fluid within the splanchnic vascular bed due to portal hypertension and a consequent decrease in the effective circulating blood

volume. The apparent decrease in the intravascular volume (under filling) is sensed by the kidney, which responds by retaining salt and water.

The overflow theory:

Suggests that the primary abnormality is inappropriate renal retention of salt and water in the absence of volume depletion.

The peripheral arterial vasodilatation theory:

According to this theory, portal hypertension results in splanchnic, arteriolar vasodilation leading to under filling of the arterial vascular space and baroreceptor mediated stimulation of renin-angiotensin sympathetic output and antidiuretic hormone release. Regardless of the initiating event a number of factors contribute to accumulation of fluid in the abdominal cavity. Increased central sympathetic outflow is found in patients with cirrhosis and ascites. This results in diminished natriuresis by activation of renin-angiotensin system and diminished sensitivity to atrial natriuretic peptide. Portal hypertension plays an important role by raising hydrostatic pressure within the splanchnic capillary bed. Hypoalbuminemia and reduced plasma oncotic pressure also favour the extravasations of fluid from plasma to the peritoneal cavity. Hepatic lymph may weep freely from the surface of the cirrhotic liver due to distortion and obstruction of the hepatic sinusoids and lymphatics and contribute to ascites formation.

To summarize, the presence of portal hypertension contributes to the development of ascites in patients who have cirrhosis. There is an increase inintrahepatic resistance, causing increased portal pressure, but there is also vasodilatation of the splanchnic arterial system, which in turn results in an increase in portal venous inflow. Both of these abnormalities result in increased production of splanchnic lymph. Vasodilating factors such as nitric oxide are responsible for the vasodilatory effect. These hemodynamic changes result in sodium retention by causing activation of the renin-angiotensin-aldosterone system with the development of hyperaldosteronism. The renal effects of increased aldosterone leading to sodium retention also contribute to the development of ascites. Sodium retention causes fluid accumulation and expansion of the extracellular fluid volume, which results in the formation of peripheral edema and ascites. Sodium retention is the consequence of a homeostatic response caused by under filling of the arterial circulation secondary to arterial vasodilatation in the splanchnic vascular bed. Because the retained fluid is constantly leaking out of the intravascular compartment into the peritoneal cavity, the sensation of vascular filling is not achieved, and the process continues.Hypalbuminaemia and reduced plasma oncotic pressure also contribute to the loss of fluid from the vascular compartment into the peritoneal cavity. Hypalbuminaemia is due to decreased synthetic function in a cirrhotic liver.

Pathogenesis of Ascites in portal hypertension

Cirrhosis and Its Complications:

Cirrhosis is a condition that is defined histopathologically and has a
variety of clinical manifestations and complications, some of which can be
life-threatening. In the past, it has been thought that cirrhosis was never

↑ Nitric oxide

PHT

Vasodilation

Renal sodium retention

↑ Sympathetic nervous activity, rennin, aldosterone Overall filling of intravascular volume

ASCITES

Reversible; however, it has become apparent that when the underlying insult that has caused the cirrhosis

has been removed, there can be reversal of fibrosis. This is most apparent with the successful treatment of chronic hepatitis C; however, reversal of fibrosis is also seen in patients with hemochromatosis who have been successfully treated and in patients with alcoholic liver disease who have discontinued alcohol use.

CAUSES OF CIRRHOSIS :

- Alcoholism
- Chronic viral hepatitis
- Hepatitis B
- Hepatitis C
- Autoimmune hepatitis
- Non alcoholic steatohepatitis
- Biliary cirrhosis
- Primary biliary cirrhosis
- Primary sclerosing cholangitis
- Autoimmune cholangiopathy
- Cardiac cirrhosis
- Inherited metabolic liver disease
- Hemochromatosis
- Wilson's disease
- α1 Antitrypsin deficiency
- Cystic fibrosis
- Cryptogenic cirrhosis.

MALIGNANT ASCITES

Most metastatic tumours originate from adenocarcinomas but lymphoid or myeloid tumors can also infiltrate the peritoneum. Tumors responsible for malignant ascites are many. The most frequent are pancreas, ovary, colon. Ascites may develop for a number of reasons – peritoneal carcinomatosis (2/3 cases), massive liver secondaries, or chylous ascites. Widespread implanation of tumor nodules on the serosal surfaces of Viscera is often found. The pathophysiologic mechanisms of malignant ascites ispoorly understood. Mechanism of fluid retention in patients with malignant related ascites may depend on the location of the tumor. Alterations in peritoneal capillary permeability may further provoke ascites formation. It is also suggested that there can be exudation of proteinaceous fluid from tumor cells lining the peritoneum and extracellular fluid enters the peritoneal cavity to re-establish oncotic pressure. Ascites in patients with primary hepatocellular carcinoma is almost always Secondary to portal hypertension. Chylous ascites due to malignant lymphoma appears to be caused by lymphnode obstruction by tumor and rupture of chyle containing lymphatics. The macroscopic appearance of malignant ascites is generally similar to that of cirrhotic ascites. Thus the differential diagnosis must be based on exploratory findings and laboratory tests. Measurement of total protein concentration of the ascitic fluid (generally over 3.0 gms/dl in malignant ascites) and its cytological examination for the malignant cells which were the laboratory tests first used to differentiate malignant ascites from that secondary to portal hypertension are still the most common methods used. Standard cytological examination is 60 to 90% accurate in the diagnosis of malignant ascites, especially when adequate volumes of fluid(atleast several hundred milliliters) and concentration techniques are used.False positive results are rare in skilled hands. The

greatest source of confusion is the differentiation of the malignant cells from atypical mesothelial cells. The use of immuno-cyto-chemical techniques with monoclonal or polyclonal antibodies against numerous tumour markers [oestrogen and progesterone receptors, cancer antigen 125, carbohydrateantigen 19_9, carcino-embryonic antigen, epithelial membrane antigen,human erythrocyte antigen , CD15, CD45, CD11c, epithelial glycoprotein 34,BW-495, tumour associated antigen 72, epithelial specific tumour associatedmonoclonal antibody (Moab), cytokeratin, vimentin, lysozyme and 83D4antigen] are useful in differentiating malignant from non-malignant ascitesin these cases. These techniques may also help differentiate primary(mesothelioma) from metastatic peritoneal malignancy. False negative results on cytological examination are the rule when the ascites is due to portal hypertension secondary to massive liver metastasis with little peritoneal involvement. In this type of ascites the total ascitic protein concentration is usually lower than 2.5 gms/dl. The differential diagnosis between cirrhotic ascites and the ascites secondary to massive liver metastasis, however, can easily be achieved by ultrasonography, computed tomography or liver scan. DNA analysis by flow cytometry or image analysis, and the detection of oncogene (c-Ha-ras) expression, are sophisticated techniques that have been explored in the differential diagnosis of effusion samples, but they do not improve the results obtained with conventional cytological examination.

The serum ascitic fluid gradient for albumin improves the diagnostic accuracy of total protein concentration in the ascitic fluid. The concentration of lactic dehydrogenase in malignant ascites is higher than the corresponding values in plasma due to leakage of enzymes from the malignant cells lining the peritoneum, whereas the reverse is the rule of cirrhotic ascitis. The concentration of lactic dehydrogenase in ascitic fluid and its ascitic fluid plasma ratio are, therefore, useful for differentiating malignant from cirrhotic ascites, although they do not improve the results obtained with the ascetic fluid-plasma albumin gradient. Other measurement in ascitic fluid that have proved to be of value in differentiating malignant from cirrhotic ascites include total lipids, free fatty acids, cholesterol, fibro-nectic, carcinoembryonic antigen and other tumour-associated antigens, urokinase, tissue plasminogen activator, plasminogen activator inhibitor, fibrin/fibrinogen degradation products and human chorionic gonadotropin-β. Of these last, the cholesterol concentration of ascitic fluid, which is higher in most malignant ascites than in cirrhotic ascites owing to higher permeability to lipoproteins, seems to the most interesting because of its simplicity and cost-effectiveness. Laparoscopy and direct biopsy of the peritoneal metastasis is a useful approach to confirm the diagnosis of malignant ascites in those cases with negative cytology.

Classification of malignancy related Ascites

- Peritoneal carcinomatosis,
- Massive Liver metastasis
- Peritoneal carcinomatosis with massive liver metastasis
- Hepatocellular carcinoma
- Malignant lymphnode obstruction
- Malignant Budd- Chiari Syndrome (tumor emboli in hepatic veins).

ASCITES IN CARDIAC FAILURE:

Heart failure is a clinical syndrome in which an abnormality of cardiac structure or function is responsible for the inability of the heart to eject or fill with blood at a rate commensurate with the requirements of the metabolizing tissues. Heart failure results in a constellation of clinical manifestations, including, in various

combinations, circulatory congestion, dyspnoea, fatigue, and weakness. Heart failure is frequently but not always, caused by a defect in myocardial contraction, and then the term myocardial failure is appropriate.

Etiology:

Two mechanisms that reduce cardiac output are recognized to cause congestive heart failure; systolic dysfunction and diastolic dysfunction. Although physical examination, chest x-ray, and echocardiogram are useful in this regard, additional diagnostic tests are usually indicated. An electrocardiogram provides information about systolic (ejection fraction) and diastolic function, and about vascular disease which may require surgery. Occult hypo- or hyperthyroidism and alcoholic cardiomyopathy may present as congestive cardiac failure; these are treatable. Ascites occurs in patients with increased pressure in the hepatic veins and in the veins draining the peritoneum. Ascites usually reflects the long standing systemic venous hypertension. In patients with organic tricuspid valve disease and chronic constrictive pericarditis ascites may be more prominent than subcutaneous edema. Protein losing enteropathy may rarely occur in patients with visceral congestion or end stage congenital heart disease, and the resultant reduced plasma oncotic pressure may lower the threshold for the development of ascites.

NEPHROTIC SYNDROME:

Another major cause of edema is nephrotic syndrome, the clinical hallmarks of which include proteinuria (>3.5 gms/day), hypoalbuminemia, hypercholesterolemia and edema. The degree of edema may range from pedal edema to total body anasarca, including ascites and pleural effusions.

The lower the plasma albumin concentration, the more likely the occurrence of anasarca; the degree of sodium intake is, however also a determinant of the degree of edema. The nephrotic syndrome is a clinical complex characterised by a number of renal and extra- renal features, the most prominent of which are proteinuria of more than 3.5gms/1.73m2/24hrs, hypoalbuminemia, edema, hyperlipidaemia, lipiduria and hypercoagulability. Nephrotic syndrome cancomplicate any disease that perturbs the negative electro-static charge orarchitecture of the GBM and the podocytes and their slit diaphragms. Recent attention has focused on several key molecules that mediate GBM-podocyte slit diaphragm interactions such as nephrin, podocin and alpha actinin-4. Six entities account for more than 90% cases of nephrotic syndrome in adults. Minimal change disease , focal and segmental glomerulosclerosis (FSGS), membranous glomerulopathy, membrano-proliferative glomerulonephritis, diabetic nephropathy and amyloidosis.

