CORRELATION BETWEEN APRI INDEX, MELD SCORE ANDCHILD PUGH SCORE IN CIRRHOSIS OF LIVER - A CROSS SECTIONAL STUDY AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI.

By

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LIST OF ABBREVIATIONS

AAR	Aspartate aminotransferase -to- alanine aminotraferase ratio
ALBI	Albumin Bilirubin Index
ALP	Alkaline Phosphatase
ANA	Antinuclear antibody
ANCA	Anti neutrophil cytoplasmic antibody
Anti-LKM	Liver Kidney Microsome
ANTI-SM	Anti- Smith
APRI	Aspartate Aminotransferase to Platelet Ratio Index
ARF	Acute Renal Failure
AST	Aspartate Aminotransferase
AUC	Area Under Curve
BP	Blood Pressure
CALD	Chronic Alcoholic Liver Disease
CNS	Central Nervous System
CO	Cardiac Output
CP	Child Pugh
CT	Computed tomography
EV	Esophageal varices
FI	FibroIndex
GB	Gall Bladder
GGT	Gamma-GlutamylTransferase
GI	Gastrointestinal
h/O	History of
HBV	Hepatitis B virus
HCC	Hepatocellular Carcinoma
HCV	Hepatitis C virus
HE	Hepatic Encephalopathy
HVPG	Hepatic vein pressure gradient
INR	International Normalized Ratio
IVC	Inferior Vena Cava
Ln	Logarithm
LSM	Liver Stiffness Measurement

MELD	Model for End Stage Liver Disease
MRI	Magnetic Resonance Imaging
NAFLD	Non-alcoholic Fatty Liver Disease
NPV	Negative predictive value
OPD	Out Patients Department
PHLF	Post hepatectomy liver failure
PHTN	Portal Hypertension
PPV	Positive predictive value
PT	Prothrombin Time
RAAS	Renin Angiotensin Aldosterone System
RBF	Renal Blood Flow
ROC	Receiver Operating Characteristic
SBP	Spontaneous Bacterial Peritonitis
TLC	Total Leucocyte Count
UGI	Upper Gastrointestinal
UGIB	Upper Gastrointestinal Bleed
UKELD	United Kingdom Model for End-Stage Liver Disease
ULN	Upper limit of normal

ABSTRACT

Background and Objectives

Cirrhosis is a condition characterised with fibrosis and formation of nodules diffusely. It is the 14th most common cause of death all over the world. Last consequence of fibrosis of the liver parenchyma is cirrhosis. Liver biopsy is the gold standard used for assessing liver necrosis, inflammation & fibrosis. But biopsy is an invasive procedure subjective to inter-observer variation and errors during sampling. A perfect non-invasive investigation for diagnosis of liver fibrosis should be easily available, uncomplicated, interpretable, less cost, and precise. Aspartate aminotransferase to Platelet Ratio Index (APRI) is considered a new index for predilection of significant fibrosis and cirrhosis. The objective of present study was to calculate APRI INDEX, Child Pugh score and MELD Score in cirrhosis of liver patients and to find a correlation between APRI Index, Child Pugh Score and MELD score in cirrhosis of liver patients.

Material and Methods-

The study was done on 102 patients attending OPD and admitted in KLE's Dr. Prabhakar Kore Hospital and MRC, Belagavi over a period of one year from January 2019 to December 2019. A thorough medical history was obtained, clinical examination and investigations were performed on the study subjects. Ultrasonography of abdomen was done with special reference to look for chronic liver disease. Complete blood counts including platelet count, liver function tests and PT/INR was done in order to calculate the APRI, Child Pugh and MELD score. The statistical analysis was done using SPSS version 20.0 software. The chi square test and the multiple logistic regression analysis were performed.

Results

The mean age in our study population was 47.32 ± 12.91 years with 86.27% being males among 102 patients enrolled in the study. Alcohol was the most common etiology for cirrhosis in our study, i.e. 72.55 %. We observed that, maximum 51subjects (50%) had APRI >1.5 which indicates advanced liver fibrosis/cirrhosis; 33subjects (32.35%) had APRI between 0.7-1.5 and 18(17.65%) had APRI <0.7. Majority of subjects belonged to Child Pugh C seen in 53(51.96%), followed by Child Pugh B seen in 40(39.22%) and Child Pugh A was in 9(8.82%). The mean Child Pugh score was 9.81 with standard deviation of 2.34 in our study. 64(62.75%) had MELD score between 18-36; followed by 32(31.37%) had MELD score <18; and 6subjects (5.88%) had MELDscore >36. Mean MELD was 23.19 with standard deviation of 9.11 in this study. APRI scores and Child Pugh scores obtained a strong correlation (r = 0.2106) and was significant as indicated by a ‘p’ value of 0.0336. APRI scores and MELD scores obtained a weak correlation (r = 0.1767) and is not significant as indicated by a ‘p’ value of 0.0756. Child Pugh scores and MELD scores obtained a strong correlation (r = 0.6779) and is significant as indicated by a ‘p’ value of 0.0001. The kappa agreement (similarity) between APRI and Child Pugh Score was 51.96% which suggests moderate strength of agreement/reliability among the two variables, the kappa agreement (similarity) between Child Pugh and MELD score was 30.39% which suggests fair strength of agreement/reliability among the two variables and the kappa agreement (similarity) between APRI and MELD score was 37.25% which suggests fair strength of agreement/reliability among the two variables.

Conclusion

Thus, we were able to conclude saying that there was significant correlation between APRI, Child Pugh and MELD scores in cirrhosis of liver. We also found that there was moderate strength of agreement/reliability among APRI and Child Pugh score. Our study also found that there was fair strength of agreement/reliability between APRI, MELD and Child Pugh, MELD scores. Currently, the Child Pugh score is being used for severity assessment.

Hence, a simple, non-invasive laboratory test such as APRI, which has a significant association with the severity of cirrhosis of liver, can be considered for use of prognostication of cirrhosis of liver in future clinical practice.

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INTRODUCTION

Cirrhosis is a condition characterisedwithfibrosis and formation of nodules diffusely.[5]It is the 14th most common cause of death all over the world.[4] Last consequence of fibrosis of the liver parenchyma is cirrhosis. Liver biopsy is the gold standard used for assessing liver necrosis, inflammation& fibrosis.[5]But biopsy is an invasive procedure subjective to among the observer variation anderrors during sampling.Length of biopsy specimen and fragmentation governsitsauthenticity and histopathological inferences. Complications such as bleeding, pneumothorax, infective peritonitis are some of the limitations for using biopsy. Other non-invasive biochemical investigations like Fibro Test, hepascore, transient elastography, Fibrospect, FornsIndex, Enhanced Liver Fibrosis are being used, needcomplex calculations and are highly costly biochemical assays.[6]

A perfect non-invasive investigation for diagnosisofliver fibrosis should be easily available, uncomplicated,interpretable, less cost, and precise. Aspartate aminotransferase to Platelet Ratio Index (APRI) is considered a new index for predilection of significant fibrosis and cirrhosis. APRI is a straightforward, easy bedside diagnostic aidwhich is being victoriouslytested in populations of Asian and western Continents. Very few studies are noted in samples of India.[6] Therefore, in our study "We have tried to evaluate APRI as a non-invasive bedside aid for cirrhosis in a subset of Indian population and statistically determine its sensitivity and specificity as a diagnostic tool and also we have tried to find the correlation between APRI, Child Pugh and MELD score in cirrhosis of liver."Various scores are available for predicting the prognosis of cirrhosis of liver.[4]

Most commonly used scores are-

1. MELD SCORE-[4]

- CALCULATED USING
- Sr. Creatinine, Sr. Bilirubin and INR.

