EVALUATION OF SERUM SODIUM LEVELS AS PROGNOSTIC MARKER IN PATIENTS WITH CIRRHOSIS OF THE LIVER

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LIST OF ABBREVIATIONS

PH	**PORTAL HYPERTENSION**
SBP	**SPONTANEOUS BACTERIAL PERITONITIS**
HRS	**HEPATORENAL SYNDROME**
HE	**HEPATIC ENCEPHALOPATHY**
GI Bleed	**GASTRO INTESTINAL BLEED**
Na	**SODIUM**
CPC	**CHILD PUGH CLASS**
CPS	**CHILD PUGH SCORE**
O	**OUTCOME**
K	**POTASSSSIUM**
Cl	**CHLORINE**
HCO3	**BICARBONATE**
Mg	**MAGNESIUM**
Ca	**CALCIUM**
PO42	**PHOSPHATE**
ADH	**ANTIDIURETIC HORMONE**
AVP	**ARGININE VASOPRESSIN**
TBW	**TOTAL BODY WATER**
ECF	**EXTRA CELLULAR FLUID**
ICF	**INTRA CELLULAR FLUID**
EABV	**EFFECTIVE ARTERIAL BLOOD VOLUME**
CLD	**CHRONIC LIVER DISEASE**
Una	**URINE SODIUM**

PT	**PROTHROMBIN**
INR	**INTERNATIONAL NORMALIZED RATIO**
SIADH	**SYNDROME OF INAPROPRIATE SECRETION OF ANTIDIURETIC HORMONE**
NaCl	**SODIUM CHLORIDE**
DH	**DILUTIONAL HYPONATREMIA**

ABSTRACT

INTRODUCTION : cirrhosis of liver is one of the leading cause of death and leads to complications like ascites, hepatic encephalopathy, portal hypertension, subacute bacterial peritonitis, hepatorenal syndrome, hepatopulmonary syndrome. It is commonly complicated by abnormalities in electrolyte levels.

MATERIAL AND METHODS : This study is conducted at shri .B.M. patil medical college and research center. The study was conducted from a period of 2020 to 2022. 50 patient admitted with cirrhosis of liver were selected and enrolled for study with consent.

RESULTS : In our study hyponatremia in patients with cirrhosis of liver showed complications like hepati c encephalopathy , hepatorenal syndrome, hepatic encephalopathy and spontaneous bacterial peritonitis.

CONCLUSION : ser um sodium levels can be used as a prognostic marker in patients with cirrhosis of liver and appropriate correction can reduce complications and morbidity and mortality

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INTRODUCTION

Cirrhosis is a condition marked by diffuse destruction of hepatocytes with fibrosis giving rise to regenerative nodules and vascular abnormalities.[1] The d evelopment of abnormalities of renal function and electrolyte levels usually complicates the clinical course of individuals with chronic liver disease (CLD), with hyponatremia being the most prevalent problem found in these patients.[2]

Child-Turcotte-Pugh classification for severity of cirrhosis: It is used to assess the severity of cirrhosis. It is divided into 3 classes depending on the scores. It includes components of Encephalopathy, Ascites, Bilirubin, Albumin, PT/INR. Class A: well compensated (5-6 points); Class B: partially decompensated (7-9 points); Class C:decompensated (10-15 points).[3–5]

Cirrhosis is leading cause of death and results in complications like ascites, hepatic encephalopathy and portal hypertension, subacute bacterial peritonitis, hepatorenal syndrome, hepatopulmonary syndrome. It is frequently complicated by development of abnormalities in renal function and electrolyte levels. Hyponatremia is common in cirrhosis it can be either hypovolemic or hypervolemic.

Reduced sodium excretion by the kidneys as a result of renal hypoperfusion, and increased secretion of anti-diuretic hormone as a result of decreased effective volemia brought on by peripheral artery vasodilation, which causes dilutional hyponatremia. Early diagnosis of hyponatremia and treatment can prevent complications like hepatic encephalop athy, hepatorenal syndrome ,spontaneous bacterial peritonitis and increase the survival rate.[6–9]

Several investigations in large groups of cirrhotic individuals have demonstrated that the kidney's capaci ty to excrete water, as determined by determining solute free water clea rance orserumsodium levels, demonstrates a strong link with regards to survival Hyponatremia patients have a Poor su rvival as compared to those without hyponatremia.[10–13] According to recent research, hyponatremia is a critical prognostic marker in patientswith CL D, and individuals with hypo natremia have a worse surviv al rate than those without

hyponatremia. Hence serum sodium levels have a role in predicting the prognosis and institutional management in patients with cirrhosis of liver.[2,14]

REVIEW OF LITERATURE

Body fluid balance

Humans are predominantly water-based organisms. It is the essence of life and the aqueous base solution in which all of the key metabolic reactions that make life take place. Humans are roughly 75% water by weight as babies and 50% to 60% water by mass as adults. Furthermore, fluid is constantly changing due to a number of regulatory processes that keep optimal concentrations in the body's different compartments. Fluid flow across spaces is generally governed by passive diffusion along concentration gradients of osmotically active solutes; nevertheless, hydrostatic forces can impact fluid movement between spaces.[15]

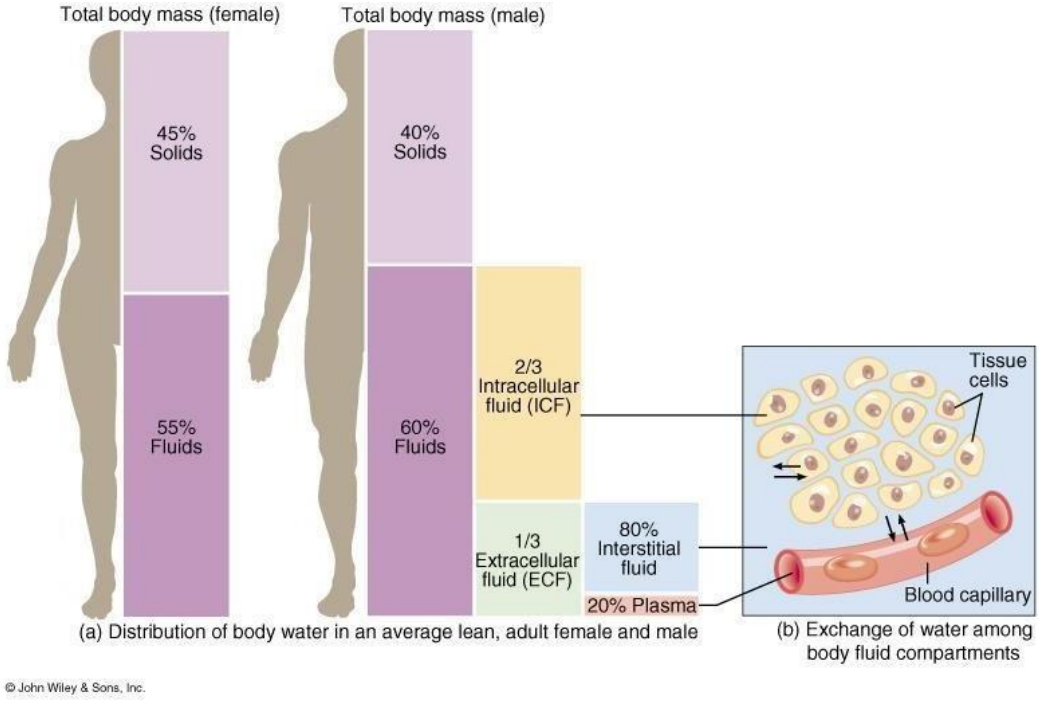

FIGURE 1: FLUID DISTRIBUTION IN THE BODY

Cellular distribution

Fluid distribution in the b ody can be divided into two categories as intracellular fluid and extracellular fluid. Intracellular fluid accounts for around 40% of total body weight. It is the entire space within cells, which isgenerally defined as cell cytoplasm. In general, intracellular fluids are stable and do not respond quickly to changes. Because here is where many chemical processes take place, it is critical to maintain an op timum osmolality. Extracellular fluid accounts for roughly 20% of total body weight and is further subdivided intoplasma, which accounts for approximately 5% of total body weight, and interstitial space, which accounts for approximately 12% of total body weight. In pathological circumstances, additional spaces are classified as transudate or exudate based.

The precise chemical makeup of bodily fluid varies greatly. This is determined by which part of the body, as well as which organ, houses the fluid. The composition of extracellular fluid and interstitial fluid is comparable. Extracellular spaces have large levels of sodium, chloride, bicarbonate, and proteins but low levels of potassium, magnesium, and phosphate. Physiologically, interstitial fluids have a low protein content. Proteins, phosphate, magnesium, and potassium are abundant in intracellular fluids, while sodium, chloride, and bicarbonate are in relatively small amounts.[16–18]

INTRACELLULAR(mEq/L)	SOLUTES	EXTRACELLULAR(mEq/L)
25	Na^+	140
150	K^+	4.5
2	Cl^-	100
6	HCO_3^-	25
15	Mg^+	1.2
0.01	Ca^{2+}	2.4
50	PO_4^{2-}	1.2

FIGURE 2 : COMPOSITIO N OF SOLUTES IN ICF AND ECF

Mechanism[15]

The physiologic osmolarity of blood plasma is around 286 mOsmoles/L. Less than this is hypoosmotic, whereas more than this is hyperosmotic. Active pumping of transmembrane ionic transport proteins maintains cellular osmotic concentration gradients. On the other hand, those components dilate or concentrate when fluid volume changes rapidly. Blood plasma osmotic gradients are maintained constant by gastrointestinal solute absorption, release into the digestive system, or urine.

Proteins and ionic components, such albumin present in serum, can affect osmolarity. Glucose is another crucial osmotically active chemical to consider. Fluid will move away from hypoosmotic compartments and toward hyperosmotic ones. An ionic net should be present in all body fluids.

electrical charge that is about zero, showing that cations and anions are in balance. Based on the presence of permeable membranes, ionic components will diffuse through fluids preferentially. A gradient with a somewhat higher concentration osmolarity occurs from a membrane that is impermeable to an ion. Membrane pumping proteins, which expend energy in the form of ATP to move constituents from low concentration places to higher concentration areas against the diffusion gradient, can create solute gradients physiologically.

These activities generate a cellular environment in which water is osmotically "pulled" into fluid compartments. Fluid mobility throughout the body is dependent on developed and maintained hydrostatic pressures in addition to the osmotic pull of fluids. The capillary membrane is particularly useful for transferring fluid from plasma in the extracellular blood space into tissue interstitial spaces.The "push" influence onfluid flow is hydrostatic pressure, which occurs when increasing pressures drive fluid out of a place. The ne t flowof fluid is caused by the "push" of hydrostaticforces mixed with the "pull" of osmotic

forces. The Starling equation is used to describe this mathematically:

$$Jv = Kfc([Pc - Pi] - n[Op-Oi])$$

Kfc stands for the capillary filtration fluid coefficient, while Jv stands for the capillary fluid flow rate, net. the osmotic reflection coefficient (n), the plasma oncotic pressure,(op) the capillary hydrostatic pressure (Pc), the interstitial hydrostatic pressure (Pi) , interstitial oncotic pressure (Oi) are all abbreviations for pressure..[16]

Clinical significance of fluid balance[16]

Fluid ba lance imbalances are caused by a clinical disease. Fluid balance disorders are characterised by an excess of fluid or a reduction in effective fluid. Edema is the medical term for fluid overflow. Edema is most typically found in the soft tissues of the extremities, but it can occur in any tissue. Dehydration is the term used to describe a decrease in fluid load.

Edema appears as swelling in the soft tissues of the limbs and face, followed by a rise in skin size and stiffness. By pressing a finger into the tissue and quickly creating a dimple in the edematous skin, one can relieve peripheral edoema by increasing the interstitial space's pressure. On the other hand, wearing compression stockings can lessen the symptoms of peripheral edoema by raising interstitial hydrostatic pressure, which forces fluid back into the capillaries.

Excess fl uid expandsinto the interstitial tissues of the lung, causing pulmonary edoema. Shortness of breath and chest discomfort are usual symptoms. Orthopnea, or difficulty breathing w hile lying flat, may happen as the extra fluid spreads throughout the entire lung. Due to the impairment of gas exchange in the lungs caused by pulmonary edoema, the situation might quickly get worse. Cardiac and renal dysfunction are both associated with pulmonary edoema. According to conventional wisdom, heart failure causes pulmonary edoema by lessening the capacity and efficacy of the left atrial and left ventricular pumps. The pulmonary veins experience back pressure as a result, increasing vessel pressure. Then, in accordance with the Starling equation, the As pulmonary capillary hydrostatic pressures rise, fluid is "pushed" into the interstitial lung space. Renal failure prevents the body from properly removing fluids and osmotic components, which results in edoema. As a result, tissues experience higher osmotic draw.[19]

Failure of the liver can also result in edoema. Lack of osmotically active proteins is the cause of this. specifically, the inability to produce albumin. In extracellular blood, albumin is primarily found in the plasma. In the interstitial space, it is unusual to notice it. Therefore, when body albumin is lowered, the "pull" of osmotic pressure into the capillaries is immediately decreased. This results in the fluid migrating into the interstitial spaces, according to Starling forces.[20]

SODIUM AND WATER HOMEOSTASIS

To funct ion and survive, every cell in the body depends on the maintenance of a healthy environment. The tonicity of ECF, which also significantly affects the composition of intracellular fluid, is a crucial

element in the preservation of this milieu. The majority of the physiological processes in our bodies, including neurona l depolarization, myocyte signalling, signalling pathways,etc., depend on the osmolarity of the air is constant. One of the most crucial factors in influencing the extracellular tonicity is the serum sodium content. In order for the cell to function normally, it needs to be closely regulated. Significant variations in serum sodium content are resulting in poor tolerance and cellular damage. Serum sodium is maintained in a restricted range due to a rigorous equilibrium between water output and consumption, despite the fact that there are considerable fluctuations in water consumption, consumed substances, and non-urinary water loss. Urinary tonicity is controlled to maintain this exact equilibrium. To ensure that variations in t otal body water correspond to changes in Na+ and K+, various renal systems carefully regulate the total body water content. Thirst management and urine dilution and concentration control the amount of water that is consumed. Typically, dysnatremias are the outcome. To appreciate the aetiology of dysnatremias with reference to water balance, it is vital to comprehend both the sides of the water balance equation.The body loses approximately 1100 ml of water every day through the skin, respiratory tract surfaces, and faeces. Additionally, water is excreted by the kidneys as part of the solute clearance process. A person consuming a typical diet ought to excrete 500 mOsm of urea and400 mOsm of electrolytes, including the equivalent anions for sodium, potassium, and each (100 m Osm from diet and 400 mOsm from protein ingestion). A normal person can reach a maximum urine concentration of 1200 mOsm. As a result, the kidneys need to excrete at least 750 mL of water in order to eliminate 900 mOsm of solutes.

The water absorbed as liquids, eaten as solid meals, and that produced by metabolism are all considered to be part of the input si de of water balance.. Fixed water consumption from solid food and metabolism is 1200 ml, or roughly the same as fixed non-urinary losses. To balance the system and match the 1850 ml of output, the remaining 650 ml of water must be consumed. As long as the person drinks this specific amount of water, water balance is simple to maintain., Even while the non-renal water losses and intake can vary greatly, the kidney can still keep this balance. This is mostly explained by the kidney's capacity to

adjust the concentration of urine in response to deviations on e ither side of the water balance. If the overall amount of water consumed exceeds the amount necessary for the excretion of solutes, the extra

Urine dilution is used to eliminate free water. The balance is kept when a person drinking a normal amount of water experiences greater water loss due to diarrhoea, increased sweating, etc. Things are maintained in check by the kidneys' concentration of urine and the stimulation of thirst.

SODIUM

Electrolytes are required for fundamental life functions such maintaining electrical neutrality in cells and producing and transmitting action potentials in nerves and muscles. Along with magnesium, calcium, phosphate, and bicarbonates, the major electrolytes are sodium, potassium, and chloride. Electrolytes are derived from our meals and bodily fluids.

These electrolytes can become unbalanced, resulting in either high or low amounts. High or low electrolyte levels disturb normal biological functioning and can lead to potentially fatal consequences.[21]

One of the most essential electrolytes in extra cellular fluid is sodium, an osmotically active cation. It is in charge of controlling extracellularfluid volume as well as regulating cell membrane potential. Active transport involves the exchange of sodium and potassium across cell membranes.

The kid neys regulate the sodium levels. The bulk of sodium reabsorption occurs in the proximal tubule. Sodium is reabsorption in the distal convoluted tubule. Sodium transport occurs via sodium-chloride symporters, which are activated by the hormone aldosterone.[21]

Hyponatremia is the common of the electrolyte d iseases. When the serum sodium level is less than 135 mmol/L, a diagnosis is made. Hyponatremia has neur ological consequences. Patients may experience headaches, disorientation, nausea, and de lirium. Hypernatremia occurs when serum sodium levels exceed 145 mmol/L. Tachypnea, trouble sleeping, and agitation are all symptoms of hypernatremia. Rapid sodium adjustments might result in significant complications such as cerebral edoema and osmotic demyelination syndrome.

The sodium level in the body is closely related to the water balance. Sodium controls the volume of extracellular fluid. The total sodium content of the body is around 4000mEq. About half of it is found in

bones, 40 percent in extracellular fluid, and 10 percent in soft tissues. Sodium is the most abundant cation in extracellular fluid. To keep sodium extracellular, sodium pumps are active in all cells. This method relies on ATP. Sodium (as sodium bicarbonate) is also vital in acid-base balance management.[22]

Normal Na+ levels in plasma are 136–145 mEq/L and 12 mEq/L in cells.

The average diet comprises 5–10 g of sodium, mostly in the form of sodium chloride. Every day, the same quantity of salt is expelled in the urine. However, the body may preserve salt to the point that urine does not contain sodium on a sodium-free diet. Sodium consumption should ideally be lower than potassium intake, yet processed meals increase sodium intake.

Normally, the kidneys are programmed to preserve salt while excretingpotassium. When urine is generated, the initial glomerular filtrate (175 litres per day) includes 800 g of sodium per day, 99 percent of which is reabsorbed. The majority of this (80%) is reabsorbed in the proximal convoluted tubules.This is an ongoing effort. Water, like salt, is reabsorbed facultatively. The distal tubules regulate sodium excretion. In the distal tubules, aldosterone enhances sodium reabsorption. Antidiuretic hormone (ADH) stimulates water reabsorption from tubules.

Hyponatremia :

A plasma sodium content of less than 135mEq/L is considered hyponatremia. When patients are ad mitted to hospitals, especially those in the intensive care unit, hyponatremia usually occurs.. At some time during their stay, 15–30% of patients have been found to have low serum salt concentrations. It typically happens when the kidneys are unable to eliminate a water load or when there is an excessive intake of water.

Dehydration, a reduction in blood pressure, sleepiness, lethargy, disorientation, stomach pains, oliguria, tremors, and coma are among clinical indications and symptoms of hyponatremia. Hyponatremia, on the other hand, is frequently asymptomatic.

In addition to consuming too much free water, hyponatremia typically happens when there is an increase in renal sensitivity to AVP7 or AVP levels. As a result, this condition may present as a rise in TBW, a fall in solute levels, or a combination of the two. Most of the time, several mechanisms are in play. Making sure that pseudohyponatremia or translocational hyponatremia is not the cause of the drop in serum sodium is the first step in evaluating a patient with hyponatremia.

Pseudo-hyponatremia:

Hyponatremia that is isotonic or normo-osmolal is another name for it. In diseases like multiple myeloma, it is caused by an increase in plasma proteins or triglycerides. Proteins and lipids make up the remaining 7% of the plasma volume in healthy humans, leaving plasma water to make up 93% of it. In hypertriglyceridemia or hyperproteinemia, plasma water makes up just 80% of the total volume of the plasma. Plasma water sodium concentration and osmolality are unaltered. The sample's lower plasma water concentration, however, causes the sodium concentration to be measured to be lower.

Translocational Hyponatremia:

Hypertonic or redistributive hyponatremia are other names for it. It results from the presence of osmotically active solutes, such as glucose or mannitol. The determination of an accurate plasma osmolality warranting direct measurement is complicated by the presence of these unmeasured solutes in plasma in considerable levels.

True hyponatremia (hypoosmolal hyponatremia) is categorised because total body sodium levels may be low, normal, or high in association with it.

• Hypovolemic hyponatremia, which is hyponatremia with a decrease in ECF volume.

• Hypervolemic hyponatremia, which is hyponatrem ia with an excess of ECF volume.

• Euvolemic hyponatremia, is with a normal ECF volume

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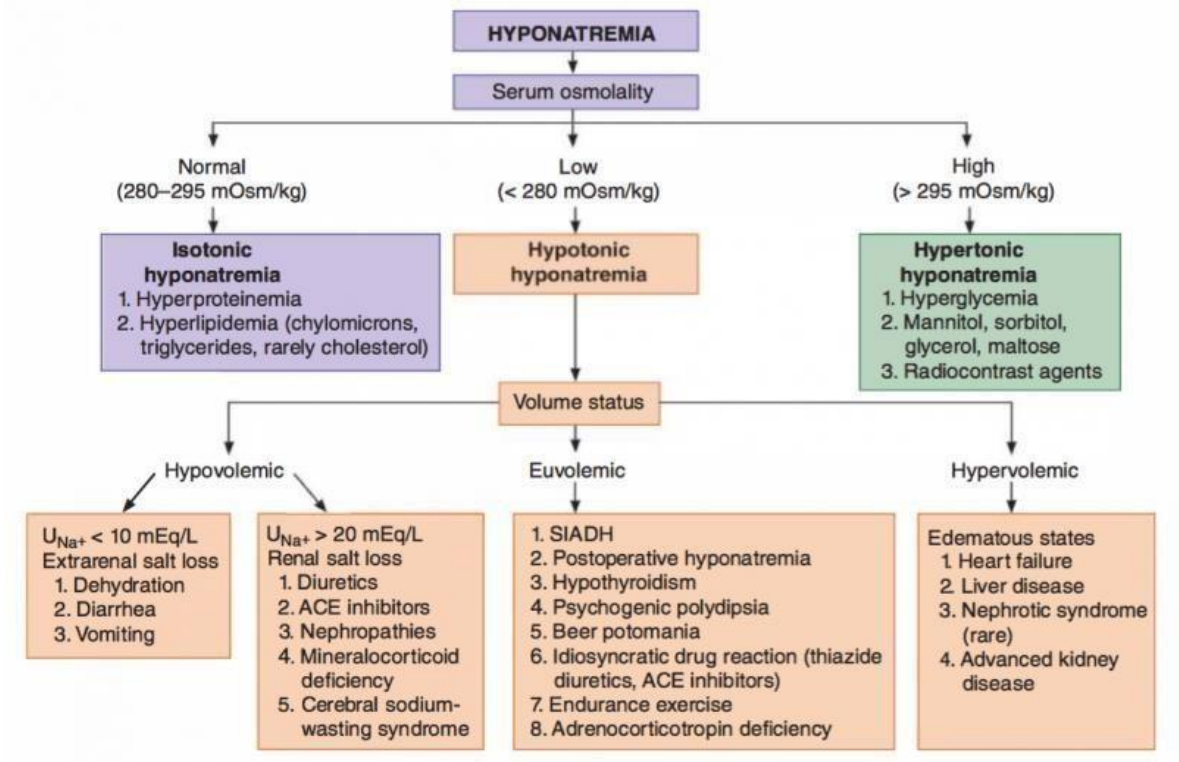


FIGURE 3: CAUSES OF HYPONATREMIA

HYPOVOLEMIC HYPONATREMIA:

It occurs when there is an imbalance between the total amount of sodium in the body and the total amount of water in the body. The fall in blood pressure and reduction in intravascular volume cause the release of AVP. The non-osmotic stimulation prevails the "Law of the Circulating Volume"-described regulation of Osmoreceptors that recognise hypoosmolality release AVP.It claims that maintaining tonicity comes last and that blood volume preservation and blood pressure maintenance come first.

In order to regulate blood pressure and boost water absorption, respectively, AVP operates on the V1Areceptors which are present in the blood vessels and the V2 receptors in the kidney. Increased intake of free water can cause hyponatremia if the V2 receptor is activated.

HYPERVOLEMIC HYPONATREMIA:

Both the amount of sodium and water in the body are elevated in this disease, but the water retention is out of proportion to the sodium retention. The severity of hyponatremia is hypothesised to function as both a predictive sign of the underlying disease and a marker of severity in diseases like cirrhosis and heart failure that cause edematous states The hormonal and intra-renal alterations brought on by reduced Effective Arterial Blood Volume(EABV) are thought to be the mechanism by which hyponatremia develops in these conditions.

Patients with advanced congestive heart failure (CHF) frequently exhibit hyponatremia. It is a sign of a bad prognosis. The left side of the arteries are underfilled as a result of congestion and diminished production. The l eft ventricle, aortic arch, carotid sinus, and afferent arterioles in the kidney all have mechanoreceptors that sense this. These receptors become active, increasing sympathetic outflow, the renin-angiotensin-

aldosterone system becoming active, the GFR decreasing, and the proximal tubular reabsorption increasing, As a result, the medullary interstitial tonicity gradient is insufficient and the distal nephron receives less water, which affects free water clearance. Non-osmotic AVP is released as a result of mechanoreceptor activation, which also increases thirst. AVP release and an increase in thirst-related free water consumption. Another illness that can cause hypervolemic hyponatremia is cirrhosis. Low EABV is caused by splanchnic and peripheral circulation vasodilation. Similar to CHF, this low EABV causes hyponatremia.

Nephrotic syndrome has also been linked to hypervolemic hyponatremia, but this association is sporadic and unrelated to the severity of the condition. Patients with chronic or acute renal failure have also shown hypervolemic hyponatremia.. Hyponatremia is 15% more common in people with chronic kidney disease (CKD), according to recent studies.

EUVOLEMIC HYPONATREMIA:

This occurs when the total body water content rises without the total body sodium content changing. Osmotic or non-osmotic factors have no control over or reason to stimulate the release of AVP. which is why this kind of hyponatremia is most frequently seen in hospitalised patients. The most common cause of euvolemic hyponatremia is syndrome of inappropriate anti-diuretic hormone (SIADH). Being the most Typical causes of hyponatremia.

HYPERNATREMIA :

Hypernatremia, which affects 1% of hospitalised patients44 and 7% of those admitted to intensive care units, is defin ed as a blood sodium concentration of more than 145 mEq/L. Up to 40 percent mortality has been noted in patients with hypernatremia, despite the fact that this is a rare occurrence.

PATHOPHYSIOLOGY:

When there is an increase of Na in ratio to water in ECF, the water balance becomes out of equilibrium, leading to hypernatremia. It is primarily caused by a water deficiency and occasionally by salt overload. Insufficient intake or excessive water loss cause a water deficit. Patients who have access to fluid intake and a sufficient thirst response can adapt to these modifications.

The osmotic load rises as a result of more salt in the ECF, and water loss from inside the cells offsets this. Du e to dehydration, this cause the cells to shrink. By moving solutes across the cell membrane in response to this shrinking, the cells change the electrically active cell membrane's resting potentials. Organic solutes prevent structural deterioration and replenish cell volume.

The CNS is where the impacts of cellular shrinkage and dehydration with changed membrane potentials are most prominent, leading to inefficient functioning. Severe neuronal atrophy can stretch bridging veins and perhaps lead to their rupture.

The carer is responsible for giving water to patients who are infants, the old, or who are disabled. They have a higher risk of hypernatremia. Osmoreceptor defects in the hypothalamus, whether congenital or acquired, lead to essential or adipic

hypernatremia. Hypernatremia and hypovolemia are the effects of this as it leads to dysregulation in the release of vasopressin and the thirst response.

Causes of Hypernatremia

Hypernatremia is defined as an increase in blood sodium levels. Hypernatremia symptoms include dry mucous membranes, fever, thirst, and agitation. Cushing's illness, extended cortisol treatment, and pregnancy, when steroid hormones increase salt accumulation in the body, are all causes of hypernatremia.