Pathophysiology:

The pathogenesis of ECF volume expansion in nephrotic syndromeappears to be due to primary renal Na retention causing nephrotic edema. In general, the greater the proteinuria, the lower the serum albumin level. Hypoalbuminemia is compounded further by increased renal catabolism and inadequate, albeit usually increased hepatic synthesis of albumin. The pathophysiology of edema in nephrotic syndrome is poorly understood. The under filling hypothesis postulates that hypoalbuminemia results in decreased intravascular oncotic pressure leading to leakage of extracellular fluid from blood into interstitium. Intravascular volume falls, thereby stimulating activation of renin-angiotensin-aldosterone axis and the sympathetic nervous system and release of vasopressin and suppressing atrial natriuretic peptide release. These neural and hormonal responses promote renal salt and water retention, thereby restoring intravascular volume, and triggering further leakage of fluid into interstium. Primary renal salt and water retention also contributes to edema formation in some cases. It appears, therefore, that nephrotic syndrome reflects a combination of primary renal NaCl retention and relative arterial under filling. In general, normal or near normal glomerular filtration rate is associated with hypovolemic, vaso-constrictive nephrotic

syndrome, whereas a diminution in glomerular filtration rate, primary renal sodium retention, and evidence of volume expansion (e.g., decreased plasma renin activity) are characteristic of hypovolemic nephrotic syndrome.

TUBERCULOUS PERITONITIS:

The differential diagnosis between cirrhotic ascites and ascites due totubercular peritonitis is particularly important since alcoholic cirrhosis may predispose to peritoneal tuberculosis. Clinically, tuberculous peritonitis is characterized by fever, abdominal pain, anorexia, weight loss, abdominal tenderness and ascites. However, none of these symptoms is invariably present. The proportion of the patient with pleural or pulmonary tuberculosis or with a reactive tuberculin skin test ranges between 21 and 78% and between 30 and 89% respectively. In females without active tuberculosis, peritoneal tuberculosis may represent the local extension of tuberculous salpingitis. However, in many cases no active focus of tuberculosis, apart from the peritoneal disease, can be detected. Ultrasonography and computed tomography may suggest the diagnosis of tuberculous peritonitis. Findings frequently seen in tuberculous peritonitis include diffuse, regular peritoneal thickening, infiltration of greater omentum, ascites with fine, mobile septations or floating debris on ultrasonography, loculations of ascites, bowel thickening, particularly in ileo-caecal area, retro-peritoneal lymph node enlargement, lesions in solid organs (pelvic , adrenal, hepatic, splenic), cold abscesses and adhesions. Results of examination of the peritoneal fluid are also suggestive of tuberculous infection if there is an increased concentration of protein (>3gms/dl) and lymphocytes. However, it has been shown that the ascetic fluid may be a transudate, particularly in cirrhotics with ascites and tuberculous peritonitis. Ziehl-nielsen stained smears usually fail to show acid fast bacilli. The proportion of cultures of ascitic fluid positive for Mycobacterium tuberculosis varies markedly from series to series (from 8-69%) probably reflecting technical differences. It has been suggested that the proportion of positive cultures may be increased upto 80% by concentrating 1 litre of the fluid by centrifugation. Nevertheless, the diagnosis of tuberculous peritonitis cannot be based on cultures of ascetic fluid since the usual techniques of culturing acid fast bacilli may require several weeks to obtain a definite result. The activity of lactic dehydrogenase in ascitic fluid is greater in tuberculous peritonitis than cirrhosis. As in malignant ascites, the concentration of this enzyme in tuberculous ascites is higher than in plasma. There are reports that the concentration of tumour antigens CA-125 in ascitic fluid may be very high in tuberculous peritonitis. The activity of adenosine deaminase in the peritoneal fluid is proven sensitive and specific test for tuberculous peritonitis. This is an enzyme in the catabolism of purine bases. It participates in the proliferation and differentiation of lymphocytes, and increases in tuberculous effusions probably as a consequence of the stimulation of cell mediated immunity and T- lymphocytes. The iso-enzyme adenosine deaminase II is a dominant component of tuberculous pleural effusions. The concentration of interferon γ in ascitic fluid is also greater in tuberculous peritonitis than cirrhotic ascites, although this does not improve on the results obtained with adenosine deaminase in the diagnosis of this condition. The concentration of adenosine deaminase in ascitic fluid in tuberculous peritonitis correlates directly with the total protein concentration in ascites. It is therefore not surprising that the number of false negative results for adenosine deaminase in tuberculous peritonitis is higher in cirrhotic patients than in patients without chronic liver disease. Open peritoneal biopsy during a laparotomy or a mini-laparotomy, blind needle biopsy of the peritoneum, and laparoscopy with direct biopsy of the affected areas have been used to confirm the diagnosis of tuberculous peritonitis. Laparoscopy with direct peritonel biopsy is the best of these methods. The peritoneum characteristically shows scattered or confluent military nodules of uniform

size, with adhesions between bowel loops, liver capsules and abdominal walls. The histological appearance is characterized by the presence of caseating granulomas. In some instances, tubercle bacilli may be seen by staining with aura mineRhoda mine and microscopy under ultra-violet light. Mycobacterium tuberculosis can be cultured from the biopsy specimen of the peritoneum. The macroscopic and microscopic appearances of tuberculous peritonitis are similar tothose of other conditions causing granulomatous peritonitis, such as sarcoidosis, crohn's disease, and iatrogenic granulomatous peritonitis. The last condition occurs after 0.15% of abdominal operations and is usually caused by a cell mediated immune response to starch, talc, cotton fibres, wool fibres originating from disposable surgical gowns and drapes. Iatrogenic granulomatous peritonitis appears 2-9 weeks post-operatively and is characterised by abdominal pain, tenderness and fever, and frequently by accumulation of ascites. The observation of starch granules in the ascitic fluid obtained by paracentesis can be diagnostic.

PANCREATIC ASCITIS:

Pancreatic ascites occurs in approximately 3% of patients with chronic pancreatitis as a result of leakage of fluid from ruptured pancreatic duct, or from a pancreatic pseudo-cyst into the peritoneal cavity. Other less frequent causes include acute hemorrhagic pancreatitis, abdominal trauma and pancreatic cancer. Since most patients with chronic pancreatitis are alcoholics and may develop massive ascites with little or no abdominal tenderness, the differential diagnosis of pancreatic from cirrhotic ascites may be difficult on clinical grounds. Laboratory analysis are therefore essential to establish a correct diagnosis. In virtually all cases, serum and especially ascitic fluid amylase and lipase are dramatically increased. The concentration of pancreatic enzymes in ascitic fluid is between 5 and 20times greater than the plasma concentrations obtained simultaneously. The protein concentration in ascitic fluid is generally over 3gms/dl and the fluid is usually serous, but can be sero-sanguineous, turbid, chylous. The concentration of methaemalbumin in ascites is markedly increased in patients with haemorrhagic pancreatitis and has prognostic significance. The concentration of leucocytes in ascitic fluid ranges between 70 and 2200/mm3,80% being lymphocytes. Ultrasonography or computed tomography are important diagnostic procedures for pancreatic ascites since they may detect the presence of pseudo-cyst. Pseudo-cyst in patients with pancreatic ascites are usually small, due to continuous leakage of the cyst fluid into the peritoneal space.

BUDD - CHIARI SYNDROME:

This syndrome comprises of Hepatomegaly, Abdominal pain, Ascites, Zone 3 sinusoidal distension and pooling. Arise from obstruction to hepatic veins at any site from the efferent vein of the acinus to the entry of the inferior vena cava into the right atrium. Constrictive pericarditis or right heart failiure – produce a similar syndrome.

Associated clinical conditions :

- Myeloproliferative diseases (polycythemiarubravera)
- Systemic lupus erythematosus.
- Disseminated intravascular coagulation.
- Antiphospholipid syndrome.
- Idiopathic granulomatous venulitis
- Deficiency of anticoagulant factors – antithrombin III , protein C
- or S deficiency.

- Paroxysmal nocturnal haemoglobinuria
- Risk factors :
- Oral contraceptives
- Pregnancy
- Trauma in those with hypercoagulable state.
- Liver transplantation and cellular rejection.
- In Neoplasm:
- Secondary to thrombosis in malignant disease – adrenal ca.,
- renal ca.,
- Invasion by hepatocellular carcinoma.
- Angiosarcoma, leiomyosarcoma of the hepatic veins.
- Testicular lesions metastatic to the right atrium.
- Wilms tumour metastasis 12.

OTHER TYPES OF ASCITES:

Other causes of ascites is easily differentiated from cirrhotic ascites include nephrogenic ascites, myxoedema, and Meig's syndrome. The pathogenesis of ascites in these patients is unknown. The protein concentration in ascitic fluid is usually over 3.0gms/dl and WBC count ranges between 30 and 1500/mm3. The amylase and lactic dehydrogenase activities in ascitic fluid are lower than the plasma concentration. High protein ascites may develop in patients with Acquired immunodeficiency syndrome in the absence of portal hypertension or any other potential cause of ascites .

ASCITES CLINICAL FEATURES AND DETECTION

Abdominal distension is a common problem in clinical medicine and may be the initial manifestation of a systemic disease or of otherwise unsuspected abdominal disease. Pain is uncommon in ascites due to cirrhosis, but when it is present ,pancreatitis, hepatoma or spontaneous bacterial peritonitis should be considered. Inspection of the abdomen is an important aspect of the abdominal examination. By noting abdominal contour, one may be able to distinguish localized from generalized swelling. The tensely distended abdomen with tightly stretched skin, bulging flanks and everted umbilicus is characteristic of ascites. Other associated features which may be present are hernia, abdominal striae, divarication of recti and occasionally meralgia paraesthetica, and scrotal oedema. Prominent abdominal venous pattern with the direction of flow away from the umbilicus often is a reflection of portal hypertension. Venous collaterals from the lower part of the abdomen towards the umbilicus suggest obstruction of Inferior venacava. Pleural effusions can be found in 10% of patients usually on the right side.

On examination small amounts of fluid may be difficult to detect in obese patients. Patients usually have flank dullness to percussion and shifting dullness. Approximately 1500ml, of fluid is required to cause flank dullness. If no flank dullness is present the patient has less than 10% chance of having ascites. Eliciting periumbilical dullness with the patient on hands and knees what is known as Puddle sign can detect fluid as little as 120ml in quantity. But the sensitivity and specificity of the puddle sign in detecting ascites have been found to be much lower than those of shifting dullness. A venous hum at the umbilicus may signify portal hypertension and an increased collateral blood flow around the liver.A very hard or nodular liver is a clue that the liver is infiltrated with tumor and when accompanied by ascites, it suggests

that the latter is due to peritoneal seeding. The presence of a hard periumbilical nodule (sister MaryJoseph's nodule) suggests, metastatic disease from a pelvic or gastrointestinal primary.
The neck veins of the patients with ascites should always be examined specifically. Constrictive pericarditis is one of the few curable causes of ascites. A pulsatile liver and ascites may be found in tricuspid regurgitation.