MELD=3.78 X ln[Sr. bilirubin (mg/dl)+11.2 Xln [INR]+9.57 X ln [Sr.Creatinine(mg/dl)] + 6.43

(ln=logarithm)

MELD- 1-<18

2-18-36

3->36

2. Child Pugh (CP) Score-[4]

This scoring system includes 5 factors:

1) Serum bilirubin,
2) Serum albumin,
3) Degree of ascites,
4) Grade of hepatic encephalopathy
5) Prothrombin time

- Child Pugh class A (score of 5-6)
- Child Pugh class B(score of 7-9)
- Child Pugh class C(score of ≥10)

3. Recently, an index comprising routinely available laboratory tests was developed to computate liver Fibrosis in cirrhosis of liver patients, namely ASPARTATEAMINOTRANSFERASE TO PLATELET RATIO INDEX (APRI).[4]

The APRI score uses a variable that is thought to be the most important predictor indetermining the presence or absence of fibrosis in patients with chronic liver disease, namely AST levels and platelet counts. Increased AST levels and reduced platelet counts can be a sign that liver damage is in the advanced stage.[3]

The main mechanism of decreasing platelets or thrombocytopenia in patients withchronic liver disease is the presence of platelet sequestration in the spleen and a decrease in the production of thrombopoietin in the liver. Platelet reduction can also be caused by various other conditions such as cirrhosis of the coagulopathy, bone marrow suppression by chronic infection of the hepatic C virus and the liver anti-cancer agents as well as antiviral treatment with interferon-based therapy can also contribute to the development of thrombocytopenia in cirrhosis patients.[3]

In addition, the development of a state of fibrosis in the liver can also reduce AST clearanceand injure mitochondria so that it will increase serum ASTlevels. AST is an enzyme that can be found in the liver that functions as a catalyst for aspartate and alpha-ketoglutarate to oxaloacetates and glutamates.[3]

ASPARTATE AMINOTRANSFERASE AND PLATELET RATIO INDEX-[4]

CALCULATEDUSING=

$$\frac{\frac{\text{AST}}{\text{ULN OF AST}}}{\text{PLATELET } (10^9/\text{L})} \times 100$$

(ULN=Upper Limit of Normal)

APRI = A-<0.7

B-0.7 TO 1.5

C->1.5

Thus, we have conducted a study in our population with 102 subjects to gauge"APRI as a non-invasive aid for cirrhosis and to establish a stronger correlation between APRI,Child Pugh score and MELD score."

OBJECTIVES

1. "To calculate APRI INDEX, Child Pugh score and MELD Score in cirrhosis of liver patients."

2. "To find a correlation between APRI Index, Child Pugh Score and MELD score in cirrhosis of liver patients."

REVIEW OF LITERATURE

EMBRYOLOGY-

The liver starts to develop as a "hollow endodermal bud from the foregut (duodenum) during the third week of gestation. The bud separates into two parts: hepatic and biliary. The hepaticpart consists bipotential progenitor cells which divide into hepatocytes or ductal cells, later form the early primitive bile duct structures."[9] This group of rapidly proliferating cells penetrates adjoining mesodermal tissue (the septum transversum) and is met by ingrowing capillary plexuses from the vitelline and umbilical veins, which further result in formation of sinusoids.

The biliarypart, forms gallbladder and extrahepatic bile ducts.The intrahepatic portion of biliary duct system development startsby8weeks from the flat surface of the epithelium that lines duct lamina abutting to tracts of portal system.[10]

Circulating monocytes and yolk sac macrophages give rise to the Kupffer cells.

Hepatic stellate cells are mesodermal derivatives from sub-mesothelial cells found below the facet of the developing liver.[11]

Haemopoiesis occurs predominantly in the fetal liverupto the 12th week; by fifth month bone marrow haemopoietic activity initiates.

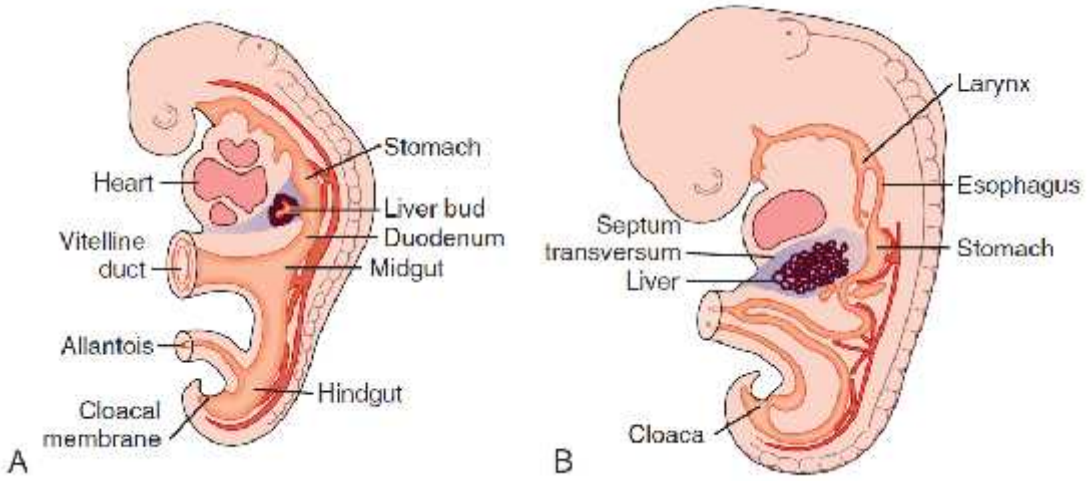


Fig 1-Embryology of liver

ANATOMY

The liver, a huge viscera in humans, is 1200–1500g in weight .[9]Is guarded by the ribs in the right upper quadrant, lies around the level of nipples.Anatomically classified into 2 lobar structures- left which is 1/6th that of right.

Caudate lobe posteriorly and the quadrate lobe inferiorly are the lower partsin right lobe. Anteriorlyfalciform ligament, posteriorly ligamentum venosum fissure, and inferiorly ligamentum teres fissure separates right and left lobes.[9]

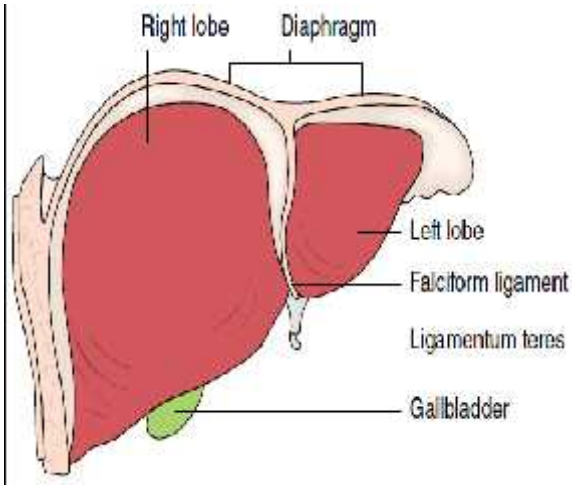


Fig 2.1- Anterior surface of liver

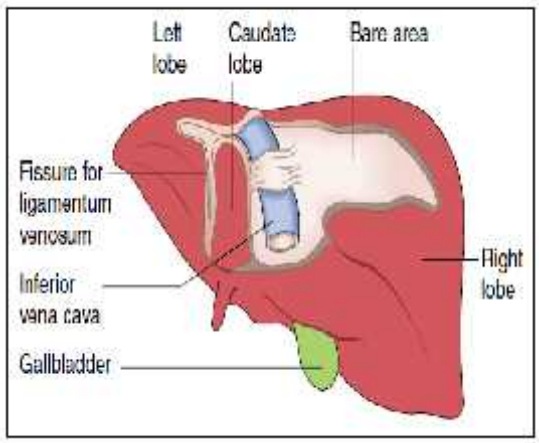
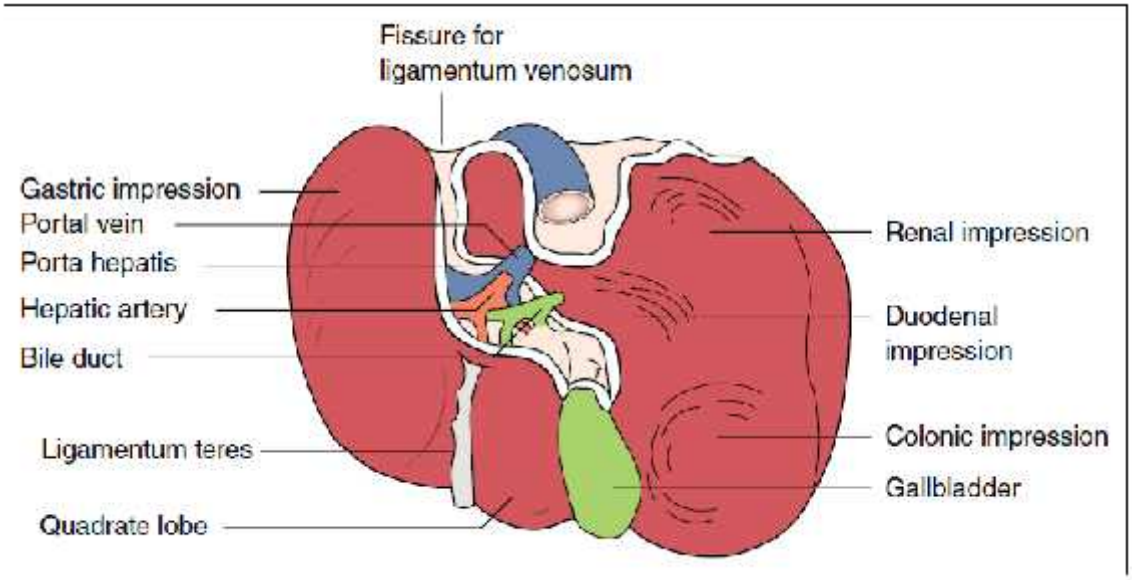


Fig 2.2- Posterior surface of liver

Fig 2.3- Inferior surface of liver



It has a dual blood supply.