Prolonged cortisone therapy

Cushing’s disease

Pregnancy

Sodium retention due to steroid hormones

Dehydration

Exchange transfusion with stored blood

Primary hyperaldosteronism

Excessive intake of salt

Elderly patients with poor water intake

Drugs (Ampicillin, tetracycline, oral contraceptives, anabolic steroids, osmotic diuretics)

CIRRHOSIS OF LIVER[23,24]

Chronic liver disease (CLD) is defined as a gradual degradation of liver functions lasting more than six months, including clotting factor production, other protein synthesis, detoxification of toxic metabolic products, and bile excretion. CLD is a continual process of theliver parenchymal inflammation, destruction, and regenerationthat leads to fibrosis and cirrhosis. Chronic liver disease has a wide range of etiologies, including toxins, long-term alcohol addiction, infection and autoimmune illnesses, genetic and metabolic problems. Cirrhosis is the laststage of chronic liver disease, characterised by disruption of hepatic architecture, the creation of extensivenodules, vascular rearrangement, neo-angiogenesis, and extracellularmatrix deposition.

Etiology

Cirrhosisis frequently the result of chronic liver disease. Cirrhosis is most commonly caused by hepatitisC virus (HCV), alcoholic liver disease, and nonalcoholic steatohepatitis (NASH) in the industrialised world, whereas hepatitis B virus (HBV) and HCV are the most prevalent causes in the poor world. Cirrhosis can also be caused by other conditions such as autoimmune hepatitis, primary biliarycholangitis, primary sclerosing cholangitis, hemochromatosis, Wilson disease, alpha-1 antitrypsin deficiency, Budd- Chiari syndrome, drug-induced liver cirrhosis, and persistent right-sided heart failure. Cirrhosis of unknown cause is referred to as cryptogenic cirrhosis.[25]

The fo llowing are the common etiologies

Cirrhosis, alcohol hepatitis (reversible due to acute drinking), and alcoholic fatty liver with or without hepatitis are all part of the spectrum of disorders known as alcoholic liver disease.

(irreversible).Chronic liver disease is the most common cause of CLD in patients with severe alcohol use disorder.

Non-alcoholic fatty liver disease (NAFLD/NASH): NAFLD has been linked to metabolic syndrome (obesity, hyperlipidemia, and diabetes mellitus). Some of these individuals develop non-alcoholic steatohepatitis, which results in liver fibrosis. All of the metabolic syndrome risk factors can exacerbate the illness process.

Chronic viral hepatitis: In East Asia and Sub-Saharan Africa, The main causes of chronic liver disease include chronic hepatitis B, C, and D infections. Hepatitis C has several genotypes. Genotypes 1a and 1b are more frequent in Europe and North America, while genotype 3 is most common in Southeast Asia. In the Sharkia governorate of Egypt, a molecularstudy discovered a considerable prevalence of HCV genoty pe 4, subtype 4a in Egyptian patients. Chronic hepatitis C can develop into hepatocellular carcinoma if left untreated.

Genetic causes include alpha-1-antitrypsin deficiency, hereditary hermochromatosis, Wilson disease.

Autoimmune cause: Autoimmune hepatitis is an uncommon condition in which autoantibodies destroy the liver tissue. The majority of individuals who appear with this condition have cirrhosis. Females are more likely to be afflicted than males. Primary biliary cirrhosis (PBC), primary sclerosing cholangitis (PSC), autoimmune hepatitis (AIH).

Epidemiology

Cirrhosis has an unknown global frequency; however, it is believed to be between 0.15 and 0.27 percent in the

United States.[26,27] CLD is a leading cause of mortality, mainly in underdeveloped countries. In recent years, there has been an increase in the prevalence of chronic liver disease. In the industrialised world, HepatitisB, C, non-alcoholic fatty liver disease (NAFLD), hemochromatosis, and alcoholic liver disease are the mojor prevalent chronic liver diseases.[24]

Pathophysiology

Cirrhosis of the liver is primarily caused by hepatocytes and the sinusoidal lining's Kupffer cells, sinusoidal endothelial cells, and hepatic stellate cells (KCs). HSCs are the cells that line the sinusoids in the liver and store vitamin A. Inflammatory cytokines excite these cells, which then trans form into myo fibroblasts and start depositing collagen, causing fibrosis. The fenestrations that SECs m ake in the wall, which allow for fluid and nutrient exchange between sinusoids and hepatocytes, serve as the endothelium lining's defining characteristic.[28]

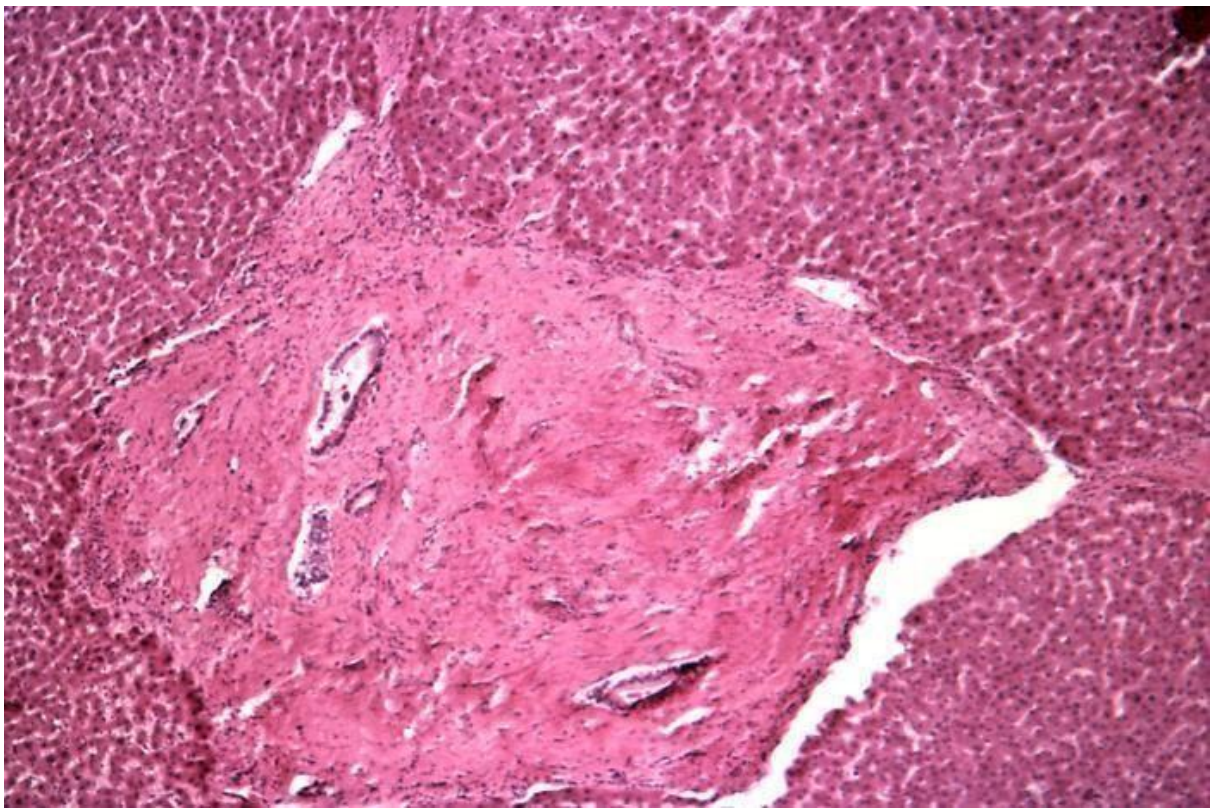


FIGURE 4: PHOTOGRAPH OF A LIVER TISSUE SPECIMEN SHOWING THE PIPE STEM CIRRHOSIS

Chronic alcohol consumption can cause defenestration of the sinusoidal wall, which promotes perisinusoidal f ibrosis. KCs are satellite macrophages that border the sinusoidal walls as well. Animal studies have demonstrated that they contribute to liv er fibrosis by generating damaging me diatorswhen exposed to hazardous substances and serving asantigen-presenting cells for viruses. The aetiology of cirrhosis is caused by the release of rea ctive oxygen species and inflammatory mediators by damaged hepatocytes, which can help in activating HSCs and liver fibrosis.[29,30]

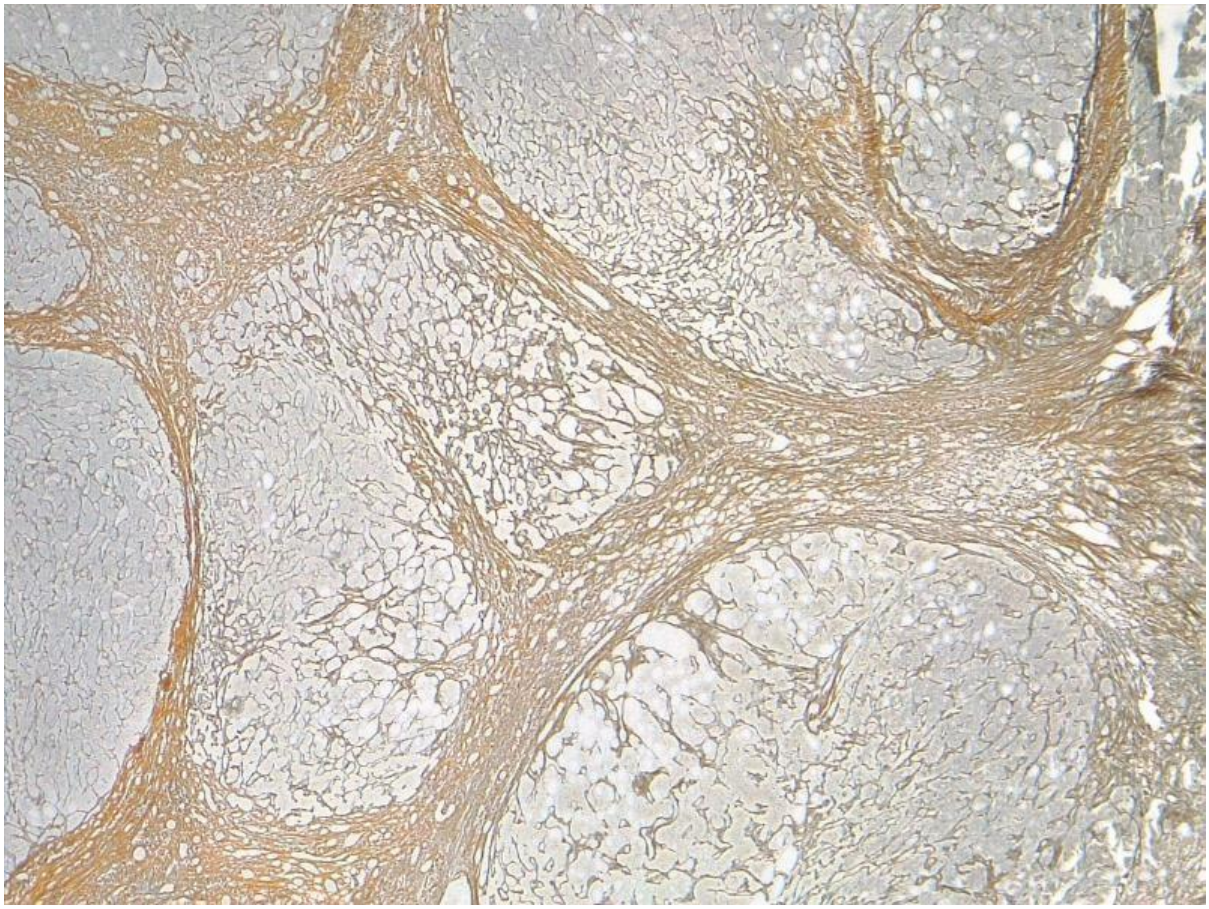


FIGURE 5: CIRRHOSISOF LIVER. RETICULIN STAIN ENHANCES THE FIBROUS SEPTA DIVIDING THE HEPATIC NODULES.

Extracellular matrix (ECM) is deposited in the liver as a result of ongoing liver injury from any source, resulting in hepatic fibrosis. The commo n route is initiated by hepatic stellate cells (HSC), latent cells that store vitamin A and are located between sinusoids and hepatocytes. Due to persistent liver

In response to damage, HSC transform into proliferative fibrogenic myofibroblasts, which increase the express ion of inflammatory receptors such as chemokine receptors, ICAM-1, and other inflammatory mediators by secreting chemokines and other leukocyte chemoattractants.. In addition to altering the gene and phenotypic expression of liver cells, this pro-inflammatory or first phase also makes them m ore sensitive to these inflammatory cytokines, and the persistence of activated HSC cells results in ECM formation and progressive fibrosis.23

Histopathology

Classification of Morphology

Cirrhosis is classified as micronodular, macronodular, or mixed. This categorization is not aseffective in clinical practise as etiologic classification.

Cirrhosis caused by a lcohol, hemochromatosis, hep atic venousoutflow blockage, chronic biliary obstruction and jejuno ileal by pass and Indianchildhood cirrhosis are all examples of micronodular cirrhosis.

Macronodular cirrhosis is the outcome of cirrhosis brought on by hepatitis B, C, alpha-1 antitrypsin deficiency, and primary biliary cholangitis (irregular nodules with a diameter fluctuation more than 3 mm).

Mixed cirrhosis: Micronodula r cirrhosis often progresses to macronodular cirrhosis over time. This condition

occurs when both micronodul ar and macronodular cirrhosis features are present.

Etiological classification

Based on the causecirrhosis, the sub-classification is derived

Viral – hepatitis B and C and D , Toxins – alcohols, drugs

Autoimmune are autoimmune hepatitis

Cholestatic are primary biliary cholangitis, primary sclerosing cholangitis

Metabolic – NASH, Wilson disease, haemochromatosis, cryptogenic cirrhosis and alpha1-antitrypsin.

Vascular - Budd-ChiariSyndrome, cardiac cirrhosis, Sinusoidal obstruction syndrome

Complications

The complications associated with cirrhosis include

Jaundice

Portal hypertension

Edema

Splenomegaly

Infections

Hemorrhage

Hepatic enxephalopathy

Differential diagnosis

Acetaminophen poisoning

Fructose intolerance

Acute fattyliver of pregnancy

Amanita phalloides mushroom poisoning

Galactosemia

HELLP syndrome of pregnancy

Drug reaction

Hemorrhagic viruses

Tyrosinemia

Neonatal iron storage disease

Evaluation[23,24]

The aetiology and consequences of chronic liver disease influence the diagnosis. A summary of the diagnosis for different CLD is provided below.

Alcohol-related liver disease: Elevated AST more than ALT values with a history of chronic alcohol use. Inalcoholic liver disease, the AST: ALT ratio is usually 2 to 1.

Serology , PCR (quantitative and qualitative) with genotyping for viral hepatitis B and C

Hemochromatosis is characterised by elevated blood iron, ferritin, reduced TIBC, and a liver biopsy. A mutation in the HFE gene, specifically C282Y, can be detected by genetic testing.

Nonalcoholic fatty liver disease: exclusion diagnosisand ALT more than AST Ultrasonography of the liveris useful.

Wilson disease is characterised by increased urine copper, reduced serum ceruloplasmin, and a liver biopsy . The ATP7B gene was tested genetically.

Hepatitis with autoimmunity: Alpha 1 antitrypsin insufficiency due to elevated ANA, ASMA, and LKM-1 Alpha one antitrypsin levels are low.

Cirrhosis of the bile duct: An antimitochondrial antibody significantly increased alkaline phosphatase levels.

Budd-Chiari and veno-occlusive disease: complete blood count (CBC), clotting profile, and imaging procedures such as ultrasonography doppler or computed tomography with contrast.

Specific lab testing to identify newlydiagnosed cirrhosis

Serology and PCR techniques for viral hepatitis may also be ordered in addition to autoimmune antibodieos (antinuclear an tibodies [ANA], anti-smoothmuscle antibodies (ASMA), anti-liver-kidney microsomal antibodies type 1 (ALKM-1) and serum IgG immunoglobulins) for autoimmune hepatitis and anti-mitochondrial antibody for primary biliary cholangitis). Other important examinations include the following: ferritinand transferrin saturation for hemochromatosis; ceruloplasmin and urine copper for Wilson disease; serum alpha-fetoprotein for hepatocellular carcinoma; alpha1-antitr ypsin level and protease inhibitor phenotype for alpha 1-antitry psin deficiency (HCC).

Imaging and liver biopsy

In addition to laboratory tests, several imagin g modalities are used to help diagnose cirrhosis. These consist of transient elastography, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound (fibroscan).

Ultrasonography is a frequently used, inexpensive, non-invasive test for cirrhosis. Although it ca n

identifynodularity and increased echogenicity of the liver, both of which are signs of cirrhosis, it is not specific because the same signs can also be found in fatty liver. The right lobe to caudate lobe width ratio, which typically rises in cirrhosis, can also be determined. It also functions well as a screening tool, for HCC in those with cirrhosis. Hepatic, portal, and mesenteric vein health is assessed using duplex doppler ultrasound.31,32

Transient elastography, also referred to as fibroscan, measures liver stiffness, which is associated with fibrosis, using high-velocity ultrasonic pulses. A technetium-99m sulphur colloid scan of the liver and spleen during cirrhosis may reveal that the bone marrow and spleen absorb more colloid than the liver. Varices on esophagogastroduodenoscopy (EGD) of the oesophagus or stomach are a sign of portal hypertension..

A liver biopsy is the gold standard for identifying the stage and grade of fibrosis as well as the presence of cirrhosis. However, sample faults could occasionally prevent it from making the correct diagnosis. For diagnosis of cirrhosis by biopsy, fibrosis and nodules must be present. Higher hepa tic venous pressure gradient (HVPG) and most serious sickness are independent risk factors for the nodul ar pattern, which can be m icronodular, macronodular, or mixed..[33]

Noninvasive diagnostics that employ direct and indirect blood indicators to distinguish individuals with substantial fibrosis/cirrhosis from those with no/mild fibrosis are used.[34–36]

Sodium disorder in cirrhosis of liver

The most frequent electrolyte imbalance detected in hospitalised patients is hyponatremia.[37] Cirrhosis Currently, a blood sodiu m level of less than 130 meq/L is considered hyponatremia.38According to one study, the prevalence of blood sodium concentrations below 135, 130, and 120 meq/L in cirrhotic and ascites patients was 49.4%, 21.6 percent, and 1.2 percent. Cirrhotic patients may develop hyponatremia as a result of hypovolemia (for example, diuretic-induced extracellular fluid loss) orhypervolemia (increased

extracellular fluid volume due to the inability of the kidneys to eliminate solute-free water according to the amount of free water consumed) (exp anded extracellularfluid volume due to the inability of the kidneys to excrete solute-free water proportionate to the amount of free water ingested).[39] Hyponatremia is common in individuals with ascites caused by severe cirrhosis and portal hypertension. Ascites develops in cirrhotic individuals for a variety of reasons. The sodiu m-retaining neurohumoral systems, including the renin-angiotensinaldosterone s ystem, sympathetic nervous system, and antidiuretic hormone, are activated by portal hypertension and the concomitant systemic vasodilation (ADH). As a result of the ardent r etention of salt, water to compensate for the decreased effective circulation volume, ascites develops. Although it is not visible in the early s tages of cirrhosis, the advancement of cirrhosis and ascites causes renal impairment in the elimination of solute-free water.[39] This causes additional compensatory mechanisms, such as non-osmotic ADH secretion, also known as arginine vasopressin, aggravating excess water retention and so hyponatremia. In patients with cirrhosis, hyponatremia is related with increased morbidity and mortality, and it is an importan t prognostic sign both before and after liver transplant. The management of hyponatremia in this environment is difficult since typical hyponatremia therapy, such as fluid restriction and loop diuretics, is usually ineffective.

Pathogenesis[39]

Systemic vaso dilation and arterialunderfilling have a substantial influence in the development of hyponatremia in patients with cirrhosis, portal hypertension. A frequent cardiovascular physiological expression of individuals with cirrhosi s and severe portal hypertension is a hyperdynamic circulation, which is characterised by enhanced car diac output, significantly decreased systemic vascular resistance, and decreased mean arterial pressure.[40,41]

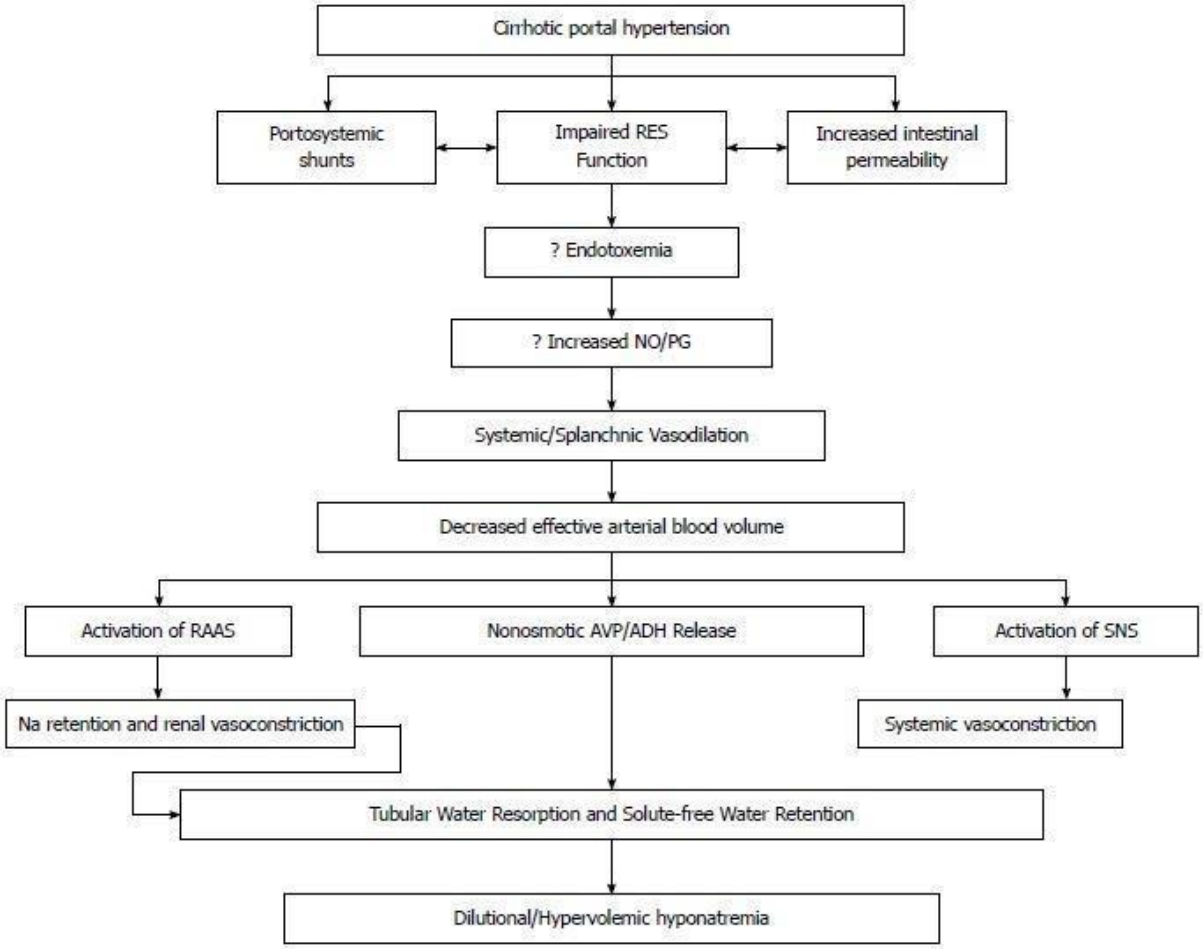


FIGURE 6: MECHANISM OF DEVELOPMENT OF HYPONATREMIA[39]

The splanchnic arterial circulation is primarily involved in the significant decrease in vascular resistance.[42] The formation of portasystemic collaterals[40] and an increase in the synthesis of circulating vasodilators such as nitric oxide (NO), glucagon, vasoactive intestinal peptide, substance P, platelet activating factor, prostaglandins, and prostacyclins all play important roles in the pathogenesis of splanchnic vasodilation.

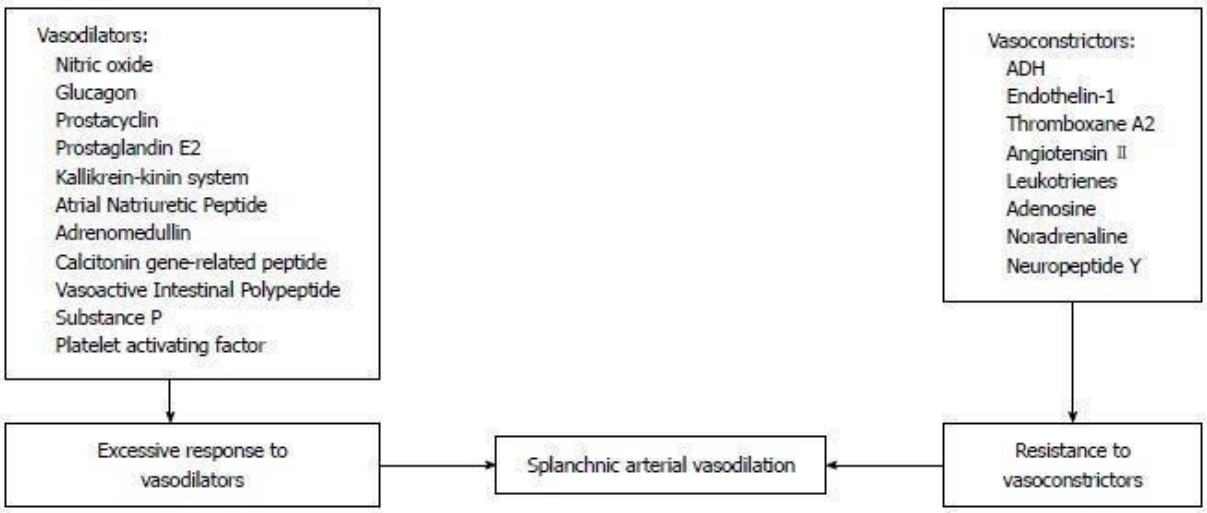


FIGURE 7: MECHANISM OF SPLANCHNIC VASODILATION IN CIRRHOSIS[39]

Nitric oxide synthase is activated in endothelial cells by mechanical stimuli from "shear stress," vascula r endothelial growth factors, tumour necrosis factor alpha, and, most importantly, endotoxins or bacterial DNA that are less efficiently cleared from the gastrointestinal tract because of portal systemic shunting and impaired reticuloendothelial cell function in cirrhosis.39

Oth er vasoactive pathways, including as angiotensin-II, norepinephrine, vasopressin, and enhanced sympathetic tone, may be up-regulated when one of the vasoact ive mediators, such as NO or prostacyclin, is blocked, impeding the correction of splanchnic vasodilation. Because of the complicated connection between these v asoactive systems, no one factor to be fully responsible for the splanchnic vasodilation found in portal hypertension patients. This might explain why discovering pharmacological medicines to inhibit splanchnic vasodilation has proven challenging.

