"EFFICACY OF SERUM ASCITES ALBUMIN GRADIENT IN ASCITES OF PORTAL AND NONPORTAL HYPERTENSIVE ASCITIC CAUSES"

By

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Dissertation submitted to BLDE University, Bijapur

In partial fulfillment of the requirements for the degree of

MD

in

General Medicine

Under the guidance of

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ABSTRACT

Background and objectives

Serum ascetic-albumin gradient is a promising diagnostic marker for early detection of portal and non portal hypertension in ascites. The present study was aimed to find serum - ascites albumin gradient in case of portal and non portal hypertension ascites patients and to compare the efficacy of serum - ascites albumin gradient (SAAG) concept in classification of ascites due to portal hypertension and non portal hypertension.

Methodology

This one and half year study was conducted from 1st October 2012- 1st april 2014 in the Department of Medicine, Shri B.M. Patil Medical College, Bijapur on a total of 60 patients with Ascites.

Results

In this study, 52 patients were males and 8 were females. The commonest age group was 31 to 40. Most of the patients were males. History of cirrhosis was found in 38 patients, cardiac failure in 5 patients, tuberculosis of abdomen in 9 patients, malignancy in 5 patients, anaemia with hypoproteinemia in 2 patients and nephrogenic cause in 1 patient. Among 60 patients 39 patients had portal hypertension and 21 patients had non portal hypertension.

Conclusion and interpretation

Serum - ascites albumin gradient (SAAG) has efficacy of 94%, and ascitic fluid total protein (AFTP) 80% in classifying ascites of portal hypertension and non portal hypertension causes. The mean of serum - ascites albumin gradient (SAAG) in

portal hypertension is 1.7 and in non portal hypertension 0.74, and is statistically significant in classifying ascites of portal and non portal hypertension causes.

Serum - ascites albumin gradient was especially useful in seperation of cardiac ascites which usually has high protien concentration.

So serum - ascites albumin gradient (SAAG) has superior discriminating power and should replace ascitic fluid total protein (AFTP) in the separation of ascites of portal and non portal hypertention causes.

Key words

Serum ascites albumin gradient, ascitic fluid total protein (AFTP), portal and non portal hypertension.

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INTRODUCTION

Ascites is defined as accumulation of fluid within the peritoneal cavity. The etiological spectrum of ascites is vast and practically includes pathology of all the systems. Careful history taking and clinical examination can provide clue to the etiology of ascites. But paracentesis remains the main stay of the investigation of a new onset ascites. In most cases ascites will appear as a part of a well recognized illness i.e. cirrhosis, congestive heart failure, nephrosis or dissiminated carcinomatosis. Few patients have more then one cause for ascites formation.[1,3]

More than 80% of cases of ascites are due to portal hypertension, mainly as a result of cirrhosis, other subset of cause includes pathology of peritoneum, which are not related to portal hypertension. This classification is important because the mode of evaluation and management is different for these two groups.[1,3]

In past portal hypertension ascites was distinguished from the non portal hypertension causes by determining whether the fluid is transudate or exudates. This concept assumed that in portal hypertension, protein poor ascitic fluid transudates from the normal peritoneal surface, Whereas in ascites associated with peritoneal diseases protein rich ascitic fluid exudates from the peritoneal surface. Ascitic fliud is termed transudate if AFTP (ascitic fluid total protein) is <2.5g/dl.[1,3]

Currently many problems and exceptions have been noted with this concept. Many infected and malignancy related samples have been reported to have transudate fluid and many samples obtained from patients with cirrhosis or heart failure had exudative ascitic fluid.[1] Hence there is a need for this study to know the efficacy of

serum ascites albumin gradient to differentiate ascites of portal and non portal hypertensive etiology.

Recently an alternative method to distinguish ascites associated with portal hypertension from non portal hypertension is proposed. This method is serum - ascites albumin gradient (SAAG), that is substracting serum fluid albumin from ascites albumin. This method is physiologically based and is a parameter of oncotic pressure gradient reflecting presence or absence of portal hypertension.[2,3] Serum ascites alubmin gradient is increased in ascites of portal hypertensive etiology and decreased in non portal hypertensive ascitic causes.

Ascites associated with portal hypertension has high gradient serum - ascites albumin gradient i.e > 1.1 gm / dl, whereas ascites associated with peritoneal inflammation or malignancy has low gradient <1.1gm/dl. In mixed ascites with more than one etiology, serum - ascites albumin gradient is high reflecting the prescence of portal hypertension .[4,5]

AIMS AND OBJECTIVES:

1. To study serum - ascites albumin gradient in case of portal and non portal hypertension ascites patients.

2. To compare the efficacy of serum - ascites albumin gradient (SAAG) concept in classification of ascites due to portal hypertension and non portal hypertension.

REVIEW OF LITERATURE

HISTORICAL ASPECT OF ASCITES

In Ayurveda literature, a mention of ascites is found as 'Jalodhar' ("water in abdomen").[6]

1500 B.C : **Egyptians** were aware of abnormal collection of abdominal fluid associated with disease of liver.

20 BC : Celsus. Advocated removal of fluid to give relief to patients with ascites.

700 A.D : Paul of aegina treated ascites by drainage through a copper tube.

1398 AD: The term '**Ascites'** was coined by **Trevesia.**

1780 AD: James Douglus described peritoneum.

1804 AD: Peritonitis was described by **Laennec**

1896 AD: James Rutherford Morrison introduced 'omentopexy' to relieve ascites due to cirrhosis of liver.

1898 AD : Sape Talma Independently described this procedure.

Henceforth known as 'Morrison-Talma operation'.

Pegot described 'Caput Medusae' pattern of veins over abdomen with a murmur heard in vicinity of umbilicus in an alcoholic soldier.

1934 AD: Joe Vincent Meigs described Meig's syndrome

1950 AD: Keith and Pleffer noted raised amylase levels in peritoneal fluid in case of Pancreatitis

1954 AD: Giegs B and **Kunkel** measured osmotic pressures of serum and. astitic fluid in patients with hepatic cirrhosis

1970 AD: Ultrasonography was used to detect as little as 100ml of ascitic fluid.

1971 AD: Witte C.L. et al explained the importance of colloid pressure in hepatic cirrhosis with ascites.

1978 AD: Hoef's JC. Proposed the idea of estimation of portal pressure by ascitic fluid analysis.

1982 AD: Pierre Pare, Jean Talbot and **John C. Hoefs** proved physiological basis of serum ascites albumin gradient.

ANATOMY AND PHYSIOLOGY

GROSS ANATOMY

The peritoneum is an areolar membrane covered by single layer of mesothelial cells. Its surface area approaches that of body surface area, approximately 1.7m^2, but the functional exchange of surface is less than 1m^2. The peritoneum is sealed in males, but communicates with the exterior via the ostia of the fallopian tubes in females. This cavity is divided into the lesser sac and the greater sac. The lesser sac is defined by the stomach and gastro colic ligament anteriorly, spleen and spleno-renal and gastro splenic ligaments *on* the left aspect, and pancreas and duodenum posteriorly. The greater sac is the larger intraperitoneal space excluding that of the lesser sac.

The peritoneum is divided into parietal and visceral components. The parietal peritoneum covers the anterior, lateral and posterior abdominal walls, the inferior surface of the diaphragm, and the pelvis.

The spread of infection within the peritoneal cavity is governed by the site of infection, the location of fibrinous and fibrous adhesions, intraperitoneal pressure gradients, and the position of the patient. Dependent recesses (e.g. paracolic gutters, pelvis, lesser sac, and subhepatic and subphrenic spaces) tend to become the sites of abscess formation after leakage of visceral contents.

A mesentry is defined as a membranous bilayer of peritoneum attaching an organ to a body wall. The term is frequently used to describe the attachment of the small bowel; however as mentioned above, other parts of the gut have a mesentry as well. An omentum is a fold of peritoneum connecting the stomach with adjacent

organs in the peritoneal cavity. The greater omentum, frequently known simply as the omentum, extends from the greater curvature of the stomach over the intestines and attaches to the transverse colon. The lesser omentum joins the lesser curvature of the stomach to the liver, this is also known as the gastrohepatic omentum. The diaphragm is the musculoaponeurotic partition separating it from the thorax. It is lined by parietal peritoneum on its inferior surface and parietal pleura on its superior surface.[7]

BLOOD SUPPLY AND INNERVATION

The visceral peritoneum is supplied by the splachnic blood vessels and the parietal peritoneum is supplied by inter costal, subcostal, lumbar, and iliac vessels. Similarly visceral peritoneum is supplied by visceral nonsomatic sympathetic nerves (without pain receptors) and the parietal peritoneum is supplied by somatic nerves. The differential innervation leads to the sharp character and precise localization of pain associated with parietal peritoneal irritation and the dull, diffuse nature of pain due to visceral peritoneal irritation.

PHYSIOLOGY

Fluid, solutes and particulate matter appear to be absorbed by different routes from the peritoneal cavities. The peritoneal mesothelial venous pores are approximately 30° A in diameter and accept molecules smaller than 2000 daltons. Materials absorbed by this route enter portal vein and then the systemic circulation. In contrast, the peritoneal mesothelial lymphatic pores are greater than 30° A in size and accommodate particles larger than 2000 daltons. These particles enter the thorasic lymphatics and then pass on to the systemic circulation.

The Particles of the size of red blood cells and bacteria preferentially escape the peritoneal cavity by way of the diaphragmatic lymphatics. The existence of

"stomata" on the inferior surface of the diaphragm was originally described by Recklinghausen in 1863 and confirmed by Leak and Rahil in 1978. These open channels, essentially funnels between the peritoneal cavities and the lumen of the diaphragmatic vessel, permit rapid bulk absorption of fluid, solutes and particles. The ball-valve like anatomy of these structures and the pressure gradients generated during respiratory motion further facilitate absorption via these channels.

ASCITES

Definition : Ascites refers to accumulation of free fluid in peritoneal cavity.

EPIDEMIOLOGY

Many large scale studies have proven that most patients with ascites have hepatic cirrhosis as the underlying cause.[8]

Cause:	**% of Patients with Ascites**
a. Parenchymal liver disease	80
b. Mixed	5
c. Malignency	10
d. Heart failure	5
e. *Tuberclulosis	2
f. Pancreatic	1
g. Nephrogenous	<1
h. Nephrotic	<1
i. Surgical peritonitis in absence of liver disease	<1

Classification of ascites by serum-ascites albumin concentration gradient

Portal hypertension	**Non- portal hypertension**
High Gradient (> 1.1 gm/dl)	Low gradient (1.1 gm/dl)
Cirrhosis	Peritoneal carcinomatosis
Alchoholic hepatitis	Tuberculous peritonitis
Cardiac ascites	Pancreatic ascites
Mixed ascites	Bowel obstruction/infarction
Massive liver metastasis	Biliary ascites
Fulminant hepatic failure	Nephrotic syndrome
Budd-chiari syndrome	Postoperative lymphatic leak
Portal vein thrombosis	Serosites in connective tissue diseases
Veno occlusive disease	
Myxoedema	
Fatty liver of pregnancy	

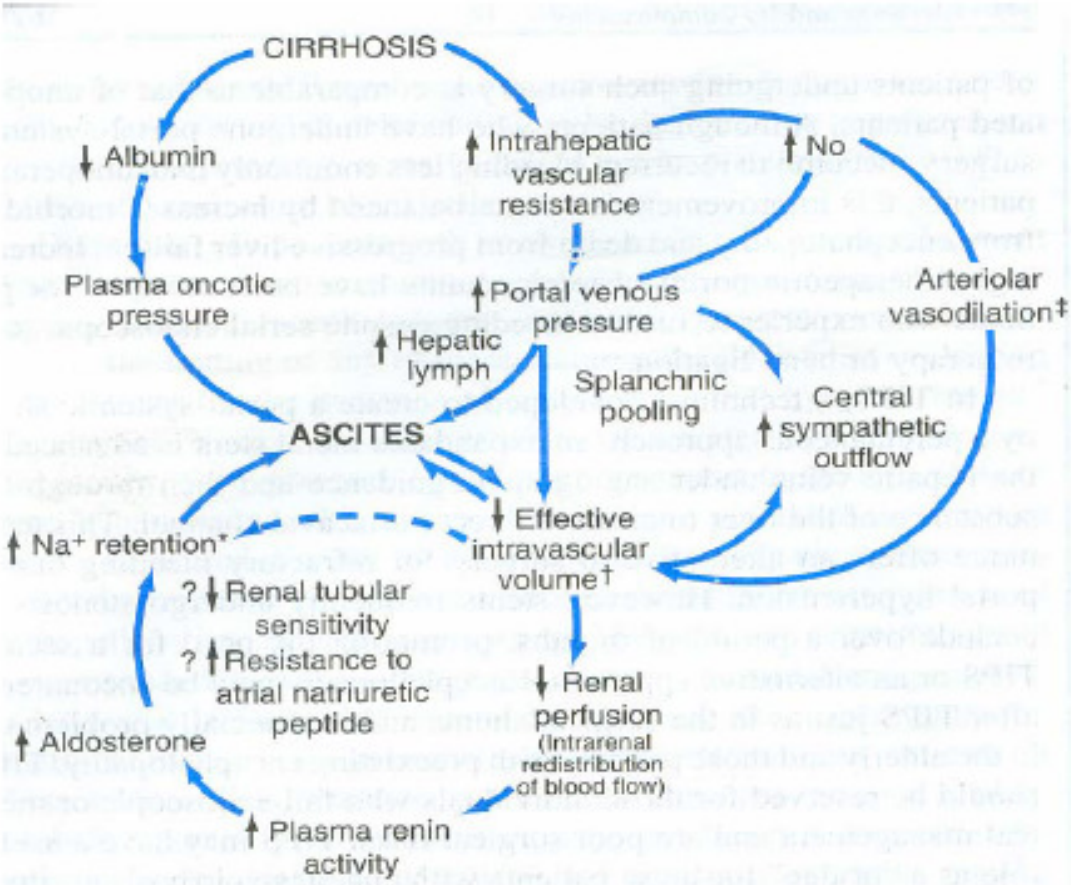
Pathogenesis

The accumulation of ascitic fluid in portal hypertension represents a state of total-body sodium and water excess, but the event that initiates this imbalance is unclear. Three theories have been proposed.