- This is a cross-sectional study being done in Department of Radio-diagnosis and cytopathology being done in Pathology Department , Shri M.P. Shah Medical College, G.G Hospital, Jamnagar.
- This study was carried out after clearance and approval of institutional ethics committee.
- A written and informed consent was obtained from all the subjects.
- Subjects will be examined by CT, USG and Cytopathology modalities.
- Results will be checked by two radiologist (PI and CO-PI) and pathologist, final comparative data will be given between CT , USG and Cytopathology with counting of various risk factors.

Study population:

Patients of various age groups presenting to medicine OPD with complaints of ascites were included.

Sample size:

A total of 50 patients are included in the study.

Inclusion criteria:

- Patients with ascites.
- Cases of all age groups irrespective of sex.

Exclusion criteria:

- Non co-operative patient.
- Haemo-dynamically unstable patient.

PROTOCOL:

After enrolment of the case in the study a detailed history was taken and clinical examination was done along with recording of demographic information. Investigations like Computed Tomography (CT), ultrasonography(USG) and cytopathology were done to confirm the diagnosis. Final diagnosis was made after correlation with results of all tests.

1. ULTRASONOGRAPHY IMAGING TECHNIQUE

- **The ultrasound examinations were performed with Philips Affiniti 50 and E-saote 60.**
- **For scanning trans abdominally a 3.5MHz to 6MHz curved linear array probe is used.**

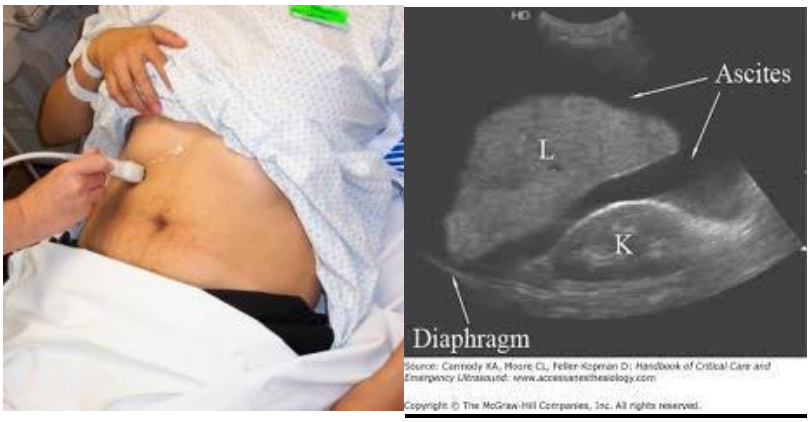
Patient position

Transabdominal technique needs the patient supine.

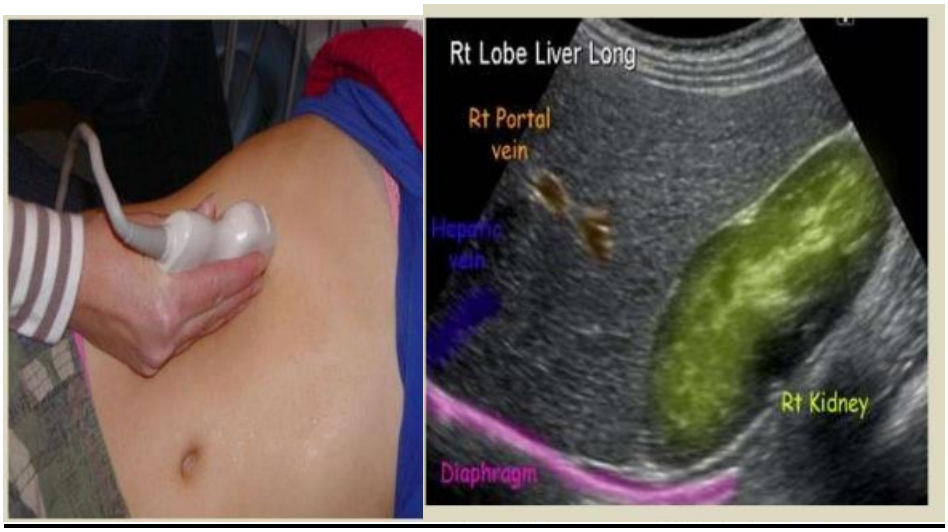
Scanning Technique

- **The patient lies supine. A urine or fluid-filled bladder is optimal for transabdominal technique to view pelvic contents.**
- **The transabdominal technique usually consists of midline sagittal and parasagittal images angled from midline.**

Transverse view



Longitudinal view



2.Computed Tomography (CT)

- Equipment- Dual source 16 slice Computed tomography scanner (Bright speed, GE health care, UK)
- Position-supine
- Coverage area- abdomen
- Scanning parameters-
  1. Helical, thin, overlapping slices.
  2. 0.625 mm thick

3. 200 mA; 120kV

" Venous ,arterial and portal algorithm

2. Cytopathology

SAMPLE COLLECTION

- The fluid is collected into a clean, dry container, which need not be sterile, and sent to the laboratory as soon as possible.
- If the fluid cannot be sent immediately, it should be stored in a refrigerator at 4 degree c and not allowed to freeze.
- We do not require anticoagulant or fixative to be added to the fluid.
- Anticoagulation by adding heparin to the receptacle before the fluid is collected does not interfere with cytologic detail.

METHODS OF PREPARATION

- Clot on standing- Exudate- High protein content, rich in fibrin.
- 1ml of 5% sodium citrate
- Centrifuged in a conical tube @ 800rpm for 12 min Lavage- 1000rpm- 10min.
- Smears made from the fluid free sediment.
- Wet fixed in 95% ethanol for Pap and H & E.
- Air dried and fixed in methanol for Romanowsky stain.
- Heavily blood stained- 1% acetic acid. Smears from buffy coat.

Types of staining smears

- PAP H & E
- Cell block for remnant sediment and histopathological examination.
- Other special stains for the most suspected diseases, to confirm diagnosis

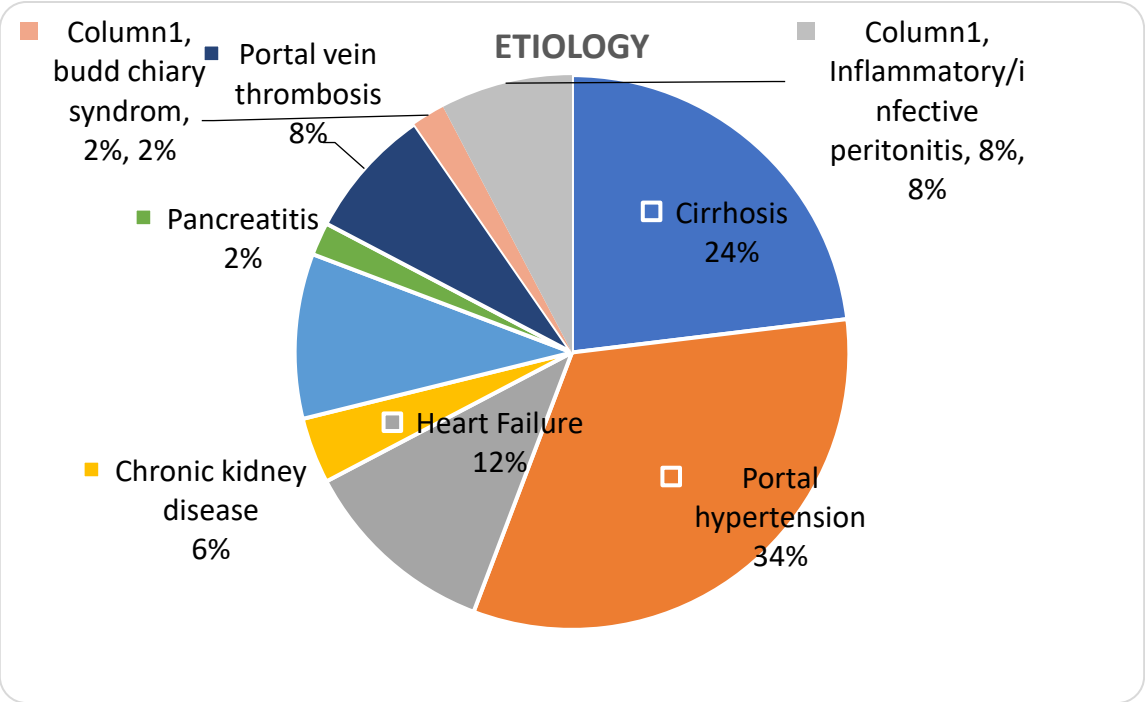
OBSERVATION AND ANALYSIS

TABLE 1

Etiological distribution of ascites

SR no	ETIOLOGY	NO. OF CASES	PERCENTAGE (%)
1.	Cirrhosis	**12**	**24**
2.	Cirrhosis with Portal hypertension	**17**	**38**
3.	Heart Failure	**5**	**10**
4.	Chronic kidney disease	**2**	**6**
5.	Malignancy	**5**	**10**
6.	Pancreatitis	**1**	**2**
7.	Portal vein thrombosis	**4**	**8**
8.	Budd- Chiari syndrome	**1**	**2**
9.	Infective/Inflammtory bowel disease	**4**	**6**

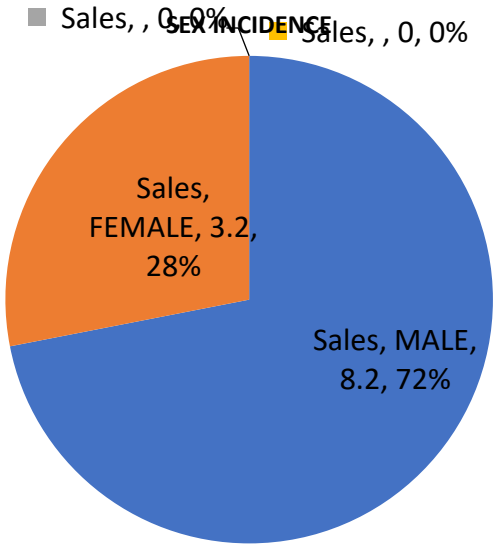
Chart -1



Above table shows cirrhosis is the most common cause for ascites.

TABLE-2
SEX INCIDENCE IN CRRHOSIS OF LIVER

SEX	No. of cases	Percentage (%)
MALE	21	72
FEMALE	8	28
TOTAL	29	100



Incidence of liver cirrhosis is more in male than female.