Role of antidiuretic hormone in water balance

Increased in water intake (typically 1.5–3 L/d; ranges from 0.5–20 L/d under extreme conditions) is followed by an increase in renal solute-free water excretion, and a decrease in water intake is followed by a decrease in free

water excretion. Total body water and osmolality are kept within normal ranges. Because antidiuretic hormone is produced primarily at the hypothalamus level, blood osmolality (and subsequently serum sodium) are tightly regulated (ADH). ADH production increases or decreases in response to changes in serum osmolality. The kidneys are in an antidiuresis state under normal physiological conditions, within 24-hour urine osmolality higher than plasma osmolality. [43]

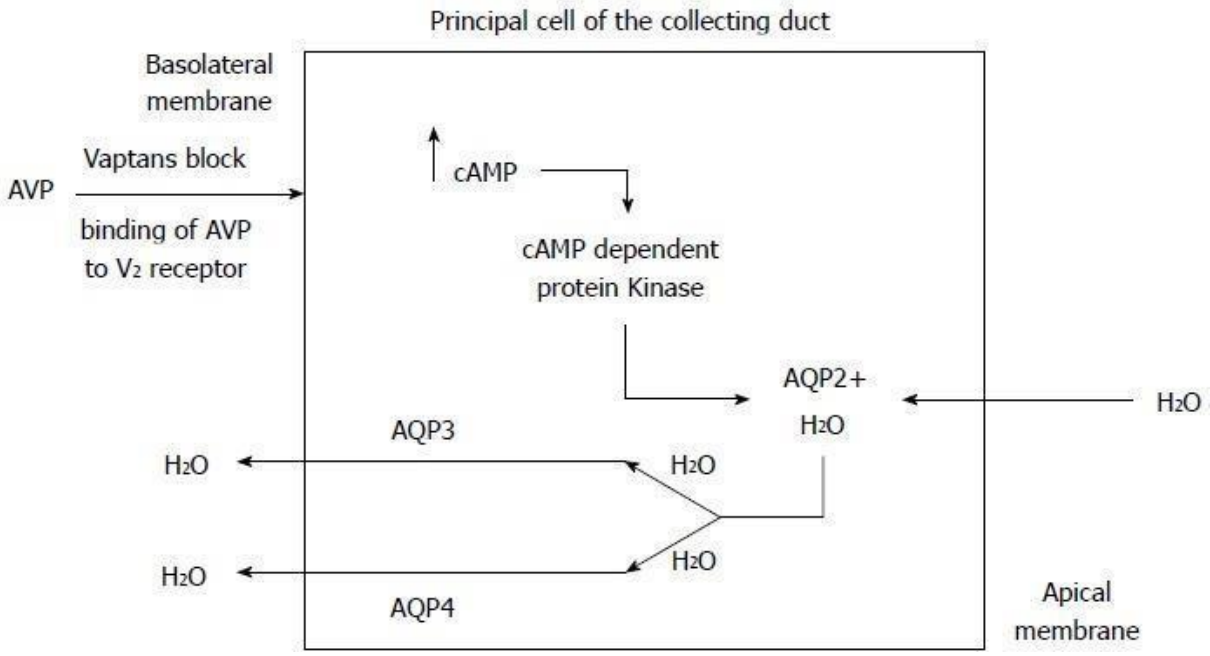


FIGURE 8: MECHANISM OF ACTION OF VAPTANS[39]

Progno stic value of hyponatremia in cirrhosis[39]

Hyponatremia in people without cirrhosis can cause a variety of s ymptoms, including moderate cognitive impairment, falls, seizures, coma, and, in rare cases, death.[44] Cirrhosis hyponatremia is a chronic condition that permits the bra in to adjust to the hypo-osmolality of the extracellular fluid. The sharpness of blood sodium decline, rather than the absolute drop of serum sodium, is the most critical determinant in defining the severity of neurologic symptoms in hyponatremia patients. As a result, individuals with cirrhosis and hyponatremia had a lower risk of developing severe neurologic symptoms. However, hyponatremia, in

addition to the a strocyte abnormality induced by elevated intracellular glutamine concentration from ammonia metabolism, may provide a sec ond osmotic blow to cerebral edoema and astrocyte swelling, precipitating hepatic encephalopathy.

Due to the need for rigorous fluid restriction, people with cirrhosis and hyponatremia have a low quality of life. Hyponatremia has been reported to be an independent predictor of poor health-related quality of life and hep atic encephalopathy. many studies have found that the degree of hyponatremia and ascites is a critical factor of cirrhosis severity and prognosis. The bloo d sodium levels before the beginning of spontaneous bacterial peritonitis (SBP) was found to be an independent renal failure predictor caused by SBP in one research.[45,46]

Although hyponatremia puts patients at a greater risk of dev eloping hepatorenal syndrome, low blood sodium in hepatorenal syndrome is caused by a combination of high ADH levels, reduced GFR, and proxim al sodium reabsorption.[47]

Patients with hyponatremia had a greater chance of dying before transplantation, regardless of cirrhosis severity as measured by MELD scores.[48] As a result, some researchers have proposed for a'sickest first' approach of liver transplantation in cir rhotic patients with MELD scores less than 21, chronic ascites, and hyponatremia. It has been proposed that serum sodium be integrated into the MELD score, which may offer a more accurate forecast of survival than MELD alone. Hyponatremia has also been reported as risk factor for increased morbidity and death following liver transplantation in other investigations.[49–51]

Various articles discussing sodium changes in patients with liver cirrhosis.

In a observational study by Qureshi MO et al., to assess the correlation between the hyponatremia with hepatic encephalopathy and severity of liver disease among the patients. 62 (30.7 percent) of the 202 patie nts had ser umsodium levels <130 meq/l. HE was found in 69 (34.15 percent) of the 202 patients, with 38 having gra de III-IV H E and 31 hav ing gra de I - II H E

57 of 69 HE patients had salt levels less than 135 ($p<0.001$). Hyponatremia was a prevalent symptom in cirrhotic individuals, and its severity increased with the severity of the liver disease. When compared to patients with blood sodium concentrations > 135 mmol/L, individuals with serum sodium concentrations <135 mmol/L had a higher prevalence of hepatic encephalopathy.[52]

In a study by Leise M et al., to assess the hyponatremia in cirrhosis of liver and implication or liver transplantation. The utility of arginine vasopressin antagonists ("vaptans") is debatable, however they may have a limited purpose. Increased usage of packed red blo od cell and fresh frozen plasma transfusions, which are frequently necessary, is a risk factor for intraoperative sodium overcorrection. More data on the use of sodium-reduced continuous venovenous hemofiltration and the use of trishydroxymethylaminomethane (Tris) to avoid excess sodium rebound are needed as intraoperative therapy evolves. This article provides a full explanation of the current therapeutic options before to and during LT.[53]

In a study by Angeli P et al., to study prevalence of hyponatremia in patients with cirrhosis. Study says that low serum sodium levels in cirrhosis are associated with severe ascites and high frequency of hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome. The prevalence of low blood sodium concentration was 49.4 percent, 21.6 percent, 5.7 percent, and 1.2 percent, respectively, for serum sodium concentrations of 135 mmol/L, 130 m mol/L, 125 mm ol/L, and 120 mm ol/L. Low blood sodium levels (135 mmol/L) were common in both inpatients and outpatients (57 percent and 40 percent , respectively). Blood sodium levels of 135 m mol/L were related with severe as cites, as evidenced by a high prevalence of refractory ascites, a high rate of fluid buildup, frequent use of large-volume paracentesis, and reduced renal function when compared to normal serum sodium levels. Furthermore, l ow blo od sodium levels were linked to an increased risk of hepa tic enceph alopathy, sp ontaneous bacterial peritonitis, and hepatorenal syndrome, but not gastrointestinal bleeding. Patients with blood sodium levels of <130

mmol/L had the highest incidence of these problems, whereas patients with minor reductions in serum sodium levels (131-135 mmol/L) also had a higher frequency. Finally, in cirrhosis, low blood sodium levels are associated with severe ascites and a high prevalence of hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome.[13]

In a study conducted by Jang CM et al., to assess the hyponatremia in patients with liver cirrhosis. study say that hyponatremia is commonly observed complication that is related to hypoalbuminemia and portal hypertension in patients who have advanced liver cirrhosis. Complications in cirrhotic individuals with hyponatremia are greatly increased, including spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatic encephalopathy. Furthermore, hyponatremia is related with increased morbidity and mortality in cirrhotic patients, and it is an important prognostic factor both before and after liver transplantation. Traditional hyponatremia treatments include albumin infusion, hydration restriction, and loop diuretics, although they are generally ineffectual. This article looks at the pathogenesis and several treatment options for hyponatremia in individuals with liver cirrhosis, including selective vasopressin receptor antagonists.[54]

In a study by Younas A et al., to assess the hyponatremia and its relation with hepatic encephalopathy and severity of the disease. The individuals' blood sodium levels varied from 115 to 142 meq/L, with a mean of 129.116.53 meq/L. It ranged from 115 to 127 meq/L in hyponatremia patients (mean 121.41 5.17 meq/L). Hyponatremia was found in 96 (36.9%) of the patients. There were 51 (53.12 percent) males and 45 (46.8 percent) females among them; 24 (9.2 percent) had mild hyponatremia, 56 (21.5 percent) had moderate, and 16 (6.2 percent) had severe hyponatremia. HE was found in 176 (67.7 percent) of the patients. HE grade I was found in 54 (20.8%) individuals, grade II in 62 (23.8%), grade III in 32 (12.3%), and grade IV in 28 (10.8%). HE was observed in 84 of 96 hyponatremia patients (p-value: 0.001). According to our findings, cirrhotic individuals with chronic hepatitis infections have varying levels of low sodium. Sodium levels of <130 meq/L were linked to increased morbidity and death. Furthermore, patients with lower salt levels had greater grades of HE.[2]

TABLE 1: SHOWING COMPLICATIONS OF DECOMPENSATED CHRONIC LIVER DISEASE

1. PORTAL HYPERTENSION • Gastroesophageal varices • Portal hypertensive gastropathy • Spleenomegaly • Ascites, Spontaneous Bacterial Peritonitis 2. HEPATIC ENCEPHALOPATHY 3. HEPATORENAL SYNDROME- TYPE I and II	4. HEPATOPULMONARY SYNDROME 5. PORTOPULMONARY HYPERTENSION 6. MALNUTRITION 7. COAGULOPATHY • FACTOR DEFICIENCY • THROMBOCYTOPENIA • FIBRINOLYSIS 8. HEMATOLOGIC ABNORMALITIES 9. BONE DISEASE

PORTAL HYPERTENSION(PH)

If the hepa tic venous pressure gradient is more than 5 mmHg, portal hypertension is said to be present. The following mechanisms contribute to its cause:

1. Cirrhotic alterations that make it difficult for blood to move through the liver

2. Splanchnic vascular bed dilatation leading to increased splanchnic blood flow

The two most frequent side effects of chronic liver illness, ascites and variceal bleeding, are caused by portal hypertension. Because there is less generation of nitric oxide (NO), endothe lial dysfunction develops in the hepatic circulation. Additionally, Nitric oxide is depleted as a result of cirrhosis' oxidative stress. Relative hypovolemia caused by increased splanchnic blood flow produces renal vasoconstriction and stimulates the renin-angiotensin system. This causes salt and water to be retained. Through portosystemic collaterals, the portal system's increased blood flow is diverted.

ASCITES:

In patients with decompensation, it is a frequent presentation. Ascites is thought to form as a result of increa sed splanchnic blood flow, decreased renal blood flow, activationsof the renin-angiotensin system, and sodium and water retention. Initial increases in cardiac output are caused by a decrease in effective arterial blood volume. Systemic hypotension, which develops as the condition worsens, triggers the release of numerous vasoconstr ictor and anti-natriuretic hormones in an effort to preserve homeostasis. Hypoalbuminemia lowers oncotic pressure and causes fluid to flow into the peritoneal cavity through capillaries.

As a resul t of vasoconstriction and ADH activation brought on by perceived hypovolemia, dilutional hyponatremia results, which reduces renal excretion of free water .

SPONTANE OUS BACTERIAL PERITONITIS(SBP):

It is a c ommon and serious ascites complication. The ascitic fluid spontaneously becomes infected, which is the cause. The development of SBP has been attributed to bacterial translocation. Bacteremia and the seeding of ascitic fluid are thought to result from gut flora travelling through the colon and reaching mesenteric lymph nodes. It can happen in up to 30 percent of patients with ascites severe enough to warrant hospitalisation and a 25% death rate. A neutrophil count of more than 250 in ascitic fluid serves as the diagnostic threshold. Fever, altered mental status**,** stomach pain, and an increased white blood cell count are their initial symptoms.

HEPATORENAL SYNDROME(HRS):

Hepatorenal syndrome refers to functional renal failure without renal disease in cirrhotic patients. The development of HRS is assumed to be caused by noticeable changes in renal circulation, manifested as increased vascular resistance in the kidneys and lower systemic vascular resistance. The precise cause of renal

vasoconstriction is unknown and presumed to be complex. Patients with significant ascites and a gradual increase in serum creatinine are diagnosed with HRS. 10% of cirrhotic patients have one of two types of HRS.

When Type I HRS occurs, serum creatinine doubles in just two weeks, and renal function rapidly deteriorates. Acute occurrences like SBP frequently serve as its catalyst. The median time to death without medical intervention

HEPATIC ENCEPHALOPATHY:

Clinical presentation can vary, however variables including infection, electrolyte imbalance, increased dietary protein load, etc. can cause encephalopathy in people with liver disease. Hepatic encephalopathy is the term used to describe the al teration in cognitive function in people with liver failure. It is an extremely dangerous side effect of chronic liver disease. Vascular shunting and a diminished hepatic bulk prevent the liver from excreting neurotoxins that are derived from the gut. Hepatic encephalopathy is brought on by their buildup in the brain. Although hepatic encephalopathy frequently has high ammonia levels, there is little connection betw een ammonia levels and the severity of liver illness. Other drugs linked to hepatic encephalopathy include mercaptans and a few fake neurotransmitters. from a lack of focus to a coma. Brain disease can range from herniation to edoema.

Hepatic Encephalopathy grading is done by WestHaven Criteria, shown in Table 13.

GRADE	CRITERIA
GRADE I	Trivial lack of awareness ; Shortened attention span; Euphoria or anxiety; Impaired performance of addition
GRADE II	Lethargy or apathy; Subtle personality change; Minimal disorientation for time or place; Inappropriate behaviour; Impaired performance of subtraction
GRADE III	Somnolence to semi-stupor, but responsive to verbal stimuli; Gross disorientation ;Confusion
GRADE IV	Coma (unresponsive to verbal or noxious stimuli)

TABLE 2: WEST HAVEN CRITERIA

CHILD - TURCOTTE PUGH SCORE: (CPS)

By Child and Turcotte in 1964, it was created. It included malnutrition53, ascites, clinically evident encephalopathy, serum bilirubin, albumin, and ascites. Points were assigned to each variable based on the severity or cut-off ranges. Three groups of escalating severity are used to group the aggregate score (A,B,C). Ten years later, Child-Pugh proposed a variation of this score to forecast the results of surgical treatments used to reduce PH. esophageal varices, and treat them54. The cut-off for albumin was lowered to 2.8 mg/dl in updated CPS, and Prothrombin Time or International Normalized Ratio (INR) was added in place of nutritional status. Later, the likelihood of survival in patients with cirrhosis was predicted using this score.

TABLE 3: MODIFIED CPS

Child-Turcotte-Pugh Classification for Severity of Cirrhosis			
Clinical and Lab Criteria	**Points***		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
International normalized ratio	<1.7	1.7-2.3	>2.3
***Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)**			
Class A = 5 to 6 points (least severe liver disease)			
Class B = 7 to 9 points (moderately severe liver disease)			
Class C = 10 to 15 points (most severe liver disease)			

AIMS & OBJECTIVES

Aim

To study serum sodium levels in determining the prognosis of patients with cirrhosis of liver.

Objective

To measure the sodium level among the patients with cirrhosis

To correlate the sodium level with severity of the liver cirrhosis

MATERIAL & METHOD

SOURCE OF DATA

Patients admitted in the medicine ICU/WARDS OF BLDEUS Shri BM Patil medical college and Research Centre, Vijayapura and who fulfil the inclusion criteria.

All investigations will be performed at the central lab of BLDEU'S Shri M Patil Medical College, Hospital and Research Centre, Vijayapura.

Period of study:

The study was conducted during the period of JANUARY 2021 to JUNE 2022.

Type of study: Cross- Sectional Study

Sample size:

With Anticipated correlation between Hyponatremia and Severity of Liver disease r= 0.39 (ref), at 95%confidence level and 80 power in the study, the sample size worked outis 50

Formula used is is $N=\left[\left(\frac{Z_\alpha+Z_\beta}{C}\right)\right]^2+3$

The standard normal deviate for $\alpha = Z_\alpha = 1.9600$The standard normal deviate for $\beta = Z_\beta = 1.6449$

$C=0.5*\ln\left[\frac{1+r}{1-r}\right]=0.4118$

N=50

INCLUSION CRITERIA

Patients who are known case of cirrhosis of liver or diagnosed case of chronic liver disease as decided by LFT AND USG.

USG CRITERIA:

Liver size

Liver morphology

Liver surface

Liver echogenicity

Portal vein diameter

Portal vein mean flow velocity

spleen

EXCLUSION CRITERIA

1) Patients with cardiovascular, renal disorders and thyroid disorders were excluded.
2) Patients who are already on drugs like diuretics
3) Patients having vomiting and diarrhea are excluded
4) Patients having burns are excluded

METHOD OF COLLECTION OF DATA:

Serum sodium levels taken on admission before the administration of fluids and during the discharge of the patient.

Depending on serum sodium levels hyponatremia is graded into 3 types:

1. Mild (131-135mmol/L)
2. Moderate (125-130mmol/L)
3. Severe (<125mmo/L)

Severity of cirrhosis is classified with the Child-Turcotte-Pugh Score.

Child-Turcotte-Pugh Classification for Severity of Cirrhosis			
Clinical and Lab Criteria	Points*		
	1	2	3
Encephalopathy	None	Mild to moderate (grade 1 or 2)	Severe (grade 3 or 4)
Ascites	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
International normalized ratio	<1.7	1.7-2.3	>2.3
*Child-Turcotte-Pugh Class obtained by adding score for each parameter (total points)			
Class A = 5 to 6 points (least severe liver disease)			
Class B = 7 to 9 points (moderately severe liver disease)			
Class C = 10 to 15 points (most severe liver disease)			

Figure 9: Child-Turcotte-Pugh Score

The data is collected according to proforma in terms of details history, clinical examination and necessary investigations of the patients who fulfilled the inclusion criteria

INVESTIGATIONS

1) Complete blood count

2) Urine complete

3) Fasting blood sugar levels

4) Post prandial blood sugar levels

5) Blood urea and serum creatinine

6) Serum sodium and serum potassium levels

(Mild hyponatremia 131-135mmol/L)

(Moderate hyponatremia 125-130 mmol/L)

(Severe hyponatremia < 125 mmol/L)

7) Liver function test

8) Coagulation profile

9) USG abdomen

10) Ascitic fluid analysis

11) ECG

12) Chest x- ray

STATISTICAL ANALYSIS

The data obtained was entered in a Microsoft Excel sheet, and statistical analysis was performed using statistical package for the social sciences (Version 20). Results are presented as Mean (Median) ± SD, counts and percentages and diagrams. For normally distributed continuous variables between two groups were compared using Independent t-test For not normally distributed variables Mann Whitney U test was used. Relationship between the variables was analyzed using Pearson's/Spearman's correlation. Categorical variables between two groups were compared using Chi- square test. ROC was used to find cutoff values and to find sensitivity specificity. A $p<0.05$ was considered statistically significant.

RESULTS

TABLE 4: SHOWING DISTRIBUTION OF AGE (Binned)

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	< 30	2	4.0	4.0	4.0
	30 - 39	16	32.0	32.0	36.0
	40 - 49	17	34.0	34.0	70.0
	50 - 59	10	20.0	20.0	90.0
	60 - 69	4	8.0	8.0	98.0
	70+	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

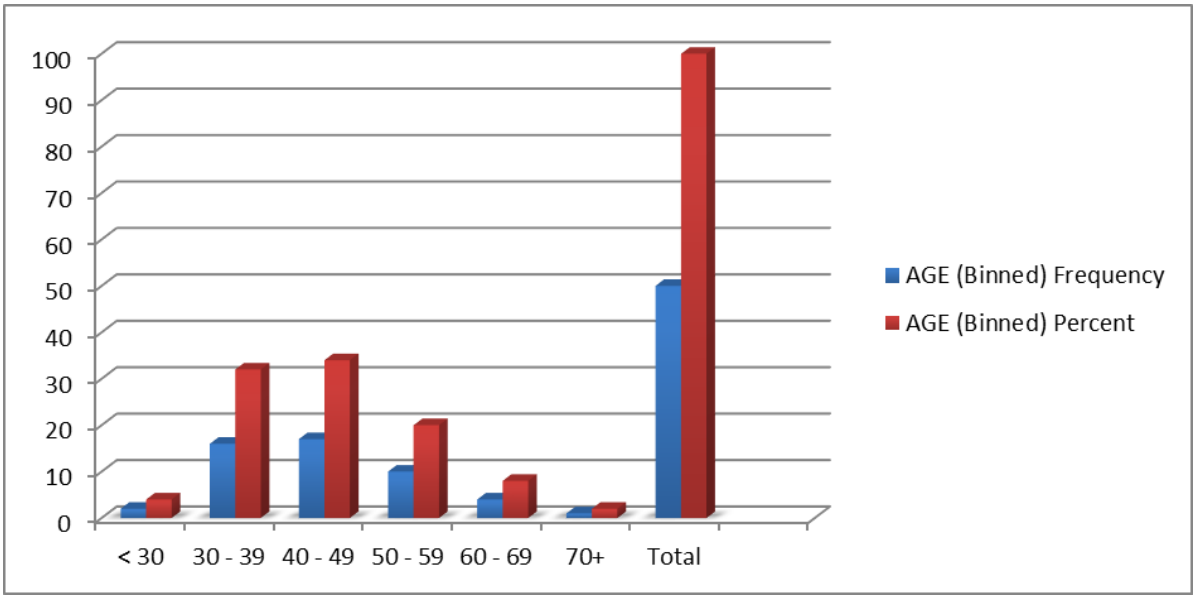


Figure 10 BAR DIAGRAM SHOWING AGE DISTRIBUTION

TABLE 5: SHOWING DISTRIBUTION OF SEX

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	M	50	100.0	100.0	100.0

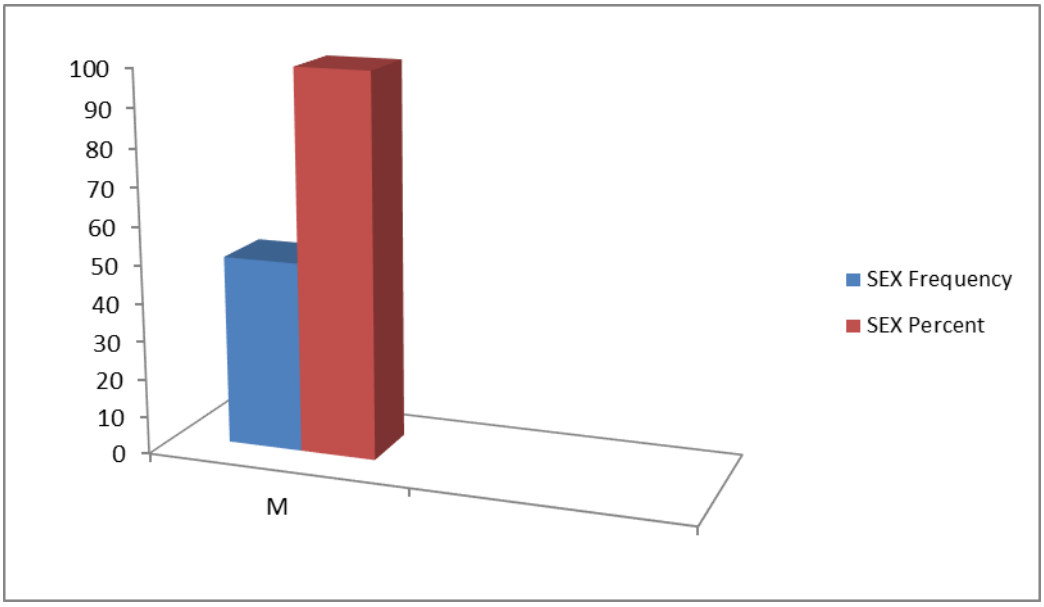


FIGURE 11 BAR DIAGRAM SHOWING GENDER DISTRIBUTION

All patients were males (100%).

TABLE 6:SHOWING SERUM SODIUM ON ADMISSION (Binned)

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	< 125	8	16.0	16.0	16.0
	125 - 130	19	38.0	38.0	54.0
	131 - 136	19	38.0	38.0	92.0
	137+	4	8.0	8.0	100.0
	Total	50	100.0	100.0	

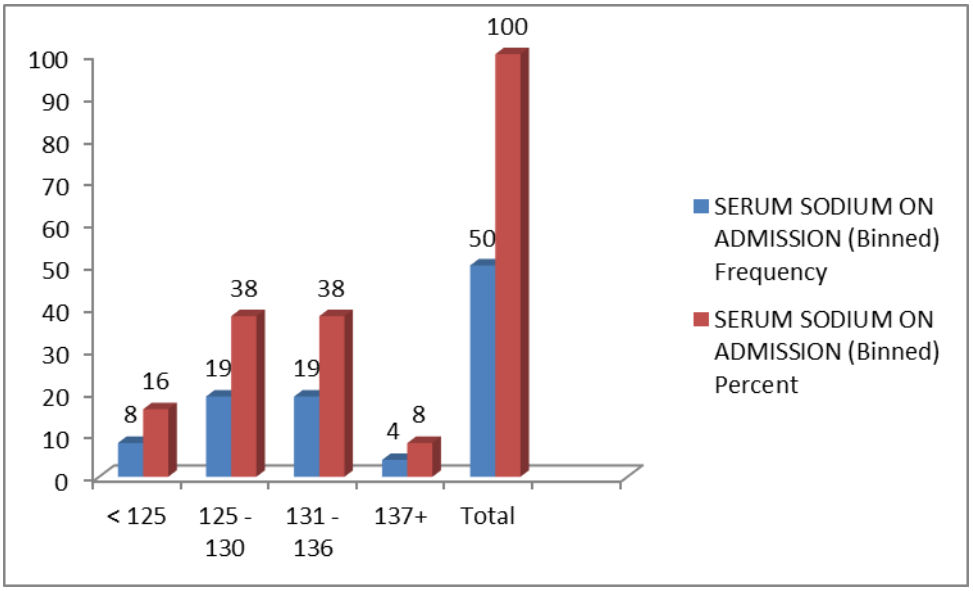


FIGURE 12 BAR DIAGRAM SHOWING SERUM SODIUM ON ADMISSION

Patients with serum sodium levels on admission were <125 were 8 (16%), sodium – 125-130 were 19 (38%) , sodium 131-136 were 19 (38%), sodium >137 were 4 (8%).

About 76% of patients were having serum sodium levels between 125-135.

TABLE 7: SHOWING SERUM SODIUM ON DISCHARGE (Binned)

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	< 125	6	12.0	12.0	12.0
	125 - 130	14	28.0	28.0	40.0
	131 - 136	24	48.0	48.0	88.0
	137+	6	12.0	12.0	100.0
	Total	50	100.0	100.0	

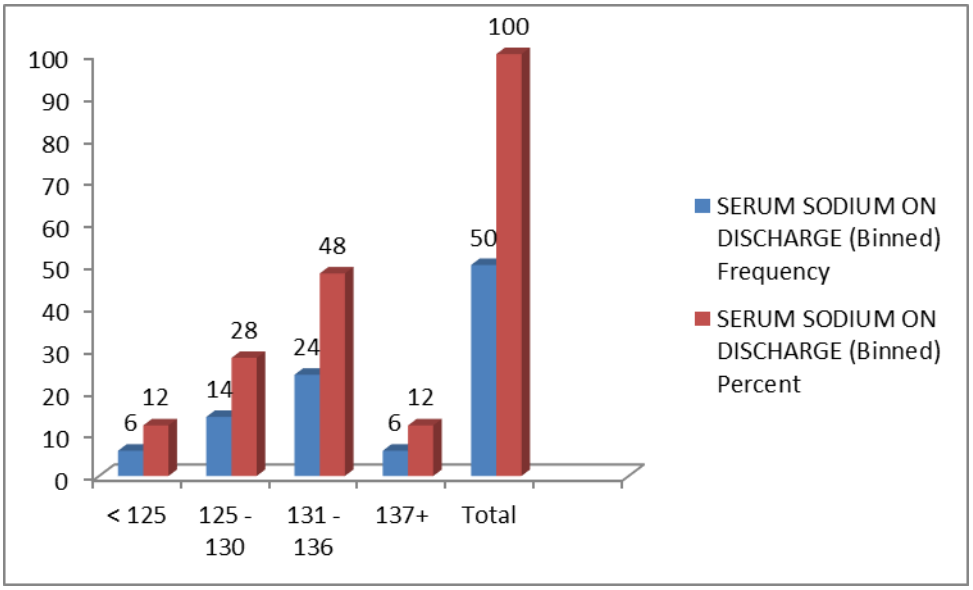


FIGURE 13 BAR DIAGRAM SHOWING SERUM SODIUM ON DISCHARGE

Patients with serum sodium levels on discharge were <125 were 6 (12%), sodium – 125-130 were 14 (28%) , sodium 131-136 were 24 (48%), sodium >137 were 6 (12%).