The "under filling" theory suggests that the primary abnormality is inappropriate sequestration of fluid, within the splanchnic vascular bed due to portal hypertension and a consequent decrease in effective circulating blood volume.

According to this theory, an apparent decrease in 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.



A third and more recent theory, the peripheral arterial vasodilation hypothesis may unify the earlier theories and accounts for the constellation of arterial hypotension and increased cardiac output in association with high levels of vasoconstrictor substances that are routinely found in patients with cirrhosis and ascites. Again, sodium retention is considered secondary to arterial vascular under filling and the result of a disproportionate increase of the vascular compartment due to arteriolar vasodilatation rather than from decreased intravascular volume. According

to this theory, portal hypertension results in splanchnic arteriolar vasodila-tion, mediated by nitric oxide, and leading to underfilling of the arterial vascular space and baroreceptor-mediated stimulation of renin-angiotensin, sympathetic output, and antidiuretic hormone release.[9,10]

Regardless of the initiating event, a number of factors contribute to accumulation of fluid in the abdominal cavity. Elevated levels of serum epinephrine and norepinephrine have been well documented. Increased central sympathetic outflow is found in patients with cirrhosis and ascites but not in those with cirrhosis alone. Increased sympathetic output results in diminished natriuresis by activation of the renin-angiotensin system and diminished sensitivity to atrial natriuretic peptide.[11,12] *Portal hypertension* plays an important role in the formation of ascites by raising hydrostatic pressure within the splanchnic capillary bed. *Hypoalbuminemia and reduced plasma oncotic pressure* also favor the extravasation of fluid from plasma to the peritoneal cavity, and thus ascites is infrequent in patients with cirrhosis unless both portal hypertension and hypoalbuminemia present. Hepatic lymph may weep freely from the surface of cirrhotic liver due to distortion and obstruction of hepatic sinusoids and lymphatics and contributes to ascites formation. In contrast to the contribution of transudative fluid from the portal vascular bed, hepatic lymph may weep into the peritoneal cavity even in absence of marked hypoproteinemia because the endothelial lining of the hepatic sinusoids is discontinuous. This mechanism may account for the high protein concentration present in the ascitic fluid of some patients with venoocclusive disease or the Budd Chiari syndrome.

Renal factors also play an important role in perpetuating Ascites. Patients with ascites fail to excrete a water load in a normal fashion. They have increased renal

sodium reabsorption by proximal and distal tubules, the latter due largely to increased plasma renin activity and secondary hyperaldosteronism. insensitivity to circulating atrial natriuretic peptide, often present in elevated concentrations in patients with cirrhosis and ascites, may be an important contributory factor in many patients. This insensitivity has been documented in those patients with the most severe impaired sodium excretion, who typically also exhibit low arterial pressure and marked overactivity of the renin-aldosterone axis.

Renal vasoconstriction, perhaps resulting from increased serum prostaglandin or catecholamine levels, may also contribute to sodium retention.[13,14] Recently a role of endothelin, a potent vasoconstrictor peptide, has been proposed. While elevated levels have been reported by some, this has not been observed by others.

Pathogenesis of non cirrhotic ascites:

The mechanism of fluid retention in patients with malignancy related ascites depends on the location of the tumour. Peritoneal carcinomatosis appears to cause ascites by "exudation" of proteinaceous fluid from tumour cells lining the peritoneum. Extracellular fluid enters the peritoneal cavity to reestablish oncotic balance.

Although the pathogenesis of ascites formation in patients with massive liver metastasis has not received much scientific investigations, fluid presumably accumulates owing to portal hypertension caused by stenosis or occlusion of portal veins by tumour nodules or tumour emboli. In patients with hepatocellular carcinoma ascites forms as a result of the underlying cirrhosis related portal hypertension and/or tumour induced portal vein thrombosis. Chylous ascites due malignant lymphoma appears to be caused by lymph node obstruction by tumour and rupture of chyle containing lymphatics.

Ascites forms in high output as well as low output heart failure. As in cirrhosis, there appears to be decreased "effective arterial blood volume" and subsequent activation of the vasopressin, renin-aldosterone and sympathetic nervous systems. This leads to renal vasoconstriction and sodium and water retention . Fluid then weeps from the congested hepatic sinusoids as lymph, as in cirrhotic ascites. Myxedema ascites is cardiac ascites.

Tuberculosis, chlamydia infection and coccidiodomycosis probably cause Ascites as a result of "exudation" of proteinaceous fluid as in peritoneal carcinomatosis. Spontaneous bacterial peritonitis does not appear to cause fluid to accumulate, infection develops in preexisting ascites.

In pancreatitis or biliary ascites fluid forms by leakage of pancreatic juice or bile into the peritoneal cavity and/or by a "chemical burn of the peritoneum. After abdominal surgery especially extensive retroperitoneal dissection, lymphatics may be transected and leak lymph for variable periods of time. The formation of ascites in this condition is similar to that of malignant chylous ascites (i.e lymphatic leak)

It is postulated that in nephrotic syndrome loss of protein in the urine leads to decreased effective arterial blood volume with activation of vasopressin, renin aldosterone and sympathetic nervous system with resulting renal sodium and water retention.

“CLINICAL APPROACH TO A PATIENT WITH ASCITES”

Approach to a patient with ascites includes a careful history taking, physical examination and investigations.

History:

Most cases of ascites are related to liver parenchymal disease- Cirrhosis. In majority of cases cirrhosis of liver is associated with alcoholism. History taking of a patient with ascites should include history of alcohol intake, dependence, past history of alcoholic liver disease.

A patient with cirrhosis presents with features peculiar to the underlying cause and results of two major events- Hepato cellular failure and portal hypertension. So a comprehensive history for a patient of ascites should include:symptomatology regarding these features.

Clinical History:

- Fatigue and weight loss
- Anorexia and flatulent dyspepsia
- Abdominal pain
- Jaundice, colour of urine and faeces
- Abdominal or leg swelling
- Hemorrhage -, nose, gums, skin, alimentary tract
- Loss of libido
- Past history of jaundice, hepatitis
- Drugs: Antitubercular drugs, Antimetabolites- methotrexate, blood transfusion
- Social history of alcohol consumption, dependence.

Patients with ascites who have history of cancer should be suspected of having malignancy related ascites. Breast, lung, colon and pancreatic primary tumors are regularly complicated by ascites.[18] Although these patients can also present with

general clinical features associated with cirrhosis- *abdominal pain* when present may be helpful in differentiating \these cases from cirrhosis. Because usually *abdominal pain* is not present in cirrhotic patients unless there is superimposed bacterial peritonitis or alcoholic hepatitis.

A past history of heart failure or obstructive lung disease can be helpful in raising the possibility of cardiac ascites. Patient usually present with ascites only at a later stage of congestive heart failure, associated with *progressive breathlessness, right hypochondrial discomfort, orthopnoea, paroxysmal nocturnal dyspnoea and swelling of legs* for a considerable duration. Abdominal distension follows leg swelling. Jaundice with features of hepatic failure can be present in these patients if there is considerable congestive liver disease.

Tubercular peritonitis is usually manifested by *fever and abdominal pain.* Patient should be questioned about risk factors associated with intra abdominal tuberculosis.

H/O Risk factors for intra abdominal tuberculosis

- Low Socioeconomic status
- Living in endemic area
- HIV infection
- Family history positive for tubercular infection
- Advanced age
- Intravenous drug abuse/alcohol
- Peritoneal dialysis

Clinical features associated with peritoneal tuberculosis

- Ascites:
- Abdominal pain
- Weight loss
- Fever
- Diarrhoea
- Abdominal tenderness

A small percentage of patients with nephrotic syndrome develop anasarca including ascites. Age group, facial puffiness preceding development of ascites and oliguria usually make the clinical diagnosis quite obvious. Known diabetic developing ascites should also be suspected of Nephrogenic ascites. Some patients with chronic renal failure undergoing chronic hemodialysis also develop - dialysis ascites associated with muscle wasting, anorexia and worsening neuropathy.

Rarely a patient of hypothyroidism can have ascites as a principal manifestation. Associated features of lethargy, weight gain, cold intolerance, constipation, menorrhagea and hair loss will indicate towards the underlying endocrinal disorder.

A small number of patients present with ascites due to acute hemorrhagic pancreatitis, there is usually a history of sudden onset severe epigastrial or hypochondria pain with nausea and vomiting. Patient is usually severely ill with possible Shock and renal failure. Ascites may develop in first few days or weeks of this type of presentation.

Physical Examination:

On the basis of history and appearence of abdominal distention the diagnosis of ascites is readily suspected usually and can be confirmed by physical examination.

General Examination:

Patient is sallow and dehydrated, poor nutritional status is usually evident. Muscle wasting is profound.

Thin limbs with Protuberent belly

Lower Limb edema or anasarca: Usually associated with ascites due to cangestive heart failure, renal ascites or ascites associated with hypoproteinemia. When patients with liver disease have peripheral edema, i.e it is usually in the lower extremeties.

Skin and nails*:* Skin is sallow, with evidence of chronic malnutrition and vitamin deficiency, specially in chronic alcoholics or other liver diseases.

Nails show pallor due to anemia. In case of hepatic cirrhosis white nails or clubbing may be present.

Examination of Neck*:* (JVP) Neck veins of patients with ascites should always be examined specifically. Most of the patients with cardiac ascites have jugular venous distention. Constrictive Pericarditis is a curable cause of ascites and characteristic jugular venous pulse changes can help in its diagnosis.

Lymph nodes :

1. Supraclavicular- Virchows gland. When palpable indicates Abdominal malignancy.
2. Palpable cervical lymph nodes: If matted indicates probability of tuberculosis, if rubbery in consistency Lymphoma can be suspected.

Signs of Hepatocellular failure:

- CIRCULATORY CHANGES

  Spider telengectesia, palmer erythema, cyanosis

- ENDOCRINE CHANGES

  Loss of libido, hair loss

  men: gynaecomastia, testicular atrophy

  women :breast atrophy,

- HAEMORRHAGIC TENDENCY

  Bruises, purpura, epistaxis

- SKIN CHANGES

  Paper money skin, diffuse pigmentation

- PORTAL HYPERTENSION

  Splenomegaly, collateral vessels, fetor hepaticus

  Cruveillhier – Baumgarten murmur in epigastrium

HEPATIC ENCEPHALOPATHY

Confusion, hypersomnolence, altered sleep rhythm, coma, flapping tremor

OTHERS

Pigmentation, low grade fever, digital clubbing .

These should be specifically looked for in all cases of ascites because liver parenchymal disease with Portal hypertension remains the major cause of ascites.

Systemic Examination: *Abdomen:*

Inspection:

Abdomen is distended with fullness particularly in flanks. The umbilicus is everted and distance between symphysis pubis and umbilicus seems diminished.

Distended abdominal wall veins: May be present

a. Portal systemic collateral channels due to portal hypertension. Veins radiate from umbilicus with blood flow away from umbilicus. Caput medusae formation may be seen.
b. Inferior vena caval colleterals - usually result from a secendary functional block of the inferior vena cava due to pressure of the peritoneal fluid . They commonly run from the groin to costal margins blood flow is towards the umbilicus. The presence of large veins on patient's back are quite characteristic of I.V.C obstruction.

Hernial sites:

The increased intraabdominal pressure favours protrusion of hernias in the umbilical, femoral or inguinal regions or through old abdominal incisional scars. *Scrotal edema* is common, testicular atrophy can also be present.

Palpation:

With tense ascites it is difficult to palpate abdominal viscera but with moderate amount of fluid the liver or spleen may be palpated.

The presence of dullness on percussion in the flanks is the earliest clinical sign. It can be detected when about 1.5 litres of peritoneal fluid is present if no flank dullness is present patient has less than 10% chance of having ascites.[19] Though *'Puddle sign'* can detect about 120 ml of fluid, sensitivity and specificity is much

lower than shifting dullness in detecting ascites. Gaseous distention of bowel, thick panniculus and an ovarian mass can mimic ascites. Gaseous distension is readily apparent on percussion and ovarian mass usually causes tympanic note over flanks with central dullness.

Other systematic examination :

Careful examination of other systems is helpful to find out the cause of ascites. Special consideration should be given to cardiovascular system.

MATERIALS AND METHODS

SOURCE OF DATA: 60 ascitis patients admitted in BLDE University Shri B.M.Patil Medical Hospital are the subjects for study from 1st Oct 2012 to 1st April 2014.

INCLUSION CRITERIA:

1. Patients with ascites proved by ultra sound
2. Patients aged more than 18 years
3. Patients not treated with diuretics

EXCLUSION CRITERIA :

1. Patients with hepatic encephalopathy and acute gastro- intestinal bleeding.
2. Patients with blunt injury abdomen.

METHOD OF COLLECTION OF DATA:

Abdominal Paracentesis

Paracentesis is a simple and safe procedure that is used to confirm the presence of ascites, to investigate the cause and to identify ascitic infection. Small amount of ascites or loculated ascites may not be identified. Paracentesis can also be used to relieve the discomfort of massive ascites.