Drained venous blood from the intestines and spleen comes into the **PORTAL VEIN**	Branch of coeliac axis- **HEPATIC ARTERY**

Porta hepatis is the entry point for the above said 2 vessels. Inside the porta hepatis the 2 major blood carrying channels divide which supply the quadrants.[9] Right side and left side of hepatic bile ducts join together and give rise to Common hepatic duct.

Nerve supply of liver consists of hepatic nerve plexus containing fibres from the sympathetic ganglia T7–T10, which synapse in the coeliac plexus, the right and left vagus and the right phrenic nerve.

The ligamentum venosum is a remainder of ductus venosus of the fetus which originates from the left branch of the portal vein and joins with inferior vena cava at the ingress of left hepatic vein.[9]

Ligamentum teres is a residue of fetal umbilical vein, runs in the free border of falciform ligament from the umbilicus to the loweredge of liver and annexes the left

branch of portal vein. Small veins going with it connect the portal vein with veins surrounding the umbilicus which become pre-eminent when the portal venous system is cloggedwithin liver.[9]

Veins from liver drain into right and left hepatic veins which originateout ofdorsal part of liver into IVCclose to its point of jotting into the right atrium. Lymphatic vessels end in small groups of glands surrounding the porta hepatis. Efferent vessels drain into glands encircling the coeliac axis. Superficial hepatic lymphatics pass into diaphragm in the falciform ligament and terminate in the glands of mediastinum.[9]

One more group of lymphatics go along theIVCthc glar thin the thoracic portion of IVC.

IVC groovesinto the right of the caudate lobe about 2 cm from the midline. Gallbladder is situated in a pit that extends from the loweredge of the liver to the right end of porta hepatis.

Liver is devoid of peritoneal covering in three places[9]-

1. Bare area- reclining medial to IVC fossa
2. IVC fossa.
3. GB fossa.

Functional anatomy of liver which is divided into sectors and segments-"**Couinaud classification**[12] defines- eight segments

- segments I–IV in the left lobe,
- V–VIII in the right lobe"

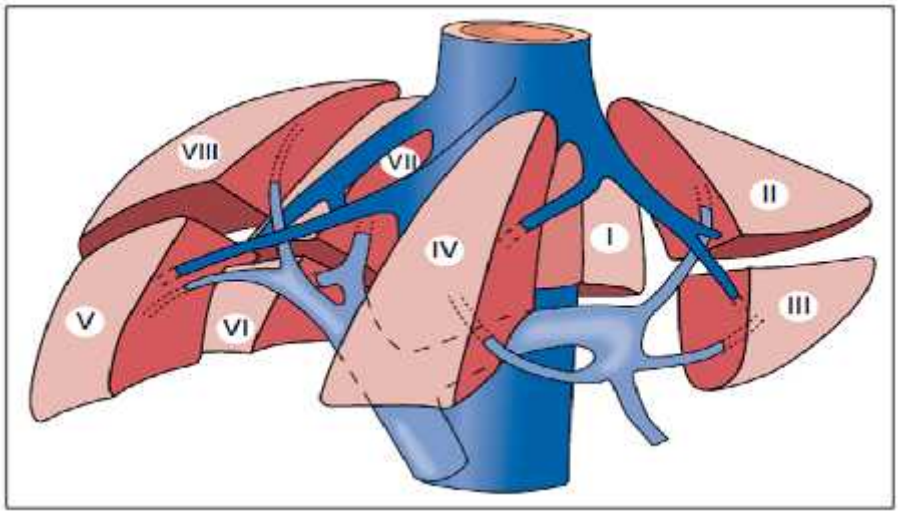


Fig 2.4 Functional anatomy of liver

The "**Bismuth classification**[13] divides the liver into four sectors.

- Right side- anterior and posterior
  - Right anterior sector contains segments V and VIII;
  - Right posterior sector contains segments VI and VII;
- Left side- medial and lateral
  - Left medial sector has segment IV;
  - Left lateral sector has segments II and III."

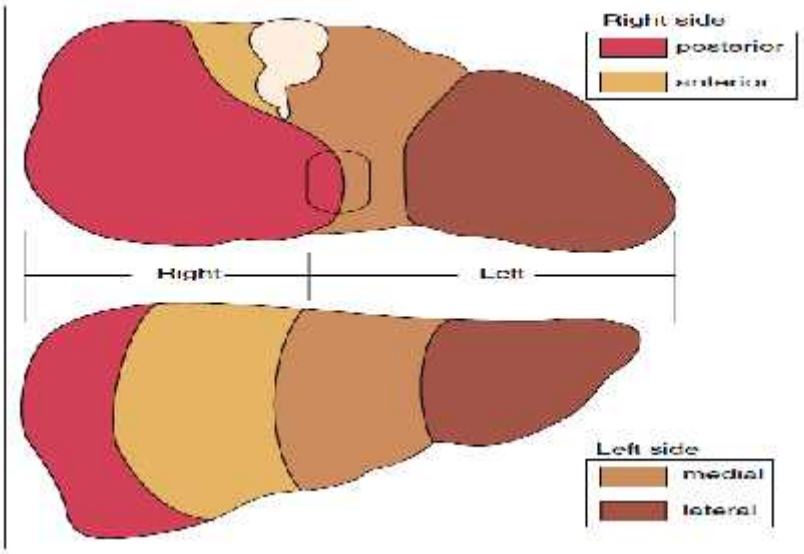


Fig 2.5 Bismuth classification

Defining according to this, “the right and left side of the liver are split along a slightly oblique line drawn from the inferior vena cava above to the gallbladder bed below. The right and left side are maverick with regard to portal and arterial blood supply, and also the bile drainage.

Three planes sunder the four sectors and contain the three major hepatic vein branches.No vascular anastomosis present between the macroscopic vessels of the segments and communication exists at the sinusoidal level. Segment I is analogous with the caudate lobe, is separate from the other segments and does not receive blood directly from the major portal branches or drain by any of the three major hepatic veins.”[9]

Liver Histology

Hepatocytesform 60% of the entire structure. [9]

They are multiangular, around30 micrometers in caliber. Kernel is sole or few of the times multiple. Division occurs by mitosis.

Time till death of hepatocytes is nearly 150 days.

The hepatocyte has three surfaces:

1. Sinusoid and space of Disse
2. Canaliculus
3. Neighbouring hepatocytes

Basement membrane is absent.

According to Wiley “Sinusoids are lined by endothelial cells with small pores for macromolecule diffusion from blood to hepatocytes.

On the vascular side of the sinusoids are the phagocytic cells of the reticulo- endothelial system (Kupffer cells) and pit cells (NK or natural killer cells) which are cytotoxic lymphoid cells.[9]

Normal human liver has nearly 202 x 10^3 cells in each milligram, out of which 171 x 10^3 areparenchymal and 31x$10^3$are sinusoidal, including Kupffer cells.[9]The Space of Disse between hepatocytes and sinusoidal endothelial cells containsfat storing cells, Ito cells, and lipocytes. These cells are known to store vitamin A and on activation in disease state become collagen synthesizing myofibroblasts.[9] Hepatic lymphatics are seen in the periportal connective tissue and are lined throughout by endothelium.