At discharge 48% of patients were having sodium values between 131-136.

TABLE 8:SHOWING FREQUENCY OF ASCITES

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	P	50	100.0	100.0	100.0

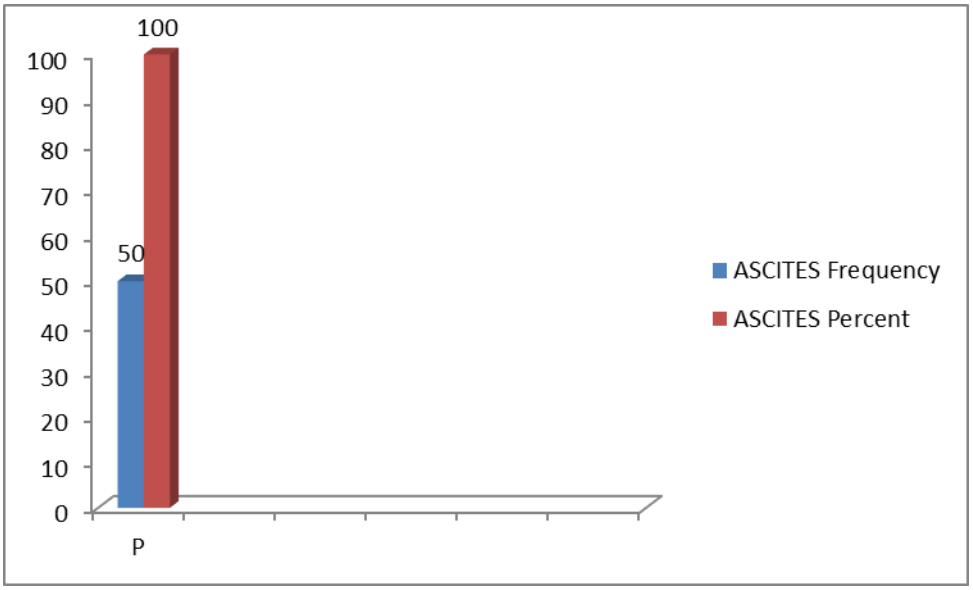


FIGURE 14 BAR DIAGRAM SHOWING FREQUENCY OF ASCITES

All 50 patients were having ascites.

TABLE 9: SHOWING FREQUENCY OF PORTAL HYPERTENSION

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	21	42.0	42.0	42.0
	P	29	58.0	58.0	100.0
	Total	50	100.0	100.0	

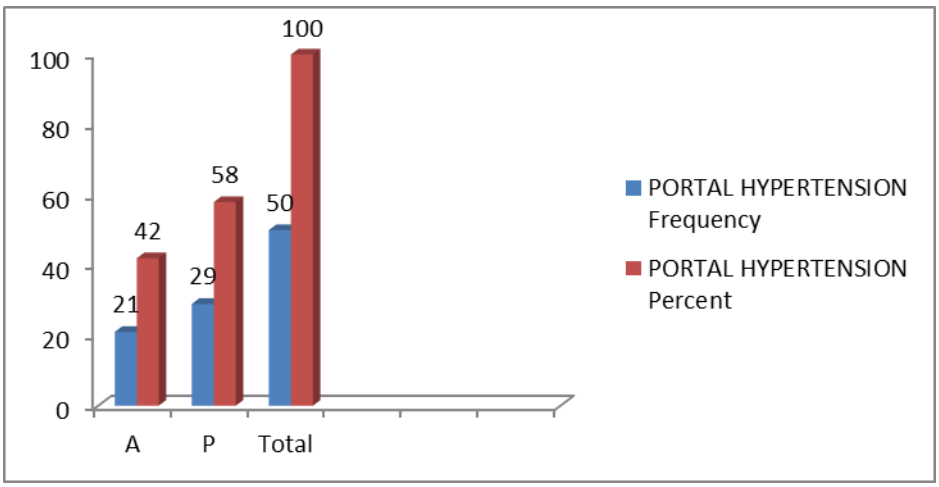


FIGURE 15 BAR DIAGRAM SHOWING FREQUENCY OF PORTAL HYPERTENSION

Portal hypertension was present in 29 (58%) patients out of 50 .

TABLE 10: SHOWING FREQUENCY OF HEPTIC ENCEPHALOPATHY

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	30	60.0	60.0	60.0
	P	20	40.0	40.0	100.0
	Total	50	100.0	100.0	

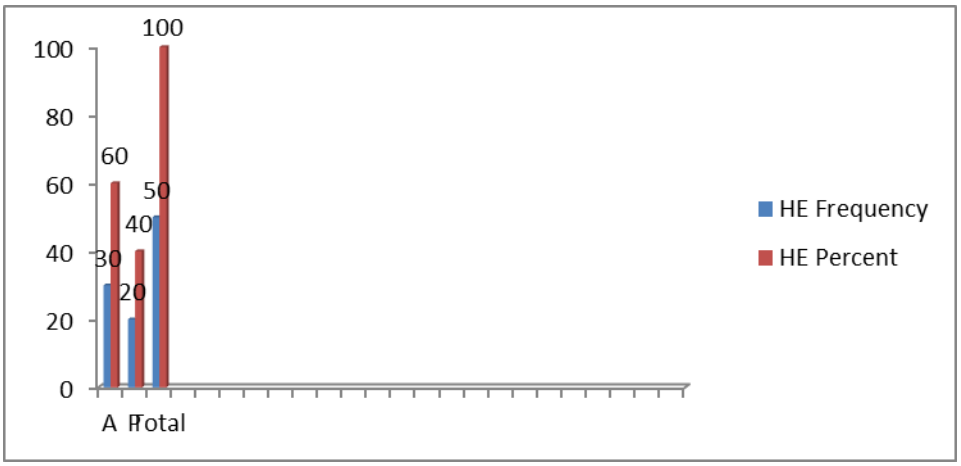


FIGURE 16 BAR DIAGRAM SHOWING FREQUENCY OF HEPATICENCEPHALOPATHY

Hepatic encephalopathy was present in 20 (40%) patients out of 50

TABLE 11: SHOWING FREQUENCY OF GI BLEED

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	34	68.0	68.0	68.0
	P	16	32.0	32.0	100.0
	Total	50	100.0	100.0	

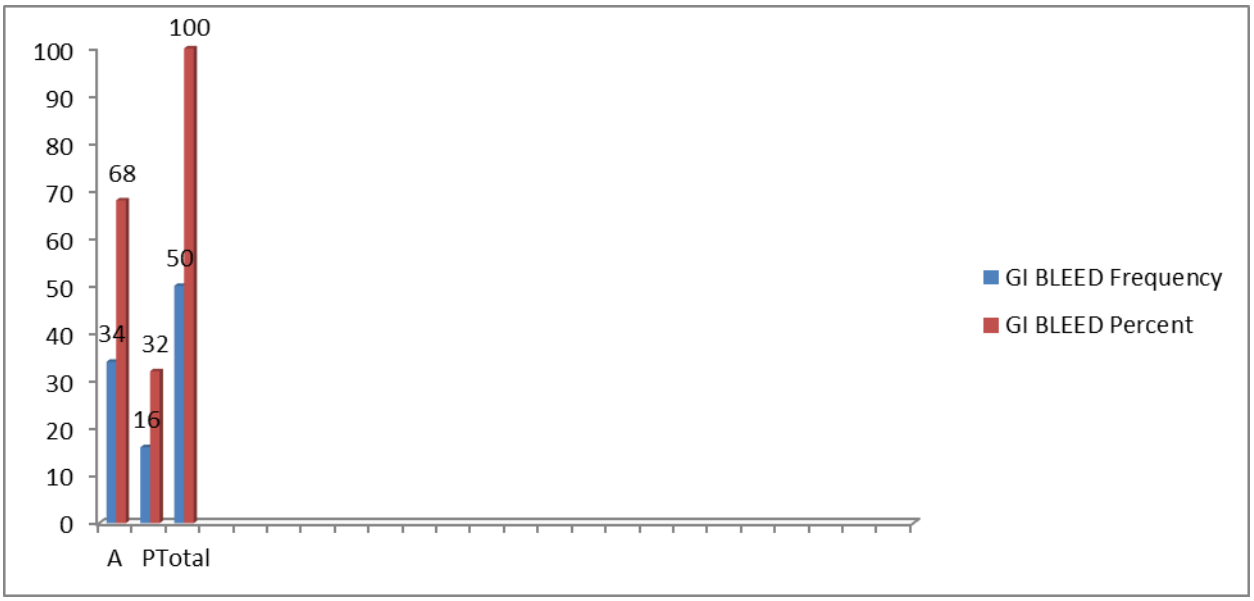


FIGURE 17 BAR DIAGRAM SHOWING FREQUENCY OF GI BLEED

GI BLEED was present in 16 (32%) patients out of 50

TABLE 12: SHOWING FREQUENCY OF HRS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	33	66.0	66.0	66.0
	P	17	34.0	34.0	100.0

Total	50	100.0	100.0	

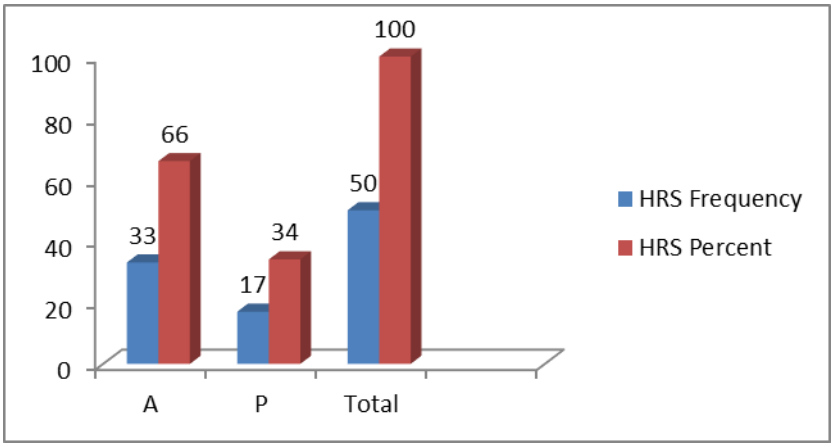


FIGURE 18 BAR DIAGRAM SHOWING FREQUENCY OF HRS

HRS was present in 17 (34%) patients out of 50

TABLE 13: SHOWING FREQUENCY OF SBP

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	45	90.0	90.0	90.0
	P	5	10.0	10.0	100.0
	Total	50	100.0	100.0	

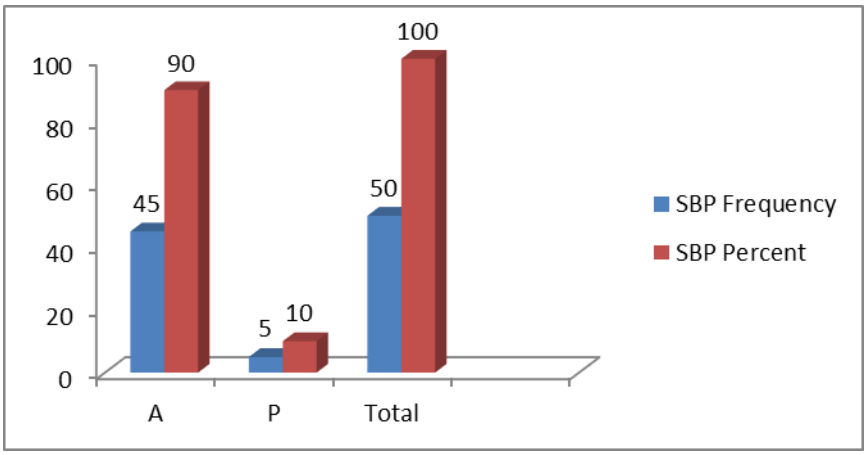
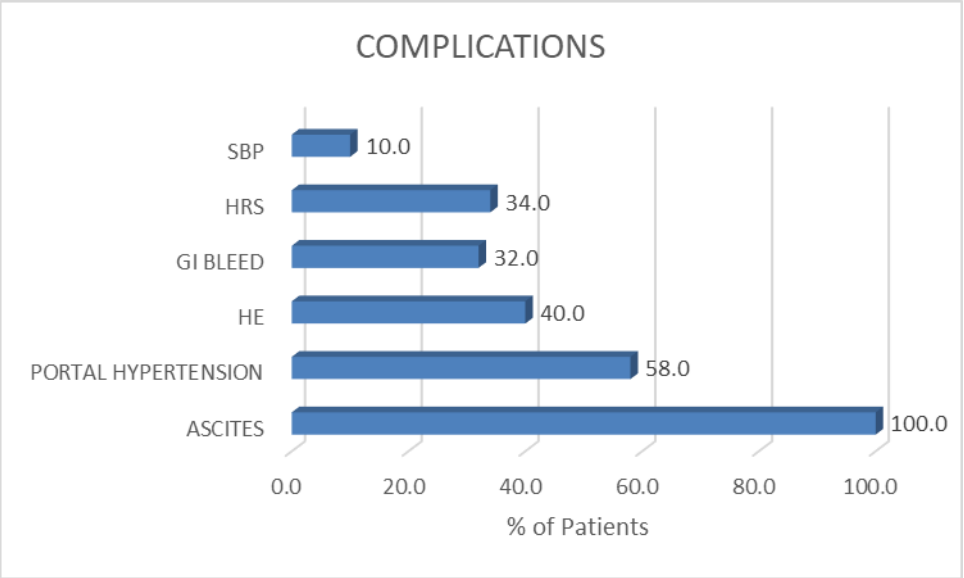


FIGURE19 BAR DIAGRAM SHOWING FREQUENCY OF SBP

SBP was present in 5 (10%) patients out of 50

FIGURE 20 BAR DIAGRAM SGOWING FREQUENCY OF COMPLICATIONS



All patients had ascites 50 (100%) , SBP was seen in 10%, HRS was seen in 34%, GI BLEED was seen in 32%, HE was seen in 40%, PORTAL HYPERTENSION was seen in 58%.

TABLE 14: SHOWING CPC

CPC	Frequency	Percent
B	17	34.0
C	33	66.0
Total	50	100.0

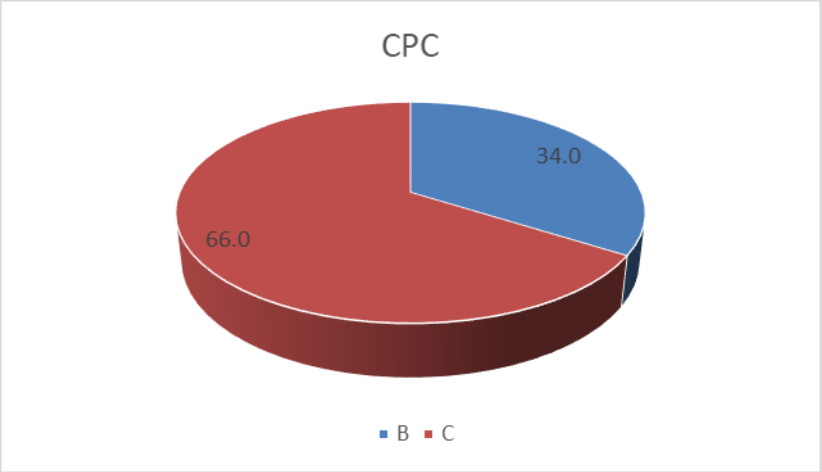


FIGURE 21 PIE CHART SHOWING CHILD PUGH CLASS DISTRIBUTION

Among 50 patients 17 (34%) of Child pugh class B and 33 (66%) patientsofclass C

TABLE 15: OUTCOME

OUTCOME	Frequency	Percent
DISCHARGED	45	90.0
EXPIRED	5	10.0
Total	50	100.0

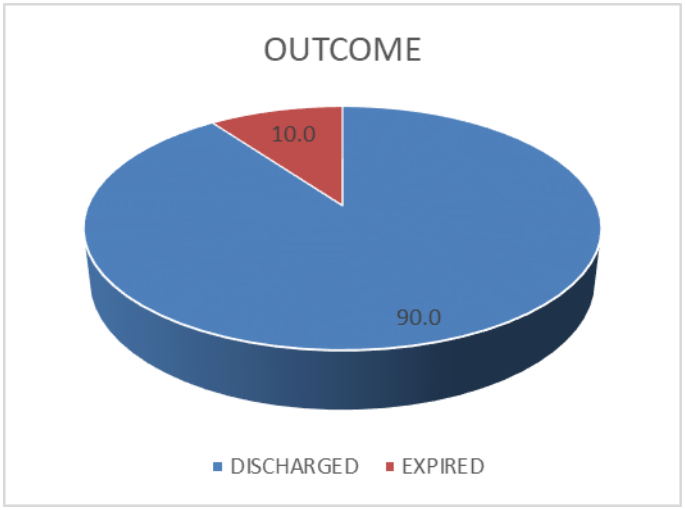


FIGURE 22 PIE CHART SHOWING OUTCOME

45 (90%) patients were discharged and 5(10%) patients expired

TABLE 16: SHOWING SERUM SODIUM ON ADMISSION AND DISCHARGE

	AGE	SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE	CPS
Minimum	22.00	117.00	117.00	7.00
Maximum	80.00	138.00	143.00	15.00
Mean	44.02	129.84	131.14	10.12
Std. Deviation	11.03	5.06	5.77	2.04

Minimum age was 22 and maximum age was 80.

Minimum sodium on admission was 117 and maximum was 138 and mean was 129.

Minimum sodium on discharge was 117 and maximum was 143 and mean was 131.

Minimum child pugh score was 7 and maximum score 15 and mean was 10.12

TABLE 17: SHOWING CHILD PUGH CLASS WITH SERUM SODIUM LEVELS

CPC		AGE	SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE	CPS
B	Mean	42.41	131.82	133.35	7.94
	Std. Deviation	9.15	4.33	3.24	0.90
C	Mean	44.85	128.82	130.00	11.24
	Std. Deviation	11.93	5.16	6.46	1.46
	p Value	0.465	0.045	0.049	<0.001
	Significance	Not Significant	Significant	Significant	Significant

Patients who belongs to child pugh class B had serum sodium levels mean of 131 at admission and 133 at discharge with child pugh score of 7.94. Patients who belongs to child pugh class C had serum sodium levels mean of 128.82 at admission and 130 at discharge with child pugh score of 11.24.

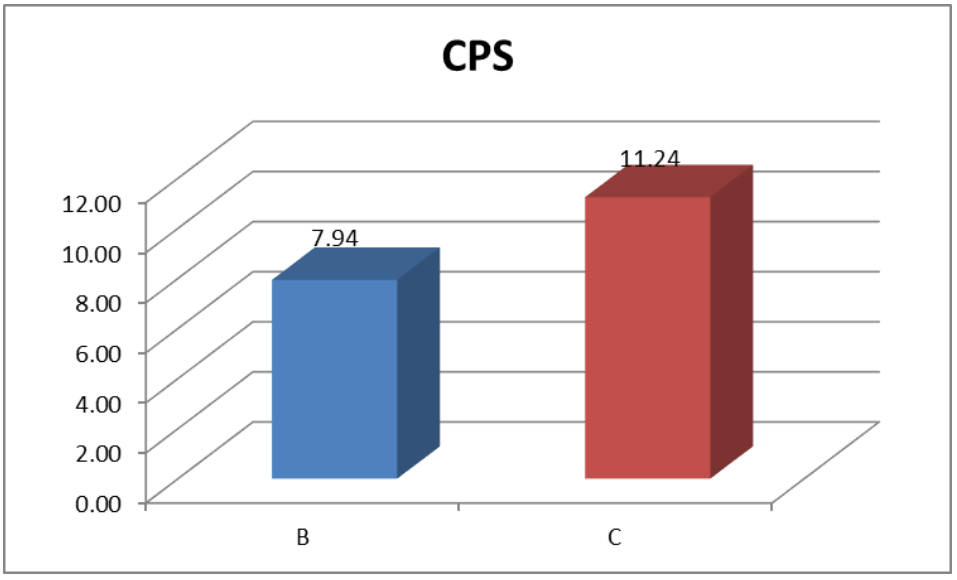


FIGURE 23 SHOWING CHILD PUGH SCORE

TABLE 18: SHOWING OUTCOME OF PATIENTS WITH SERUM SODIUM LEVELS

OUTCOME		AGE	SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE	CPS
DISCHARGED	Mean	43.67	130.51	132.09	9.82
	Std. Deviation	11.50	4.63	4.89	1.90
EXPIRED	Mean	47.20	123.80	122.60	12.80
	Std. Deviation	4.44	5.17	6.58	1.10
	p Value	0.502	0.004	<0.001	0.001
	Significance	Not Significant	Significant	Significant	Significant

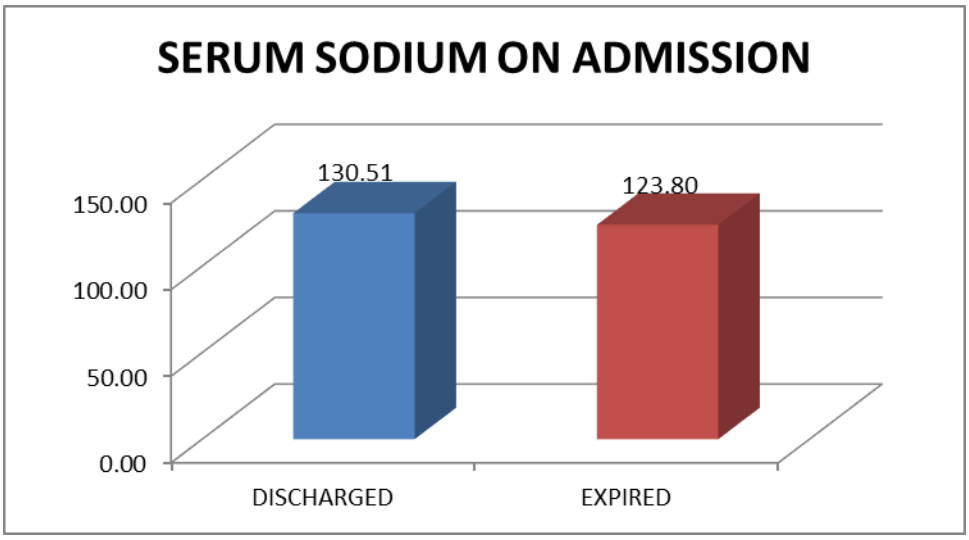


FIGURE 24 OUTCOME OF PATIENTS WITH SERUM SODIUM LEVELS ON ADMISSION

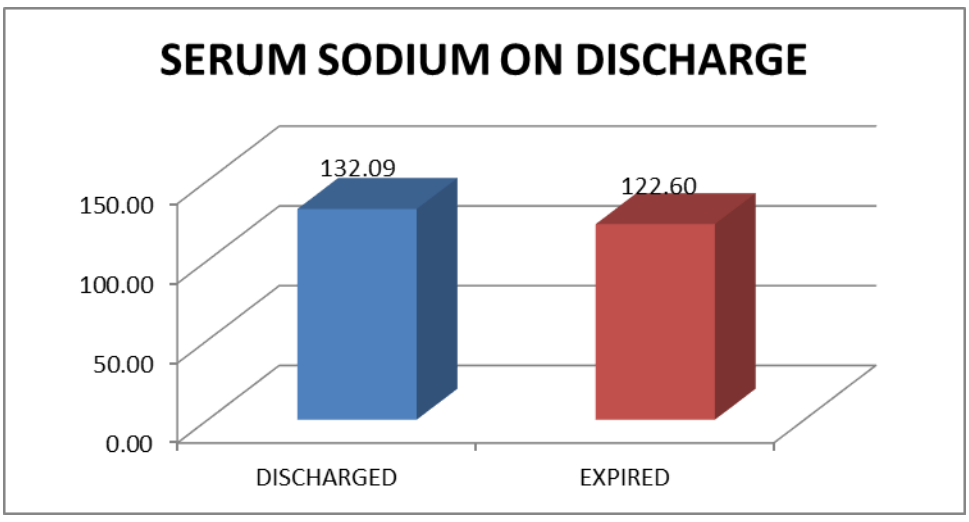


FIGURE 25 OUTCOME OF PATIENTS WITH SERUM SODIUM LEVELS ONDISCHARGE

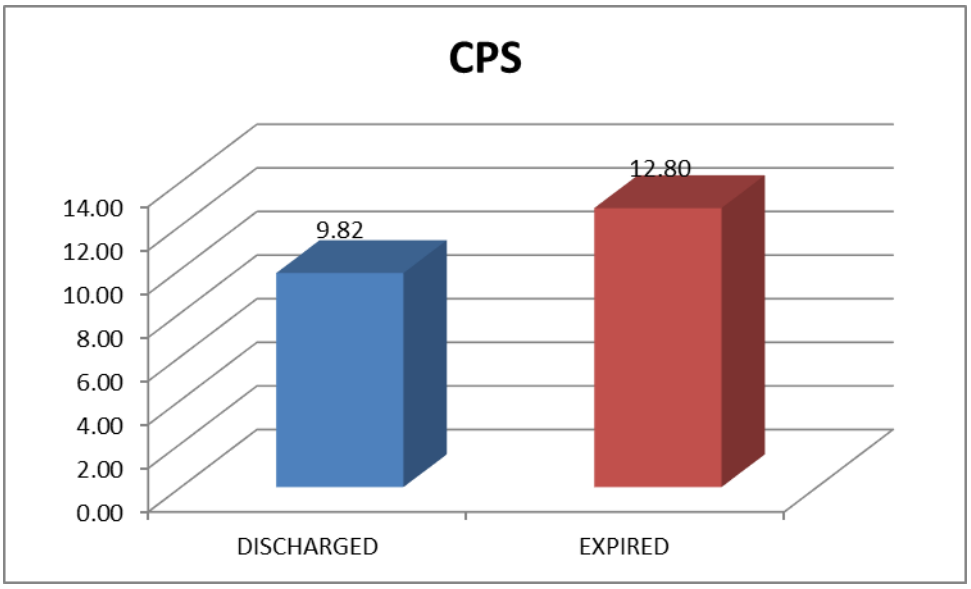


FIGURE 26 OUTCOME OF PATIENTS WITH CHILD PUGH SCORE

Among discharged patients mean serum sodium levels on admission were 130.51 and mean serum sodium levels on discharge were 132.09 with mean child pugh score of 9.82.

Among expired patients mean serum sodium levels on admission were 123.80 with mean child pugh score of 12.80.

TABLE 19: SHOWING CHILD PUGH CLASS

		CPC		Total		
		B	C		p Value	Significance
ASCITES	PRESENT	17(100)	33(100)	50(100)	NA	NA
Total		17(100)	33(100)	50(100)		

Among total of 50 patients with ascites 17 patients ofchild pugh class B and 33of child pugh class C.

TABLE 20: SHOWING OUTCOME

		OUTCOME		Total		
		DISCHARGED	EXPIRED		p Value	Significance
ASCITES	PRESENT	45(100)	5(100)	50(100)	NA	NA
Total		45(100)	5(100)	50(100)		

Among 50 patients with ascites 45 were discharged and 5 patients were expired.