Procedure

The patient should empty the bladder and then lie comfortably in the supine position. The iliac fossa away from the inferior epigastric vessels or the midline (linea alba) can be used for the puncture. The skin is cleaned and 3 - 6 ml of lidocaine (1 - 2%) is infiltrated into the anterior abdominal wall down to the peritoneum. A fine bore needle (19 - 23 gauge) attached to a large syringe is used to minimize the chance of subsequent leakage of ascites and the needle is inserted into the peritoneal cavity.

Fluid can be aspirated easily, but if none comes the needle should be repositioned or the patient rolled slightly to the side of the puncture. At least 50 ml should be taken for biochemical and bacteriological investigations, and at least 100 ml for cytology. The scars of previous operations should be avoided as puncture sites; they may have caused adhesions that can be vascular, they may be the site of varices or there may be adherence of the loops of bowel. Bleeding or infections are rare complications even when the bowel has been punctured, and leakages is minimized by using a small bore needle.

A midline insertion site may be preferable to a lower quadrant insertion site for a diagnostic tap in a non obese patient, unless there is a midline surgical scar. The midline caudad to the umbilicus is avascular unless there is an unusual collateral vein there. A large collateral is frequently located in the midline *cephalad* to the umbilicus in the setting of portal hypertension; this area should be avoided. Therefore, needles inserted in the midline should be placed *caudad* to , the umbilicus.

Obese patients pose special problems. The abdominal wall may be thicker than the length of even a 3.5 inch paracentesis needle. Also. it is frequently diffcult to be certain of the presence or absence of ascites in the obese patient. Ultrasound guidance

with determination of the thickness of the abdominal wall in the midline versus the lower quadrants is helpful in successfully obtaining ascitic fluid.

Appearance

Ascitic fluid due to liver disease is usually clear and straw or light green in colour. It can be differentiated *from* urine in a patient who does not have diabetes mellitis by its glucose contents. Bloody ascites, in the absence of trauma is usually due to malignancy. Pronounced bile staining indicates a communication with the biliary system, usually due to gall stones, neoplasms or trauma.[23] Infection may cause the fluid to be cloudy

Cell Count:

The mean white cell count (WBC) in uncomplicated cirrhotic ascites is reported to be 281± 25 cells/cumm; the upper limit is said to be 500 cell/cu mm.[24] However, during diuresis in patients with cirrhosis and ascites, the cells concentrate to more than 1000 cells/cu mm.[25] However, before a patient can be diagnosed has having a diuresis-related elevation of ascitic fluid WBC count a prediuresis count must be available and must be normal, there must be a predominance of lymphocytes and there must be no unexplained clinical signs or symptoms of fever or abdominal pain.

The upper limit of absolute polymorphonuclear neutrophil (PMN) leukocyte count in uncomplicated cirrhotic ascitic fluid is usually stated to be 250/cu mm. The short survival of polymorphonuclear neutrophils results in relative stability in the absolute PMN count during the diuresis. Therefore the 250 cells/cu mm "cut off' remains reliable even at the end of diuresis.

Any inflammatory process can result in an elevated ascitic fluid WBC count. Spontaneous bacterial peritonitis is the most common cause of inflammation of ascitic fluid and the most common cause of an elevated WBC count. The total WBC count as well as the absolute PMN count is elevated with spontaneous bacterial peritonitis; PMN is usually compose greater than 70% of the total WBC. Also in tuberculous peritonitis and peritoneal carcinomatosis there is frequently an elevated total WBC count, but with a predominance of lymphocytes.[26]

Most bloody ascites is the result of a slightly traumatic tap. Leakage of blood into the peritoneal cavity leads to an elevated ascitic fluid WBC count. Because neutrophils predominate in blood, the ascitic fluid differential count may be altered by contamination of ascitic fluid with blood.

Chylous ascites :

Chylous ascites is rare and has a milky white appearance due to chylomicrons that pass into the supernatant on centrifugation. It is usually due to malignant disease, often a lymphoma, or trauma including surgery in adults, but there are other causes. Fat droplets are seen on ascites microscopy, the ascites triglyceride concentration exceeds the serum concentration, and no organisms grow on culture. The ascites protein exceeds 30 gm/l and hypoproteinemia and lymphopenia may be present. Chylous ascites occasionally occurs in cirrhosis even clear ascites contains small chylomicrons, probably due to intestinal lymphatic hypertension, but where there is chylous ascites the number and size of the chylomicrons entering the ascites *from* the lymphatics is increased greatly. *Whether* this results from increased permability or from rupture of the lymphatic is unknown.

Indications

Abdominal paracentesis with appropriate ascitic fluid analysis is probably the most rapid and cost-effective mcthod of diagnosing the cause of ascites. Also, because of the 10% to 27% prevalence of ascitic fluid infection at the time of the patient's admission to the hospital, a surveillance tap performed at that time may detect an unexpected infection.[20] Not all patients with ascitic fluid infection are symptomatic ; many have very subtle symptoms (e.g., mild hepatic encephalopathy so that only the family notices the patient's change in mental status). Detection of infection at an early asymptomatic stage may reduce mortality. So sampling of ascitic fluid in all inpatients and outpatients with *new onset* ascites and in all patients admitted to the hospital with ascites (i.e., a tap at the time of each hospitalization). Paracentesis should be *repeated* in patients (whether in the hospital or not) who develop signs or symptoms or have ubnormal laboratory values suggestive of infection (e.g.. hypotension, abdominal pain or tenderness, fever, encephalopathy, renal failure, acidosis, or peripheral leukocytosis).

Contraindications

There are few contraindications to paracentesis. Coagulopathy is a potential contraindication; however. most patients with cirrhotic ascites have coagulopathy. If mild coagulopathy were considered to be a contraindication to paracentesis, few cirrhotic patients would undergo this procedure. coagulopathy should preclude paracentesis only when there is clinically evident fibrinolysis or clinically evident dissmenated intravascular coagulation. These conditions occur in fewer than one in 1000 taps. There is no data-supported cut-off of coagulation parameters beyond which paracentesis should he avoided. Even after multiple paracenteses, patients with severe prolongation of prothrobin time do not usually develop bloody ascites. Cirrhotic

patients who do not have clinically obvious coagulopathy simply do not bleed excessively from needlesticks unless a blood vessel is entered.

Studies regarding complications of paracentesis in patients with ascites documented no deaths or infections caused by the paracentesis.[21,22] There were no episodes of hemoperitoneum or bowel entry by the paracentesis needle. Complications included only approximately 2% abdominal wall hematomas, despite the fact that 71% of the patients had an abnormal prothrombin time; 21 % had a prothrombin time that was prolonged by 5 seconds more. Complication rates may be higher when paracentesis is performed by inexperienced operators.

Some physicians give blood products (fresh-frozen plasma or platelets) routinely before paracentesis in cirrhotic patients with coagulopathy, presumably in order to prevent hemorrhagic complications. This policy is not supported by data. Since a hematoma that requires a transfusion develops in only approximately I % of patients who undergo paracentesis without prophylactic transfusions of plasma or platelets, approximately 100 to 200 U of fresh-frozen plasma or platelets would have to be given to prevent transfusion of approximately 2 U of red blood cells. Also, despite our screening efforts, there remains a finite risk of post-transfusion hepatitis. Since most patients with cirrhosis and ascites die when they develop superimposed acute hepatitis, a small but real risk or death from hepatitis superimposed on cirrhosis is not an acceptable alternative to a trivial reduction in the risk of bleeding.

On the basis of the complications of paracentesis performed with large bore trocars, many physicians have avoided diagnostic paracentesis in the evaluation of the patient with ascites. However, because of the documented safety of this procedure and

the frequency of ascitic fluid infection, paracentesis is now being performed more frequently.

SERUM - ASCITES ALBUMIN GRADIENT

*Be*fore the 1980' s the ascitic fluid total protein concentration was used to classify ascites into exudate (>2.5 gl /dl) and transudate (<2.5 gl/dl) Unfortunately this form of classification does not work well in ascitic fluid, and these terms, as applied to ascitic fluid, were never carefully defined or validated. Attempts at using combinations of lactate dehydrogenase (LDH) and serum/ascitic fluid ratios of LDH or protein have not been shown to classify ascitic fluid into exudates and transudates either.[27,28]

The serum ascites albumin gradient (serum ascites albumin - gradient) has been provided in multiple studies to categorize ascites better than the total protein concentration and better than other parameters.[29,30]

CALCULATION OF SERUM - ASCITES ALBUMIN GRADIENT

Serum - ascites albumin gradient

= (Serum albumin concentration - Ascitic fluid albumin concentration)

SAMPLE SIZE:

With the prevalence rate of ascites 2.5% [3] at 95% confidence interval and at ± 5 margin of error the sample size is 60

$$N = \frac{(1.96)^2 \times P \times Q}{d^2}$$

Hence 60 cases will be included in the study

STATISTICAL ANALYSIS:

1. Diagramatic presentasion
2. Mean ± SD

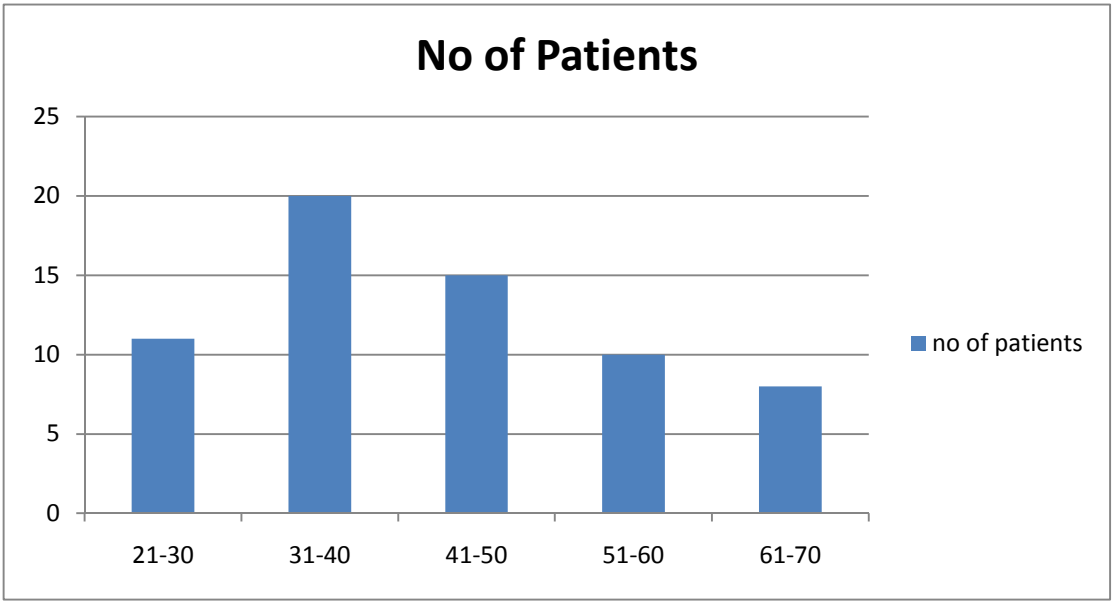
RESULTS

60 consecutive patients with ascites were taken for the study irrespective of the etiology of ascites during time period from 1st Oct 2012 to 1st April 2014.

Table no :1

Age wise distribution of cases AGE (in years)	No.of.Patients
21-30	11
31-40	20
41-50	15
51-60	10
61-70	8

Graph no - 1

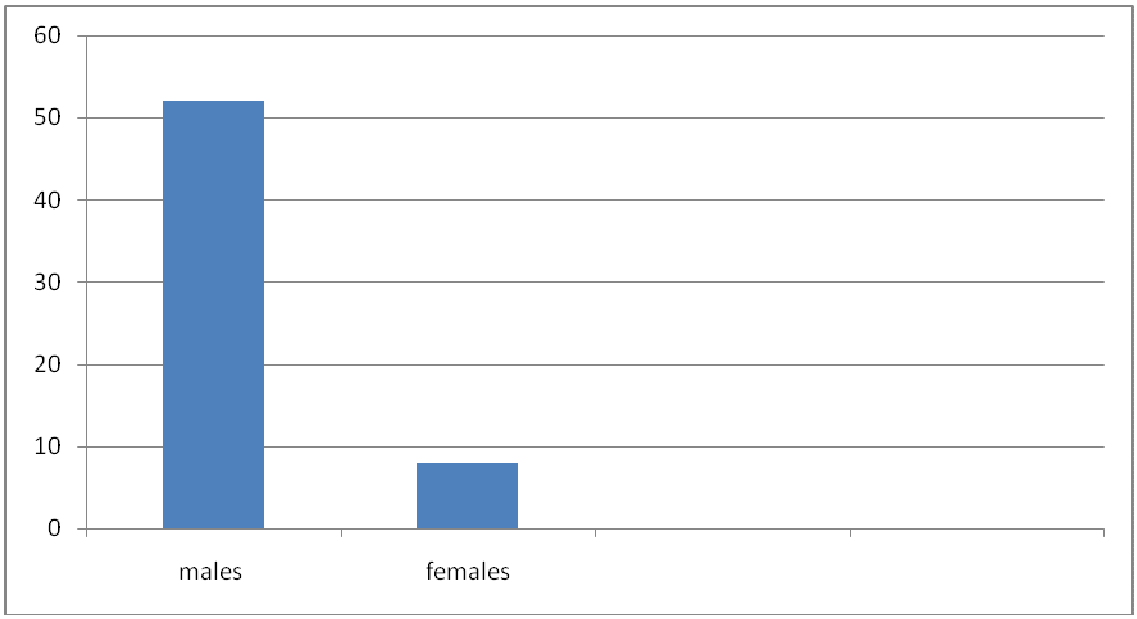


Maximam patients who had ascitis were under the age group of 31-40yrs, 20 patients were admitted, 15 patients were between 41-50yrs,11 patients were between 21-30yrs,10 patients between 51-60yrs and 8 were between 61-70yrs of age group.