Tissue fluid exudes through the endothelium into the lymph vessels."[9]

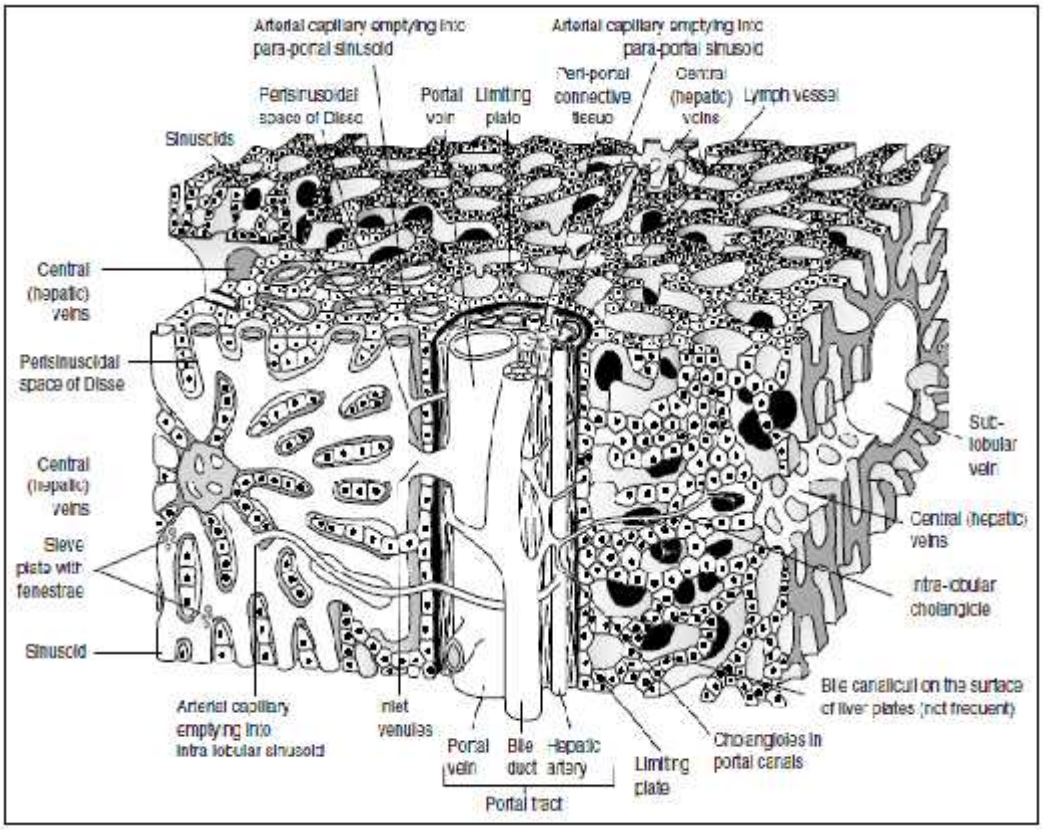
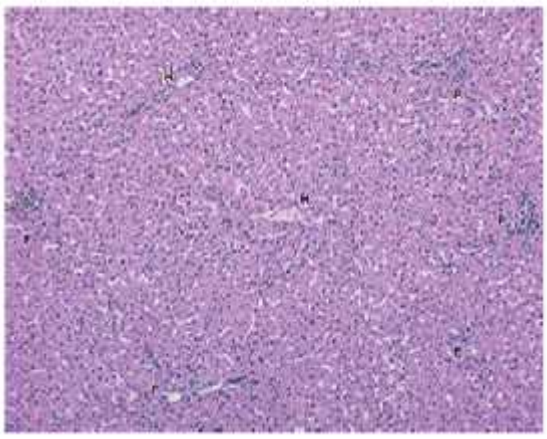


Fig 2.6 Structure of normal human liver



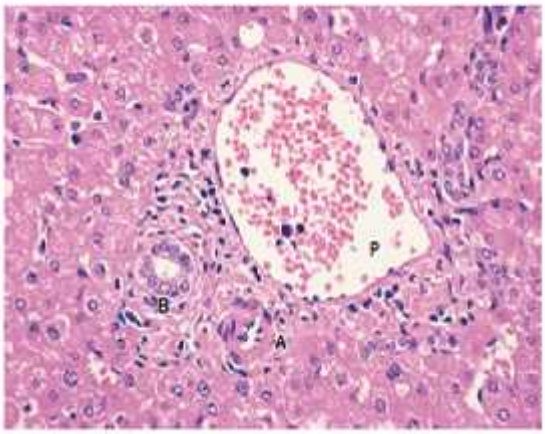


Fig 2.7 Normal Liver Histology **Fig2.8 Normal Portal Tract**

CIRRHOSIS OF LIVER

ETIOLOGY OF CIRRHOSIS-[9]

1. Viral Hepatitis (B, C, D)
2. Alcohol
3. Non alcoholic steatohepatitis
4. Metabolic- Iron overload (hemochromatosis)

   Wilson's disease

   Alpha 1 Antitrypsin deficiency

   Tyrosinemia

   Type 4 Glycogenesis

   Galactosemia
5. Primary biliary cirrhosis
6. Primary sclerosing cholangitis
7. Hepatic venous outflow obstruction
8. Autoimmune hepatitis
9. Toxins and drugs- Methotrexate, Amiodarone

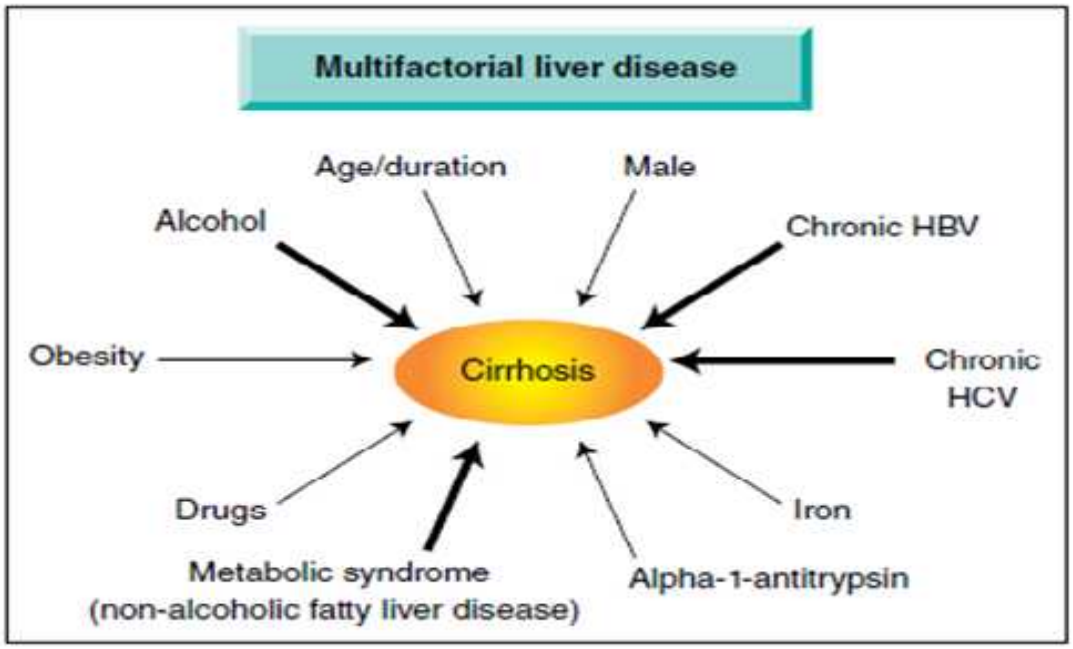


Fig 2.9

PATHOGENESIS OF CIRRHOSIS-

The liver cells involvedin pathogenesisof cirrhosis include both parenchymal and non parenchymal cells. The hepatic sinusoids have three different nonparenchymal cell lining:

a) Liver sinusoidal endothelial cells (LSECs),

b) Kupffer cells (KCs), and

c) Hepatic stellate cells (HSCs).

The cells which are most commonly implicated in the formation of cirrhosis are stellate cells. The stellate cell is activated into myofibroblast due to the action of inflammatory cytokines and this process leads to collagen deposition, which is the main reasonfor development cirrhosis of liver.[9]

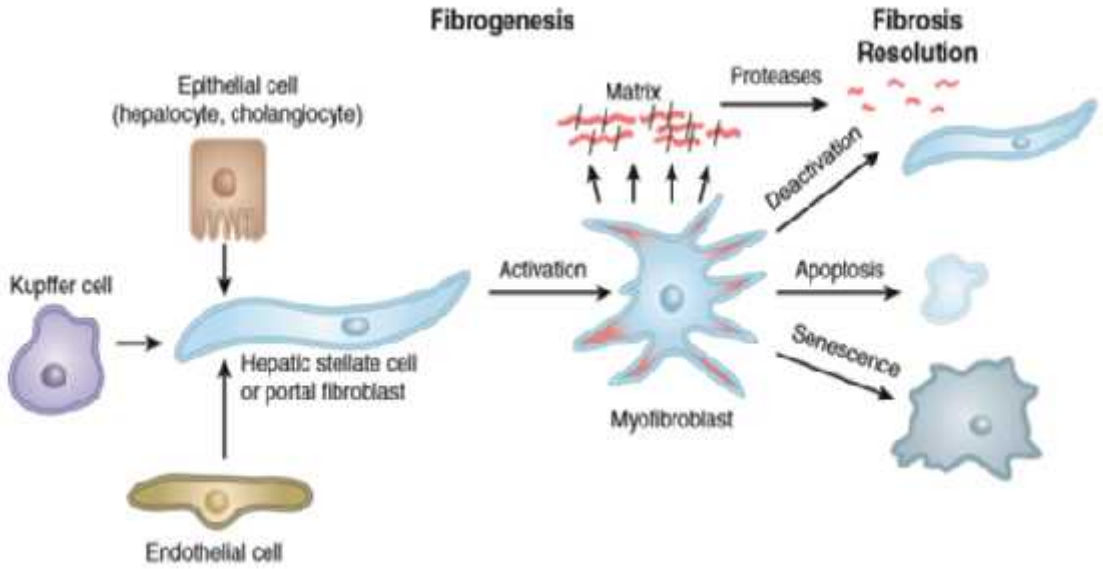


Fig 3.0

APPROACH TO A PATIENT WITH CIRRHOSIS-[9]