FIGURE 27 BAR DIAGRAM SHOWING PORTAL HYPERTENSION WITH CHILD PUGH CLASS

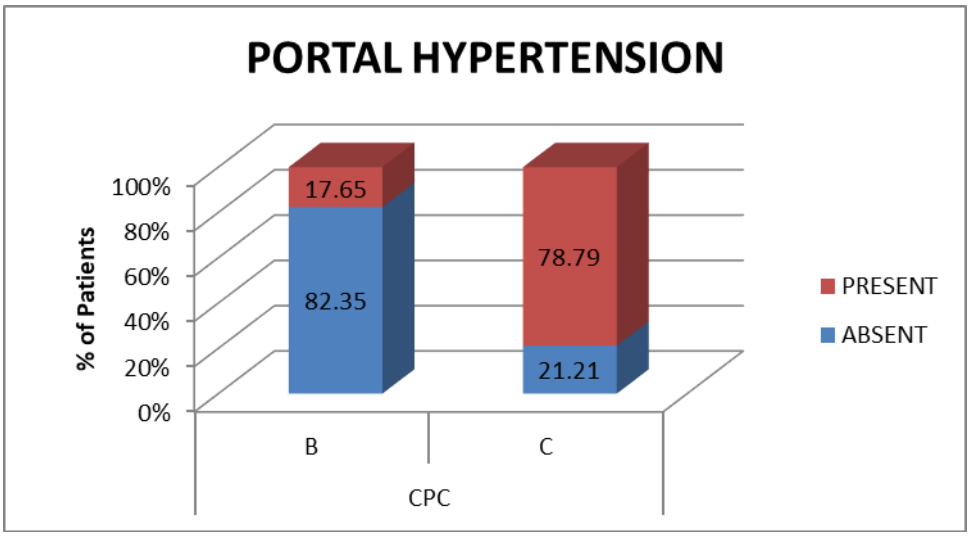


TABLE 21: SHOWING PORTAL HYPERTENSION WITH CPC AND OUTCOME

		CPC		Total		
		B	C		p Value	Significance
PORTAL HYPERTENSION	ABSENT	14(82.35)	7(21.21)	21(42)	<0.001	Significant
	PRESENT	3(17.65)	26(78.79)	29(58)		
Total		17(100)	33(100)	50(100)		
		OUTCOME		Total		
		DISCHARGED	EXPIRED		p Value	Significance
PORTAL HYPERTENSION	ABSENT	21(46.67)	0(0)	21(42)	0.045	Significant
	PRESENT	24(53.33)	5(100)	29(58)		
Total		45(100)	5(100)	50(100)		

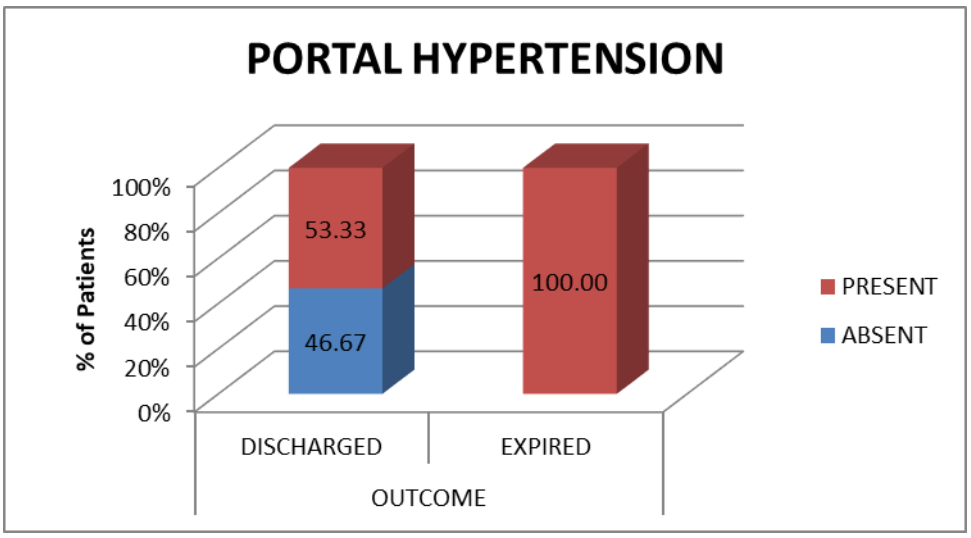


FIGURE 28 BAR DIAGRAM SHOWING OUTCOME WITH PORTAL HYPERTENSION

Among total of 50 patients portal hypertension was present in 29 patients . among them 3 belonged to child pugh class B and 26 belonged to class C.

Among 29 patients with portal hypetension 24 were discharged and 5 were expired.

TABLE 22: SHOWING HEPATIC ENCEPHALOPATHY WITH CPC

		CPC		Total		
		B	C		p Value	Significance
HE	ABSENT	16(94.12)	14(42.42)	30(60)	<0.001	Significant
	PRESENT	1(5.88)	19(57.58)	20(40)		
Total		17(100)	33(100)	50(100)		

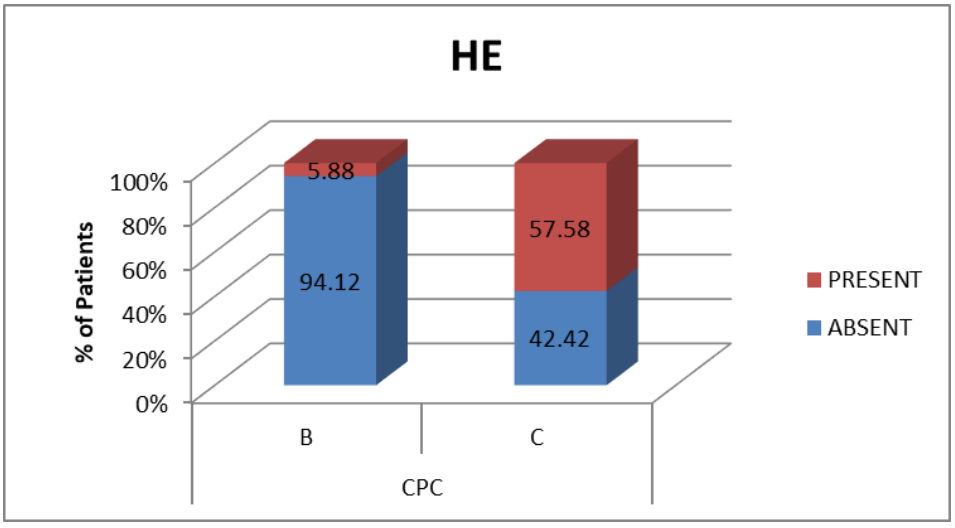


FIGURE 29 BAR DIAGRAM SHOWING HEPATIC ENCEPHALOPATHY WITH CPC

TABLE 23: SHOWING OUTCOME WITH HEPATIC ENCEPHALOPATHY

		OUTCOME		Total		
		DISCHARGED	EXPIRED		p Value	Significance
HE	ABSENT	30(66.67)	0(0)	30(60)	0.004	Significant
	PRESENT	15(33.33)	5(100)	20(40)		
Total		45(100)	5(100)	50(100)		

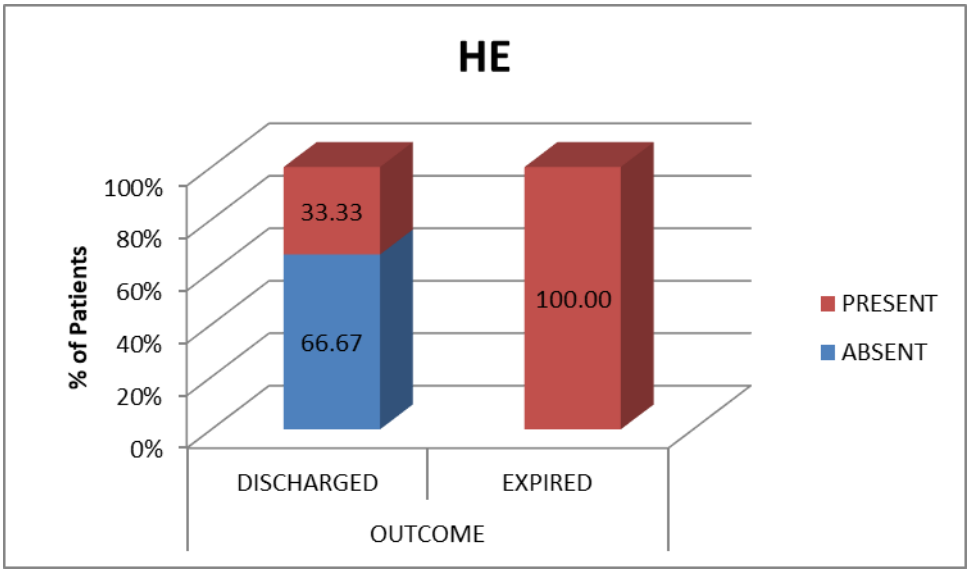


FIGURE 30 BAR DIAGRAM SHOWING OUTCOME WITH HEPATIC ENCEPHALOPATHY

Among total of 50 patients hepatic encephalopathy was present in 20 patients among them 1 belonged to child pugh class B and 19 belonged to child pugh class C and 15 were discharged and 5 were expired.

TABLE 24: SHOWING GI BLEED WITH CPC

		CPC		Total		
		B	C		p Value	Significance
GI BLEED	ABSENT	17(100)	17(51.52)	34(68)	<0.001	Significant
	PRESENT	0(0)	16(48.48)	16(32)		
Total		17(100)	33(100)	50(100)		

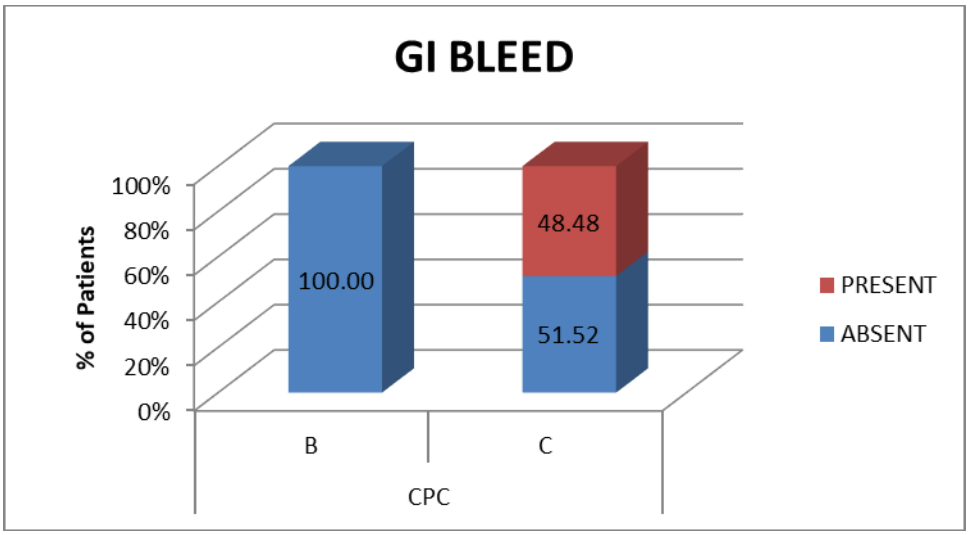


FIGURE 31 BAR DIAGRAM SHOWING GI BLEED WITH CPC

TABLE 25: SHOWING GI BLEED WITH OUTCOME

		OUTCOME		Total		
		DISCHARGED	EXPIRED		p Value	Significance
GI BLEED	ABSENT	34(75.56)	0(0)	34(68)	0.001	Significant
	PRESENT	11(24.44)	5(100)	16(32)		
Total		45(100)	5(100)	50(100)		

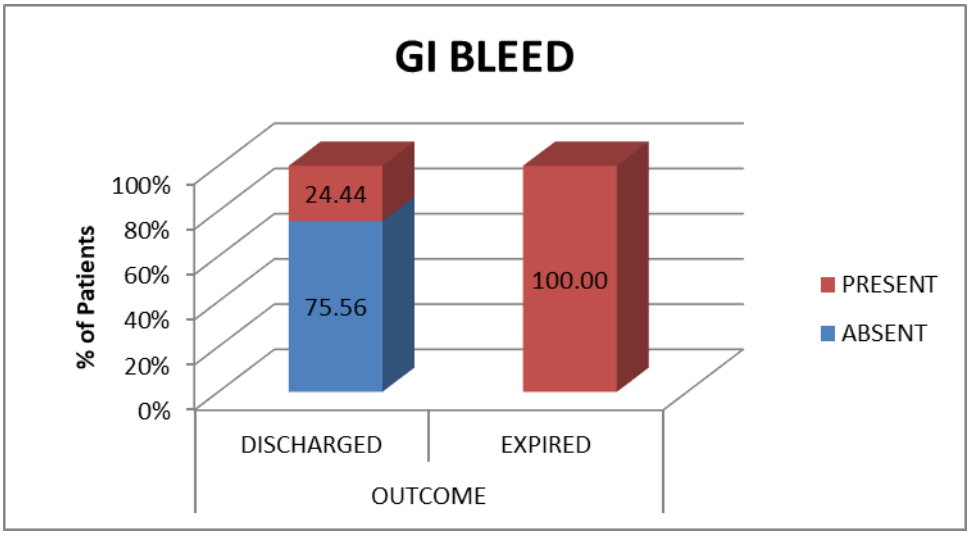


FIGURE 32 BAR DIAGRAM SHOWING GI BLEED WITH OUTCOME

Among 50 patients GI BLEED was present in 16 patients among them all 16 belonged to child pugh class C and 11 were discharged an 5 were expired.

TABLE 26: SHOWING HRS WITH CPC

		CPC		Total		
		B	C		p Value	Significance
HRS	ABSENT	16(94.12)	17(51.52)	33(66)	0.003	Significant
	PRESENT	1(5.88)	16(48.48)	17(34)		
Total		17(100)	33(100)	50(100)		

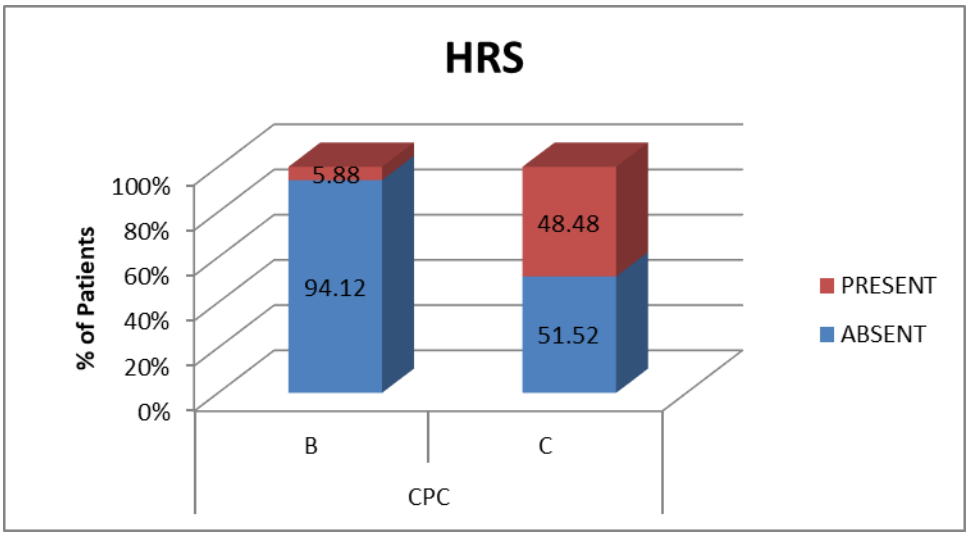


FIGURE 33 BAR DIAGRAM SHOWING HRS WITH CPC

TABLE 27: SHOWING HRS WITH OUTCOME

		OUTCOME		Total		
		DISCHARGED	EXPIRED		p Value	Significance
HRS	ABSENT	33(73.33)	0(0)	33(66)	0.001	Significant
	PRESENT	12(26.67)	5(100)	17(34)		
Total		45(100)	5(100)	50(100)		

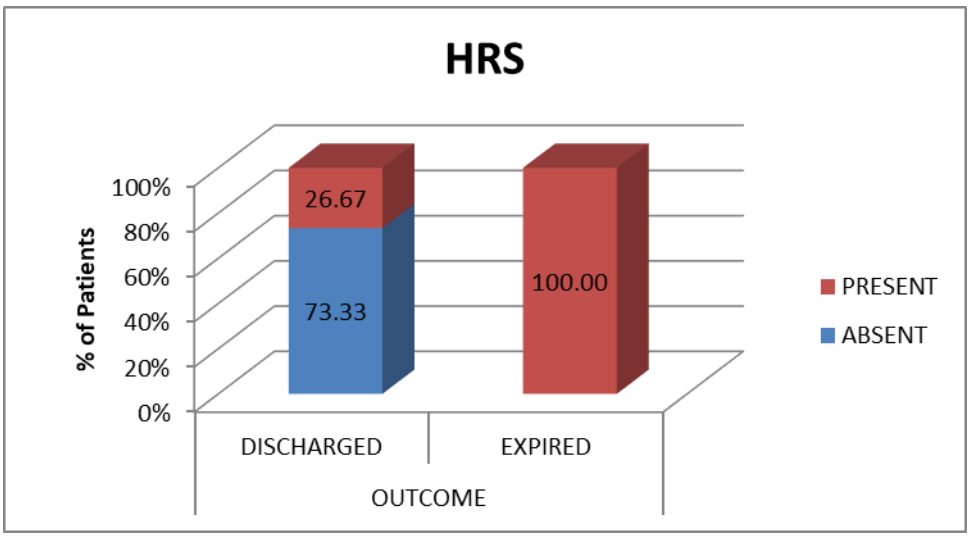


FIGURE 34 BAR DIAGRAM SHOWING HRS WITH OUTCOME

Among 50 patients HRS was seen in 17 patients among them 1 belonged to child pugh class B and 16 belonged to child pugh class C, and 12 patients were discharged 5 patients were expired.

TABLE 28 : SHOWING SBP WITH CPC

		CPC		Total		
		B	C		p Value	Significance
SBP	ABSENT	16(94.12)	29(87.88)	45(90)	0.486	Not Significant
	PRESENT	1(5.88)	4(12.12)	5(10)		
Total		17(100)	33(100)	50(100)		

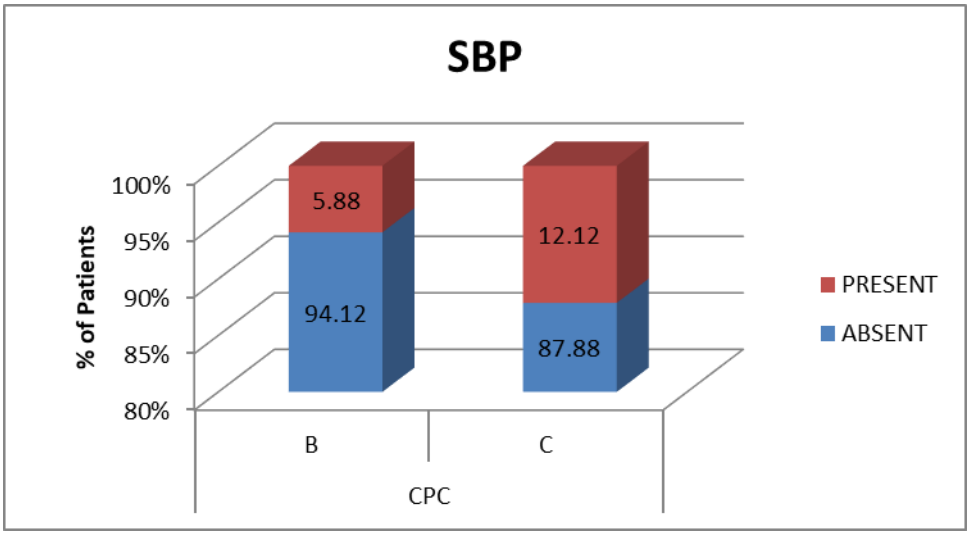


FIGURE 35 BAR DIAGRAM SHOWING SBP WITH CPC

TABLE 29 : SHOWING SBP WITH OUTCOME

	OUTCOME		Total		
	DISCHARGED	EXPIRED		p Value	Significance

SBP	ABSENT	42(93.33)	3(60)	45(90)	0.018	Significant
	PRESENT	3(6.67)	2(40)	5(10)		
Total		45(100)	5(100)	50(100)		

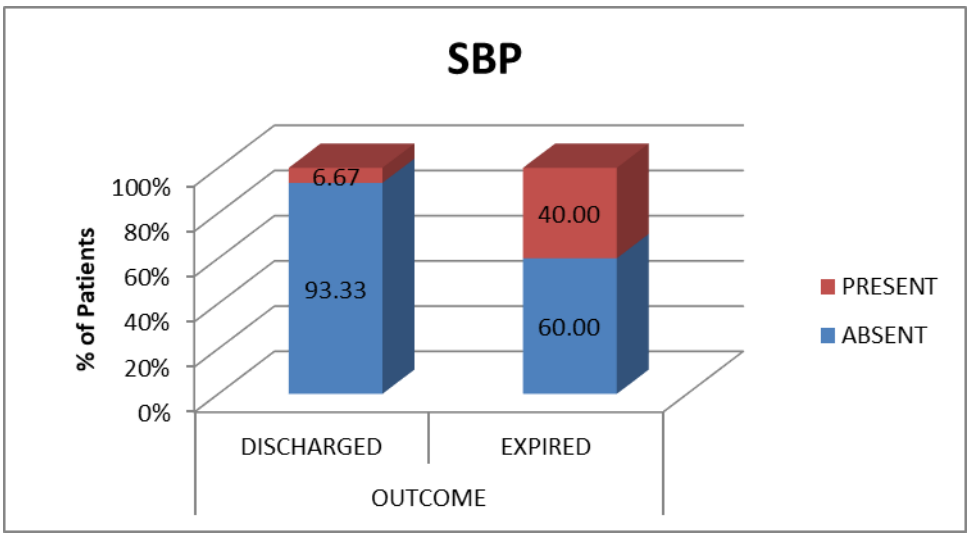


FIGURE 36 BAR DIAGRAM SHOWING SBP WITH OUTCOME

Among 50 patients SBP was seen in 5 patients among them 1 patients belonged to child pugh class B and 4 belonged to child pugh class C and 3 patients were discharged and 2 were expired.

TABLE 30: SHOWING COMPLICATIONS WITH SERUM SODIUM LEVELS

ASCITES		SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE
PRESENT	Mean	129.84	131.14
	Std. Deviation	5.06	5.77
	p Value	NA	NA
	Significance	NA	NA
PORTAL HYPERTENSION		SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE
ABSENT	Mean	133.14	134.43
	Std. Deviation	3.37	3.93
PRESENT	Mean	127.45	128.76
	Std. Deviation	4.75	5.76
	p Value	<0.001	<0.001
	Significance	Significant	Significant
HE		SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE
ABSENT	Mean	131.40	132.43
	Std. Deviation	4.83	4.52
PRESENT	Mean	127.50	129.20
	Std. Deviation	4.55	6.93
	p Value	0.006	0.049
	Significance	Significant	Significant
GI BLEED		SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE
ABSENT	Mean	131.29	132.88
	Std. Deviation	4.06	4.07
PRESENT	Mean	126.75	127.44
	Std. Deviation	5.69	7.13
	p Value	0.002	0.001
	Significance	Significant	Significant
HRS		SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE
ABSENT	Mean	130.73	132.70
	Std. Deviation	4.49	4.09
PRESENT	Mean	128.12	128.12
	Std. Deviation	5.75	7.33
	p Value	0.084	0.007
	Significance	Not Significant	Significant
SBP		SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE
ABSENT	Mean	130.44	131.98
	Std. Deviation	4.76	5.20
PRESENT	Mean	124.40	123.60
	Std. Deviation	4.83	5.55
	p Value	0.010	0.001
	Significance	Significant	Significant

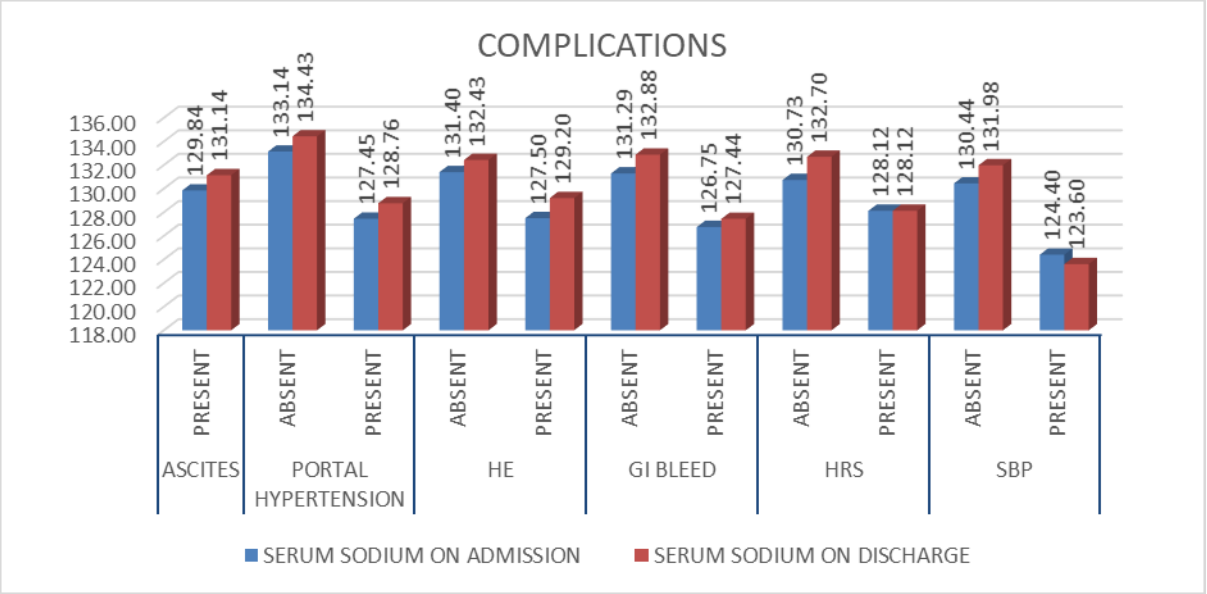


FIGURE 37 BAR DIAGRAM SHOWING COMPLICATIONS WITH SERUM SODIUM LEVELS

Patients with ascites had a mean serum sodium levels of 129.84 on admission and on discharge of 131.14.

Patients with portal hypertension had mean serum sodium levels of 127.45 and on discharge of 128.76.

Patients with hepatic encephalopathy had mean serum sodium levels of 127.50 and on discharge of 129.20.

Patients with GI bleed had mean serum sodium levels of 126.75 and on discharge of 127.44.

Patients with hepatorenal syndrome had mean serum sodium levels of 128.12 and on discharge of 128.12.

Patients with spontaneous bacterial peritonitis had mean serum sodium levels of 124.40 and on discharge of 123.60.