Table no 2

Sex distribution of patients	No.of patients
Males	**52**
Females	**8**

Graph no - 2

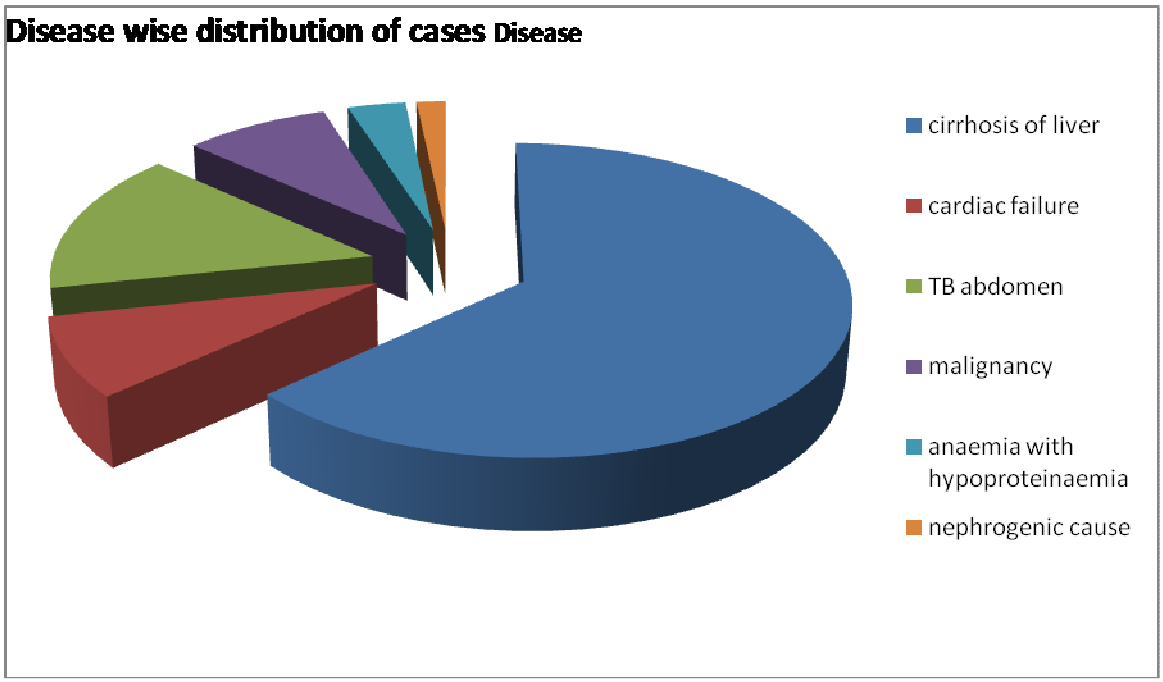


Among all 60 patients maximum patients were males 52and females were 8 patients**.**

Table no 3

Disease wise distribution of cases Disease	No.of. Cases
Cirrhosis	38
Cardiac failure	5
Tuberculosis Abdomen	9
Malignancy	5
Anemia with Hypoproteinemia	2
Nephrogenic cause	1
Total	**60**

Graph no – 3



Out of 60 patients 38 patients had cirrhosis of liver,9 patients had TB abdomen,5 patients had cardiac failure,5 patients had malignancy,2 patients had anaemia with hypoproteinaemia and 1 patient had nephrogenic cause for ascites.

Table : 4

Symptomatology of the patients Disease	Symptoms					
	Distension of abdomen	Fever	Pain abdomen	Loss of appetite	Bowel disturbances	Yellowish discolouration of sclera
Cirrhosis (38)	35	7	8	16	9	17
Cardiac failure (5)	3	-	-	1	-	1
Tuberculosis Abdomen (9)	3	6	4	4	2	-
Malignancy (5)	2	1	-	4	1	-
Anemia with Hypoproteinemia (2)	2	-	-	2	-	-
Nephrogenic cause (1)	1	-	-	1	-	-

Graph -4

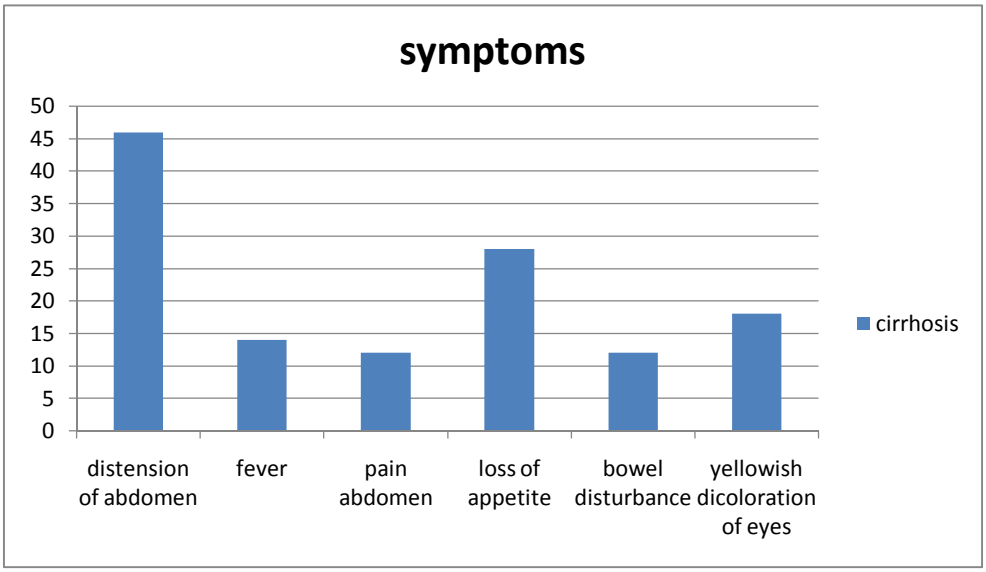


Table no 5

Cirrhosis (38)	No of patients	Percentage
Distension of abdomen	35	92%
Fever	7	18%
Pain abdomen	8	21%
Loss of appetite	16	42%
Bowel disturbance	9	23%
Icterus	17	44%

Graph no 5

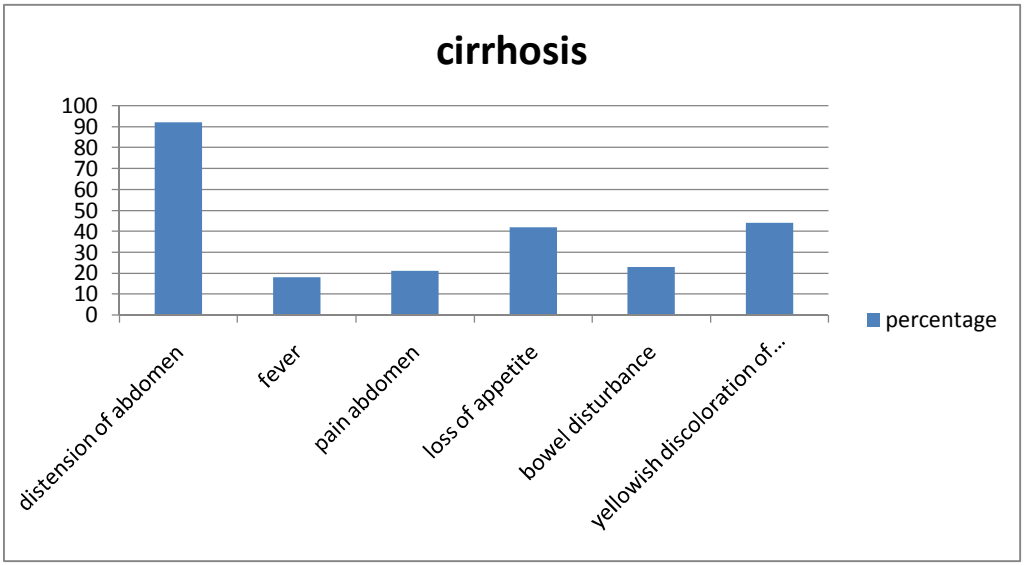


Table no 6

Cardiac failure	No of patients	Percentage
Distension of abdomen	**3**	**60**
Fever	**0**	**0**
Pain abdomen	**0**	**0**
Loss of appetite	**1**	**20**
Bowel disturbance	**0**	**0**
Icterus	**1**	**20**

Graph 6

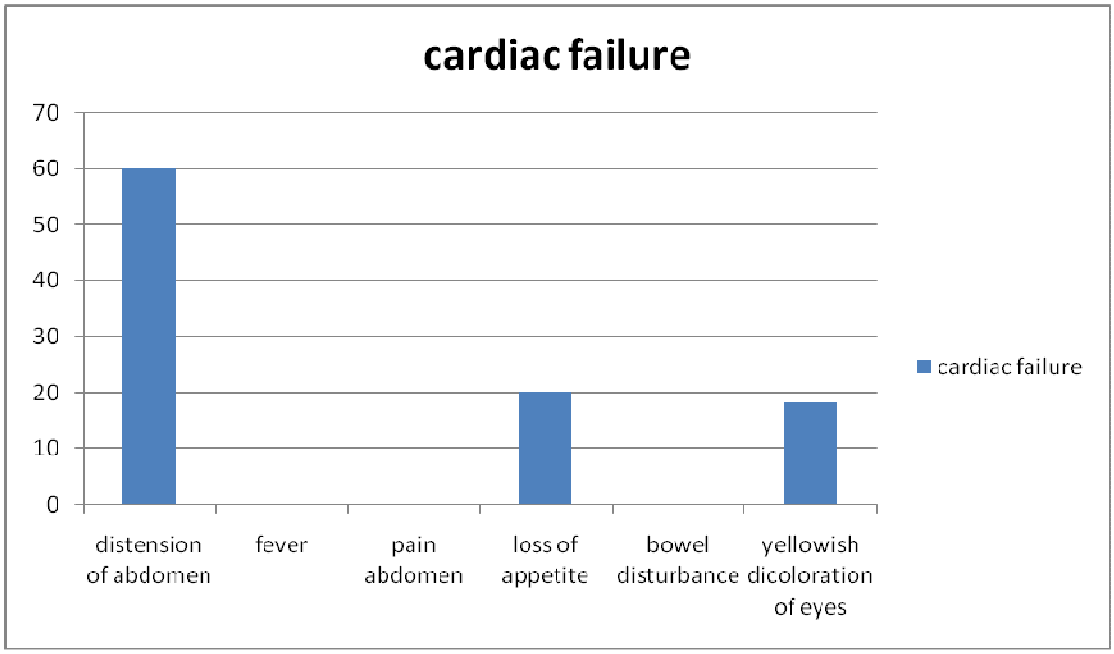


Table no 7

Tuberculosis of abdomen	No of patients	Percentage
Distension of abdomen	3	33
Fever	6	66
Pain abdomen	4	44
Loss of appetite	4	44
Bowel disturbance	2	22
Yellowish disclouration of eyes	0	0

Graph 7

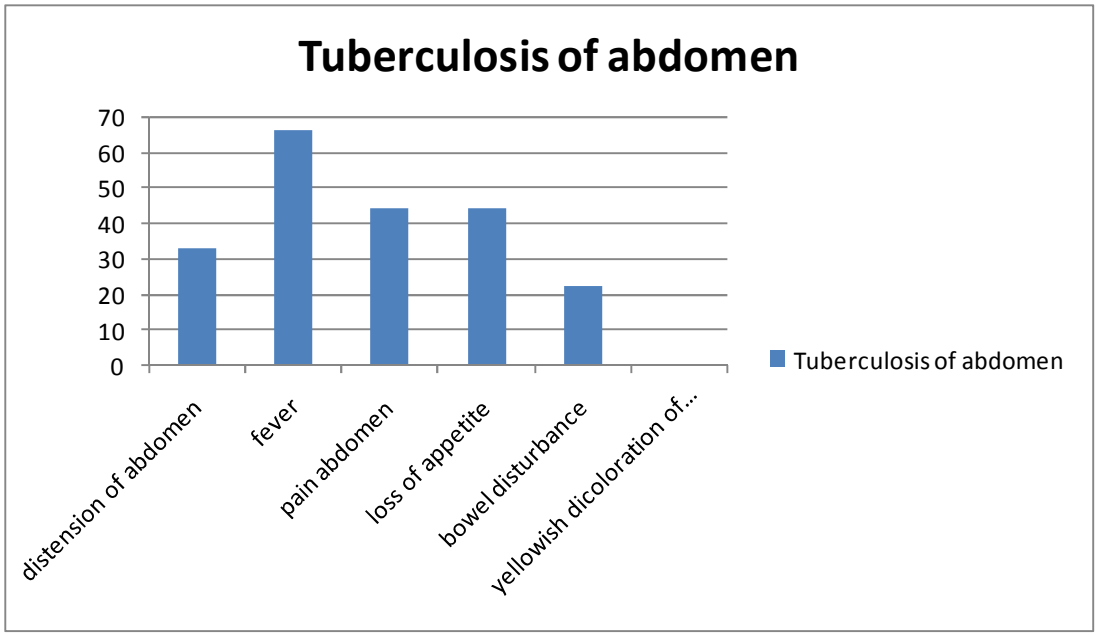


Table no 8

Malignancy	No of patients	Percentage
Distension of abdomen	2	40
Fever	1	20
Pain abdomen	0	0
Loss of appetite	4	80
Bowel disturbance	1	20
Yellowish disclouration of eyes	0	0