TABLE 3

Distribution of ascites with internal septa and free fluid

Sr.no	Etiology	Ascites with septae	Ascites without septae
1.	Cirrhosis	**0**	**12**
2.	Cirrhosis with Portal hypertension	0	**17**
3.	Heart Failure	**0**	**6**
4.	Chronic kidney disease	**0**	**2**
5.	Malignancy	2	**3**
6.	Pancreatitis	**0**	**1**
7.	Portal vein thrombosis	**0**	**4**
8.	Budd- Chiari syndrome	**0**	**1**
9.	Infective/Inflammtory bowel disease	3	**1**

CHART 3

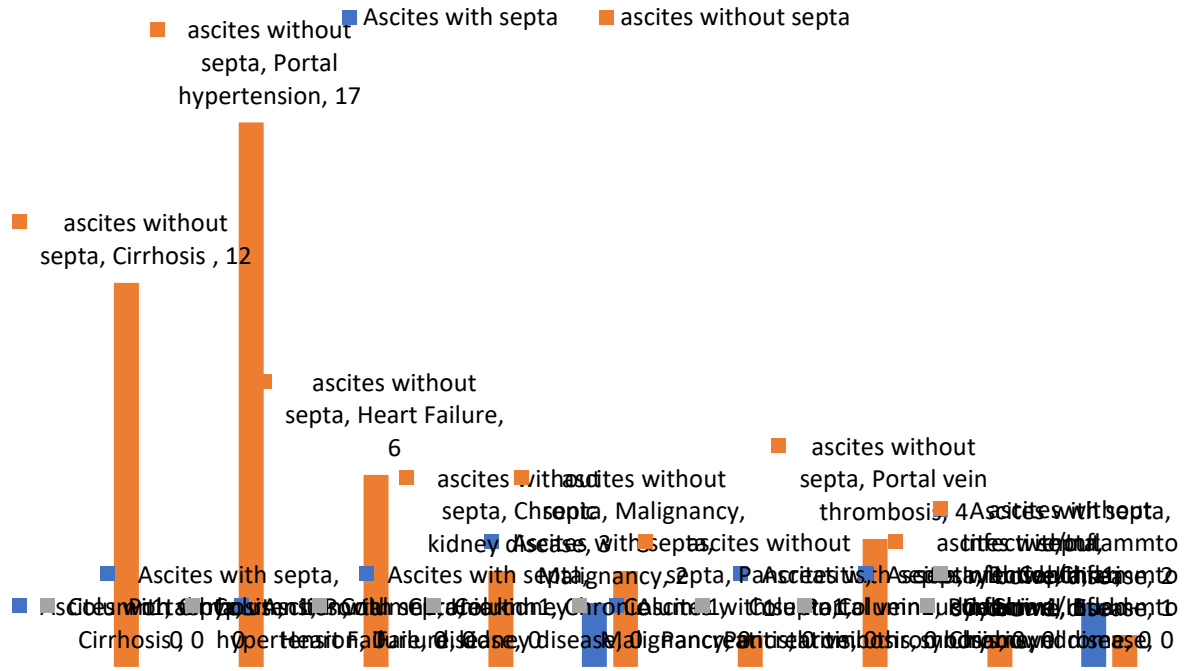


Table 4

Distribution of ascites with internal echoes and clear fluid in USG

Sr.no	etiology	Ascites with internal echoes	Clear fluid
1.	Cirrhosis	**0**	**12**
2.	Cirrhosis with portal hypertension	0	**17**
3.	Heart Failure	**0**	**6**
4.	Chronic kidney disease	**0**	**2**
5.	Malignancy	4	**1**

.6.	Pancreatitis	**1**	**0**
7.	Portal vein thrombosis	**0**	**4**
8.	Budd- Chiari syndrome	**0**	**1**
9.	Infective/Inflammtory bowel disease	3	**1**

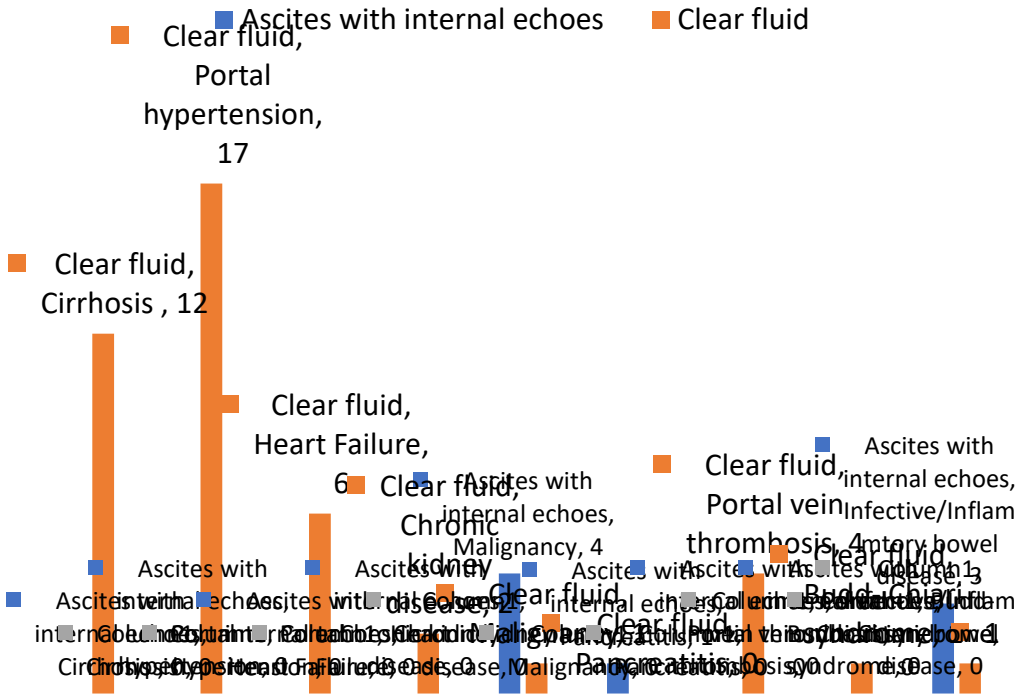


TABLE NO.5 Serum ascites albumin gradient (SAAG) in patient Cirrhosis with portal hypertension

SAAG value	No. of cases	Percentage (%)
>1.1	15	87
<1.1	2	13

CHART 5

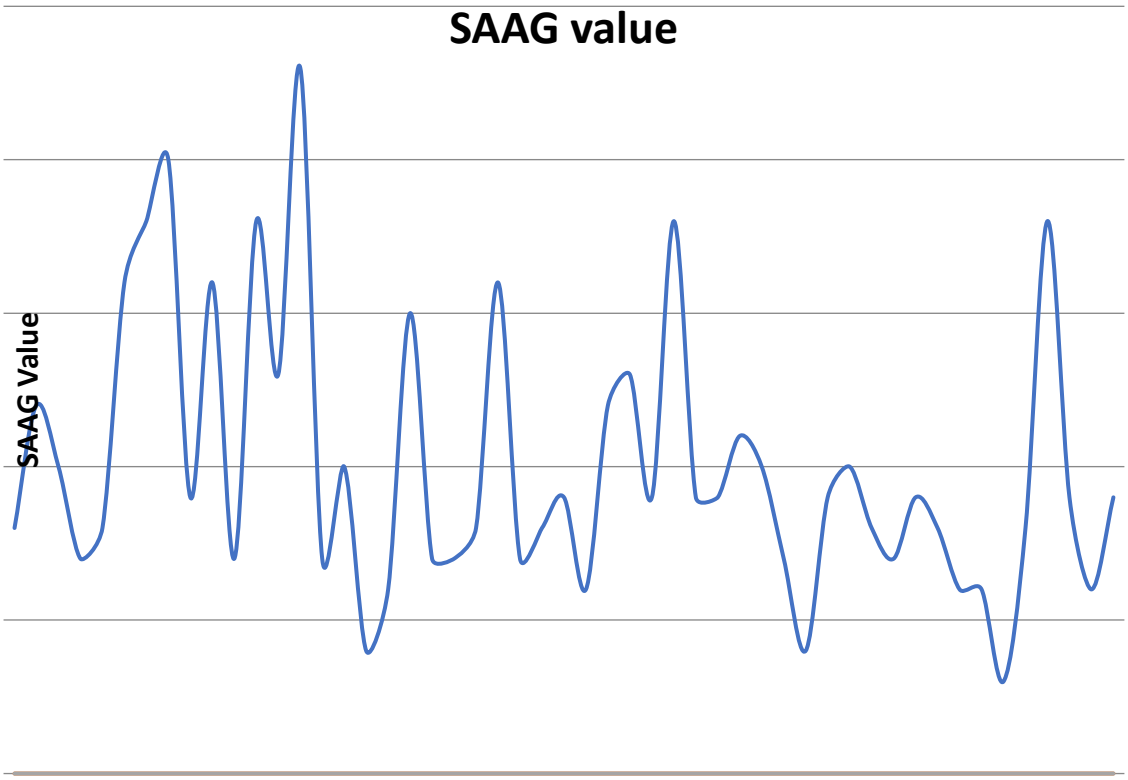


TABLE-6

Sr no.	Relation of alcohol with cirrhosis	
1.	Number of cases with cirrhosis	29
2.	Number cirrhosis with alcoholism	21
3.	Percentage of cirrhosis with alcoholism	72

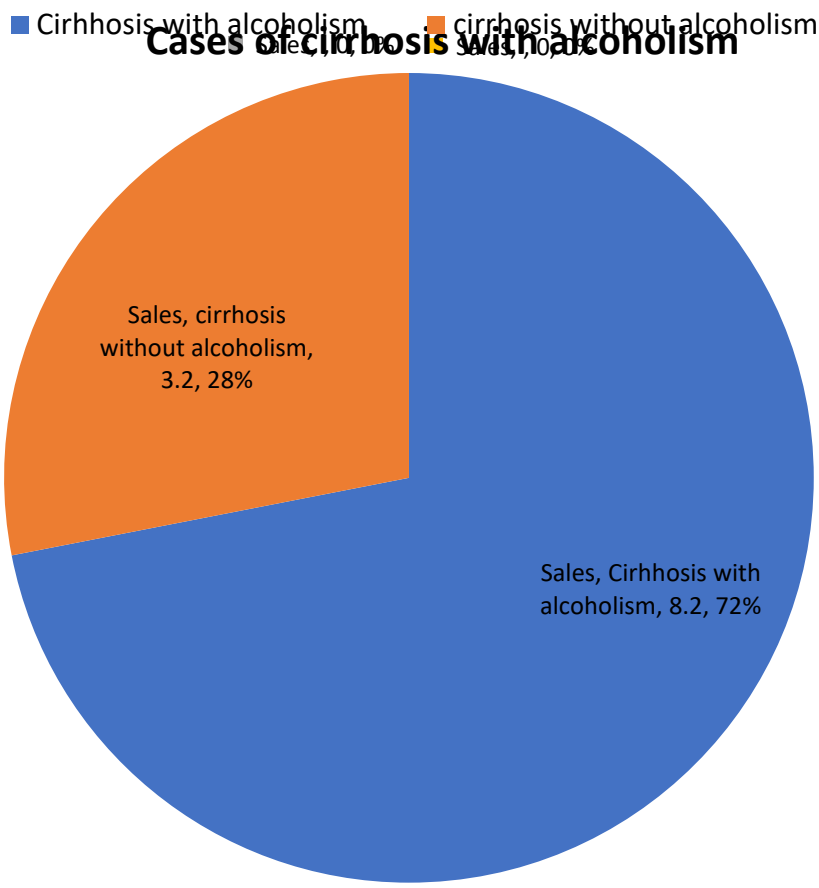
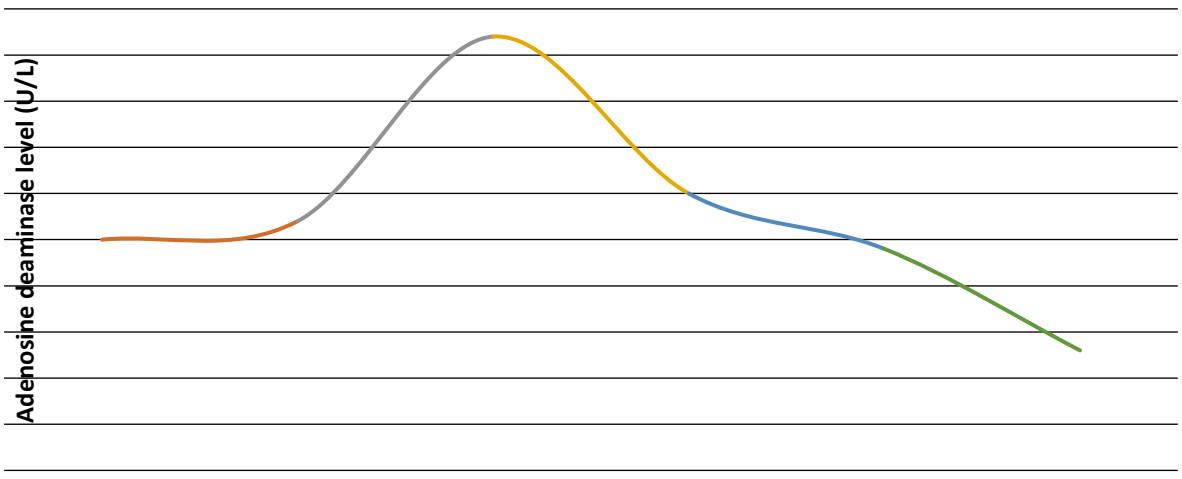