Serum sodium levels on admission of less than 125

TABLE 31: SHOWING FREQUENCY OF ASCITES IN PATIENTS WITH SERUM SODIUM LEVELS LESS THAN 125

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	P	8	100.0	100.0	100.0

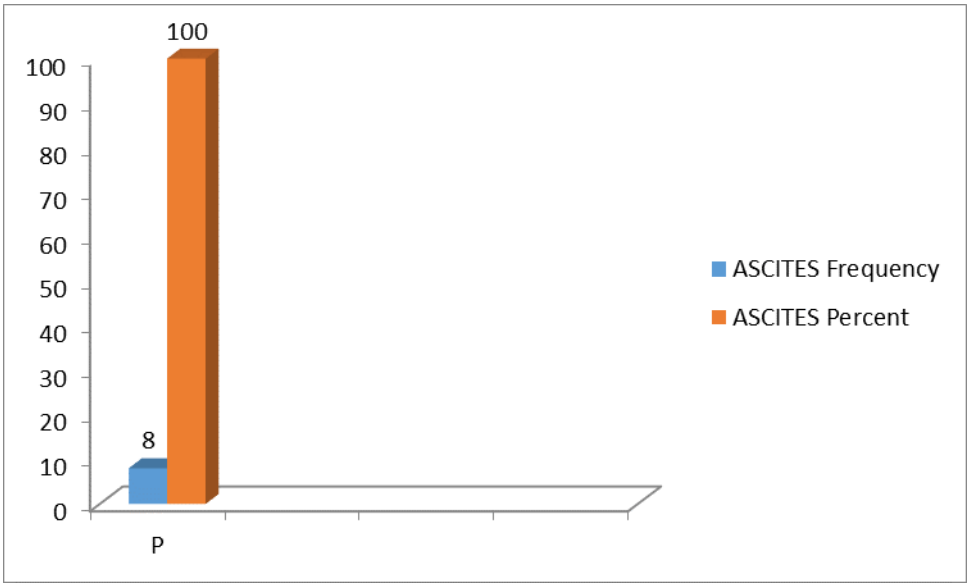


FIGURE 38 SHOWING FREQUENCY OF ASCITES IN PATIENTS WITH SERUM SODIUM LEVELS LESS THAN 125

With seru m sodiu m levels less than 125 , 8 (100%) patients had ascites

TABLE 32: SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTS WITHSERUM SODIUM LEVELS LESS THAN 125

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	P	8	100.0	100.0	100.0

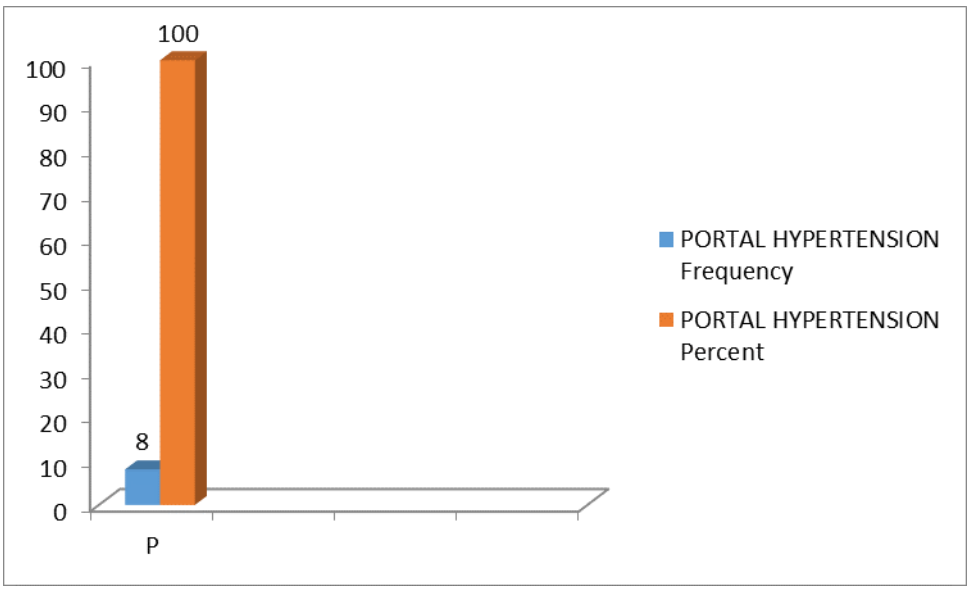


FIGURE 39 SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTS WITHSERUM SODIUM LEVELS LESS THAN 125.

Wit seru m sodium levels less than 125 , 8 (100%) patients had portal hypertension.

TABLE 33: SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTSWITH SERUM SODIUM LEVELS LESS THAN 125.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	3	37.5	37.5	37.5
	P	5	62.5	62.5	100.0
	Total	8	100.0	100.0	

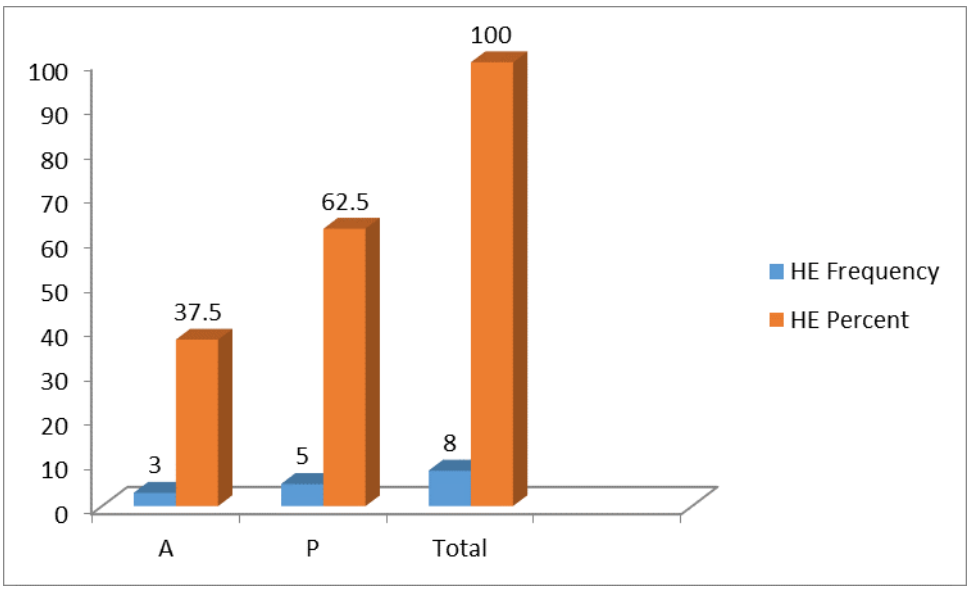


FIGURE 40 SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTSWITH SERUM SODIUM LEVELS LESS THAN 125.

Wit serum sodium levels less than 125 , 5 (62.5%) patients had hepatic encephalopathy.

TABLE 34: SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITH SERUMSODIUM LEVELS LESS THAN 125.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	2	25.0	25.0	25.0
	P	6	75.0	75.0	100.0
	Total	8	100.0	100.0	

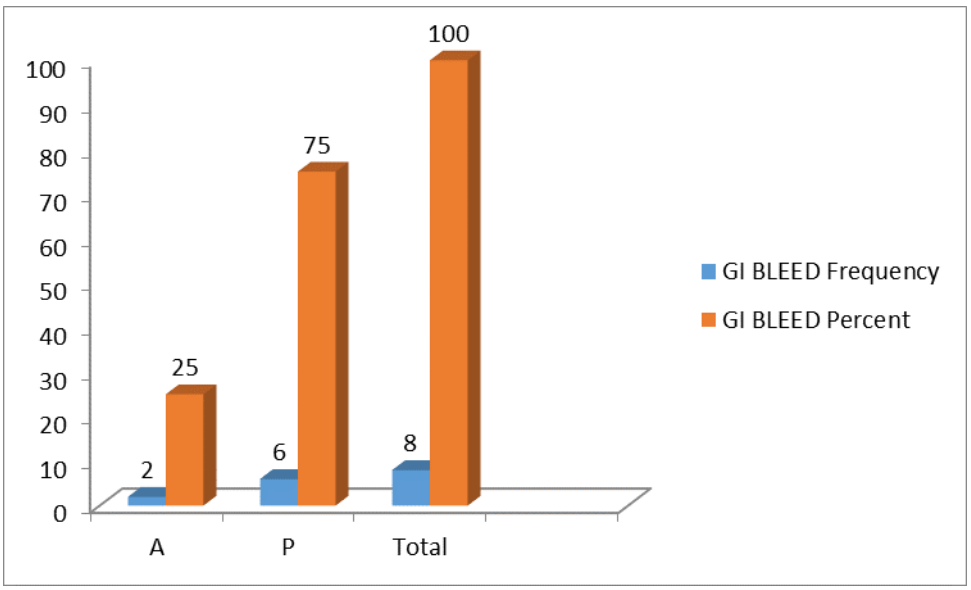


FIGURE 41 SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITH SERUMSODIUM LEVELS LESS THAN 125.

With serum sodium levels less than 125 , 6 (75%) patients had GI bleed.

TABLE 35: SHOWING FREQUENCY OF HEPATORENAL SYNDROME IN PATIENTSWITH SERUM SODIUM LEVELS LESS THAN 125.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	3	37.5	37.5	37.5
	P	5	62.5	62.5	100.0
	Total	8	100.0	100.0	

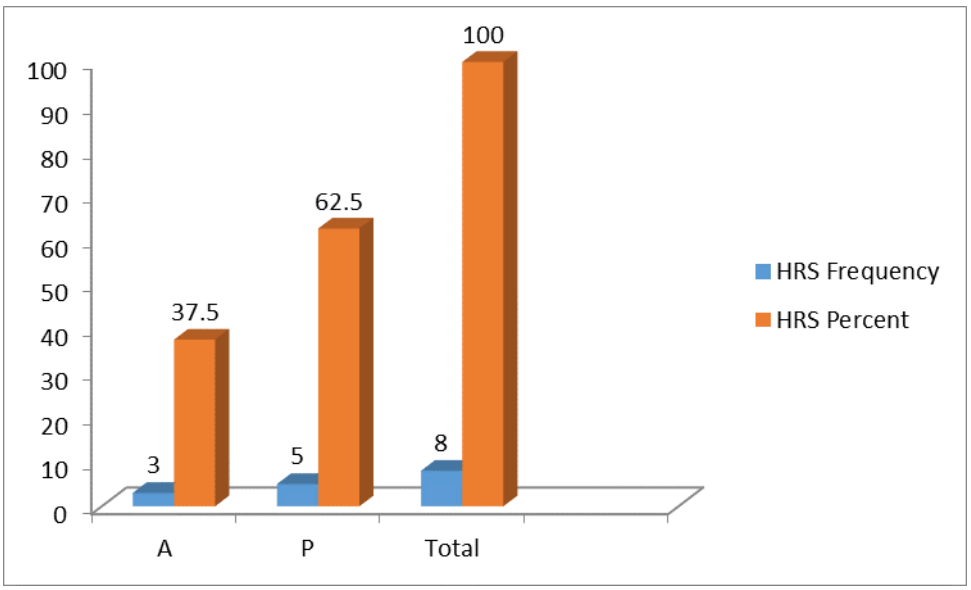


FIGURE 42 SHOWING FREQUENCY OF HEPATORENAL SYNDROME IN PATIENTSWITH SERUM SODIUM LEVELS LESS THAN 125.

Withserum sodium levels less than 125 , 5 (62.5%) patients had hepatorenal syndrome.

TABLE 36: SHOWING FREQUENCY OF SBP IN PATIENTS WITH SERUM SODIUMLEVELS LESS THAN 125.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	6	75.0	75.0	75.0
	P	2	25.0	25.0	100.0
	Total	8	100.0	100.0	

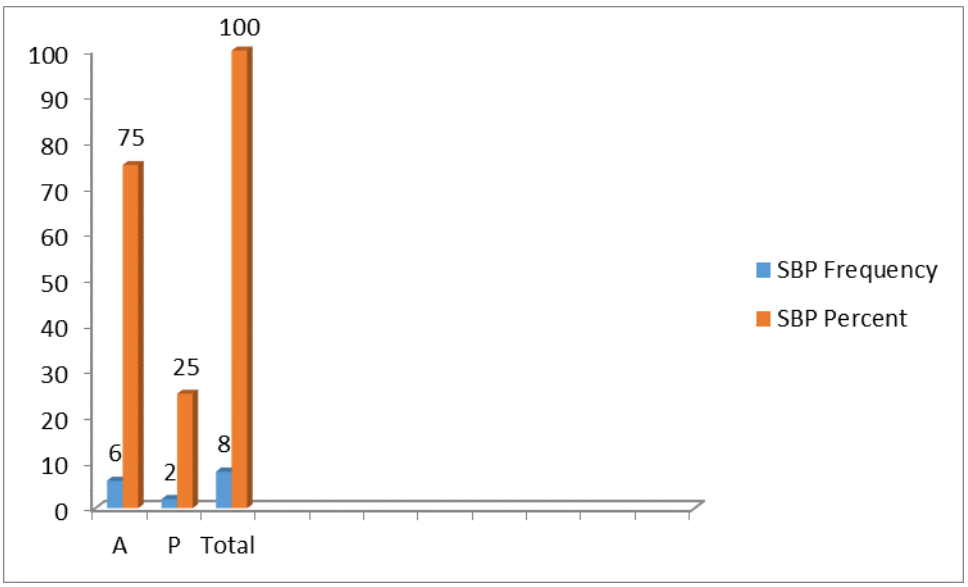


FIGURE 43 SHOWING FREQUENCY OF SBP IN PATIENTS WITH SERUM SODIUMLEVELS LESS THAN 125.

With serum sodium levels less than 125 , 2 (25%) patients had SBP.

TABLE 37: SHOWING FREQUENCY OF PATIENTS BELONGS TOCHILD PUGH CLASS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	B	1	12.5	12.5	12.5
	C	7	87.5	87.5	100.0
	Total	8	100.0	100.0	

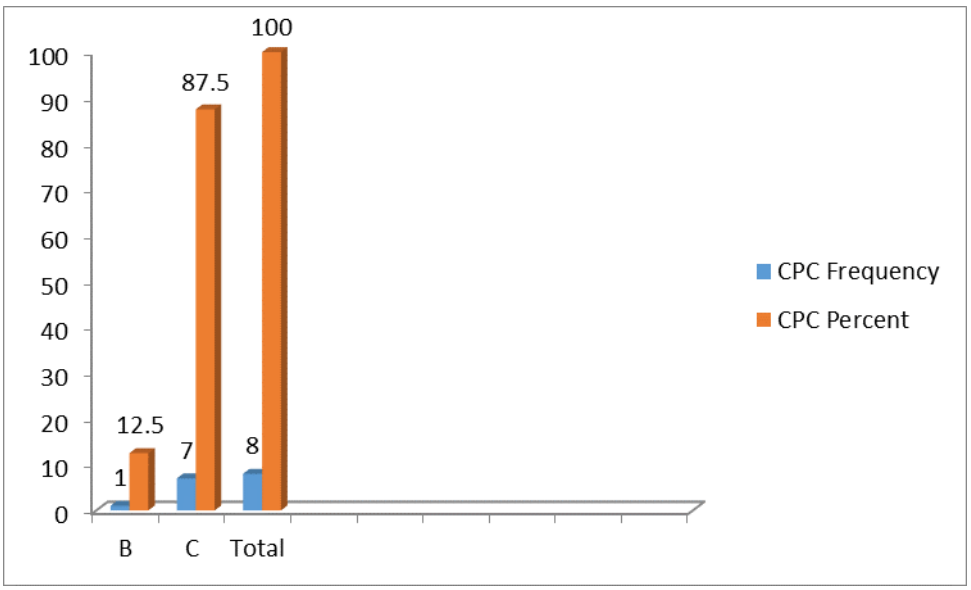


FIGURE 44 SHOWING FREQUENCY OF PATIENTS BELONGS TOCHILD PUGH CLASS

Among 8 patients with serum sodium levels less than 125 , 1(12.5%) patient of childpugh class B and 7 (87.5%) patientsof class C.

TABLE 38: SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	D	5	62.5	62.5	62.5
	E	3	37.5	37.5	100.0
	Total	8	100.0	100.0	

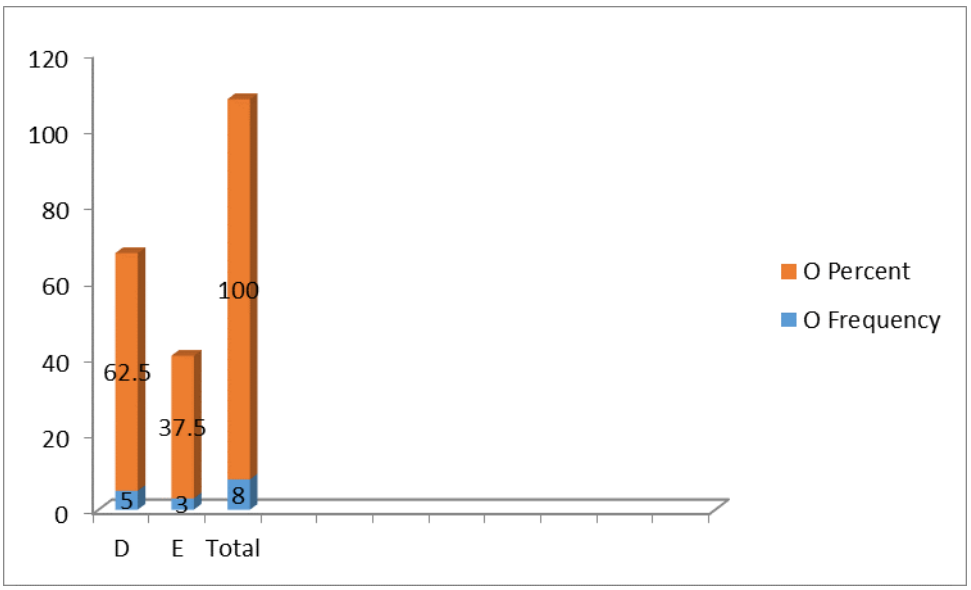


FIGURE 45 SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

Among 8 patients with serum sodium less than 125 , 5 patients were discharged and 3 were expired.

Serum sodium levels on admission between 126-130

TABLE 39: SHOWING FREQUENCY OF ASCITES IN PATIENTS WITH SERUM SODIUM LEVELS BETWEEN 126-130.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	P	19	100.0	100.0	100.0

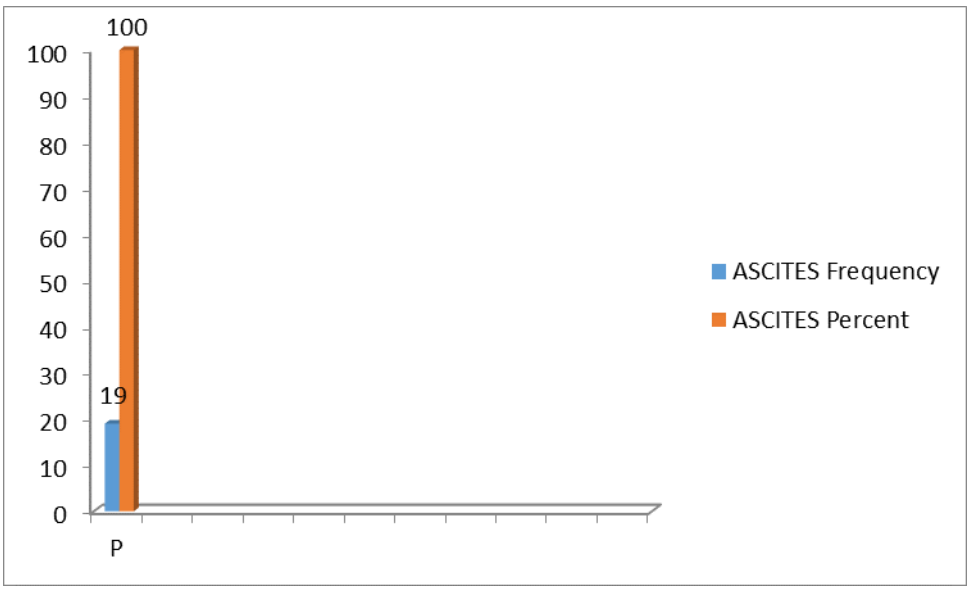


FIGURE 46 SHOWING FREQUENCY OF ASCITES IN PATIENTS WITH SERUM SODIUM LEVELS BETWEEN 126-130.

Among 19 patients with serum sodium levels between 126-130 all 19(100%) patients had ascites.

TABLE 40: SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	5	26.3	26.3	26.3
	P	14	73.7	73.7	100.0
	Total	19	100.0	100.0	

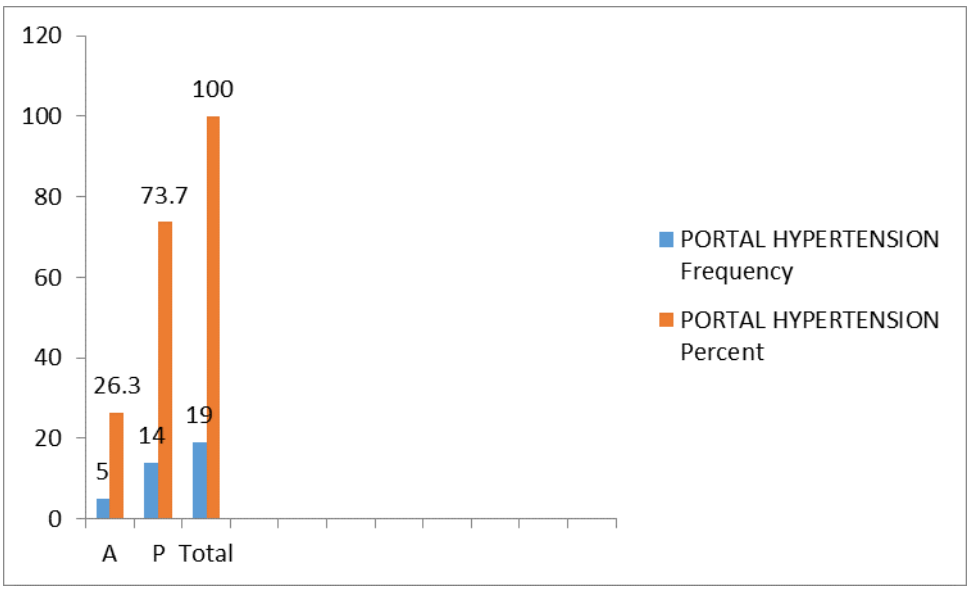


FIGURE 47 SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

Among 19 patients with serum sodium levels between 126-130 all 14(73.7%) patients had portal hypertension.

TABLE 41: SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	9	47.4	47.4	47.4
	P	10	52.6	52.6	100.0
	Total	19	100.0	100.0	

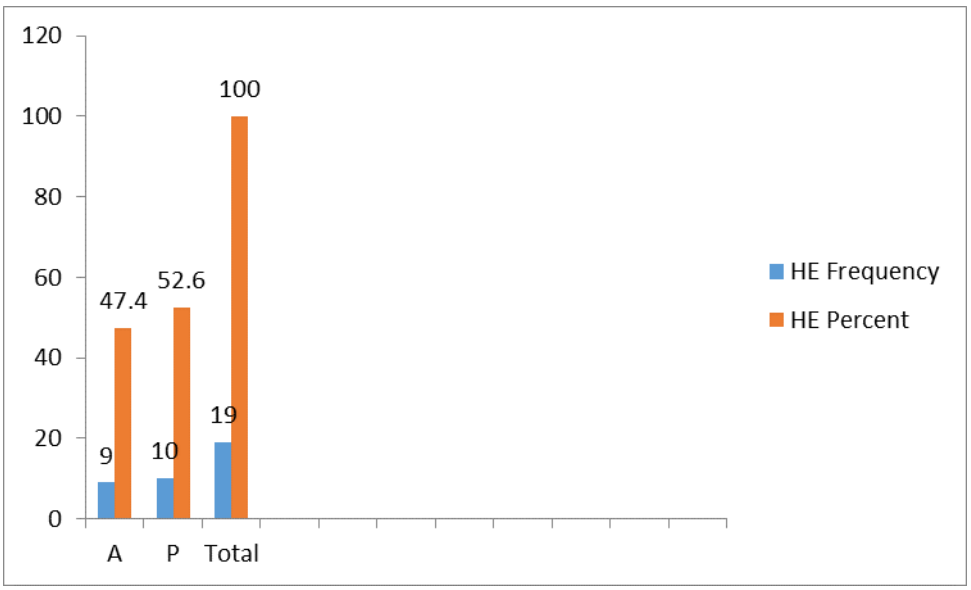


FIGURE 48 SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

Among 19 patients with serum sodium levels between 126-130 all 10(52.6%) patients had hepatic encephalopathy.

TABLE 42: SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITH SERUMSODIUM LEVELS BETWEEN 126-130.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	13	68.4	68.4	68.4
	P	6	31.6	31.6	100.0
	Total	19	100.0	100.0	

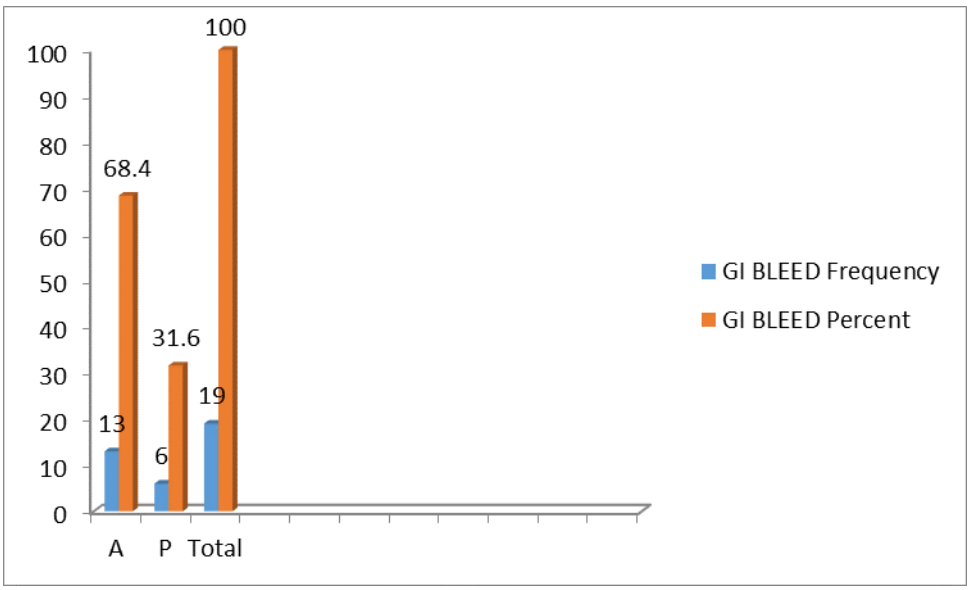


FIGURE 49 SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITH SERUMSODIUM LEVELS BETWEEN 126-130.

Among 19 patients with serum sodium levels between 126-130 all 6(31.6%) patients had GI BLEED.

TABLE 43: SHOWING FREQUENCY OF HEPATORENAL SYNDROME IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	13	68.4	68.4	68.4
	P	6	31.6	31.6	100.0
	Total	19	100.0	100.0	

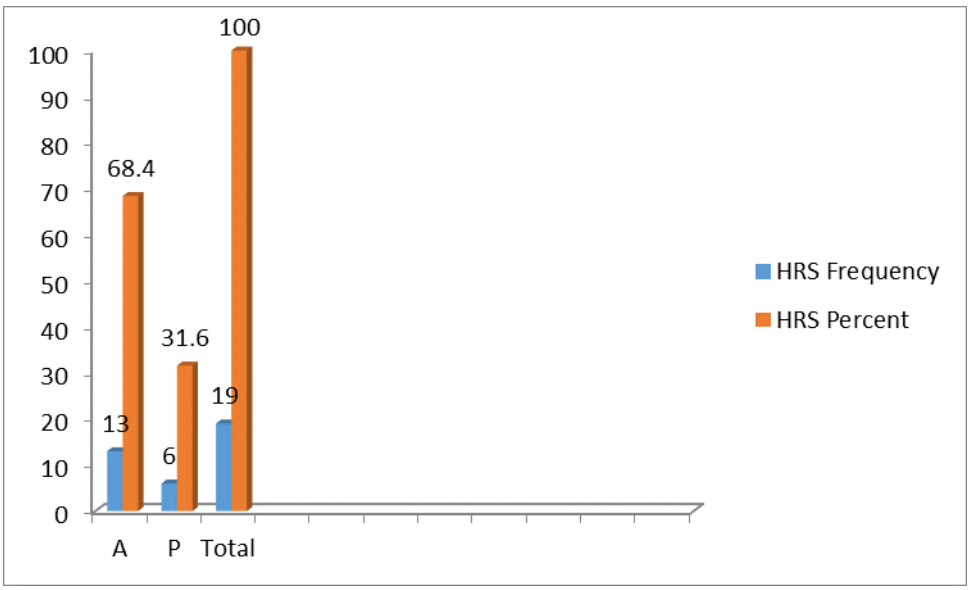


FIGURE 50 SHOWING FREQUENCY OF HEPATORENAL SYNDROME IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

Among 19 patients with serum sodium levels between 126-130 all 6 (31.6%) patients had hepatorenal syndrome.