Graph 8

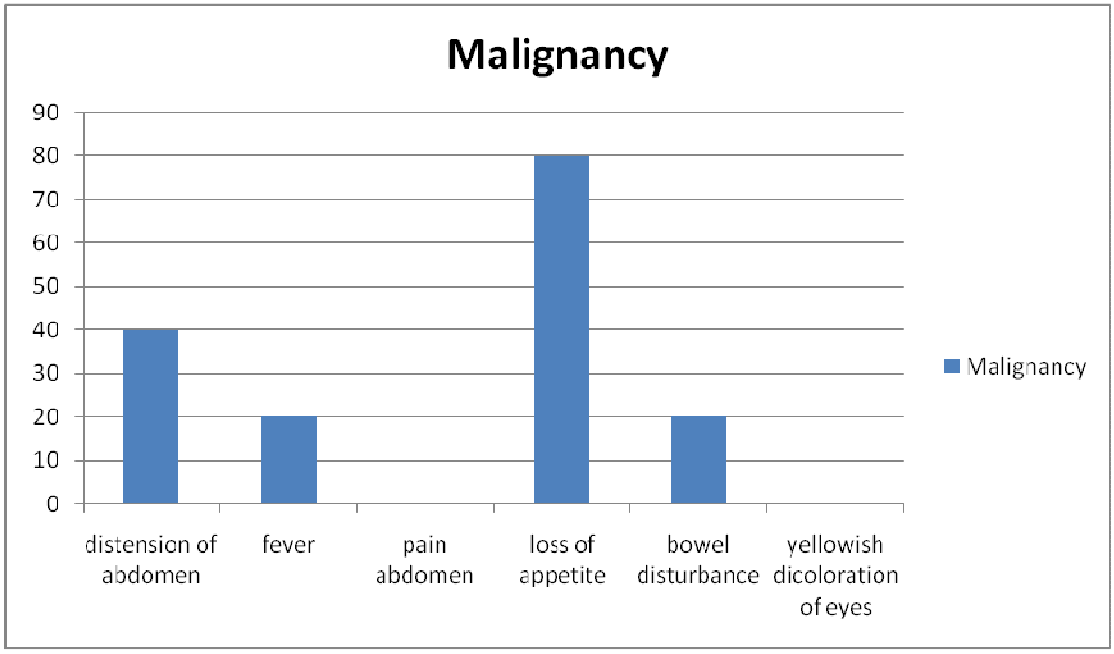


Table no 9

Anaemia with hypoproteinaemia	No of patients	Percentage
Distension of abdomen	2	100
Fever	0	0
Pain abdomen	0	0
Loss of appetite	2	100
Bowel disturbance	0	0
Yellowish disclouration of eyes	0	0

Graph 9

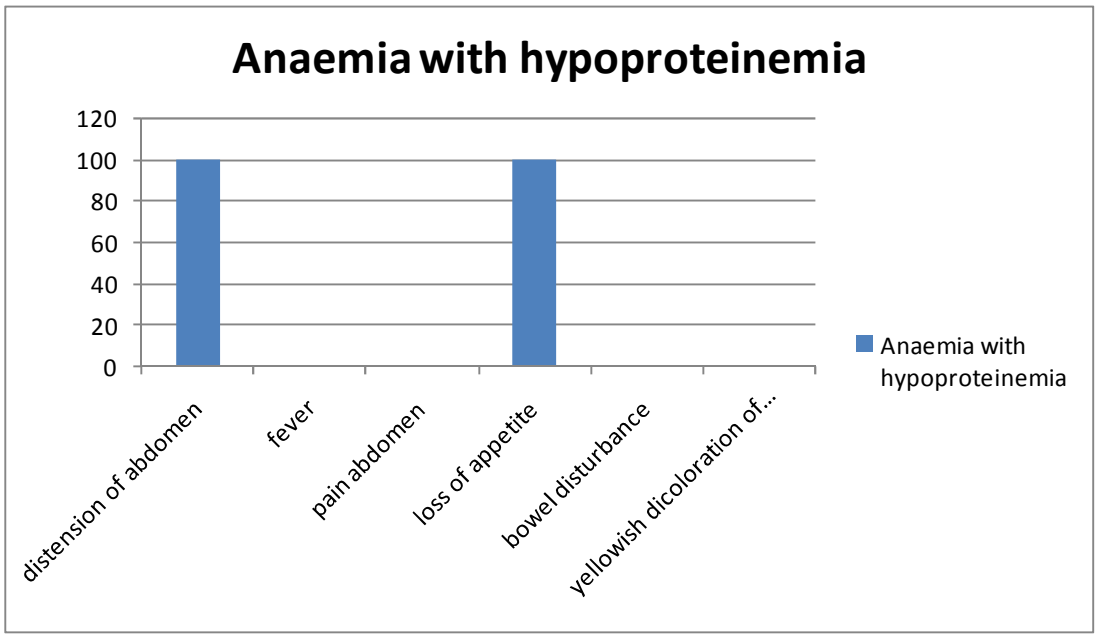


Table no 10

Nephrogenic cause	No of patients	Percentage
Distension of abdomen	1	100
Fever	0	0
Pain abdomen	0	0
Loss of appetite	1	100
Bowel disturbance	0	0
Yellowish disclouration of eyes	0	0

Graph 10

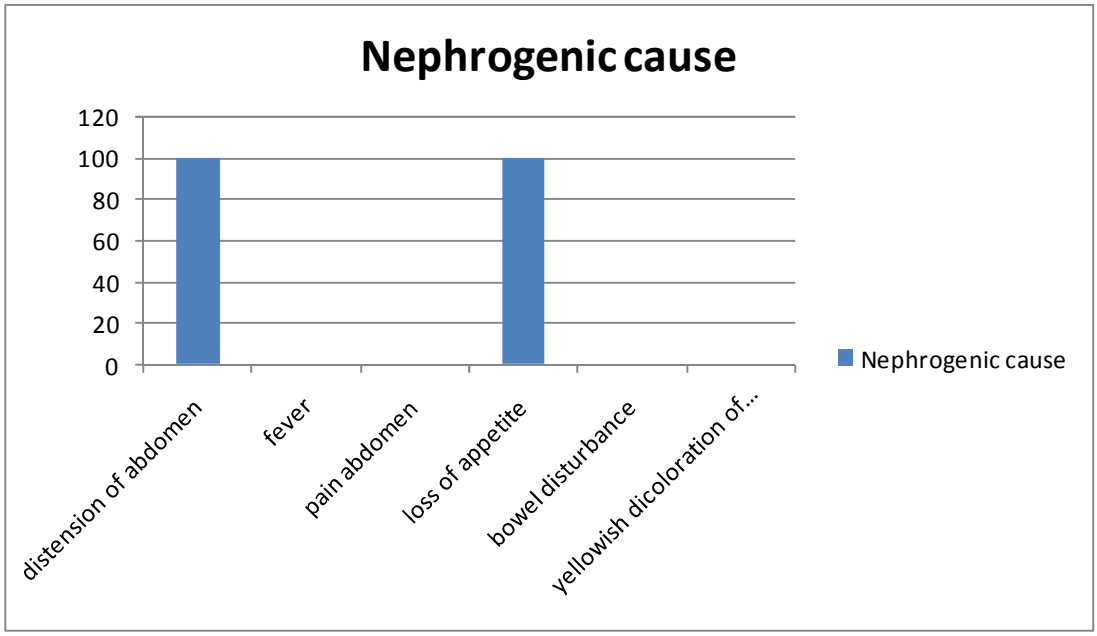


Table :11

Signs of the patients. Diseases (No of Patients)	Signs					
	Icterus	Pallor	Hepato megaly	Ascites	Spleno megaly	Pedal edema
Cirrhosis (38)	24	15	10	38	16	8
Cardiac failure (5)	1	1	5	4	2	5
Tuberculosis Abdomen (9)	-	3	-	2	1	-
Malignancy (5)	1	3	1	4	-	-
Anemia with Hypoproteinemia (2)	-	2	-	2	-	2
Nephrogenic cause (1)	-	1	-	1	-	1

Graph 11

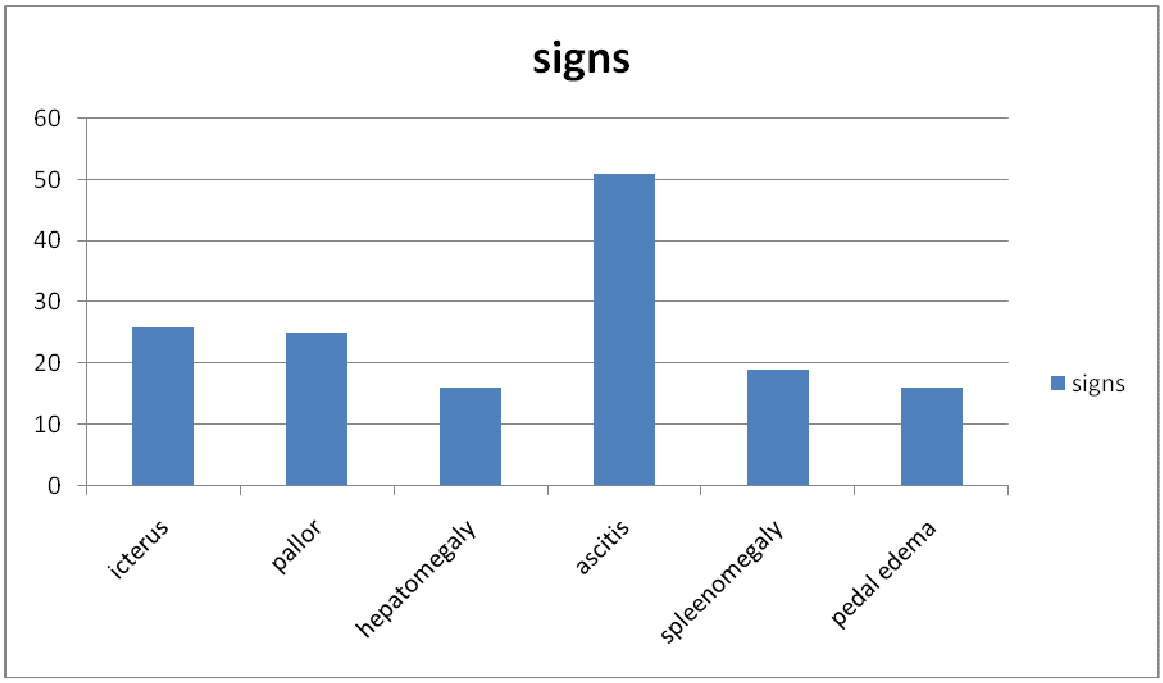


Table no 12

Cirrhosis	No of patients	Percentage
Icterus	24	63
pallor	15	39
Hepatomegaly	10	26
Ascites	38	100
Splenomegaly	16	42
Pedal edema	8	21

Graph no 12

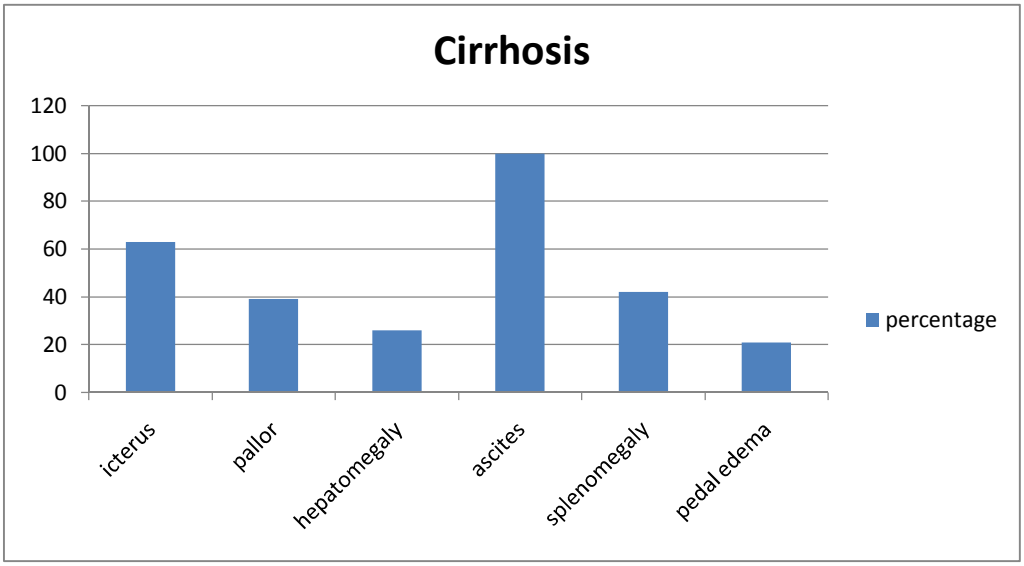


Table no 13

Cardiac failure (5)	No of patients	Percentage
Icterus	1	20
pallor	1	20
Hepatomegaly	5	100
Ascites	4	80
Splenomegaly	2	40
Pedal edema	5	100