TABLE-7

Adenosine deaminase level (U/L)	Number of patients with bowel wall thickening
>43	1
< 43	3
Total	4

CHART- 7

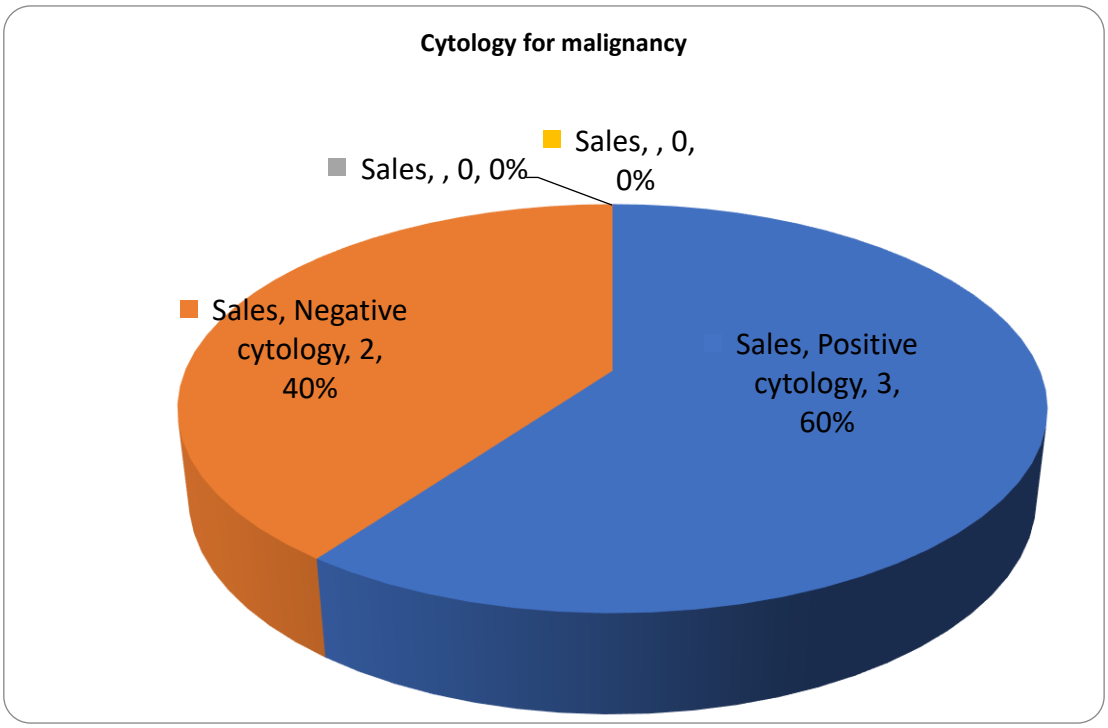


Patient

Adenosine-de aminase level is increased (> 43) with tuberculous ascites.

TABLE-8

No. cases of Malignancy	Positive Cytology	Negative cytology
5	3	2

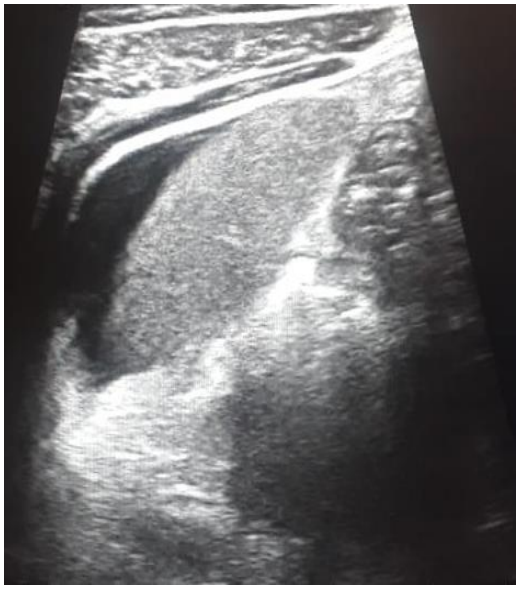


There is association of cytology positive with malignancy shows 60 %.

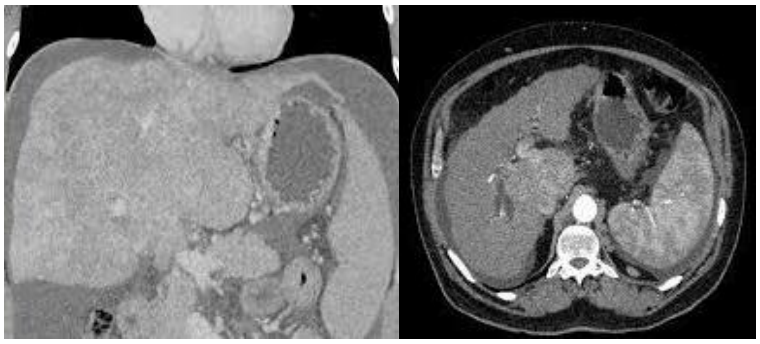
CASES

Case 1: 56 year male with complain of abdominal distension and pedal edema, history of alcoholism.

USG image :



CT images :



Cytopathology :

Time

FLUID EXAMINATION

Test Name	Observed Value	Reference Value
FLUID R-M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	07 ml	
COLOUR	Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Absent	
SUPERNATANT FLUID	Clearhtly	
CHEMICAL EXAMINATION		
PROTEINS	2.6 G/dl	Up to 3.0 G/dl
SUGAR	70 mg/dl	60 TO 100 mg/dl
CYTOLOGICAL EXAMINATION		
WBC COUNT	270 WBC/mm3	
POLYMORPHS	48 %	
LYMPHOCYTES	64 %	
RBC COUNT	72 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA'		
GRAM'S STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

TUBERCULOSIS PROFILE

Test Name	Observed Value	Reference Value
enosine-Deaminase activity	15 U/L	SERUM/PLASMA/BODY FLUID U Normal : <43 U/L Suspected for MTB : 43-62 U/L Strong suspect for MTB : >62 U/L CSF U/L Normal : <11 U/L Suspect for MTB :11-12.35 U/L Strong suspect for MTB : >12.35 U/
Material	Ascitic Fluid	

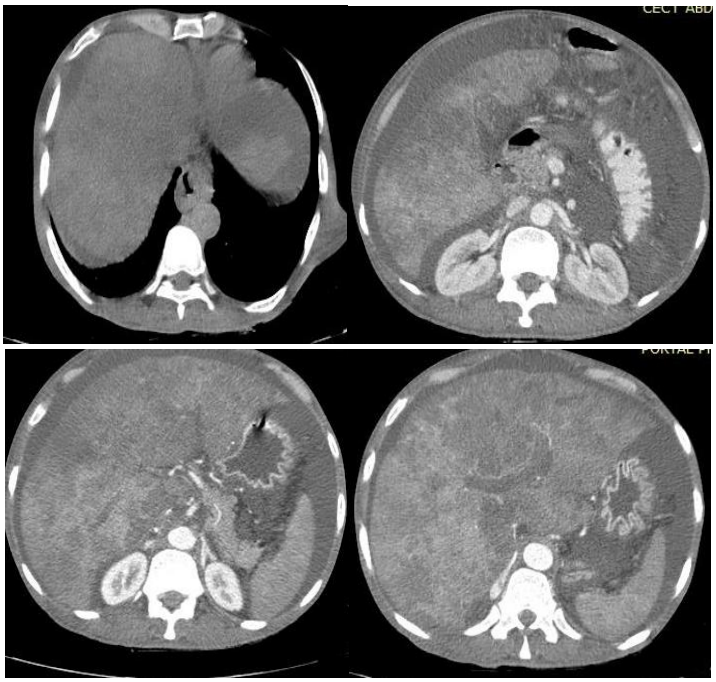
bove results are subject to variations due to technical limitations, hence corelate with clinical findings & other investigations.

k You For Your Reference.