TABLE 44: SHOWING FREQUENCY OF SBP IN PATIENTSWITH SERUM SODIUM LEVELS BETWEEN 126-130.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	16	84.2	84.2	84.2
	P	3	15.8	15.8	100.0
	Total	19	100.0	100.0	

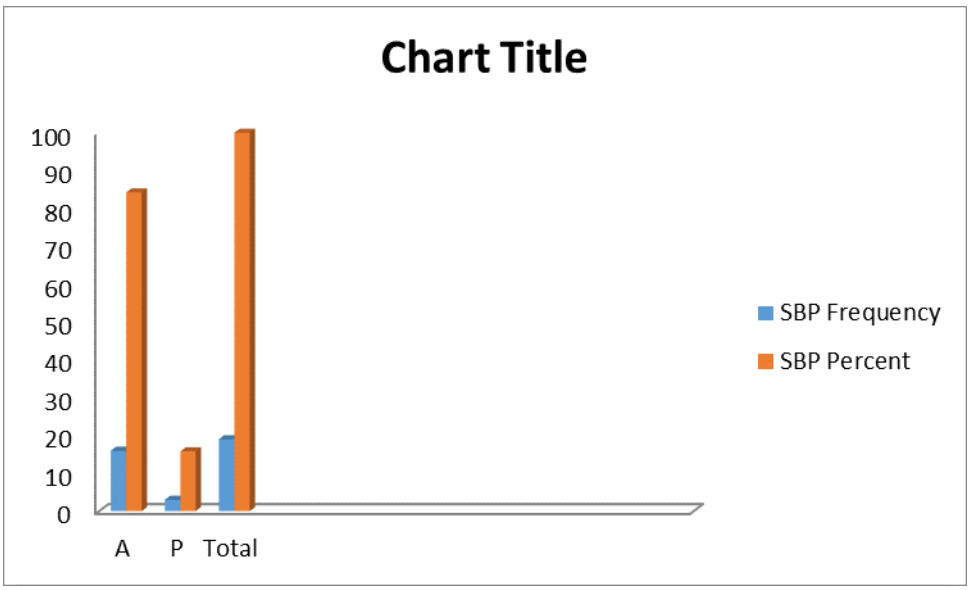


FIGURE 51 SHOWING FREQUENCY OF SBP IN PATIENTS WITH SERUM SODIUMLEVELS BETWEEN 126-130.

Among 19 patients with serum sodium levels between 126-130 all 3(15.8%) patients had SBP.

TABLE 45: SHOWING FREQUENCY OF PATIENTS BELONGS TO CHILD PUGHCLASS.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	B	6	31.6	31.6	31.6
	C	13	68.4	68.4	100.0
	Total	19	100.0	100.0	

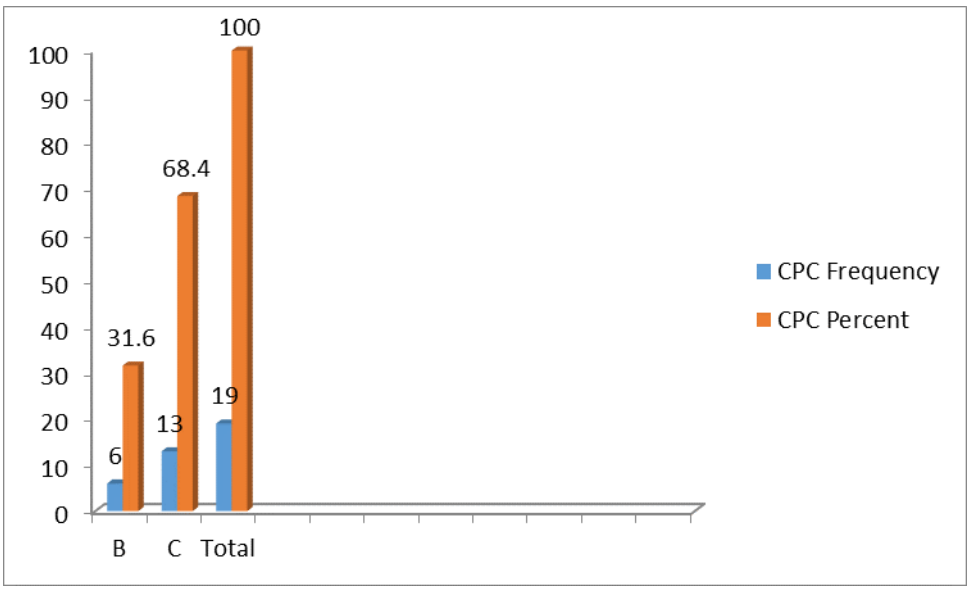


FIGURE 52 SHOWING FREQUENCY OF PATIENTS BELONGS TO CHILD PUGHCLASS.

Among 19 patients with serum sodium levels between 126-130 , 6(31.6%) patient of child pugh class B and 13 (68.4%) patients belongs to class C.

TABLE 46: SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	D	18	94.7	94.7	94.7
	E	1	5.3	5.3	100.0
	Total	19	100.0	100.0	

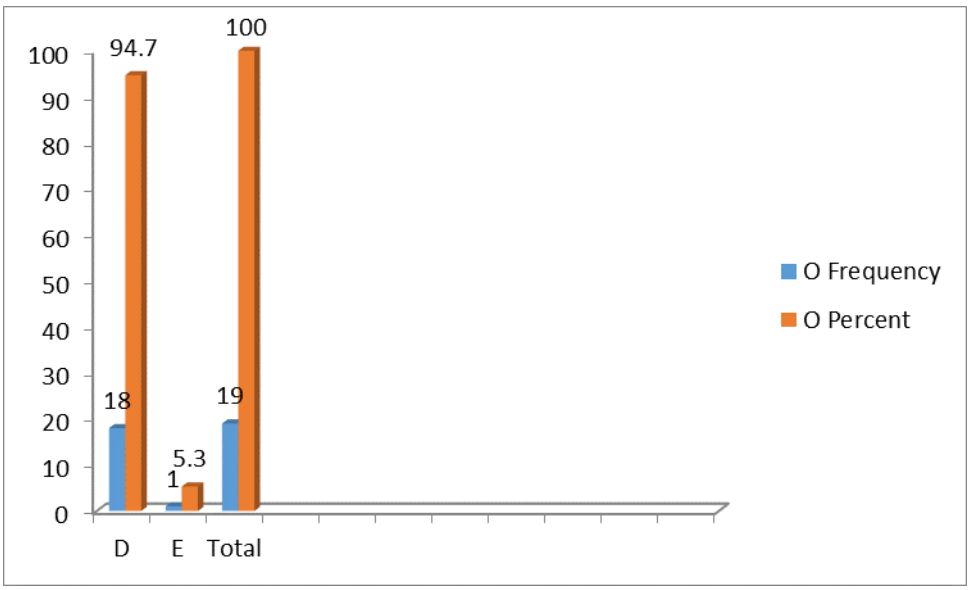


FIGURE 53 SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

Among 19 patients with serum sodium less between 126-130 , 18 patients were discharged and 1 patient expired.

Serum sodium levels on admission between 131-135

TABLE 47: SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	12	66.7	66.7	66.7
	P	6	33.3	33.3	100.0
	Total	18	100.0	100.0	

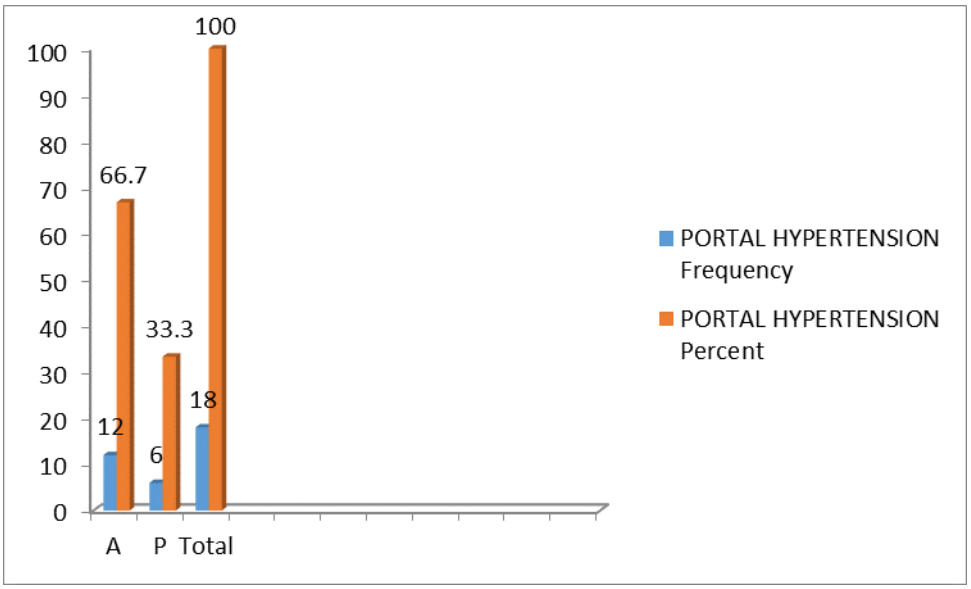


FIGURE 54 SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

Among total of 18 patients 6 (33.3%) patients had portal hypertension.

TABLE 48: SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTS WITH SERUM SODIUM LEVELS BETWEEN 131-135

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	13	72.2	72.2	72.2
	P	5	27.8	27.8	100.0
	Total	18	100.0	100.0	

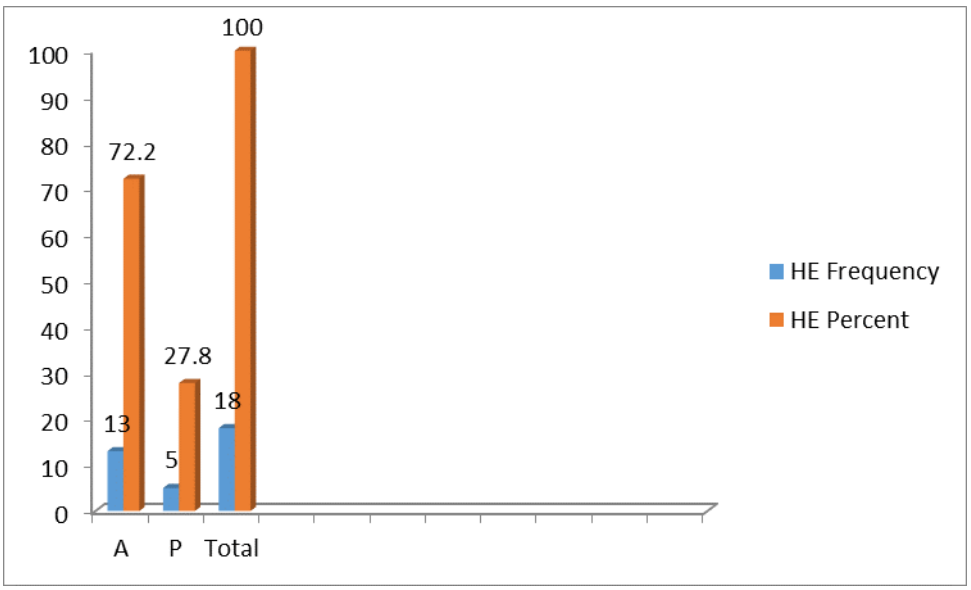


FIGURE 55 SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTS WITH SERUM SODIUM LEVELS BETWEEN 131-135.

Among total of 18 patients 5 (27.8%) patients had HEPATIC ENCEPHALOPATHY.

TABLE 49: SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	15	83.3	83.3	83.3
	P	3	16.7	16.7	100.0
	Total	18	100.0	100.0	

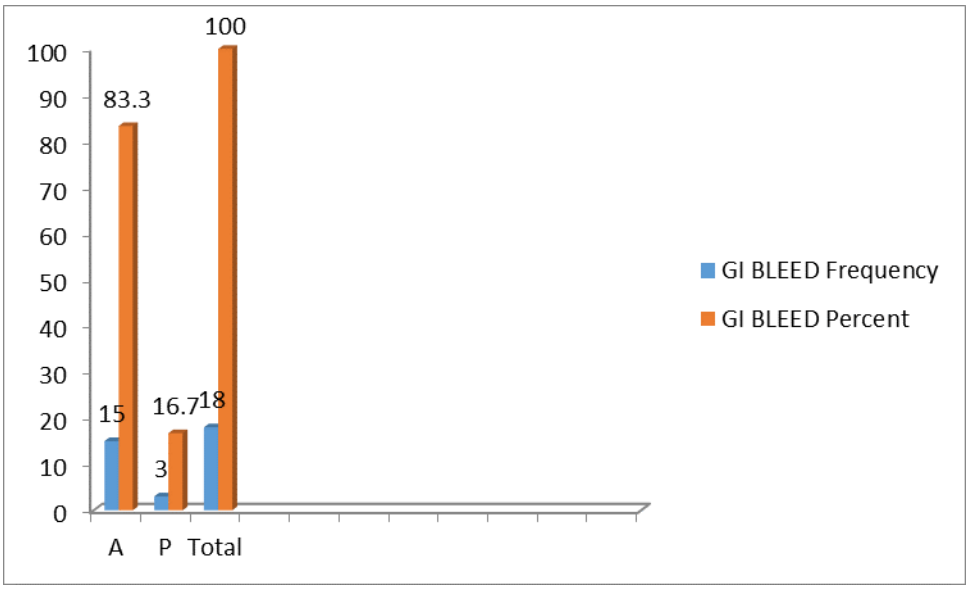


FIGURE 56 SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

Among total of 18 patients 3 (16.7%) patients had GI BLEED.

TABLE 50: SHOWING FREQUENCY OF HRS IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	14	77.8	77.8	77.8
	P	4	22.2	22.2	100.0
	Total	18	100.0	100.0	

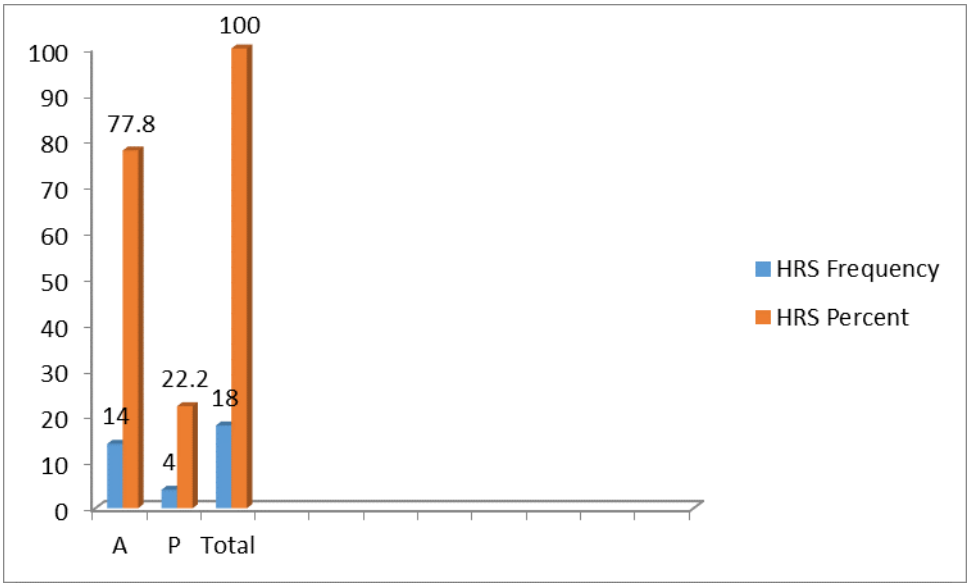


FIGURE 57 SHOWING FREQUENCY OF HRS IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

Among total of 18 patients 4 (22.2%) patients had HRS.

TABLE 51:SHOWING FREQUENCY OF SBP IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	18	100.0	100.0	100.0

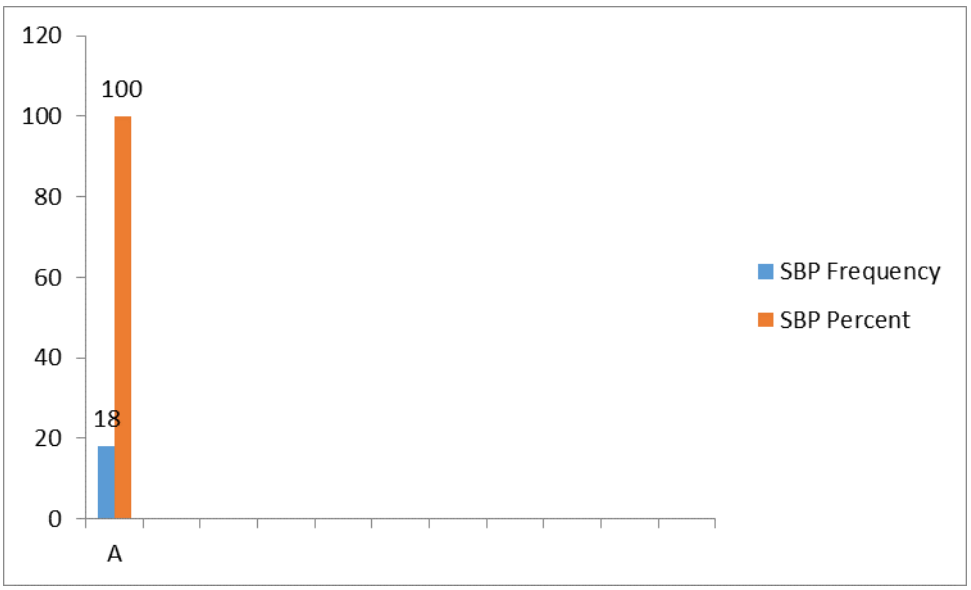


FIGURE 58 SHOWING FREQUENCY OF SBP IN PATIENTS WITHSERUM SODIUM LEVELS BETWEEN 131-135.

Among total of 18 patients no patient had SBP.

TABLE 52: SHOWING FREQUENCY OF PATIENTS BELONGS TO CHILD PUGHCLASS.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	B	7	38.9	38.9	38.9
	C	11	61.1	61.1	100.0
	Total	18	100.0	100.0	

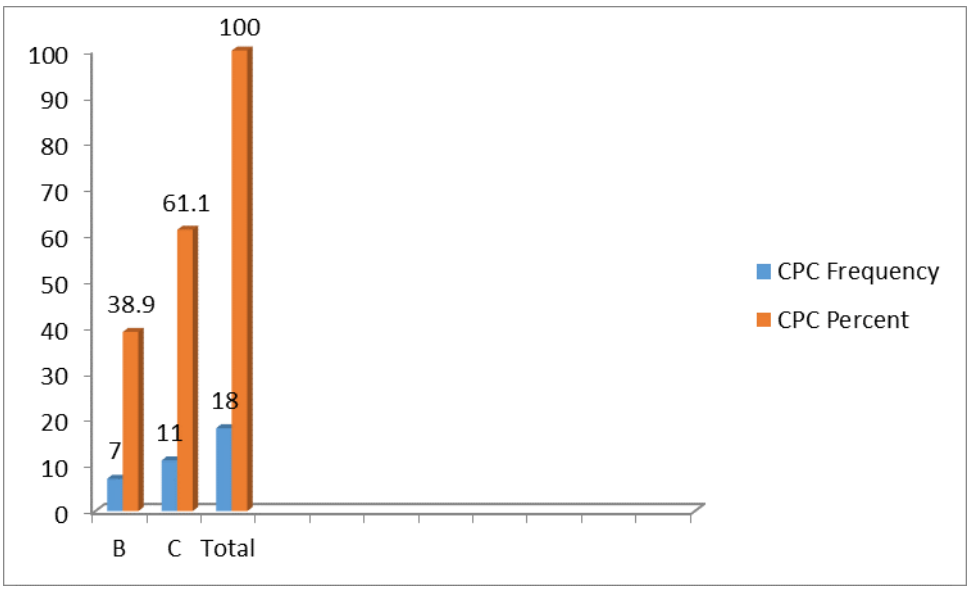


FIGURE 59 SHOWING FREQUENCY OF PATIENTS OF CHILD PUGHCLASS.

Among 18 patients with serum sodium levels between131-135 , 7(38.9%) patient belongs to child pugh class B and 11 (61.1%) patients of clas C.

TABLE 53: SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	D	17	94.4	94.4	94.4
	E	1	5.6	5.6	100.0
	Total	18	100.0	100.0	

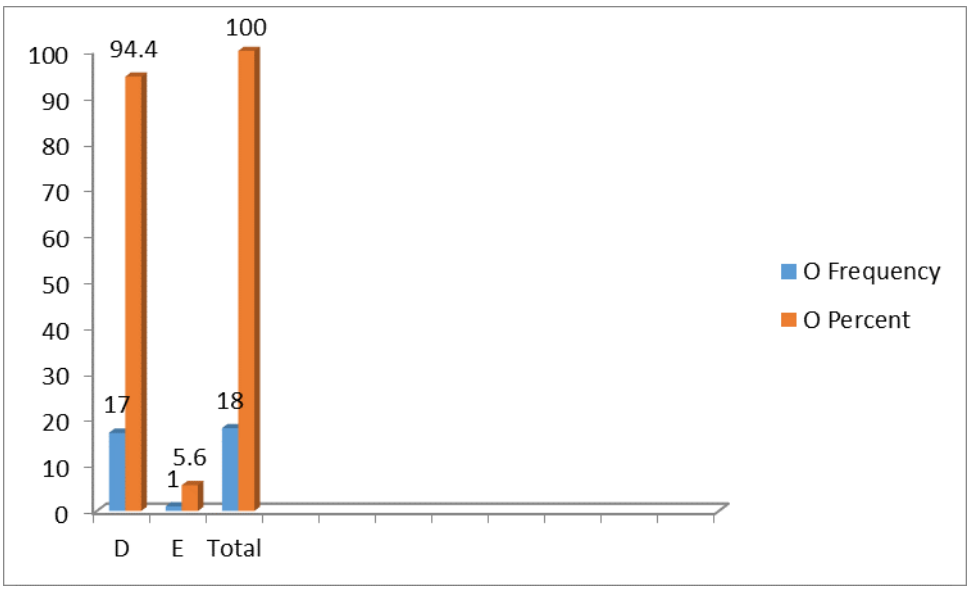


FIGURE 60 SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

Among 18 patients with serum sodium less between 131-135 , 17 patients were discharged and 1 patient expired.

Serum sodium levels on admission >135

TABLE 54: SHOWING FREQUENCY OF ASCITES IN PATIENTS WITHSERUM SODIUM LEVELS >135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	P	5	100.0	100.0	100.0

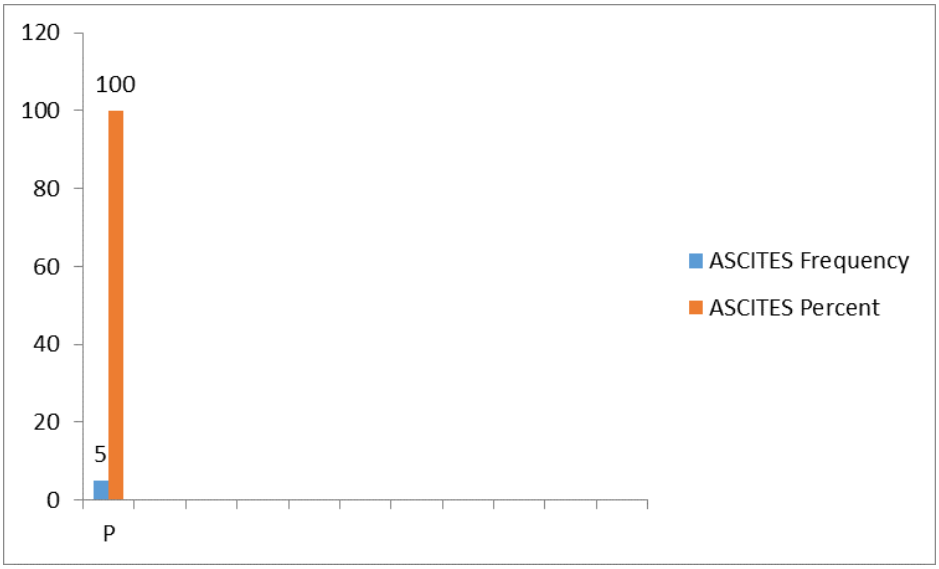


FIGURE 61 SHOWING FREQUENCY OF ASCITES IN PATIENTS WITHSERUM SODIUM LEVELS >135.

All 5 (100%) patients had ascites.

TABLE 55: SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTS WITHSERUM SODIUM LEVELS >135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	4	80.0	80.0	80.0
	P	1	20.0	20.0	100.0
	Total	5	100.0	100.0	

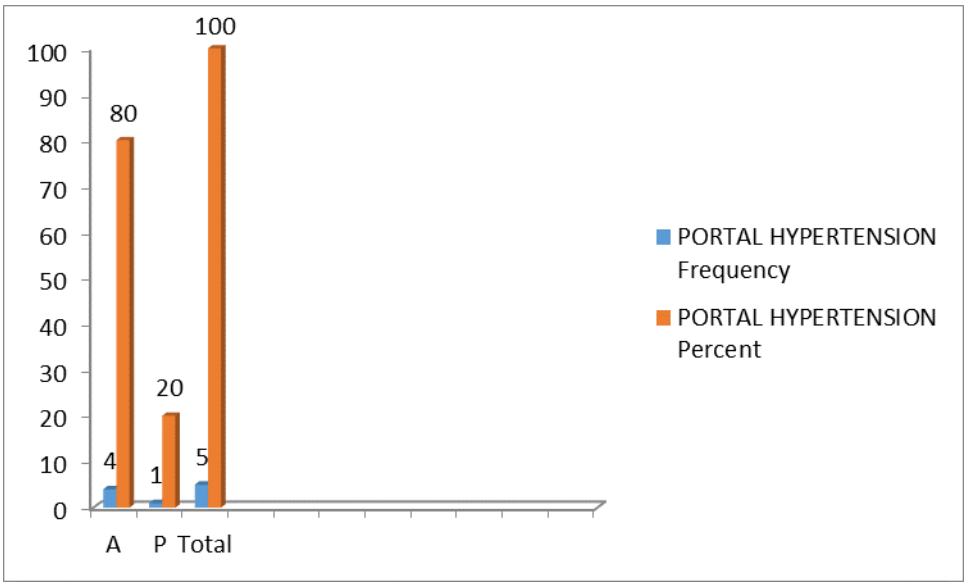


FIGURE 62 SHOWING FREQUENCY OF PORTAL HYPERTENSION IN PATIENTS WITHSERUM SODIUM LEVELS >135.

Among total of 5 patients 1 (20%) patients had portal hypertension.