1. Ask for the patient's demographic proflie.
2. Take appropriate CLINICAL HISTORY and enquire about complaints of-
    - fatigue,
    - loss of weight,
    - loss of appetite,
    - pain abdomen,
    - yellowish discolouration of sclera and skin,
    - pruritis,
    - colour changes in urine, feces,
    - edema of legs
    - abdomen distension
    - bleeding from gums, epistaxis, ecchymoses, purpura, hematemesis, melena.
    - sexual abnormalities, menstrual history

3. Enquire about history of past illnesses such as
    - jaundice
    - hepatitis
    - drug ingestion
    - blood transfusion
4. History of alcohol consumption
5. Any family history of liver disease or autoimmune disorders.
6. ON EXAMINATION look for
    a. Nutritional status, hyperthermia, foul smelling breath, icterus, hypo or hyper pigmentation, purpuric rash, clubbing of fingers, leukonychia, spider angioma, erythema over the palms, glandular enlargement of male breast tissue, atrophy of testes, alopecia, contracture deformities of hand and BP.
    b. On per abdomen examination see for fluid accumulation in abdomen, prominent or engorged veins in the epigastrium, flanks, palpation of abdominal organs.
    c. In CNS, examine for evidence of asterixis, higher mental functions, stupor and tremors.

INVESTIGATIONS-

a) In Haematology - Haemoglobin, TLC, Platelet count, PT/INR.
b) In Biochemistry- Serum Bilirubin, Transaminase, Albumin, Globulin.
    Alkaline Phosphatase
    GGT
    Serum Iron, Ferritin, Transferrin saturation
    Serum Ceruloplasmin, Serum Copper

Alpha 1 Antitrypsin

Renal function Tests

c) If ascites present do- diagnostic paracentesis, daily weight monitoring, 24hr urine volume and sodium excretion.

d) Autoimmune profile which includes-

anti- Sm antibody

Anti-mitochondrialantibody

ANA

Anti-LKMantibody

ANCA

e) Viral markers- HBV, HCV

f) Tumour marker- Alpha fetoprotein

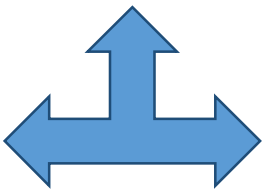
g) Upper GI Endoscopy

h) Ultrasonography of hepatic system, CT,MRI abdomen

i) Liver biopsy if not contraindicated

j) Electroencephalography for hepatic encephalopathy grading

CLINICAL CIRRHOSIS[9]



COMPENSATED CIRRHOSIS

- ✓ Usually asymptomatic and maybe found as a result of abnormalities found on routine biochemical assays or clinically by thorough examination.
- ✓ On examination if liver is palpable in the epigastrium then it is an important sign for considering cirrhosis diagnosis.
- ✓ Confirmation of cirrhosis is by liver imaging, fibroscan.
- ✓ If signs and symptoms suggestive of portal hypertension are present then it suggests worst outcome.

DECOMPENSATED CIRRHOSIS

- ✓ Patients usually seeks advise because of ascites, jaundice, GI bleeding or altered sensorium(hepatic encephalopathy).
- ✓ Decompensated cirrhosis precipitated by bacterial infection, surgery, trauma or medications.
- ✓ Wasting of muscles and loss of weight are routine.
- ✓ Most common cause of hepatic encephalopathy is cirrhosis.
- ✓ If Jaundice present- indiactes that destructive capacity of liver is more than the regenerative capacity.
- ✓ Purpura over arms, shoulder and shins indicate thrombocytopenia.
- ✓ Circulation is overactive which is indicated by warm peripheries, tachycardia, hypotension, easily palpable pulses.
- ✓ Ascites, hepatomegaly and splenomegaly is present; peripheral edema present.
- ✓ Other features of decompensation-

a) Hepatorenal syndrome
b) Spontaneous bacterial peritonitis
c) Hyponatremia

PROGNOSTICATION OF LIVER

CIRRHOSIS IS BY-[9]

A) CHILD PUGH SCORE

B) MELD SCORE

C) UKELD SCORE

A) CHILD PUGH SCORE

TABLE 1. CRITERIA FOR CHILD-TURCOTTE CLASSIFICATION

Group designation	A	B	C
Serum bilirubin[a] (mg·%)	Below 2.0	2.0–3.0	Over 3.0
Serum albumin (gm·%)	Over 3.5	3.0–3.5	Under 3.0
Ascites	None	Easily controlled	Poorly controlled
Neurological disorder	None	Minimal	Advanced "coma"
Nutrition	Excellent	Good	Poor, "wasting"

Original CP Score classification

The following table of modified CP Score Classification was used in our study-

Parameters	Value	Point
Encephalopathy	none Grade I-II Grade III-IV	1 2 3
Ascites	none mild uncontrolled	1 2 3
Bilirubin (mg/dL)	< 2 2-3 >3	1 2 3
Albumin(g/dL)	> 3.5 2.8-3.5 < 2.8	1 2 3
Prothrombin time (INR)	< 1.7 1.7-2.3 >2.3	1 2 3

Group A= 5–6 points; Group B= 7–9 points; Group C=10–15 points

B) MELD (MODEL FOR ENDSTAGE LIVER DISEASE)

Used to determine prognosis in patients undergoing TIPS.

Is calculated using Sr. Creatinine, INR, Sr. Bilirubin

Now widely used criterion for liver transplant listing and to determine priority for organ allocation.

C) UKELD - similar to MELD

Signs suggestive of poor prognosis-[9]

a) Prolonged prothrombin time

b) Marked ascites

c) Advanced age

d) GI bleeding

e) High daily alcohol consumption

f) Hyperbilirubinemia

g) Increased ALP

h) Hypoalbuminemia

i) Poor nutrition.

CLINICAL RULES OF THUMB FOR CIRRHOSIS INCLUDE-[9]

a) If precipitating factors such as haemorrhage, infection, alcoholism are present for decompensation to occur suggests better prognosis as they are correctable causes.

b) Response to therapy- If patient failed to improve within 1 month stipulate below par augury.

c) Jaundice if present is an ominous indicator.

d) Ascites compounds the prognosis, explicitly if resistant to diuretic therapy.

e) Better prognosis is indicated by larger liver size.

f) Variceal hemorrhage - PHTN must be taken into account together with the state of liver cells.

- ✓ liver functioning fair enough- hemorrhage condoned
- ✓ liver function bad- hemorrhage not well condoned results in hepatic coma and death.

g) Hypoalbuminemia and hyponatremia designate bad prognosis.

h) Persistent hypotension would specify sepsis and should be treated instantly.

PATHOPHYSIOLOGY OF CIRRHOSIS

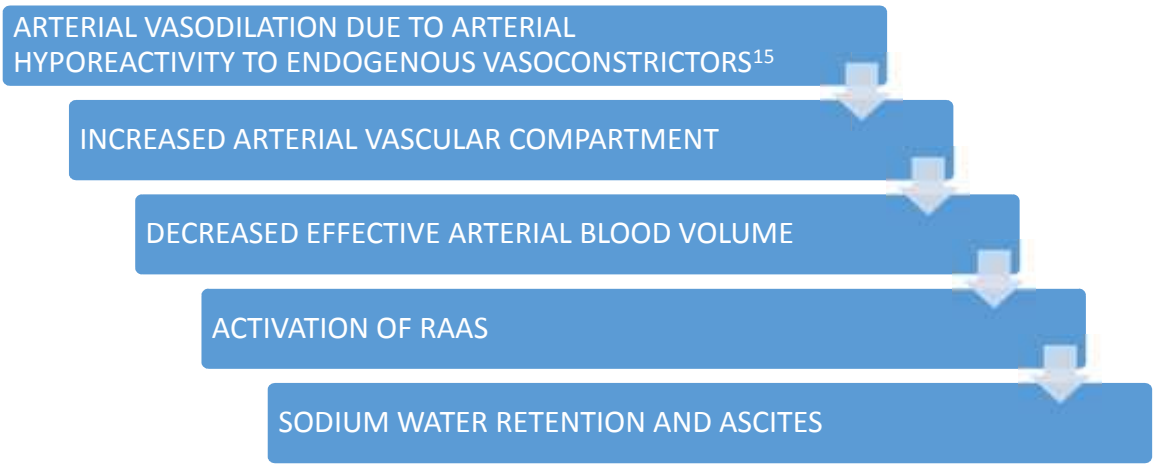
Complications in decompensated cirrhosis are a result of vasodilatation and hyperdynamic circulation.