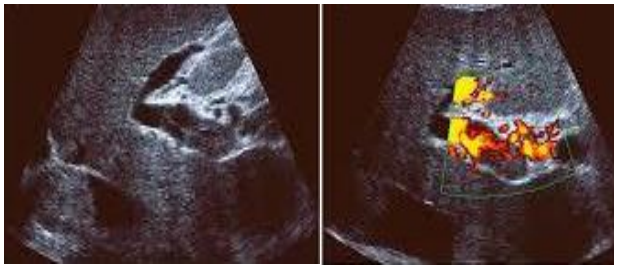
Dr. Tejal P. Kotecha (M.D)

USG findings: Liver shows irregular margin with nodular echotexture and Moderate acsites.
CT findings:Liver shows heterogenous enhancement with irregular margin and moderate ascites.
Cytopathology: Clear fluid and low protien content.
Diagonsis: Cirrhsosis of liver

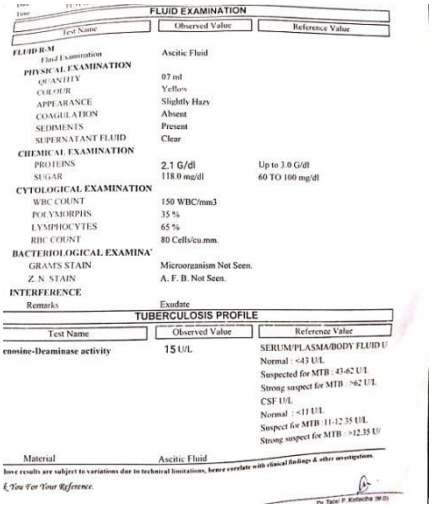
Case2 : 52 year male presented with abdominal pain, fever and jaundice



USG images



Cytopathology :



FLUID EXAMINATION

Test Name	Observed Value	Reference Value
FLUID R/M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	07 ml	
COLOUR	Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Present	
SUPERNATANT FLUID	Clear	
CHEMICAL EXAMINATION		
PROTEINS	2.1 G/dl	Up to 3.0 G/dl
SUGAR	118.0 mg/dl	60 TO 100 mg/dl
CYTOLOGICAL EXAMINATION		
WBC COUNT	150 WBC/mm3	
POLYMORPHS	35 %	
LYMPHOCYTES	65 %	
RBC COUNT	80 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA'		
GRAMS STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

TUBERCULOSIS PROFILE

Test Name	Observed Value	Reference Value
enosine-Deaminase activity	15 U/L	SERUM/PLASMA/BODY FLUID U/ Normal : <43 U/L Suspected for MTB : 43-62 U/L Strong suspect for MTB : >62 U/L CSF U/L Normal : <11 U/L Suspect for MTB :11-12.35 U/L Strong suspect for MTB : >12.35 U/

Material Ascitic Fluid

Above results are subject to variations due to technical limitations, hence correlate with clinical findings & other investigations.

Thank You For Your Reference.

USG findings: Near complete thrombosis of main portal vein with altered echotexture of liver.

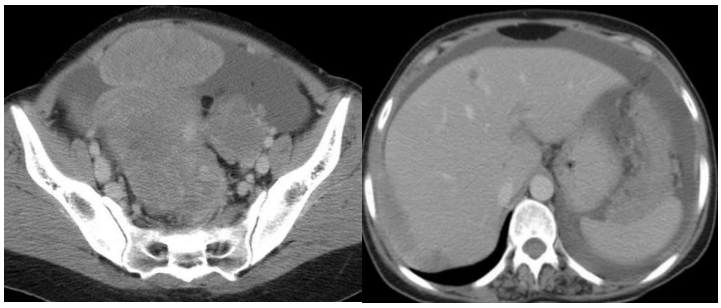
CT findings: Near complete thrombosis involving main portal vein with heterogeneous enhancement of liver.

Cytopathology : Clear fluid, low protien level, no malignant cells.

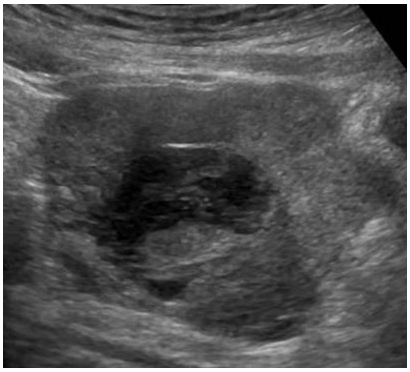
Diagnosis : Portal thrombosis with liver parenchymal disease

CASE 3 61 year female presented with pelvic pain and weight loss since 4 months.

CT images



USG image



Cytopathology:

Dr. Gupta Pathology Laboratory

Dr. (Mrs.) V. Gupta M. D., DCP
HON. PATHOLOGIST &
EX. CHIEF PATHOLOGIST : N.P. CANCER INSTITUTE, RAJKOT.
SPECIAL TRAINING IN U.S.A., U.K. & FRANCE

SHIVAM COMPLEX, 104, GROUND FLOOR,
DR. YAGNIK ROAD, RAJKOT - 360 001.
TELE : 0281 - 2463833
LMC No. 327

Pt.'s Name : PUSHPABEN B. THAKKAR Age : 70 Years Sex : F
Ref. By : Dr. JAY MISTRY Date 17/11/2017

CYTOLOGY REPORT

C.D. No : 1218/2017
Clinical : Pelvic Mass with Peritoneal Fluid
Material : Peritoneal Fluid for Cytology

Microscopy : The aspirate reveals papillaroid clusters of atypical epithelial cells showing mild nucleomegaly with hyperchromasia. In between are seen benign mesothelial cells in sheets. The haemorrhagic background shows mild inflammatory cell infiltrate.

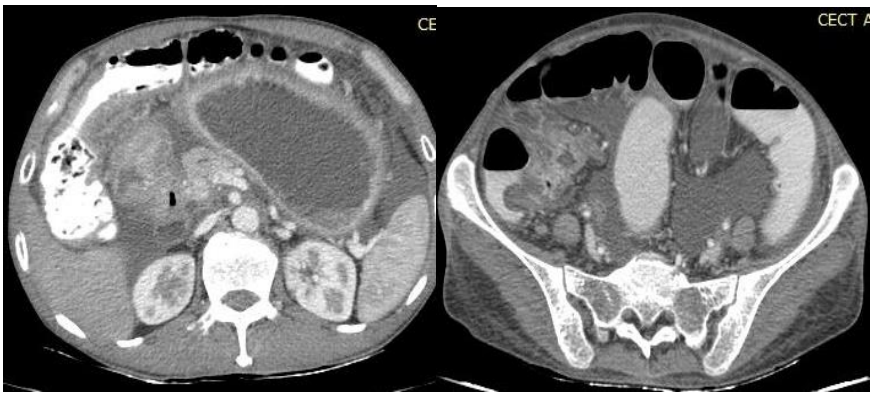
Opinion : **Malignant Ascites**
Cell Type - Serous Papillary Carcinoma

Please collect the Slides within 3 months.

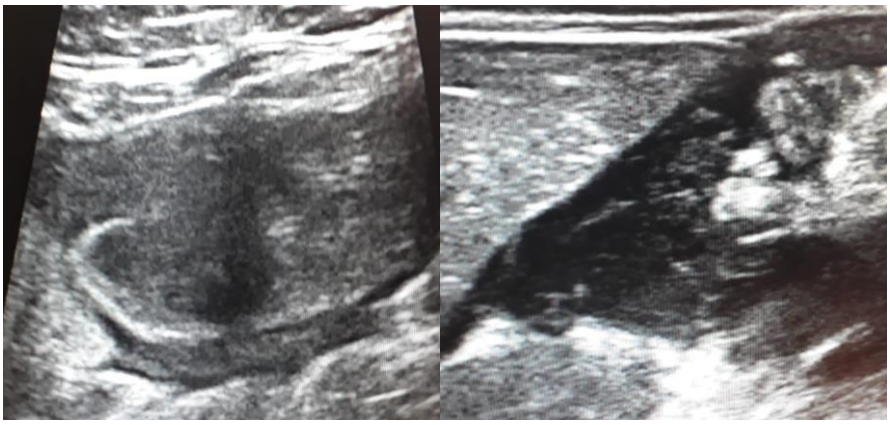
Note : The above results are subject to variations due to technical limitations, hence correlation with clinical findings and other investigations should be done.

USG finding: Well defined heterogeneously hypoechoic solid cystic lesion (predominantly solid) involving right adnexa, mild ascites.
CT findings : Well defined heterogeneously enhancing solid cystic lesion (predominantly solid) involving right adnexa, mild ascites.
Cytopathology: Hazy fluid and shows Atypical cells.
Diagnsosis : Serous papillary carcinoma of right ovary.

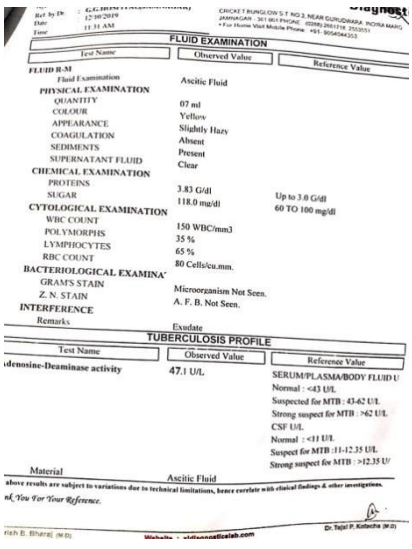
Case 4: 43 year male presented with abdominal pain and diarrhea since 3 months
CT images:



USG images:



Cytopathology



FLUID EXAMINATION

Test Name	Observed Value	Reference Value
FLUID R-M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	07 ml	
COLOUR	Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Present	
SUPERNATANT FLUID	Clear	
CHEMICAL EXAMINATION		
PROTEINS	3.83 G/dl	Up to 3.0 G/dl
SUGAR	118.0 mg/dl	60 TO 100 mg/dl
CYTOLOGICAL EXAMINATION		
WBC COUNT	150 WBC/mm3	
POLYMORPHS	35 %	
LYMPHOCYTES	65 %	
RBC COUNT	80 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA		
GRAM'S STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

TUBERCULOSIS PROFILE

Test Name	Observed Value	Reference Value
idenosine-Deaminase activity	47.1 U/L	SERUM/PLASMA/BODY FLUID U Normal : <43 U/L Suspected for MTB : 43-62 U/L Strong suspect for MTB : >62 U/L CSF U/L Normal : <11 U/L Suspect for MTB : 11-12.35 U/L Strong suspect for MTB : >12.35 U/

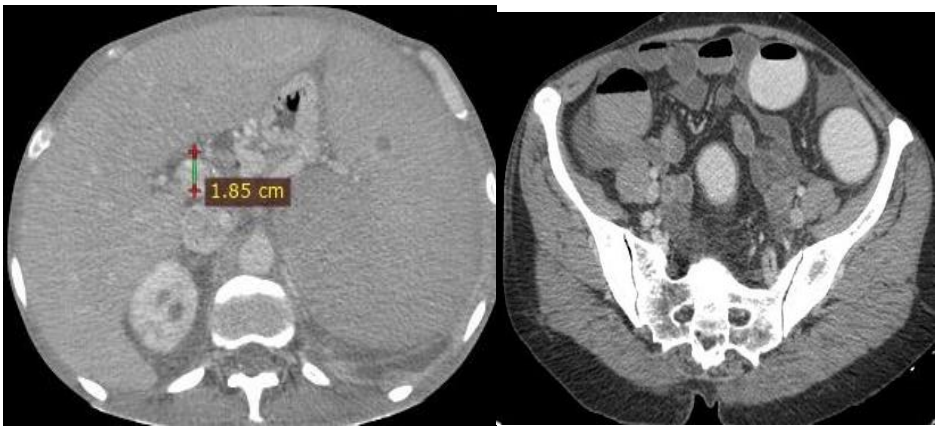
Material: Ascitic Fluid

above results are subject to variations due to technical limitations, hence correlate with clinical findings & other investigations.
nk You For Your Reference.