Graph no 13

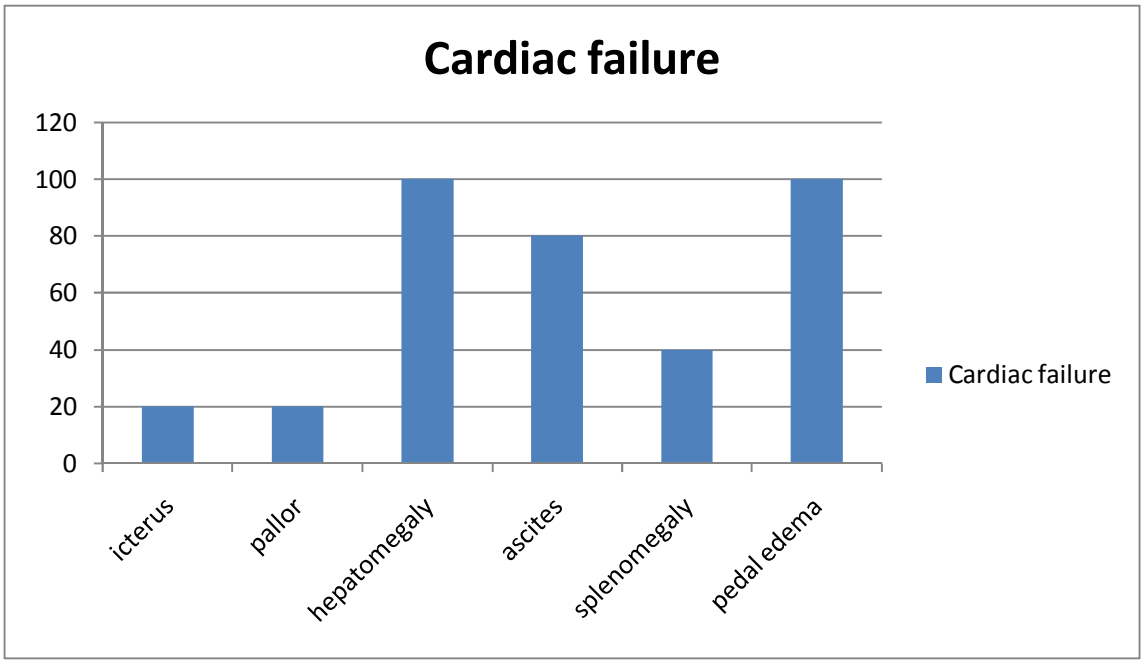


Table no 14

Tuberculosis abdomen(9)	No of patients	Percentage
Icterus	0	0
pallor	3	33
Hepatomegaly	0	0
Ascites	2	22
Splenomegaly	1	11
Pedal edema	0	0

Graph no 14

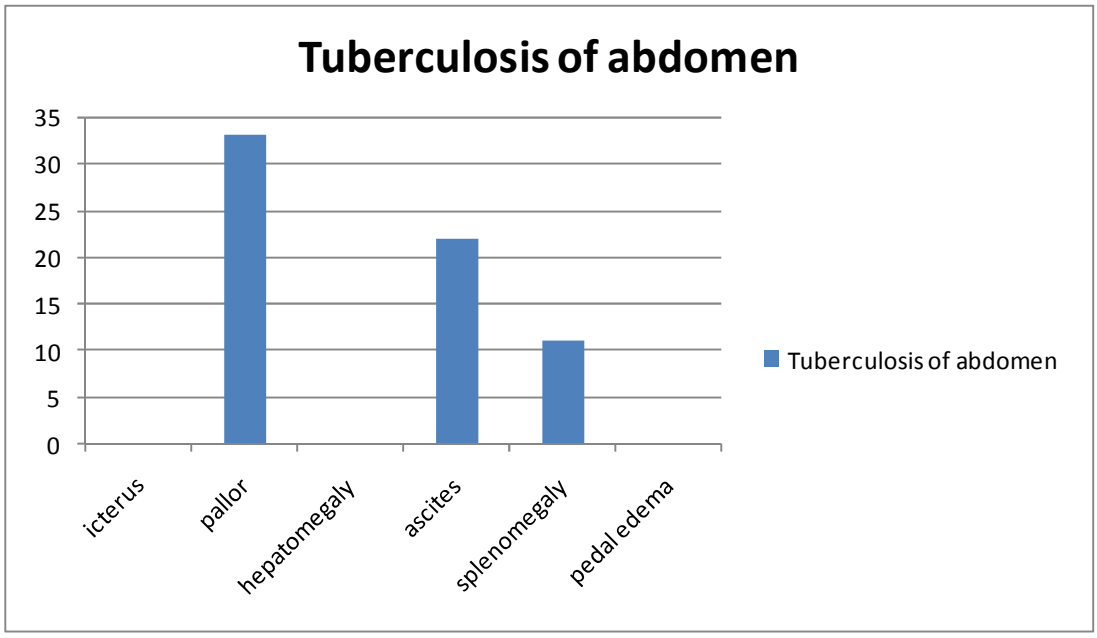


Table no 15

Malignancy (5)	No of patients	Percentage
Icterus	1	20
pallor	3	60
Hepatomegaly	1	20
Ascites	4	80
Splenomegaly	0	0
Pedal edema	0	0

Graph no 15

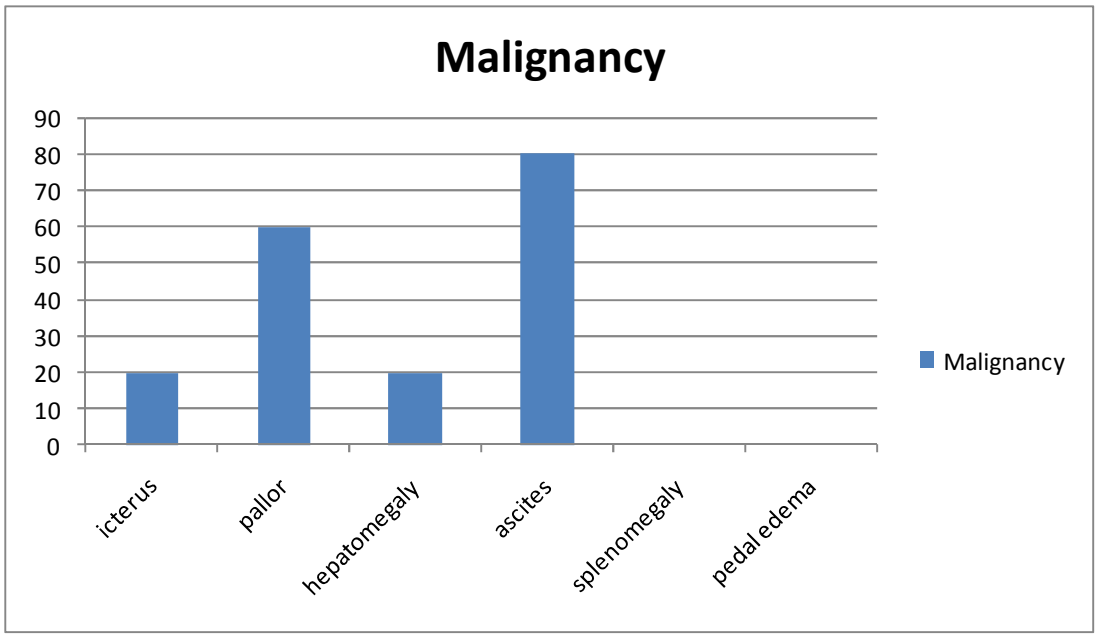


Table no 16

Anaemia with hypoproteinemia(2)	No of patients	Percentage
Icterus	0	0
pallor	2	100
Hepatomegaly	0	0
Ascites	2	100
Splenomegaly	0	0
Pedal edema	2	100

Graph 16

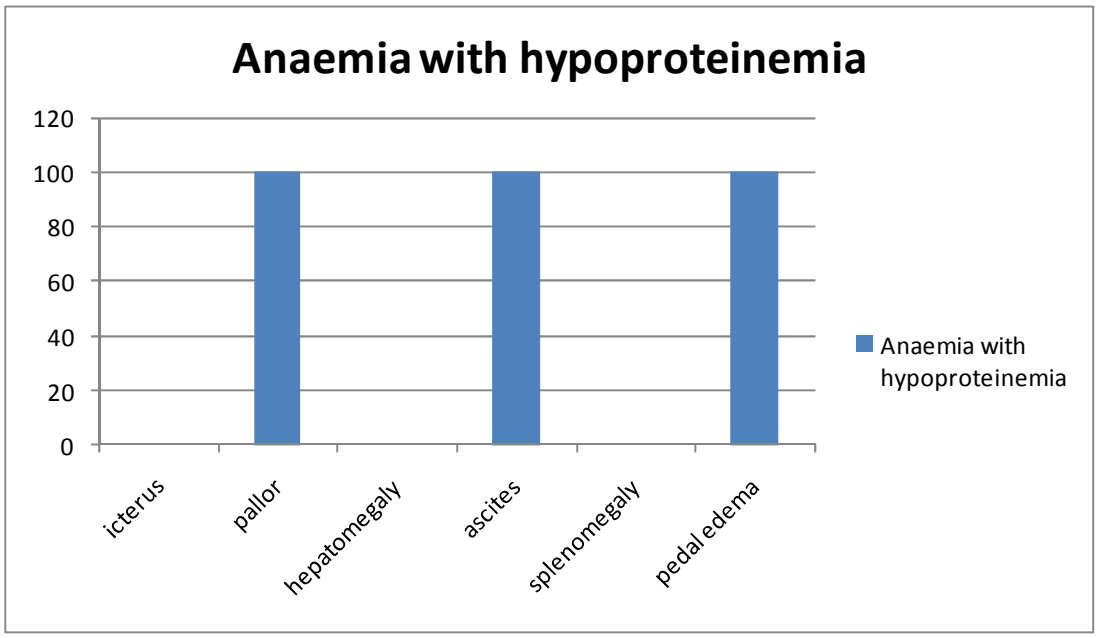
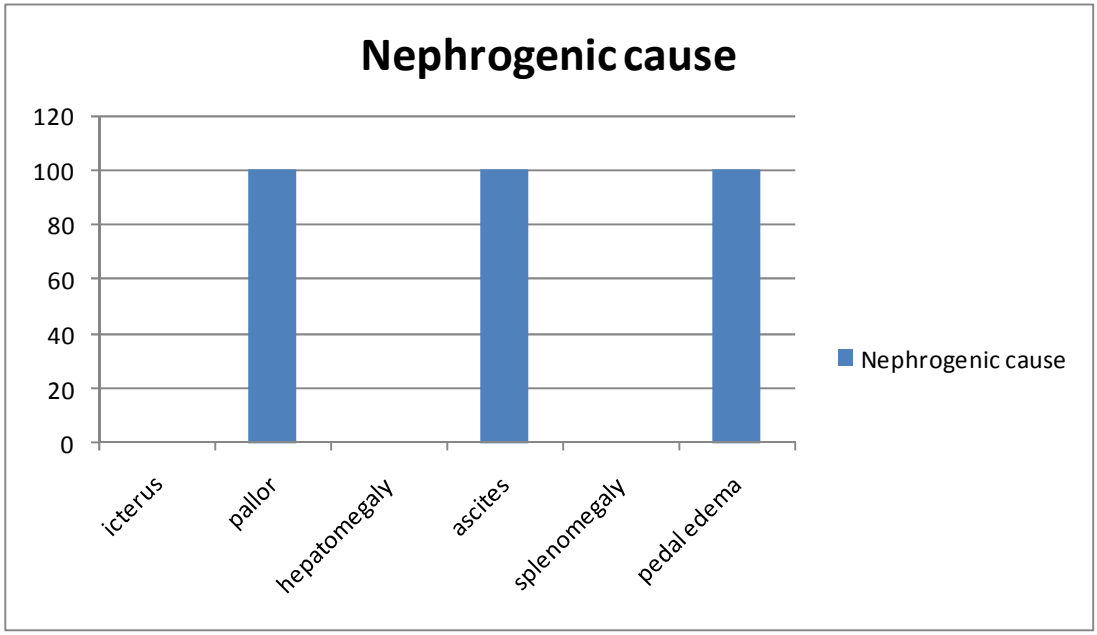


Table no 17

Nephrogenic cause(1)	No of patients	Percentage
Icterus	0	0
Pallor	1	100
Hepatomegaly	0	0
Ascites	1	100
Splenomegaly	0	0
Pedal edema	1	100

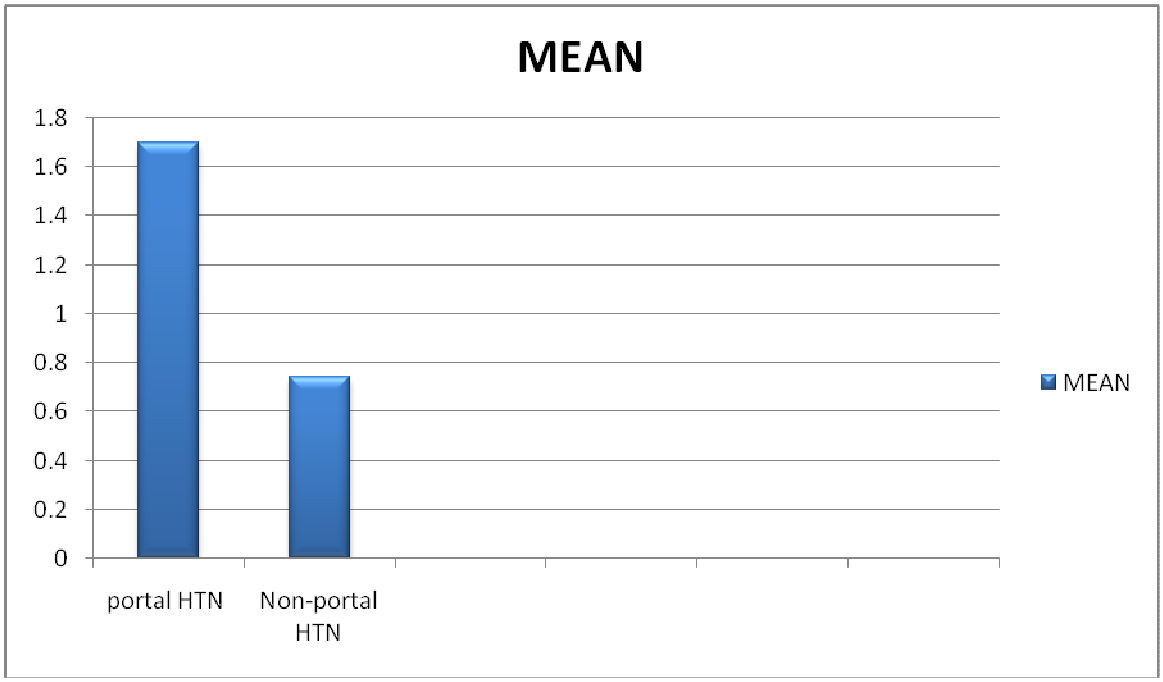
Graph no 17



Out of total 60 patients 39 patients SAAG was under portal hypertensive range and in 21 patients SAAG value was in non hypertensive range. The mean value for serum - ascites albumin gradient in portal hypertension is 1.7, greater that of non portal hypertension 0.74.