Flow of blood in peripheral arteries and flow of portal vein blood= (raised)

CO = (raised)[14]

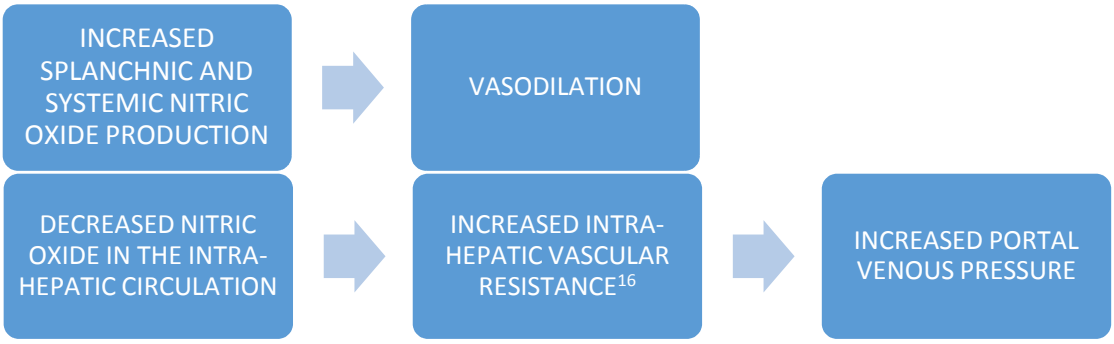
RBF mainly perfusion of renal cortex= (decreased)

a) VASODILATION-



b) HYPERDYNAMIC CIRCULATION-

Nitric Oxide is the most important molecule for hyperdynamic circulation.



EXTRAHEPATIC MANIFESTATIONS OF CIRRHOSIS-

a) Hepato-Pulmonary syndrome[17]

b) Hepato-Renal syndrome[18]

c) Cirrhotic cardiomyopathy[19]

d) GI Manifestations of cirrhosis- splenomegaly, peptic ulcer[20], small bowel bacterial overgrowth,[21] gallstones, chronic calcific pancreatitis.

e) Foetor hepaticus[22]

f) Spider angioma[23]

g) Palmar erythema

h) Leukonychia

i) Madelung disease[24]

j) Clubbing[25]

k) Dupuytren's contracture

l) Sarcopenia[26]

m) Gynaecomastia[27]

MANAGEMENT OF CIRRHOSIS[9]

COMPENSATED CIRRHOSIS	DECOMPENSATED CIRRHOSIS
a) Adequate balanced diet- 35-40kcal/kg/day 1.2-1.5g/kg/day Avoid fatty foods, salt restriction.	Treatment of precipitating factors such as hepatic encephalopathy, ascites and variceal haemorrhage.
b) Avoidance of alcohol and obesity	
c) Avoid fluid retention	
d) Avoid encephalopathy	
e) Functioning of the renal system to be maintained	
f) Haemorrhage from the varices should be prevented	

RELEVANT STUDIES

B. C. Prakash et al (2020), performed a cross-sectional study, on 100 patients of cirrhosis of liver. Cirrhosis as a result of alcohol, Hepatitis B and C, autoimmune, Cryptogenic, NAFLD, were appended in the study. APRI Index, MELD Score and CP Score were premeditated, and the correlation was prevailed. Mean age of 46.14 was observed with a standard deviation of 10.45. There were 93 males and 7 females who were diagnosed with liver cirrhosis.

Etiology of cirrhosis were as follows , 75 subjects developed alcohol related cirrhosis, 13 of them had HBV infection, 9 subjects were diagnosed with HCV infection, and 1 each hadAutoimmune, NAFLD and Cryptogenic cirrhosis. Diagnosis was confirmed bybiochemical and radiological investigations. The study showed,"significant 'p' value(less than 0.05) between APRI index and CP score, MELD Score and CP Score, MELD Score and APRI index. This demonstrated significant correlation between APRI index, CP score, MELD Score. Therefore APRI index can be used as substituteinvestigation for assessing severity and prognosis of liver cirrhosis. The study exhibited that,more the APRI index, higher MELD scores and CP scores wereseen in patients who had complication of cirrhosis like ascites, thus stating that as the severity of the disease increased, all the three scores also increased."

CP score of 13±2.8, MELDscoreof 26.8±9.5, and APRI of 4.371±2.4 was seen among the patient who had refractory ascites.

APRI of 3.4±2, CP score of 13.2±1.5, and MELD score of 36.08±6was seen in subjects with grade 3 or 4 hepatic encephalopathy.[1]

Rong-Yun Mai Jiaet al[2] (2019), conducted a study, which aimed to probe the effectiveness of aspartate aminotransferase to platelet-ratio index (APRI) calculated preoperatively, to foretell the risk of PHLF(post hepatectomy liver failure) in patients

with HCC after liver resection, and to collate relation of APRI with MELD score ,CP score, and albumin–bilirubin (ALBI) score. According to Rong et al study, "1,044 consecutive patients with HCC who underwent resection of liver were included. CP, MELD, ALBI, and APRI scores for predicting PHLF were gauged according to area under the ROC curve.ROC analysis was used to find the cut-off value of APRI which predicted PHLF. 213 (20.4%) patients showed PHLF. CP, MELD, ALBI, and APRI scores were significantly associated with PHLF. This study proved that CP, MELD, or ALBI scores were less accurate than APRI in prognosticating PHLF. With an optimal cut-off value of 0.55, APRI had a sensitivity of 72.2% and specificity of 68% for predicting PHLF.Incidence and grade of PHLF in patients with high risk APRI score of >0.55 was significantly higher than in the low-risk cohort APRI score of 0.55)."[2]

A study conducted by **Adelia Muhlifa Saputri et al[3]** (2019), was intended to discover the association between APRI score, CP score and degree of varices in esophagus in cirrhotics. It was a retrospective analytic study with cross sectional approach. The data was analyzed using ANOVA.The study included 48 cirrhosis of liver patients. 81.3% were male, with mean age 49.98 years. Frequent cause of cirrhosis was hepatitis B in 33 patients (68,8%). The study showed APRI score was found to have a weak correlation (r = 0.044) and not significant 'p' value (p = 0.868) with the CP score. The APRI score was not able to assess the prognosis in patients with advanced liver cirrhosis.[3]

Dr Jithin George et al[4] (2018), conducted a study on 50 patients with liver cirrhosis. Ultrasound abdomen and biochemical reports were used for diagnosis. Cirrhosis as a consequence of alcohol, Hepatitis B, Hepatitis C,NAFLD were included. APRI Index, CP Score, MELD Score were assessed and the relation was found. 47 males and 3

females present in the study. Out of 50 subjects; 10, 18 and 22 had mild ascites, moderate ascites and severe ascites respectively. Hepatic encephalopathy features were not seen in 15 patients, grade 1 or 2 hepatic encephalopathy features seen in 7 patients and grade 3 or 4 hepatic encephalopathyfeatures wereobserved in 28 patients. CP class A had 2 patients, CP class B had 14 Patients and CP class C had 26 patients.

Significant positive correlation was found among MELD Score and CP Score, and MELD Score and APRI and APRI Index and CP Score, with significant 'p' value. The study proved that there was increased mortality in patients with raised APRI Index, higher CP classification and higher MELD score during the course of treatment. APRI-median value of 12.58; CP score-median value 15 and MELD score-36.08 with standard deviation of 5.946 was seen in patients who died. Hence APRI index can also be used for forecasting the mortality of liver cirrhosis patients and to prognosticate like other investigations.[4]

Ajay Pratap Singh et al[5] (2018), conducted a study including 64 patients of CLD and 64 healthy subjects. FIBROSCAN was done and APRI score was calculated. Fibrosis stage has to be determined as it is important to decide whether the patient is having mild or advanced liver disease. Advanced fibrosis patients are known to progress rapidly into cirrhosis and hence abstinence or antiviral therapy must be advised so as to delay the progression. Liver biopsy is the gold standard and provides exact information. Liver biopsy had only about 80% accuracy in fibrosis staging and is knownto miss advanced fibrosis in 30%. Non-invasive approaches are considered to provide information about diagnosis of cirrhosis. In the present study, FIBROSCANwas used to assess fibrosis. It is known to have a sensitivity of 70% and specificity of 84%.