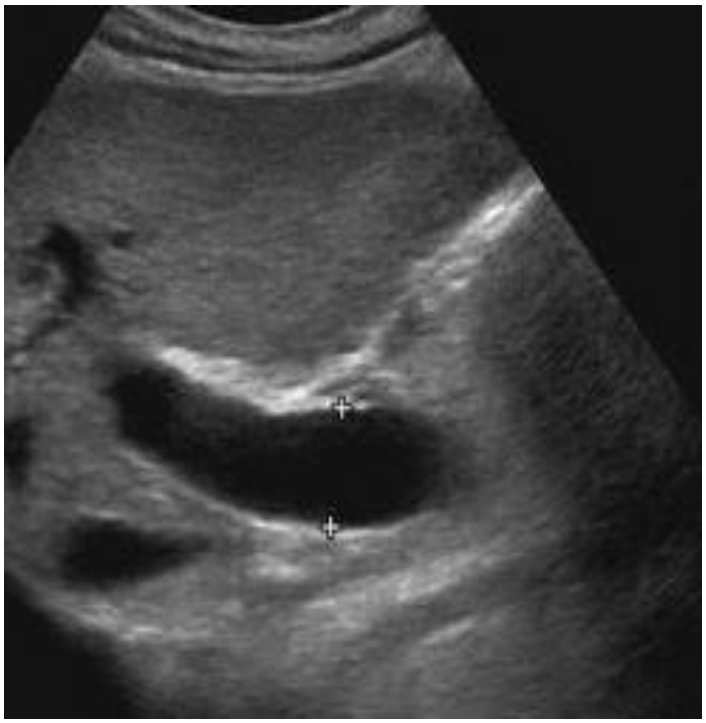
Dr. Tejal P. Kotecha (M.D)

CT findings: symmetrical bowel wall thickening involving caecum, IC junction and terminal ileum with preserved mural stratification, moderate ascites.
USG findings: Symmetrical bowel wall thickening with preserved gut signature, moderate septated ascites.
Cytopathology : Increased level adenosine de aminase activity.
Diagnosis : Possibility of tuberculous etiology

Case 5 62 year male presented with abdominal pain, abdominal distention since 2 months
CT images:



USG images:



Cytopathology:

FLUID EXAMINATION

Test Name	Observed Value	Reference Value
FLUID R-M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	07 ml	
COLOUR	Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Present	
SUPERNATANT FLUID	Clear	
CHEMICAL EXAMINATION		
PROTEINS	2.1 G/dl	Up to 3.0 G/dl
SUGAR	118.0 mg/dl	60 TO 100 mg/dl
CYTOLOGICAL EXAMINATION		
WBC COUNT	150 WBC/mm3	
POLYMORPHS	35 %	
LYMPHOCYTES	65 %	
RBC COUNT	80 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA'		
GRAM'S STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

TUBERCULOSIS PROFILE

Test Name	Observed Value	Reference Value
enosine-Deaminase activity	15 U/L	SERUM/PLASMA/BODY FLUID U Normal : <43 U/L Suspected for MTB : 43-62 U/L Strong suspect for MTB : >62 U/L CSF U/L Normal : <11 U/L Suspect for MTB :11-12.35 U/L Strong suspect for MTB : >12.35 U/
Material	Ascitic Fluid	

lose results are subject to variations due to technical limitations, hence correlate with clinical findings & other investigations.
k You For Your Reference.

Test Name	Observed Value	Reference Value
S. Protein		
Total Protein	6.08 G/dl	6.00 To 7.8 G/dl
S. Albumin	4.06 G/dl	3.00 To 4.50 G/dl
S. Globulin	2.02 G/dl	2.30 To 3.50 G/dl
A:G Ratio	2.01 %	0.9 TO 2.90 %

The above results are subject to variations due to technical limitations, hence correlate with clinical findings & other investigations.
Thank You For Your Reference.

USG findings : Dilatation of portal vein, varieces formation, splenomegaly, moderate acsites and altered liver echotexture.

CT findings : Dilatation of portal vein, varieces formation splenomegaly, moderate acsites and heterogeneous enhancement of liver.

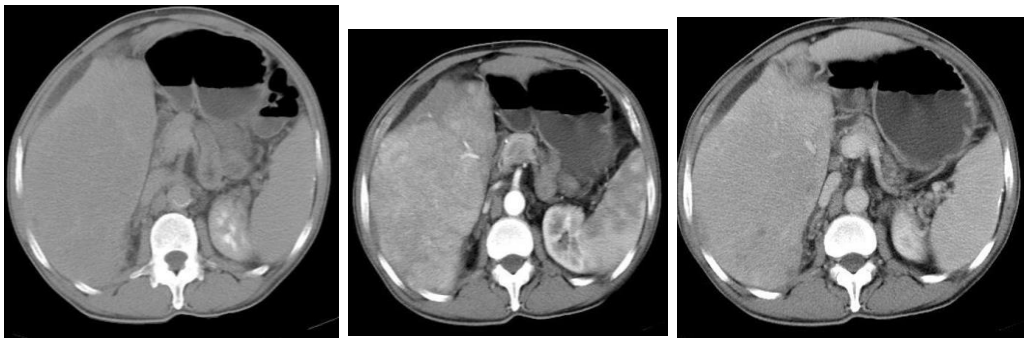
Cytopathology : Clear fluid, serum ascites albumin ratio (SAAG) = (albumin concentration of serum) – (albumin concentration of ascitic fluid) = 4-2.1 =1.9

Diagnosis : Portal hypertension with cirrhosis

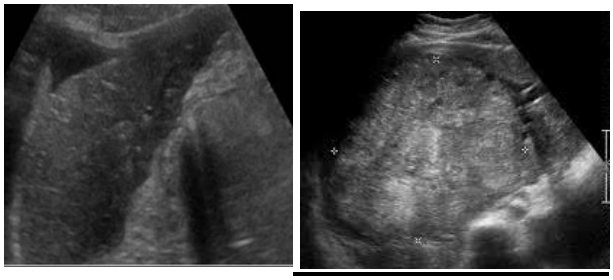
Case 6 58 year male presented with abdominal distension and abdominal pain with weight loss since 4months.

CT images:

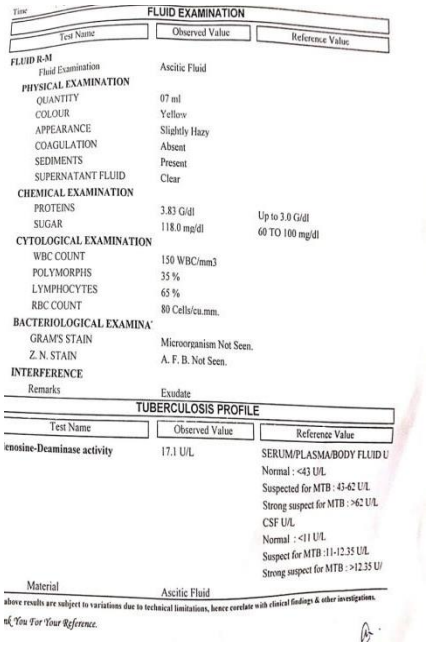
Plain **Arterial** **delayed**



USG images :



Cytopathology:



FLUID EXAMINATION

Test Name	Observed Value	Reference Value
FLUID R-M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	07 ml	
COLOUR	Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Present	
SUPERNATANT FLUID	Clear	
CHEMICAL EXAMINATION		
PROTEINS	3.83 G/dl	Up to 3.0 G/dl
SUGAR	118.0 mg/dl	60 TO 100 mg/dl
CYTOLOGICAL EXAMINATION		
WBC COUNT	150 WBC/mm3	
POLYMORPHS	35 %	
LYMPHOCYTES	65 %	
RBC COUNT	80 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA		
GRAM'S STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

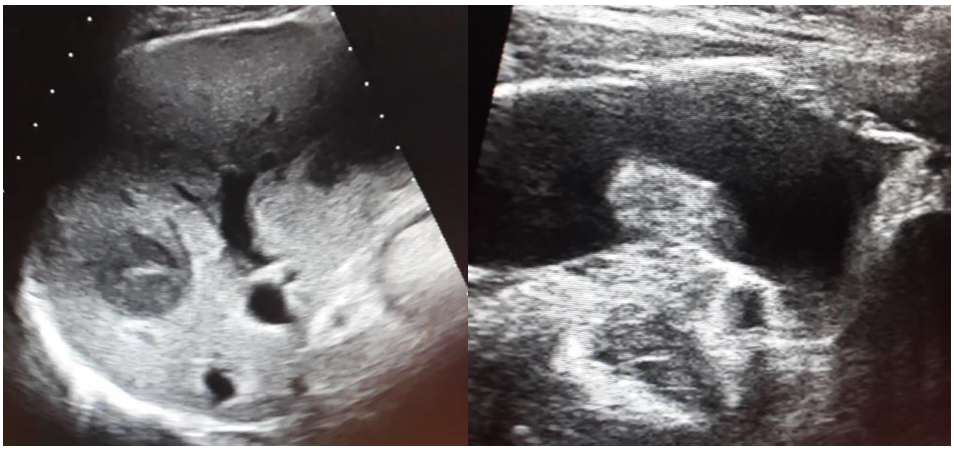
TUBERCULOSIS PROFILE

Test Name	Observed Value	Reference Value
ienosine-Deaminase activity	17.1 U/L	SERUM/PLASMA/BODY FLUID U Normal : <43 U/L Suspected for MTB : 43-62 U/L Strong suspect for MTB : >62 U/L CSF U/L Normal : <11 U/L Suspect for MTB :11-12.35 U/L Strong suspect for MTB : >12.35 U/
Material	Ascitic Fluid	

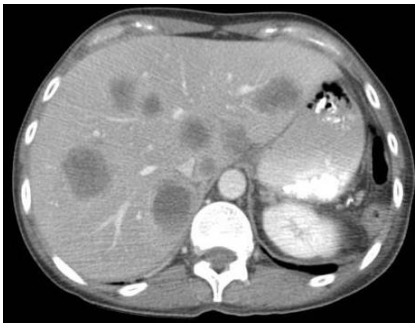
above results are subject to variations due to technical limitations, hence corelate with clinical findings & other investigations.
nk You For Your Reference.