Graph Showing mean value of serum ascites albumin-gradient in portal hypertension and non portal hypertension

Graph no 18



The t value is 4.6 and p value is <0.0001 . The P value clearly suggests that serum - ascites albumin gradient is highly effective in classifying ascites of portal and non portal hypertensive etiology liver, out of 38 cases, serum - ascites albumin gradient correctly classified it as a portal hypertensive etiology in 35 cases while AFTP (Ascitic fluid total protein) did so in 30 cases .

In cardic failure , serum - ascites albumin gradient correctly classified it under portal hypetensive etiology in 4 cases

Table no 19

Disease	Portal HTN	Non portal HTN
Cirrhosis(38)	**35**	**3**
Cardiac failure(5)	**4**	**1**
Tuberculosis of abdomen(9)	**0**	**9**
Malignancy(5)	**0**	**5**
Anaemia with hypoproteinamia(2)	**0**	**2**
Nephrogenic cause(1)	**0**	**1**

TABLE : 20

Comparing sensitivity , Specificity , Positive and Negative predictive values of the two parameters studied

	serum –ascites albumin gradient (%)
Sensitivity	92.1
Specificity	84
Positive Predictive value	89.7
Negative predictive value	87.5

The Serum – Ascites albumin gradient (SAAG) has the highest sensitivity 92.1%.

The specificity of serum - ascites albumin gradient is 84%.

The positive predictive value of serum - ascites albumin gradient is 89.7 %.

The negative predictive value of serum - ascites albumin gradient is 87.5%.

Efficacy means percentage of all results that are true results.

It is expressed as true positive and true negative tests over the number of tests.

Efficacy[46] for serum - ascites albumin gradient is 94%and that of AFTP is 80%. Comparing mean of serum - ascites albumin gradient in portal and non portal hypertension cases.

	Portal hypertension	Non portal hypertension
SAAG	**1.7**	**0.74**

DISCUSSION

Comparing efficacy of different parameters studied

The efficacy of serum - ascites albumin gradient (SAAG) and ascetic fluid total protein (AFTP) to classify portal hypertension and non portal hypertension etiology is 94% and 80% respectively.

These values are comparable to the results obtained by Akrividis EA et al in 1996 Goyal AK[47] et al in 1999 and Runyon B A et al in 1992.[48]

The serum - ascites albumin gradient correctly differentiated Ascites of portal hypertension and non portal hypertension causes in 94%of the cases in the present study ,96.7% as studied by Runyon et al, and 97% as studied by Mc Hutchison JG .

In Ascites of liver disease, 35 out of 38 patients of liver diseases serum - ascites albumin gradient was increased, i.e in portal hypertensive range.

This correlated well with the previous studies by Pierre pare,Talbot and Hoefs[52] who studied 51 patients with Ascites, reported 28 out of 29 patients with liver disease serum - ascites albumin gradient in the predicted range.

The differential diagnosis of Ascites remains a clinical problem unless a positive diagnosis of malignancy or infection is confirmed by cytology or culture. Such a definite cause cannot be firmly established by conventional analysis of ascitic fluid Moreover these possibilities may be suspected inappropriately in patients with ascites related to liver diseases. The earlier approach used in the differetial diagnosis consisted of separating ascitic fluid based on the concentration of protein. Defining fluid with protein level <2.5or 3g/dl as transudate usually caused by liver diseases and fluid with higher protein level as exudate usually found in neoplasms and tuberculosis

or other inflammatory diseases.However high protein ascites occurs in 15-20% of patients with liver diseases.[49]

The present study was undertaken to evaluate the reliability of serum - ascites albumin gradient ,a parameter reflecting the oncotic pressure gradient between the vascular bed and the interstitial splanchnic or ascitic fluid. According to starling hypothesis the fluid movement across the capillaries is controlled by the balance of hydrostatic and colloidal osmotic forces across the capillary wall. These forces tend to achieve a dynamic equilibrium so that the increased portal pressure is counter balanced by increased oncotic pressure gradient across the capillary membrane .This physiological event is the basis for postulated serum - ascites albumin gradient as the true indicator of presence or abscence of increased portal pressure.[50]

Lt col AK Sethi et al who studied 50 patients with ascitis reported 33 patients had portal hypertension out of 39 patients with high SAAG value and 2 patients had portal hypertension out of 11 patients with low SAAG value.

B A Runyon et al who studied 931 patients with ascites reported accuracy of serum - ascites albumin gradient in 96.7% of the cases and 55.6% for AFTP.

Kundu et al studied 51 patients and reported accuracy of 97.8% for serum - ascites albumin gradient and 42% for AFTP.[51]

Study by Lauden DM et al the accuracy was 95.7% for serum - ascites albumin gradient and 65.5% for AFTP.

Kundu et al reported increased serum - ascites albumin gradient in 100% of patients with liver diseases and portal hypertension causing Ascites.

In contrast to serum ascites albumin - gradient, AFTP retained accuracy in 26 out of 32 patients (81.2%) with liver diseases. It is due to the fact AFTP is influenced by other factors than portal pressure, serum protein concentration being one.

This results correlated with the previous studies, 55.6% patients had AFTP in transudative range in study conducted by Runyon et al, Kundu et al reported 70% efficacy for AFTP.

In Ascites of congestive heart failure serum - ascites albumin gradient is in the portal hypertension range i.e >1.1gm % in all the 4 Patients studied, where as AFTP was in transudative range only in two patients.

Pierre-pare et al reported 100% accuracy of serum - ascites albumin gradient in these patients.

BA Runyon et al and Kundu et al have reported 96.7% and 97% accuracy respectively for serum - ascites albumin gradient in cardiac failure.

In tuberculosis serum - ascites albumin gradient placed it under non portal hypertension etiology in all 9 patients where as AFTP in 7 out of 8 patients.

Marshal JB[53] reported SAAG, 1.1gm/dl, all in the Non portal Hypertension range in all the patients he studied.

In malignancy related ascites serum - ascites albumin gradient was in non portal hypertensive range in all 5 patients , whereas AFTP in exudative range in 3 patients.

In Pierre pare et al study serum - ascites albumin gradient retained accuracy in 14 out of 15 patients (93.3%) with malignancy related Ascites.

B.A Runyon and kundu et al reported accuracy of 96.7% and 100% respectively for serum - ascites albumin gradient in malignancy related Ascites.

AFTP is in exudative range in malignancies in 55.67% of cases in study by BA Runyon et al and 73.3% of the 15 patients of Pierre pare et al.

This study further substantiates that SAAG can be used classify ascites of portal and non portal hypertensive causes.

In this study for diagnostic purpose invasive procedures were not done (liver is peritoneal biopsy), chances of missing ascites of mixed etiology is present. However in ascites of mixed etiology SAAG will be in portal hypertensive range.

Study done by entesar H Shargawy eman M fathy at faculty of medicine Benaha university. In there study non alcoholic cirrhotic cases with ascites were included. They had mesaurment of SAAG computed. An upper GI endoscopy was done in all cases to asess the presence and size of EV.

36 patients (80%) had EV (osophagcal varices) The mean SAAG was 1.40+0.27 SAAG score (>1.55gm/di) was useful predictor of the presence of EV in cirrhotic patients with ascites [54].

Study done by M Beg, S.Husain, N.Ahmed, N Aktar at JN medical college Aligarh muslim university.

The study was done to differentiate ascites on the basis of secum/ascites albumin gradient. 100 patients were examined 76 patients having ascites related to portal hypertention (cirrhosis 54, cardiac 10, secondary bacterial peritonitis-6, Liver metastasis 6) and 24 patients of tubercular ascites not related to portal hypertension.

The diagnostic accuracy of SAAG & AFTP were 96% and 68% respectively[55]

Study done at second xiangya hospital central south university,chaysha china. In their study 136 patients ascites were divided into 5 groups cirrhotic group (Group A), hepatocellular carcinoma group(Gp B) peitoneal carcinoma (GpE) spontaneous peritonitis (Gpc), tubercular peritonitis (GpD).

Group A,B,C, had portal hypertention SAAG was more than 1.1mg/dl in group A,B & C but less than 1.1mg/dl in Group D&E.

SAAG demonstrates that patient ascites fluid possess the basis of portal hypertention[56].

Study done by Pazhanivel Mohan, Jayanthi Venkataraman at Stanly Medical College Chennai. Study was done between Jan 2008 and December 2009 patients with cirrhosis of liver undergoing therapeutic paracentesis, cirrhosis of liver (55.5%) hepatites B (21.8%), hepatites C (9.1%) and others (13.6%). In 7 patients ascitic fluid infection was found[57].

SUMMARY AND CONCLUSION

In this study 60 patients of ascitic proved by ultrasound were included. They were categorised into 2 groups based on the presence or absence of portal hypertension by ultrasound criteria.

Ascitic fluid total protein, Serum and Ascitic fluid albumin was done in all patients.

SAAG was in portal hypertensive range in 34 of the 36 patients with portal hypertensive and in non-portal hypertensive range in 5 of the 20 patients in non portal hypertension causes.

AFTP was in portal hypertensive range in 28 of 36 patients hypertension and in non portal hypertensive range in 13 of the 14 patients non portal hypertension causes.

Serum - ascites albumin gradient (SAAG) has efficacy of 94%, and ascitic fluid total protein (AFTP) 80% in classifying ascites of portal hypertension and non portal hypertension causes.

The mean of serum - ascites albumin gradient (SAAG) in portal hypertension is 1.7 and in non portal hypertension 0.74, and is statistically significant in classifying ascites of portal and non portal hypertension causes.

The mean of ascitic fluid total protein (AFTP) in portal hypertension is 2.23 and in non portal hypertension 3.41 and is statistically significant but lesser than serum- ascites albumin gradient.

Serum - ascites albumin gradient was especially useful in seperation of cardiac ascites which usually has high protien concentration.

So serum - ascites albumin gradient (SAAG) has superior discriminating power and should replace ascitic fluid total protein (AFTP) in the separation of ascites of portal and non portal hypertention causes.

A serum ascites albumin gradient >1.1g/dl does suggest of portal hypertension not only in patient with transudate type of ascites but also in cases with high protein concentration.

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RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT : EFFICACY OF SERUM ASCITIC ALBUMIN GRADIENT IN ASCITES OF PORTAL AND NONPORTAL HYPERTENSIVE ASCITIC CAUSES

PRINCIPAL INVESTIGATOR : DR. SUHASINI.S.SHENDGE
P.G. DEPARTMENT OF GENERAL MEDICINE

P.G.GUIDE : DR. R. M. HONNUTAGI MD
PROFESSOR,
DEPT OF GENERAL MEDICINE.

CHAIRMAN : Dr. A. A. NAIKWADI
Prof. of PHARMACOLOGY
9342355742

PURPOSE OF RESEARCH:

I have been informed that the present study is conducted to SAAG in ascites patients with portal and non portal hypertention.

PROCEDURE:

I understand that I undergo detailed history and after which necessary investigations will be done.

RISK AND DISCOMFORTS:

I understand that, there is no risk involved in the procedures performed.

BENEFITS:

I understand that my participation in the study will help to know the SAAG in ascites patients with portal and non portal hypertention.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of the hospital. If the data is used for publications the identity of patient will not be revealed.

REQUST FOR MORE INFORMATION:

I understand that I may ask more information about the study at any time.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw from the study at any time without prejudice.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me during the study I will get medical treatment but no further compensations.

I have read and fully understood this consent form. Therefore I agree to participate in the present study.

_____ _____

Participant / Guardian Date:

_____ _____

Signature of Witness Date:

I have explained the patient the purpose of the study, the procedure required and possible risk and benefit to the best of my ability in the vernacular language.

_____ _____

Investigator / P.G. Date:

_____ _____

Witness to Signature Date

PROFORMA

Name:

Age:

Sex:

Inpatient No:

Occupation:

Date of admn:

Date of Discharge:

Address:

Chief complaints

History of Presenting Illness

- Abdominal distension - Sudden / Gradual.
- Pain - discomfort in abdomen - generalized or localized,
- Swelling - lower limb / upper limb / face / sacral region.
- Breathlessness / cough - dry or productive (color, amount, smell, blood)
- Chest pain - Site, Character, Radiation, Aggravating / Relieving factors,
- Palpitations
- Fever
- Jaundice
- Hacmaturia / Malena / Haematemesis / Haemoptysis
- Oliguria or Polyuria, Polydipsia
- Loss of appetite, weight gain / loss of weight, Fatigue.
- Blood transfusion, alcoholic.

Past History

Jaundice / haemetemesis

Bleeding per rectum / change in bowel habits

TB / HTN ; Epilepsy / Head Injury - loss of consciousness

Blood Transfusions

Breathlessness, Palpitations.

Personal History ;

Appetite

Diet

Sleep

Bowel and Bladder Habits

Habits - Alcohol - type, quantity

Smoking / Tobacco chewing

Menstrual and Obstetric history.

Family History:

Similar history in the family members.

Chronic illness in the family.

Socio - economic status, Housing.

General Physical Examination :

General appearance Built and nourishment

Pallor Icterus Cyanosis Clubbing Pedal edema

Koilonychia Lymphadenopathy

Skin - Dry / Dehydration - Skin turgor

/ oedema / pigmentation / pallor / cyanosis / jaundice / / hair changes / spider naevi .