In this study, FIBROSCANshowed that, 75% (n=48) had grade F4 as per color coding scale of fibrosis which suggests that most of the patient came to hospital in a relatively advanced stage of liver disease, followed by 9.4%(n=6) patients in F2 grade and 7.8%(n=5) patients in each F1 and F3 grade. The mean value of fibrosis as per transient elastography in the cases were 36.06±24.18 kPa and in controls it was 3.96±0.97 kPa. The FIBROSCAN value was statistically significantly raised(p value<0.05) in F4 grading of fibrosis than F1, F2 and F3 grade fibrosis with mean values of 44.97±21.32 kPa in F4 and 7.14±1.73 kPa , 8.08±0.65 kPa and 12.94±5.05 kPa in F1, F2 and F3 respectively. This showed that the FIBROSCAN fibrosis grading increased with the grade of Fibrosis according to biopsy.Mean value of FIBROSCAN fibrosis was calculated in each group of disease(CALD , HEP B and HEP C) in the cases and it came out that fibrosis value (kPa) was maximum in patients with chronic alcoholic liver disease 41.70±25.30 kPa followed by Hepatitis B 32.40±24.57 kPa and Hepatitis C 26.62±16.23 respectively. Thus, showing that, cirrhotic patients with alcoholic liver disease had higher liver stiffness values than cirrhotic patients with chronic hepatitis C. They found that FIBROSCAN was able to detect fibrosis with a sensitivity and specificity of 98.4 and 70.3%. The diagnostic accuracy of FIBROSCAN was AUC 0.99 (0.98-1.01) for prediction of cirrhosis at cut-off value of 4.5kPa which was obtained comparing the FIBROSCAN values within cases and controls.

Serum biomarkers are being used as an alternative to liver biopsy for grading of liver fibrosis. One of them is combined score of APRIin cirrhotics. The mean value of APRI in cases was 2.47±2.05 and in controls was 0.45±0.16.This showedstatistical significance (p value <0.001) telling that values of APRI were increased in the case group than the control group. 45.84±13.20 and 41.23±11.08 respectively was the

mean age for cases and controls. Majority of patients were males in cases (76.6%; n=49) and controls (89.1%; n=57). The predominant etiology in the cases group was CALD (n=32; 50%), HEPATITS B (n=21; 32.8%) and HEPATITS C (n=11; 17.2%). The mean liver stiffness was 36.06±24.18 and 3.96±0.97 kPa for cases and controls respectively. 75% (n=48) of cases were of F4 grade. Mean LSM was premediated in each of thecategory which showed that it was maximum in CALD (41.70±25.30) followed by HEP B(32.40±24.57) and HEP C(26.62±16.23). A cutoff value of 4.5 kPa was obtained in this study and it had AUC (95%CI) of 0.99 (0.98-1.01) and was able to diagnose significant fibrosis with sensitivity, specificity, PPV, and NPV of 98.4%, 70.3%, 76.8% and 97.8% respectively. The mean APRI score for cases and controls was 2.47±2.05 and 0.45±0.16. APRI score was maximum in F4 (3.03±2.06) grade fibrosis followed by F3 (1.27±0.85), F2(0.66±0.24) and minimum in F1(0.48±0.23). A cutoff value of 0.70 was obtained in our study and it had AUC (95%CI) of 0.91 (0.85-0.96) and diagnosed significant fibrosis with sensitivity, specificity, PPV, and NPV of 81.3%, 90.6%, 89.7% and 82.9% respectively.A positive significant correlation was obtained between transient elastography fibrosis grading (FIBROSCAN) and APRI score with R value = +0.379.[5]

Princi Jainet al[6] (2015), conducted a study including 51 patients with cirrhosis, diagnosed on USG abdomen. APRI was premediated for every patient using the formula {(AST / ULN) x 100}/platelet count (10^9/L). Receiver-operating characteristics (ROC) curve predictive accuracy was evaluated.According to this study, “out of 51 patients, 9(17.64%)wereHepatitis B positive, 7 (13.72%) were Hepatitis C positive & 25 (49.01%) were chronic alcoholics.There wasgood amount of difference in the platelet count, SGOT/PT,INR,prothrombin time, albumin and serum bilirubin of the casesand controls. Mean APRI of the cases and controls

were2.178±1.224 and 0.364±0.137. The results showed statistically significant p-value ≥0.001.A cut-off point was chosento predict the absence (APRI<0.65) or presence (APRI>0.65) ofcirrhosis. For patients with APRI of 0.65 or less, 48 out of 50(96%) patients did not have cirrhosis in the control cohort. On the other hand, for patients withAPRI > 0.65, 49 out of 51 (96.1%) patients in the case cohort hadcirrhosis. TheArea under the curve (AUC) of APRI for predicting cirrhosis was0.973. APRI accuracy in predilicting cirrhosis was 96.1% sensitiveand 96% specific."

Thus,this study found that APRI was an accurate marker for cirrhosis in Indians which hadhighest specificity and sensitivity.[6]

Wai et al[6]., conducted a study which stated that,"using the cut-off APRI values of 1.00 and2.00, which was decided by the ROC curves, significant fibrosis couldbe predicted accurately in 51% and cirrhosis in 81% of patients.The AUC of APRI for predicting significant fibrosis and cirrhosis inthe validation set were 0.88 and 0.94, respectively."

Khan et al[6], revealed that "cut-offs of APRI were 0.90 and 1.75 basedon ROC for advanced fibrosis. An APRI value ≤0.90 to rule outadvanced fibrosis was 90% sensitive and 70% specific with negative predictive value of 95% and positive predictive value of 49%."

Han Deng et al[7] (2015), studied that esophageal varices in liver cirrhosis can be diagnosed by use of King scores, FIB-4, AAR, FI and APRI. They had an objective to checkthe reliability of above said parameters in predilection ofwhether there is presence of EVs and also see if it is severe in cirrhotics. They stated,"In-patients of hospital and and all those who had undergoneUGI scopy between January 2012 and June 2014 were considered for this retrospective study. AUC was jotted. Based on

history of UGI bleeding and splenectomy they were divided into small groups. Sample size was 650; 81.4% had moderate-severe EVs. AUCs of the above scores for prediction of moderate to severe EVs and presence of EVs was 0.506–0.6 and 0.539–0.612, respectively. In the subgroup analysis, without UGIB, AUCs for moderate-severe varices was 0.601–0.664 and EVs were present in 0.596–0.662.APRI, AAR, FIB-4, FI, and King scores had modest diagnostic accuracy of EVs in liver cirrhosis. They might not be able to replace the utility of upper gastrointestinal endoscopy for the diagnosis of EVs in liver cirrhosis."[7]

Study done by **Vipin Verma et al**[8] (2014), included 74 patients whose APRI, HVPG were calculated in all cirrhotics among the age group of 18–75 years, with serumbilirubin<5 mg/dl, CTPscore >12, and no evidence of ACLF. 222 was the sample size. 148 patients were not includedbecause of,HE,SBP, ARF with creatinine >2 mg/dl, AST >5 times ULN,h/O cancer, h/o surgical treatment taken for portal hypertensionor TIPSS,presence of comorbidities, h/o medical line of management forpreventing bleeding from varices and if they did not want to be a part of this survey. Thus, only 74 patients wereconsidered for survey. Survey said, "there was significantcorrelation between HVPG andAPRI (Spearman's rho 0.365; p¼0.001).APRI in patients with HVPG<12mmHgwas 0.70 (range 0.17–7.92) and in those withHVPG >12 mm HgAPRI was 1.38(range 0.34–6.49); p¼0.004,respectively. The study has showed that,APRI correlates well withHVPG in patients ofcirrhosis. The APRI scores inpatients with high portal pressure (HVPG>12 mmHg) were significantly higher than APRIscores in patients with lower HVPG. An APRI scoreof >1.09seems to have an acceptable accuracy for predictionof high portal pressure and can be usedas surrogatemarker in settings where HVPG measurement isnot available."[8]

METHODOLOGY

A cross-sectional study was conducted in patients who attended the Medicine OPD and in those who were admitted in the wards and ICU of "Department of General Medicine at KLE's Dr. Prabhakar Kore Hospital and Research Centre, Belagavi."

STUDY POPULATION:

SOURCE OF DATA:

Our surveyincluded 102 patients attending Medicine OPD and admitted in the wards and ICU of General Medicine at "KLE's Dr. Prabhakar Kore Hospital, Belagavi" fulfilling the inclusion criteria, during January 2019 to December 2019.

Inclusion criteria:

- Subjects with liver cirrhosis.
- Age group of 18-75years.

Exclusion criteria:

- Acute infectious diseases.
- Primary hematological disorders.

DATA COLLECTION PROCEDURE

Informed consent was taken and then patient was enrolled for the study.All patients fulfilling inclusion criteria were subjected to a questionnaire and thorough clinical examination was done, to identify possible aetiology of liver cirrhosis and to identify presence of complications of liver disease.

Routine workup for chronic liver disease was done.

Complete blood count, renal function tests was done.

Liver function tests including coagulation profile was done.