CT findings: Ill defined large heterogeneously enhancing lesion involving right lobe of liver which shows early enhancement in arterial phase and wash out in delayed phase, moderate ascites.
USG findings : Ill defined mix echogenic lesion involving right lobe of liver, moderate ascites.
Diagnosis: Hepatocellular carcinoma

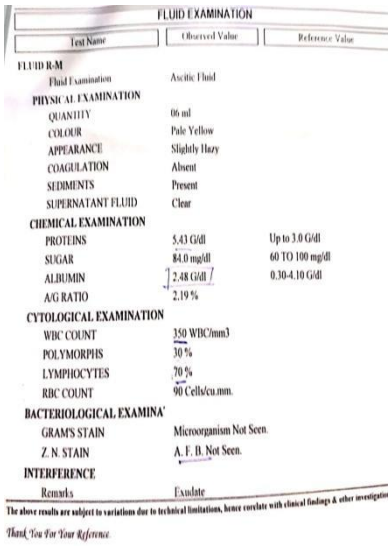
Case 7. 68 year male known case of primary lung carcinoma presented with abdominal pain and distention.
USG images:



CT images:



Cytopathology:



FLUID EXAMINATION

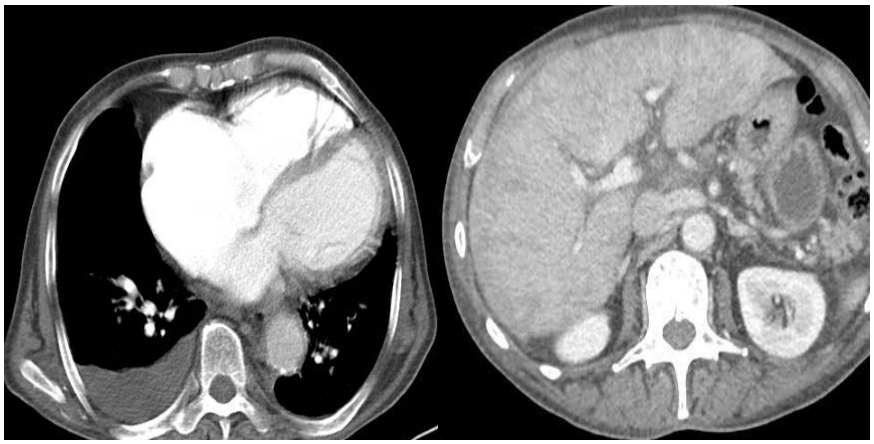
Test Name	Observed Value	Reference Value
FLUID R-M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	06 ml	
COLOUR	Pale Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Present	
SUPERNATANT FLUID	Clear	
CHEMICAL EXAMINATION		
PROTEINS	5.43 G/dl	Up to 3.0 G/dl
SUGAR	84.0 mg/dl	60 TO 100 mg/dl
ALBUMIN	2.48 G/dl	0.30-4.10 G/dl
A/G RATIO	2.19 %	
CYTOLOGICAL EXAMINATION		
WBC COUNT	350 WBC/mm3	
POLYMORPHS	30 %	
LYMPHOCYTES	70 %	
RBC COUNT	90 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA'		
GRAM'S STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

The above results are subject to variations due to technical limitations, hence correlate with clinical findings & other investigation

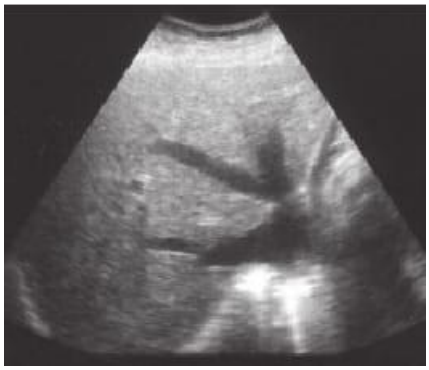
Thank You For Your Reference

USG findings : Multiple hypoechoic lesions involving liver, moderate ascites
CT findings : Multiple heterogeneously enhancing lesions involving liver, moderate ascites
Cytopathology : Hazy fluid, no malignant cells
Diagnosis : Primary lung carcinoma with liver metastases.

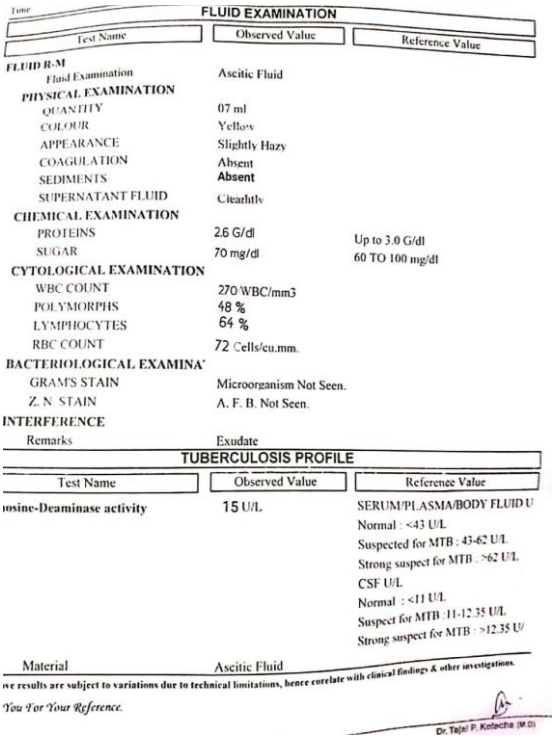
Case 8 : 54 year male presented with chest pain and breathlessness since 15 days
CT images:



USG images:



Cytopathology :



FLUID EXAMINATION

Test Name	Observed Value	Reference Value
FLUID R-M		
Fluid Examination	Ascitic Fluid	
PHYSICAL EXAMINATION		
QUANTITY	07 ml	
COLOUR	Yellow	
APPEARANCE	Slightly Hazy	
COAGULATION	Absent	
SEDIMENTS	Absent	
SUPERNATANT FLUID	Clearhtly	
CHEMICAL EXAMINATION		
PROTEINS	2.6 G/dl	Up to 3.0 G/dl
SUGAR	70 mg/dl	60 TO 100 mg/dl
CYTOLOGICAL EXAMINATION		
WBC COUNT	270 WBC/mm3	
POLYMORPHS	48 %	
LYMPHOCYTES	64 %	
RBC COUNT	72 Cells/cu.mm.	
BACTERIOLOGICAL EXAMINA'		
GRAM'S STAIN	Microorganism Not Seen.	
Z. N. STAIN	A. F. B. Not Seen.	
INTERFERENCE		
Remarks	Exudate	

TUBERCULOSIS PROFILE

Test Name	Observed Value	Reference Value
iosine-Deaminase activity	15 U/L	SERUM/PLASMA/BODY FLUID U Normal : <43 U/L Suspected for MTB : 43-62 U/L Strong suspect for MTB : >62 U/L CSF U/L Normal : <11 U/L Suspect for MTB : 11-12.35 U/L Strong suspect for MTB : >12.35 U/
Material	Ascitic Fluid	

ive results are subject to variations due to technical limitations, hence correlate with clinical findings & other investigations.
You For Your Reference.
Dr. Tejal P. Kotecha (M.D)

<u>USG findings</u> : Right sided pleural effusion moderate acsites and hepatomegaly with altered echotexture.
<u>CT findings</u> : Cardiomegaly, right sided pleural effusion, moderate acsites and hepatomegaly.
<u>CYtopathology</u>: Clear fluid, Low protein content.
Diagnosis: Congestive cardiac failure.

DISCUSSION

1. In this study of 50 cases of ascites, the etiology and its incidence is observed as,

- Cirrhosis 58 %
- Heart Failure 10 %
- Chronic kidney disease 2 %
- Nephrotic syndrome 2 %
- Peritoneal calcinomatosis 4 %
- Liver malignancy : 4 %
- Chronic pancreatitis 1 %
- Portal vein thrombosis 2 %
- Budd-Chiari syndrome 1 %

Cirrhosis with portal hypertension was the most common cause for Ascites and Heart failure was the second most common cause of ascites.

This coincides well with the following two studies, a) The study of Runyon BA, Montano AA, Akriviadis EA et al 35, the etiology and its incidence for ascites is as follows,

- Cirrhosis with portal Hypertension 85 %
- Miscellaneous portal hypertension 8 %

- Cardiac disease 3 %
- Peritoneal Carcinomatosis 2 %
- Miscellaneous normal HT related disorders 2 %

The Study of Vicente Arroyo, Pere Gines, Ramon planas, Juan Roodes et al2 the etiology and its incidence for ascites is as follows, hepatic cirrhosis 88 % neoplasms 6% and to a lesser extent congestive heart failure 3 %, tuberculous peritonitis 2 % and other 1 %.

In this study, of the 28 cases of cirrhosis with portal hypertension, 21 cases were male and 8 cases were female. Of the 21male cases 18 cases were alcoholics – alcoholism is the commonest cause for cirrhosis with portal HT in male. This coincides well with the study of the Tuyns A PequignotG: Greatest risk of ascitic cirrhosis in males in relation to alcohol consumption Int J Epidemiol 13:53, 1984 40.

Chronic pancreatitis was the cause for ascites in one case of Ascites, and alcoholism was the cause for pancreatitis. In the study of Norton J Green berger, alcoholism was the commonest cause for pancreatitis 11.

In this study along with ascites, pleural effusion was present in 4 cases (8 %) of ascites.

Of the 8 cases, 6 cases presented with Right sided effusion (75 %), 1 case presented with Left sided effusion (12.5 %), 1 case presented with Bilateral effusion (12.5 %). In the study of Leuallen EC, Carr DT, 4.8 % cases of cirrhosis with portal HT were having Pleural effusion and majority of cases 90 % were having Right sided Pleural effusion, 7 % were having Bilateral effusion 3 % having Left sided pleural effusion.

In cirrhosis with Portal hypertension, serum protein ranges between 2 – 6 grams. 75.6 % have 4 – 6 grams, 19.5 % have 2 – 4 grams, 5 % have > 6 grams, and 5 % cases have normal protein value. In the study of Runyon total ascitic protein concentration ranges between 0.5 grams and more than 6 grams and is greater than 3 grams in up to 30 % of patients with other uncomplicated ascites 37. In the study of Runyon, the proportions of albumin and globulin in the total protein concentration are approximately 45 and 55 % respectively and the value ranges between 0.225 grams to 2.7 grams 37.

In this study in cirrhosis with Portal hypertension 8 cases (93.7 %) were having SAAG value more than 1.1, cases (6 %) was having SAAG value less than 1.1 . This coincides with the study of Runyon BA, Montano AA, Akriviadis EA et al where SAAG value was more than 1.1 in 97 % cases of Cirrhotic ascites and less than 1.1 in Non cirrhotic ascites 35.

In this study, 8 cases of ascites were caused by heart failure. Of which 6 cases (62.5 %) were caused by CAHD 2 were caused by RHD.

In the study of Eugene Braunwald, CAHD followed by RHD are the common causes of heart failure 9.

In heart failure, 6 cases (75 %) had Ejection fraction < 60 % and 2 cases (25 %) had an ejection fraction > 60 % and both of these cases were caused by RHD.

In the study of Rick a Nishimura, Raymond J Gibbons, James F, Glockner, A Jamil Tajik, Ejection fraction usually less than 60 % in Left ventricular failure 38.

CONCLUSION

- This study shows Cirrhosis with portal hypertension was the most common cause for ascites (78 %) and the next common cause for ascites was heart failure (8 %) followed by renal diseases (5 %) – chronic kidney disease, nephrotic syndrome and other causes including peritoneal carcinomatosis, portal vein thrombosis, budd-chiari syndrome all together were 9 % only.
- In this study 67 % of ascites were found to be high gradient ascites and 11 % were low gradient ascites.

- In this study portal hypertension was present in all the case of ascites due to cirrhosis where as hypoproteinaemia was present only in 95 % which shows portal hypertension is the major cause for ascites in Cirrhosis.
- In cirrhosis with portal hypertension alcoholic liver disease was the commonest cause (64 %).
- Among alcoholics in one case the ascites was due to pancreatitis and not due to cirrhosis of liver.
- Among the renal causes for ascites which was 5 %, the incidence of chronic kidney disease (4 %).
- In this study malignant ascites was only 4 %.

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