Face - Puffiness / Flushing .

Conjunctiva and Cornea - Bitot's spots Xerosis Conjunctivitis Icterus Keratomalacia

Tongue - Pale / Cyanosed / Dry.

Mouth - Angular stomatitis / Glossitis / Chelitis / Apthous Ulcers.

Neck - Lymph node swelling / Thyroid Swelling

Hands & Legs - Pale / Koilonychia / Cyanosis / Oedema / Clubbing / Icterus,

Scrotum / Testis: Size Sensation Hydrocele

Systemic Examination :

Abdominal Examination :

Inspection :

Shape - Scaphoid *I* Distension. *I* Uniform

Abdominal Scars

Skin over the abdomen *I Dry I* Tense

Glistening -Iinea alba ,Pigmentation

Umblicus - Normal/Transverse *I* Vertical *I* Everted.

Divarication of Recti

Veins - Caput medusae *I* Prominence in Flanks.

Movement : Normal *I* Restricted.

Hernia - Abdominal / Inguinal *I* Umbilical / lncisional

Visible Peristalsis.

Palpation :

Temperature of the body. .

Muscle Guarding / rigidity *I* Tenderness.

Edema of the abdominal wall

Doughy *I* Resilient feeling.

Liver - Size Shape Surface Borders

Consistency Movement with respiration Tenderness.

Spleen - Size

Any other Mass

Measurement of Abdominal Girth at the level of Umbilicus

Distance between :-

Umbilicus and Pubic Symphysis

Umbilicus and Xiphi Sternum

Umbilicus and ASIS.

Palpation after abdominal paracentesis.

Percussion Fluid Thrill & Shifting Dullness

Minimal Ascitis - Knee Elbow Position

Liver Dullness and Splenic, Dullness

Auscultation Splenic Rub and Liver, -Rub

-Bruit

- Venous Hum

- Bowel Sounds

Cardio Vascular System

Pulse B.P JVP

Cardiac Examination

Respiratory System

Central Nervous System

For evidence of peripheral tremors.

For evidence of Pre-Hepatic Coma and Hepatic Coma - Flapping Tremors

Investigations

Hb%

TC

DC

ESR

B. Urea

S. Creatinine

HBs Ag

B. Sugar - FBS & PPBS

Liver Function Tests

Serum bilirubin

Serum direct bilirubin

Serum Total Protein

SGPT

SGOT

SALP

Serum Albumin

Serum Globulin

A : G Ratio

Urine

---Specific Gravity

---Albumin

---Sugar

---Microsopy

Bile Salts & Pigments

Stool

Ova

Cyst

Ocult Blood

Ascitic Fluid

Total Protein

Albumin

Cell Count & Type

AFB AND Gram stain

USG Abdomen

ECG

Chest X-ray PA view

Endoscopy

Treatment

Summary

ETHICAL CLEARANCE CERTIFICATE

Institutional Ethical Committee
BIJAPUR-586 103.
OUTWARD
No. 046/12
18/10/12
Shri B. M. Patil Medical College.

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-2012 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title Efficacy of Serum ascites albumin gradient in ascites of portal and non portal hypertensive ascitic causes – A Prospective Study

Name of P.G. student Dr. Sulasini. S. Shendge
Medicine

Name of Guide/Co-investigator Dr R.M. Honnutagi
Professor of Medicine

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

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

KEY TO MASTER CHART

CIRR	:	Cirrhosis
Nep	:	Nephrogenic cause
Card	:	Cardiac failure
Ca	:	Carcinoma
TB	:	Tuberculosis of abdomen
An & Hyp	:	Anaemia with hypoproteinemia

Sl.No	Name of the Patient	Age in years	Sex	IP No	Date of Admission	Distension of abdomen	Fever	Pain abdomen	Loss of appetite	Bowell disturbance	Yellow discoloration of sclera	Pallor	Icterus	Hepatomegaly	Ascites	Splenomegaly	Pedal edema	Serum albumin	Ascitic albumin	Serum Ascitic albumin Gradient	Ascitic fluid Total Protein	Cause
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
1	Sharanu	28	M	19049	30/6/2014	+	-	-	+	-	+	-	+	-	+	-		2.5	0.3	2.2	0.6	CIRR
2	Guruhindappa	45	M	20565	14/07/2014	+	+	-	-	+	-	+	-	+	+	+	-	1.2	o.2	1	2.8	CIRR
3	Laxman	55	M	23400	08/08/2014	+	-	+	+	+	+	+	-	-	+	-		1.8	0.9	0.9	2.1	AN & HYP
4	Sadashiv	30	M	13959	12/03/2014	-	+	-	-	+	-	-	-	-	-	-	-	3.4	2.6	0.8	3.8	TB
5	Balaram	50	M	8463	26/03/2014	-	+	-	+	+	-	-	+	-	-	-	-	3.2	2.2	1	4	TB
6	Manjunath	30	M	7001	12/03/2014	+	-	-	-	-	-	-	-	-	+	-	-	2.8	1.2	1.6	1.8	CIRR
7	Vivekanand	38	M	4599	16/02/2014	+	+	-	-	-	+	+	+	-	+	+	-	3	1.4	1.6	2.8	CIRR
8	Suresh	26	M	3861	08/02/2014	+	-	-	-	-	-	-	-	-	+	-	-	2	1.2	0.8	2.2	CIRR
9	Mahantsh	35	M	2032	12/11/2013	+	-	-	+	-	-	-	+	-	+	-	-	3.2	2.5	0.7	4.2	CA
10	Laxmi	45	F	4674	21/12/2013	+	-	+	-	+	-	-	-	+	+	+	-	3	1.1	1.9	2.4	CIRR
11	Subhash	53	M	3052	04/12/2013	+	-	-	-	-	-	+	+	-	+	-	+	2.1	0.6	1.5	1.2	CIRR
12	Prakash Chandru	62	M	2941	07/12/2013	+	-	+	-	-	-	-	-	-	+	-	-	2.1	0.9	1.2	2	CIRR
13	Saiprasad	73	M	1851	22/11/2013	+	-	-	+	-	-	+	-	-	+	+	-	3	0.9	2.1	1.9	CIRR
14	Prabhu	38	M	1566	19/11/2013	-	-	-	+	-	-	-	-	-	+	-	-	2.4	2.2	0.2	3.9	CA
15	Mohan	50	M	13513	06/11/2013	+	-	-	-	-	-	-	-	-	-	-	-	3.5	2.5	1	3	TB
16	Kalyani	16	F	20014	08/08/2014	+	-	-	-	+	+	-	+	+	+	-	-	2.6	1.2	1.4	1.8	CIRR
17	Bagavantaraya	40	M	30016	29/10/2013	+	+	-	+	-	+	+	+	-	+	+	-	3	1.1	1.9	3.3	CIRR
18	Mallikarjun	42	M	13149	05/05/2014	+	-	-	-	-	-	-	-	+	+	+	-	3.4	2	1.4	2.9	Card
19	Shantosh	40	M	197829	07/07/2014	-	-	-	+	-	-	-	-	-	+	-	-	3.5	2.7	0.8	4	TB
20	Malkarji	28	M	22887	29/07/2014	+	-	-	-	+	+	-	+	-	+	-	-	2	0.6	1.4	0.8	CIRR
21	Chandrakant	52	M	1639	15/12/2013	+	+	-	+	-	-	+	-	-	+	+	+	2.1	1.2	0.9	2.3	CIRR
22	Chandrakanth	35	M	339	09/11/2013	+	-	-	-	+	+	-	+	+	+	-	-	3	1.2	1.8	2.1	CIRR
23	Vittal	32	M	10199	12/04/2013	+	-	-	+	-	+	+	+	-	+	+	-	2.1	0.6	1.5	1.9	CIRR
24	Madiwalappa	55	M	17608	03/10/2013	+	+	-	-	-	-	-	+	-	+	-	+	1.6	0.4	1.2	2.6	CIRR
25	Saiprasad	73	M	5018	25/02/2014	+	-	+	+	+	+	-	+	-	+	-	-	3.4	1.2	2.2	2.1	CIRR
26	Shrishail	30	M	16326	14/06/2013	+	-	-	-	-	-	+	-	-	+	+	-	3	1.1	1.9	2.4	CIRR
27	Vishwanath	30	M	19472	15/07/2013	+	+	-	+	-	-	-	-	-	+	-	-	2.6	1.9	0.7	2.4	TB
28	Sagareppa	55	M	43	01/01/2013	+	-	-	+	-	+	-	+	+	+	+	-	2.6	0.8	1.8	2.7	CIRR
29	Nimbanna	51	M	3372	06/02/2013	+	-	-	+	-	-	-	-	-	+	-	-	2.6	2	0.6	3	Nep
30	Ravasab	45	M	1916	21/02/2013	+	-	-	-	-	-	+	-	-	+	+	-	2.8	1.4	1.4	3	CIRR
31	Bhimaraya	64	M	15393	23/07/2013	+	-	+	-	+	-	-	+	+	+	-	+	3	1.5	1.5	2.8	CIRR

32	Manawwa	46	F	5077	23/02/2013	+	-	-	+	-	-	+	-	-	+	-	-	3.4	0.2	3	1.6	CIRR
33	Manikchand	44	M	4076	16/12/2013	+	-	-	-	-	+	-	+	-	+	+	-	2	0.6	1.4	1.2	CIRR
34	Basavaraj	40	M	1127	27/11/2013	+	-	+	-	+	-	-	-	+	+	-	-	3.3	1.9	1.4	2.4	CIRR
35	Ganapati	54	M	20877	29/07/2013	+	-	-	+	-	+	-	+	-	+	+	+	2.8	1.8	1	1.8	CIRR
36	Ningappa	70	M	3455	09/12/2013	-	+	-	+	-	-	-	-	-	-	-	-	2.9	1.4	1.5	2.1	Ca
37	Basavaraj	35	M	11277	22/04/2013	-	+	-	-	-	-	-	-	-	-	-	-	3.9	3	0.9	5.7	TB
38	Basappa	50	M	6366	06/03/2013	+	-	-	-	-	-	+	+	-	+	-	-	2.9	1.4	1.5	2.3	CIRR
39	Shanta	48	F	3416	06/02/2013	+	+	-	+	+	+	-	+	+	+	+	+	3	1.2	1.8	2.1	CIRR
40	Malkari	40	M	20316	10/09/2012	-	-	-	+	-	+	-	+	+	+	-	-	3.4	1.6	1.8	2.3	Card
41	Danappa	55	M	15808	10/06/2013	+	-	-	-	+	-	-	-	-	-	-	-	2.5	1.7	0.8	3.9	ca
42	Ashok	48	M	25948	19/09/2013	+	+	-	+	-	-	-	-	-	+	-	-	3.7	3	0.7	4.9	TB
43	Kajesab	70	M	1097	12/01/2013	-	-	+	+	-	+	+	+	-	+	-	-	2.9	1.3	1.8	1.6	CIRR
44	Mallamma	50	F	9565	06/04/2013	+	-	-	-	-	-	-	-	+	-	+	-	4	2.3	1.7	3.4	Card
45	Shanta	18	F	11458	08/10/2013	+	-	-	-	-	-	-	-	+	+	+	-	2.9	0.5	2.4	0.8	CIRR
46	Channamma	30	F	14554	28/05/2013	+	+	-	-	-	+	-	+	-	+	-	-	2.2	0.9	1.3	2.3	CIRR
47	Mallanna	40	m	15515	07/06/2013	+	-	-	+	-	-	+	+	+	+	+	+	3	1.5	1.5	2.5	CIRR
48	Jakkawwa	46	F	4555	12/12/2013	+	-	+	+	-	-	-	-	-	-	-	-	2.9	2	0.9	2.4	TB
49	Manjunath	30	M	7001	03/11/2014	-	-	-	-	-	-	-	-	+	+	-	-	3.2	1.9	1.3	2.4	Card
50	Raju	40	m	6411	05/03/2014	+	-	-	+	-	+	-	+	-	+	-	-	2.4	0.5	1.9	3	CIRR
51	siddaram	32	m	8705	27/03/2014	+	-	-	+	-	+	+	+	-	+	+	-	2	O.3	1.7	1.4	CIRR
52	madiwalappa	55	m	8449	25/03/2014	+	-	-	-	-	-	-	-	+	+	-	+	2.2	1	1.2	2.6	Card
53	lalsab	55	m	24863	21/02/2014	+	-	-	+	-	-	-	-	+	+	+	-	1.6	O.4	2	2.4	CIRR
54	Abdul	41	m	20987	18/05/2014	+	+	-	+	-	-	-	-	+	-	-	-	1.2	O.3	0.9	1.6	TB
55	shantesh	40	m	19829	07/04/2014	+	-	-	+	-	+	-	-	-	+	+	+	2.6	1.1	1.5	2.5	CIRR
56	santosh	35	m	16541	06/06/2014	-	-	+	+	-	+	-	-	-	-	-	-	1.2	O.6	0.6	1.4	CA
57	Manjunath	30	m	7001	11/03/2014	+	+	-	-	-	+	-	+	-	+	-	-	2.4	O.8	1.6	3.2	CIRR
58	Ambresh	41	m	8567	26/03/2014	-	-	-	+	-	-	+	-	-	-	-	+	O.8	O.2	O.2	O.6	AN & HYP
59	Kallappa	65	m	9716	06/04/2014	+	-	-	-	-	+	-	-	-	+	+	-	3	O.9	2.1	2.4	CIRR
60	Neelakumar	29	m	12501	30/04/2014	+	-	-	-	-	+	-	-	-	+	+	+	2.4	O.8	1.6	2.8	CIRR