TABLE 56: SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTS WITHSERUM SODIUM LEVELS >135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	5	100.0	100.0	100.0

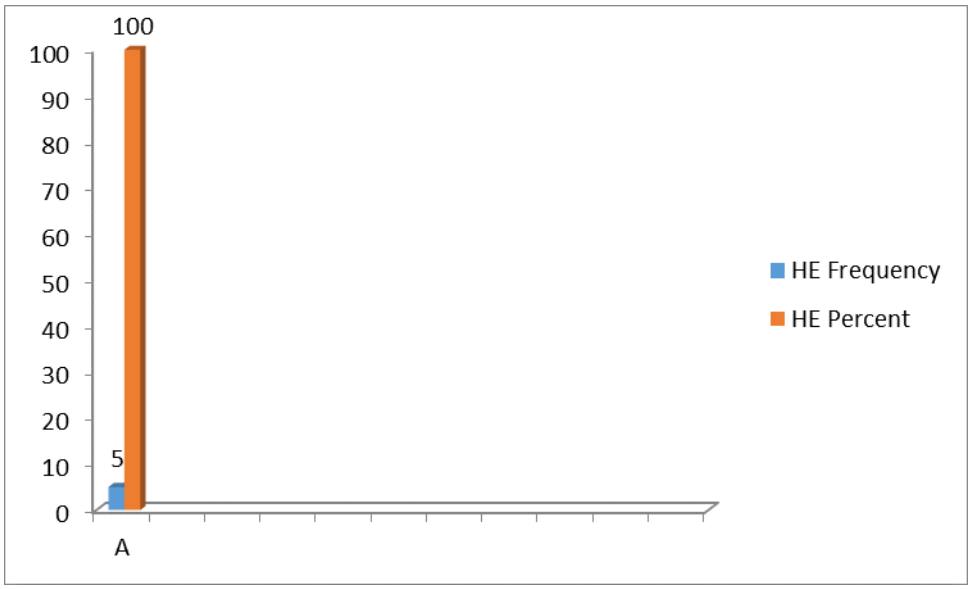


FIGURE 63 SHOWING FREQUENCY OF HEPATIC ENCEPHALOPATHY IN PATIENTS WITHSERUM SODIUM LEVELS >135.

Among total of 5 patients no patient had HE.

TABLE 57: SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITHSERUM SODIUM LEVELS >135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	4	80.0	80.0	80.0
	P	1	20.0	20.0	100.0
	Total	5	100.0	100.0	

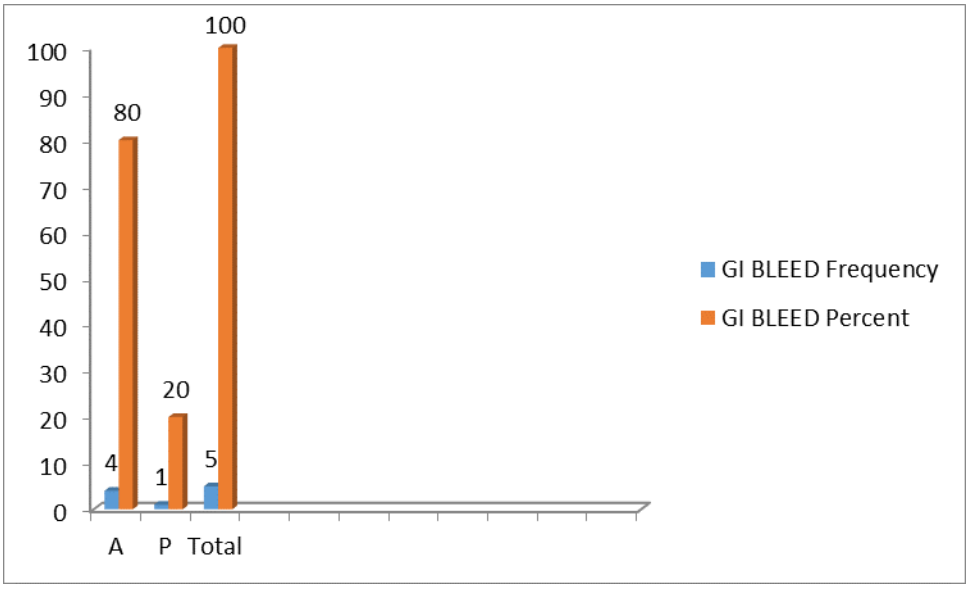


FIGURE 64 SHOWING FREQUENCY OF GI BLEED IN PATIENTS WITHSERUM SODIUM LEVELS >135.

Among total of 5 patients 1 (20%) patients had GI BLEED.

TABLE 58: SHOWING FREQUENCY OF HRS IN PATIENTS WITHSERUM SODIUM LEVELS >135.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	3	60.0	60.0	60.0
	P	2	40.0	40.0	100.0
	Total	5	100.0	100.0	

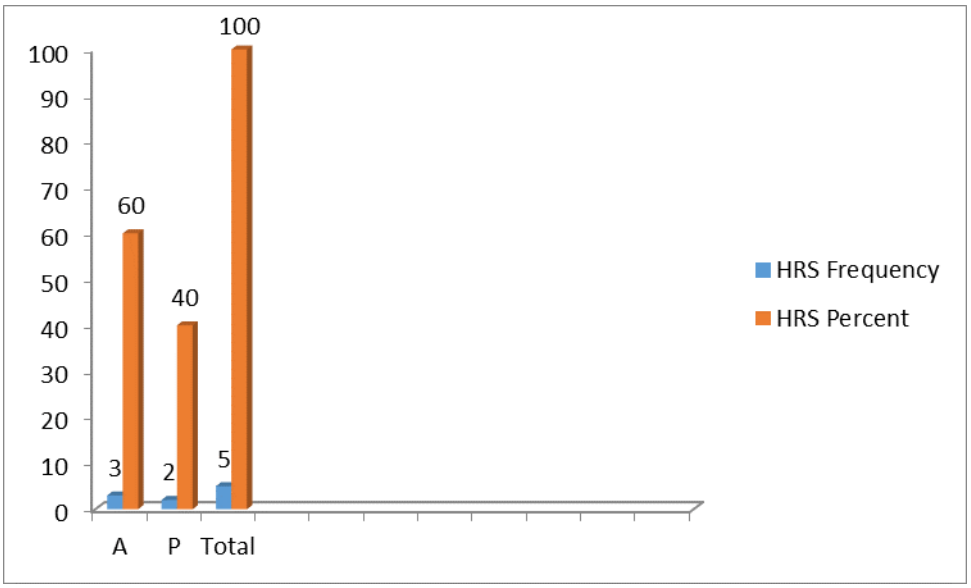


FIGURE 65 SHOWING FREQUENCY OF HRS IN PATIENTS WITHSERUM SODIUM LEVELS >135.

Among total of 5 patients 2 (40%) patients had HRS.

TABLE 59: SHOWING FREQUENCY OF SBP IN PATIENTS WITHSERUM SODIUM LEVELS >135

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	5	100.0	100.0	100.0

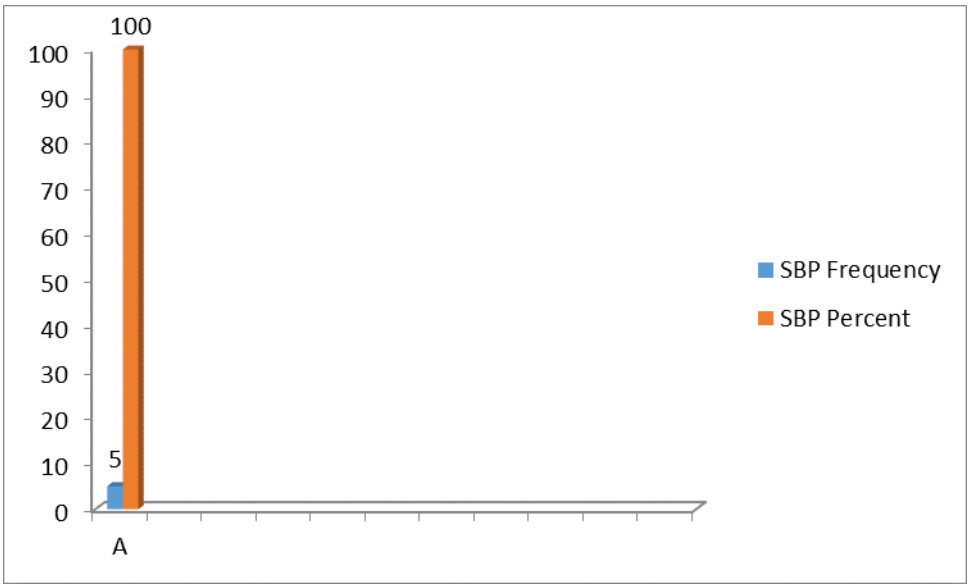


FIGURE 66 SHOWING FREQUENCY OF SBP IN PATIENTS WITHSERUM SODIUM LEVELS >135.

Among total of 5 patients no patient had SBP.

TABLE 60: SHOWING FREQUENCY OF PATIENTS BELONGS TO CHILD PUGHCLASS.

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	B	3	60.0	60.0	60.0
	C	2	40.0	40.0	100.0
	Total	5	100.0	100.0	

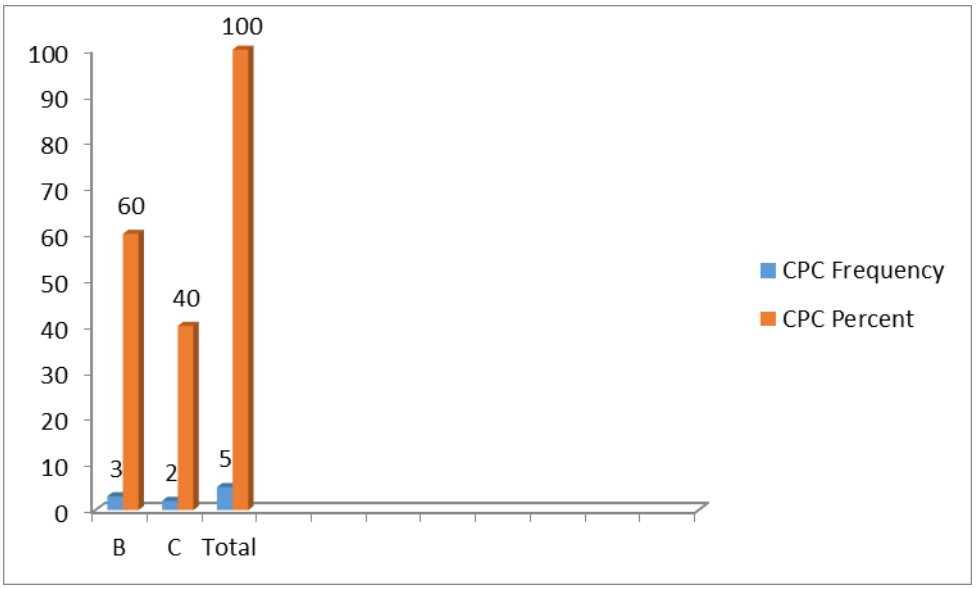


FIGURE 67 SHOWING FREQUENCY OF PATIENTS BELONGS TO CHILD PUGHCLASS.

Among 5 patients with serum sodium levels between >135 ,3(60%) patient belongs tochild pugh class B and 2 (40%) patients belongs to class C.

TABLE 61: SHOWING FREQUENCY OF OUTCOME OF THE PATIENTS

		Frequen cy	Percent	Valid Percent	Cumulative Percent
Valid	D	5	100.0	100.0	100.0

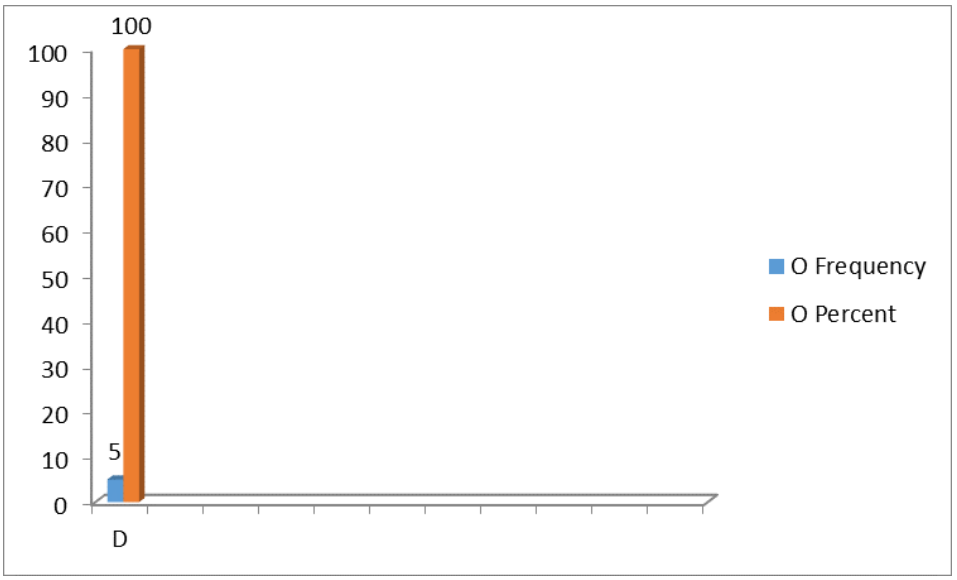


FIGURE 68 SHOWING FREQUEN CY OF OUTCOME OF THE PATIENTS

Among 5 patients with serum sodium less >135 , all 5 patients were discharged.

DISCUSSION

- Most of the patients with cirrhosis of liver showed abnormal sodium levels.
- 16% of the patients had serumsodium levels on admission <125, 38% had serum sodium levels between 125-130, 38% had 131-136, 8% ha d serum sodium levels >137.
- 12% of the patients had serumsodium levels on discharge <125, 28% had serum sodium levels between 125-130, 48% had 131-136, 12% had serum sodium levels >137.
- Age of the patients ranged between 30-70, maximum patients were between 30-50 age groups.
- All 50 (100%) patients had ascites, 29 (58%) had portal hypertension, 20(40%) had hepatic encephalopathy, 16 (32%) had GI BLEED , 17 (34%) had HRS , 5(10%) had SBP.
- Maximum seen complications were ascites, portal hypertension and least seen were SBP and HRS.
- All patients belonged to child pugh class of B and C. among 50 patients 17 (34%) belonged to child pugh class B and 33(66%) belonged to class C.
- Among 50 patients 45 patients were discharged and 5 were expired.
- Minimum sodium levels on admission were 117 and maximum were 138 and mean was 129.
- Minimum sodium levels on discharge were 117 and maximum were 143 and mean was 131.
- Minimum child pugh score was 7 and maximum score was15 and mean was 10.12
- Patients who belonged to child pugh class B had serum sodium levels mean of 131 at admission and 133 at discharge with child pugh score of 7.94.
- Patients who belonged to child pugh class C had serum sodium levels mean of 128.82 at admission and 130 at discharge with child pugh score of 11.24.

- Among discharged patients mean serum sodium levels on admission were 130.51 and mean serum sodium levels on discharge were 132.09 with mean child pugh score of 9.82
- Among expired patients mean serum sodium levels on admission were 123.80 with mean child pugh score of 12.80
- Among total of 50 patients with ascites, 17 patients of child pugh class B, 33 patients of child pugh class C.
- Among total of 29 patients with portal hypertension , 3 patients of child pugh class B, 26 patients of child pugh class C.
- Among total of 20 patients with hepatic encephalopathy , 1 patient ofchild pugh class B, 19 patientsof child pugh class C.
- Among total of 16 patients with GI BLEED , all 16 patients belonged to child pugh class C.
- Among total of 17 patients with HRS , 1 patients ofchild pugh class B, 16 patients of child pugh class C.
- Among total of 5 patients with SBP, 1 patients belonged to child pugh class B, 4 patients belonged to child pugh class C.
- Among total of 50 patients with ascites 45 were discharged and 5 were expired.
- Among total of 29 patients with portal hypertension 24 were discharged and 5 were expired.
- Among total of 20 patients with HE 15 were discharged and 5 were expired.
- Among total of 16 patients with GI BLEED 11 were discharged and 5 were expired.
- Among total of 17 patients with HRS 12 were discharged and 5 were expired.
- Among total of 5 patients with SBP 3 were discharged and 2 were expired.
- patients with serum sodium levels less than 125 were 8, among them 8 (100%) patients

had ascites, 8 (100%) had portal hypertension, 5 (62.5%) had HE, 6 (75%) had GI BLEED, 5(62.5%) had HRS, 2 (25%) had SBP, and 1 patient belonged to child pugh class B and 7 patients belonged to child pugh class C, 5 patients were discharged and 3 were expired.

- patients with serum sodium levels 126-130 were 19 , among them 19 (100%) patients had ascites, 14 (73.7%) had portal hypertension, 10 (52.6%) had HE, 6 (31.6%) had GI BLEED, 6(31.6%) had HRS, 3 (15.8%) had SBP, and 6 (31.6%) patient belonged to child pugh class B and 13 (68.4%) patients belonged to child pugh class C, 18 patients were discharged and 1 patient expired.
- patients with serum sodium levels 131-135 were 18, among them 18 (100%) patients had ascites, 6 (33.3%) had portal hypertension, 5 (27.8%) had HE, 3(16.7%) had GI BLEED, 4(22.2%) had HRS, no patients had SBP, and 7(38.9%) patient belonged to child pugh class B and 11 (61.1%) patients belonged to child pugh class C, 17 patients were discharged and 1 patient expired.
- patients with serum sodium levels >135 were 5, among them 5 (100%) patients had ascites, 1 (20%) had portal hypertension, no patient had HE, 1(20%) had GI BLEED, 2 (40%) had HRS, no patients had SBP, and 3(60%) patient belonged to child pugh class B and 2 (40%) patients belonged to child pugh class C, 5 patients were discharged.
- Maximum patients serum sodium levels were between 126-135.
- Patients with serum sodium levels less than 125 showed maximum mortality and maximum belonged to child pugh class C.
- No patients had hypernatremia.
- 92% patients had hyponatremia with serum sodium levels less than 135.

CONCLUSION

Cirrhosis of liver isassociated with abnormality in serum sodium levels. Hyponatremia is the abnormality seen in this study. Age and gender doesnot show any association withserumsodium levels. Serum sodium levels less than 135 meq/l showed increased frequency of complications like spontaneous bacterial peritonitis, hepatorenalsyndrome, hepaticencephalopathy, gastrointestinal bleed when compared to patients with normal sodium levels. Patients with serum sodium levels less than 130meq/l had more complications. Lowserumsodium levels were associated with increased child pugh score and class and increased mortality indicating inverse relation between serum sodium levels and severity of disease. Thus lower serum sodium levels acts as marker of poor prognosis in patients with cirrhosis of liver. Thus patients with hyponatremia in case of cirrhosis of liver should be treated as high risk population for complications and should be treated accordingly to reduce morbidity and mortality.

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ANNEXURE

SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE,VIJAYAPUR-586 103

INFORMED CONSENT FORM

TITLE OF THE PROJECT **:** EVALUATION OF SERUM SODIUM LEVELS AS PROGNOSTIC MARKER IN PATIENTS WITH CIRRHOSIS OF LIVER

PG GUIDE **:** DR. R. C. BIDRI

PG STUDENT **:** DR. MARINENI VICTORIA

PURPOSE OF RESEARCH:-

BENEFITS:-

I understand that my participation in this study will help the investigator to diagnose the disease better and will help in the management of the disease.

PROCEDURE:-

I understand that relevant history will be taken and I will undergo detailed clinical examination after which necessary investigations will be done and accordingly treatment will be given.

RISK AND DISCOMFORTS:-

I understand there is no risk involved and I will experience no pain during the procedures performed.

CONFIDENTIALITY:-

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

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

REQUEST FOR MORE INFORMATION:-

I understand that I may ask more questions about the study at any time Concerned. The researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which may influence my continued participation.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:-

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in this study at any time without prejudice. I also understand that the researcher may terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician, if this is appropriate.

INJURY STATEMENT:-

I understand that in the unlikely event of injury to me resulting directly from my participation in this study and if such injury were reported promptly, then medical treatment will be available to me, but no further compensation will be provided. I understand that by my agreement for my participation in this study, I am not waiving any of my legal rights.

I have explained to (patient's / relevant guardian's name) the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability in patient's own language.

Investigator / P. G. Guide	Date

I confirm that …….(Name of the PG guide / chief researcher) has explained to me the research, the study procedures that I undergo and the possible risks and discomforts as well as benefits that I may experience. I have read and I understand this consent form. Therefore, I agree to give my consent for my participation as a subject in this research project.

Participant / guardian	Date
Witness to signature	Date

SCHEME OF CASE TAKING

B.L.D.E.U'S SHRI B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA.

DEPARTMENT OF MEDICINE

PROFORMA

Name IP number

Age: Sex

Address : Occupation :

Date of Admission : Date of discharge:

Chief Complaints :

History of present illness:

Past history:

Treatment History:

Personal History:

Physical Examination:

On Examination :

VITALS:

Temperature:

Pulse:

Respiratory rate:

Blood pressure:

GENERAL CONDITION:

Pallor:	Yes/ No
Icterus:	Yes/ No
Cynosis:	Yes/No
Clubbing:	Yes/No
Lymphadenopathy:	Yes/No
Edema:	Yes/No
Pt condition on discharge	Improved/Worsened/same/Expired

SYSTEMIC EXAMINATION:

PER ABDOMEN EXAMINATION:

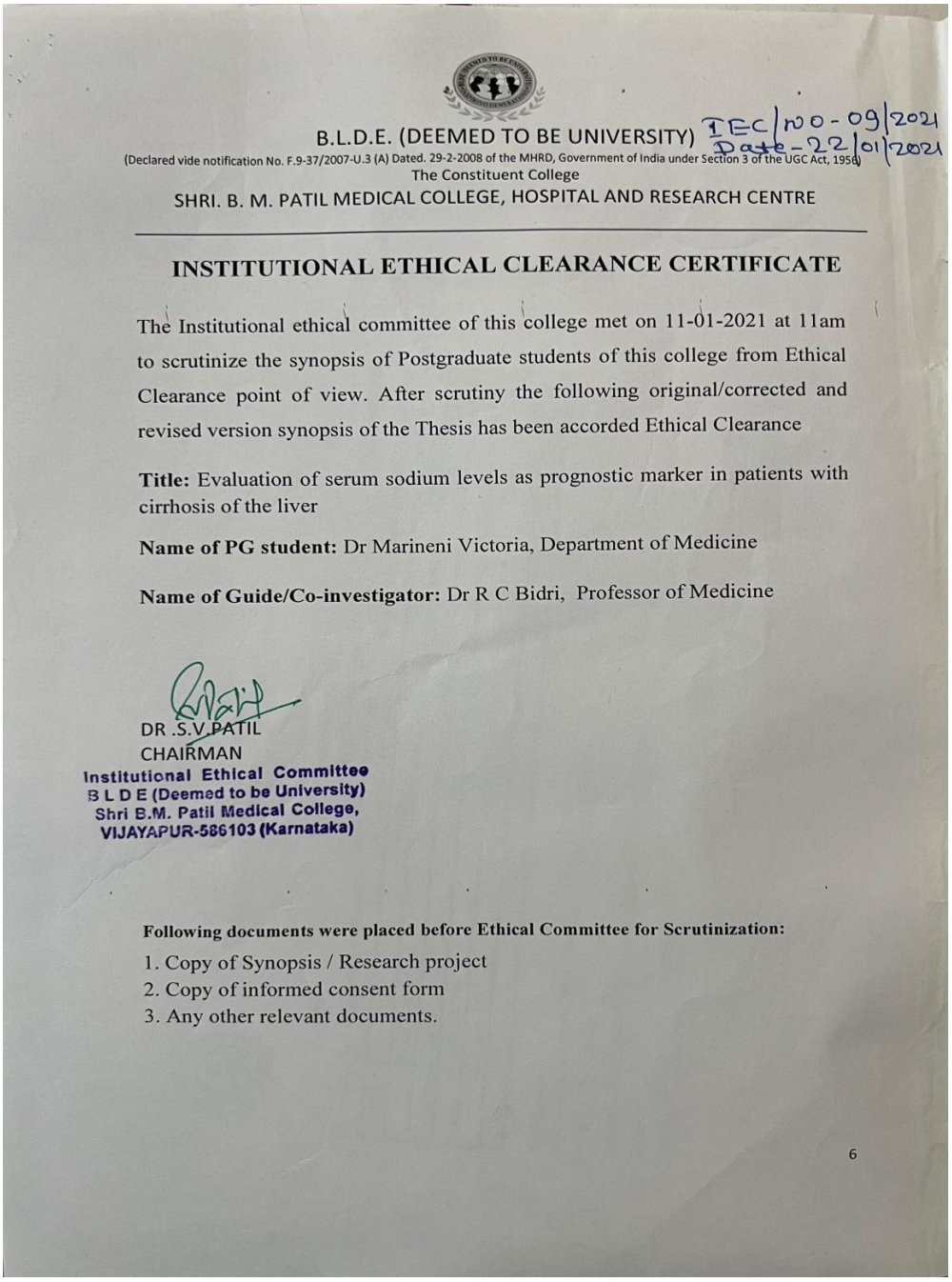
1. Inspection

2. Palpation:

3. Percussion:

4. Auscultation:

ETHICAL CLEARENCE CERTIFICATE



B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/NO-09/2021
Date-22/01/2021
(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)
The Constituent College
SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Evaluation of serum sodium levels as prognostic marker in patients with cirrhosis of the liver

Name of PG student: Dr Marineni Victoria, Department of Medicine

Name of Guide/Co-investigator: Dr R C Bidri, Professor of Medicine

DR .S.V.PATIL
CHAIRMAN
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

6

MASTERCHART

AGE	SEX	SERUM SODIUM ON ADMISSION	SERUM SODIUM ON DISCHARGE	ASCITES	PORTAL HYPERTENSION	HE	GI BLEED	HRS	SBP	CPS	CPC
51	M	137	138	P	A	A	A	A	A	8	B
52	M	126	121	P	P	P	P	P	P	11	C
41	M	135	140	P	A	P	A	P	A	11	C
38	M	123	123	P	P	A	P	P	P	14	C
35	M	135	143	P	P	A	P	A	A	11	C
40	M	137	132	P	P	A	P	P	A	10	C
45	M	120	126	P	P	A	P	A	A	11	C
80	M	126	125	P	P	P	P	P	P	13	C
28	M	123	126	P	P	P	P	P	A	15	C
22	M	130	134	P	P	P	P	A	A	11	C
45	M	129	132	P	A	A	A	A	A	9	B
31	M	137	132	P	A	A	A	P	A	10	C
60	M	130	128	P	P	P	A	A	A	12	C
64	M	122	134	P	P	P	A	A	A	7	B
50	M	135	136	P	A	A	A	A	A	10	C
56	M	122	126	P	P	A	A	A	A	10	C
38	M	135	138	P	A	A	A	A	A	7	B
55	M	129	125	P	P	P	A	P	A	10	C
40	M	129	133	P	P	P	P	A	A	10	C
35	M	135	136	P	P	A	P	A	A	10	C
49	M	117	117	P	P	P	P	P	P	14	C
43	M	131	132	P	A	P	A	A	A	11	C
30	M	133	129	P	A	A	A	A	A	7	B
57	M	128	127	P	A	A	A	A	A	9	B
42	M	125	126	P	P	A	P	A	A	10	C
35	M	130	132	P	P	A	A	A	P	9	B
43	M	123	134	P	P	P	P	P	A	13	C
36	M	128	129	P	A	A	A	A	A	9	B
30	M	135	136	P	A	A	A	A	A	9	B
42	M	128	132	P	P	A	A	P	A	9	B
45	M	135	136	P	A	A	A	A	A	7	B
47	M	135	143	P	A	P	A	P	A	10	C
32	M	128	129	P	P	A	A	A	A	10	C
42	M	131	121	P	P	P	P	P	A	13	C
30	M	128	133	P	A	P	A	P	A	11	C
30	M	132	132	P	P	P	A	P	A	12	C
36	M	136	135	P	A	A	A	A	A	8	B
42	M	132	128	P	P	A	A	A	A	10	C
50	M	122	120	P	P	P	P	P	A	13	C
54	M	128	132	P	P	A	A	A	A	10	C
46	M	127	135	P	A	A	A	A	A	8	B
35	M	135	136	P	A	A	A	A	A	7	B
39	M	128	134	P	P	P	A	A	A	11	C
45	M	132	133	P	A	A	A	A	A	7	B
55	M	126	122	P	P	P	P	P	A	13	C
62	M	134	138	P	A	A	A	A	A	10	C
39	M	138	135	P	A	A	A	A	A	7	B
47	M	133	130	P	A	A	A	A	A	8	B
52	M	132	133	P	P	A	A	A	A	10	C
60	M	127	130	P	P	P	A	A	A	11	C