USG abdomen was done to look for portal vein diameter, echo texture and dimensions of liver and Spleen.

Based on the patient's investigation reports and clinical features the Child Pugh Score, MELD Score and APRI Index was calculated using the formula.

LABORATORY INVESTIGATIONS

1. Liver Function tests.
2. Complete blood count including Platelet count
3. Blood Urea Nitrogen.
4. Serum.Creatinine.
5. Serum electrolytes.
6. PT/INR.
7. USG ABDOMEN.
8. Ascitic fluid analysis for glucose, proteins, cytology.

9. Viral markers- HCV, HBV.

Using the above laboratory investigations APRI, MELD score and Child Pugh score are calculated.

Sample size

Calculated by the following formula:

N= 4PQ/D2 Where N=Sample size

P = Prevalence of the disease Q= 100- P

D = Absolute error taken as 12%of P

(P = 73; Q = 27; D=76.73)

Sampling Method: Cross-sectional Study

Statistical methods-

All consecutive patients fulfilling the inclusion criteria were added and the data was coded and entered into Microsoft excel spread sheet and data was analysed using SPSS version 21 and Statistica 21. The categorical data was expressed in terms of rates, ratio and percentage and the continuous data was expressed in terms of mean +- standard deviation. The association between the outcome, clinical and demographic characteristics was tested using chi-square test. Co-relation among the continuous and categorical variables were compared using Karl Pearson scatter diagram and kappa agreement method. A probability (p) value of <= 0.05 was considered as statistically significant.

RESULTS

Methods used: Data analysis was done using SPSS version 21 and Statistica 21. Categorical variables are represented using percentages. Correlation between 2 variables are studied using Karl Pearson scatter diagram. P-value <0.05 is considered as significant.

Summary:

102 subjects of age group 47.32±12.91years (range: 24-87)were enrolled for the study.

In the study population, 88were male and 14were female subjects.

Table 1: Age wise distribution of patients:

Age groups	No of patients	% of patients
<=30yrs	7	6.86
31-40yrs	26	25.49
41-50yrs	35	34.31
51-60yrs	19	18.63
>=61yrs	15	14.71
Total	102	100.00
Mean age	47.32	
SD age	12.91	

Figure: Age wise distribution of patients

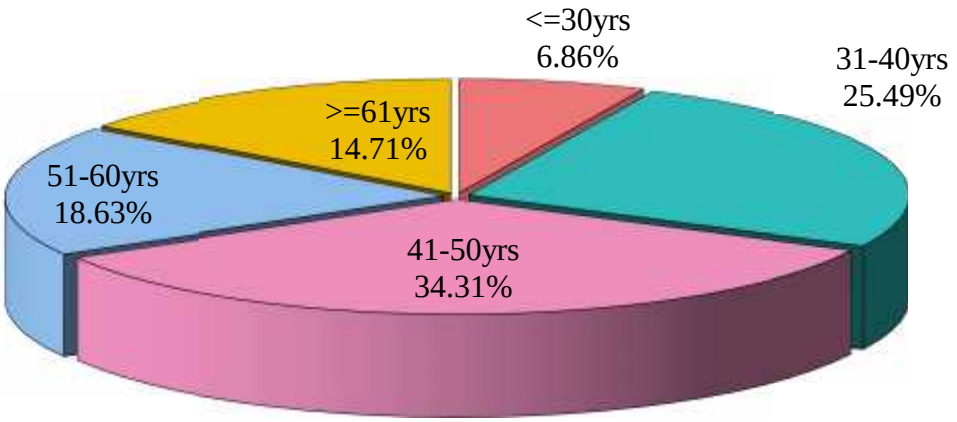


Figure 1: Pie diagram for Age group

From table 1, we observe that majority of subjects 35(34.31%) are in the age group "41-50"yrs followed by 26(25.49%) in the age group of "31-40"yrs and 15(14.71%) subjects are in the age group ">=61yrs".The Minimum and Maximum age of subjects was 24 and 87 years respectively.

Table 2: Distribution of subjects by Gender

Sex	No of patients	% of patients
Male	88	86.27
Female	14	13.73
Total	102	100.00

Figure: Sex wise distribution of patients

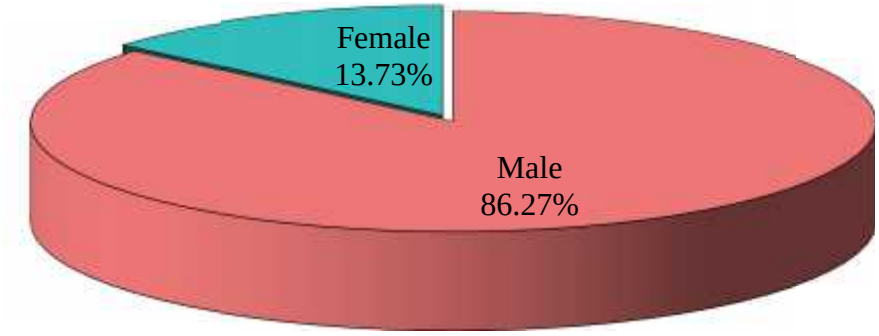


Figure 2: Pie diagram of Gender

From this pie diagram, 88(86.27%) are male subjects and 14(13.73%) female subjects are included in the sample.

Table 3: Distribution of male and females by age groups

Age groups	Male	%	Female	%	Total	%
<=30yrs	6	85.71	1	14.29	7	6.86
31-40yrs	22	84.62	4	15.38	26	25.49
41-50yrs	34	97.14	1	2.86	35	34.31
51-60yrs	15	78.95	4	21.05	19	18.63
>=61yrs	11	73.33	4	26.67	15	14.71
Total	88	86.27	14	13.73	102	100.00
Mean age	46.44		52.86		47.32	
SD age	11.99		17.14		12.91	

Figure: Distribution of male and females by age groups

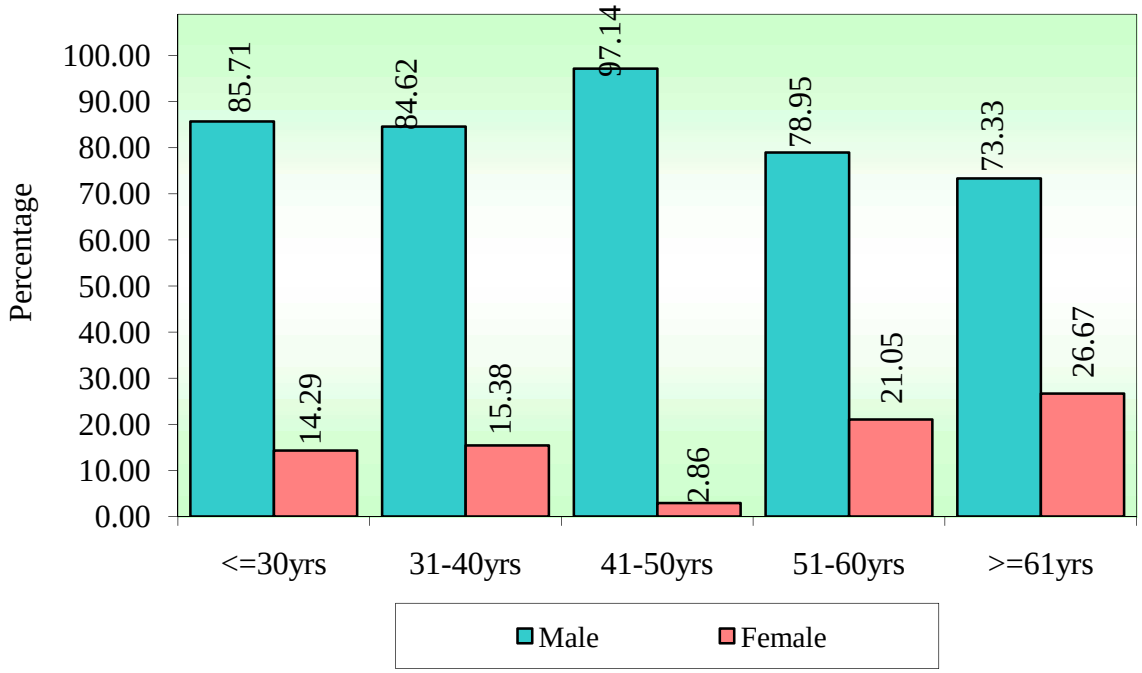


Figure 3: Bar diagram of distribution of males and females by age groups.

According to the above table and bar diagram, 97.14% males were seen in the age group between 41-50 years and 26.67% females were seen in age group >= 61years.

Table 4: Distribution of patients based on history of alcohol intake (N=102)

Alcohol habit	No of patients	% of patients
Alcoholic	74	72.55
Non-Alcoholic	28	27.45
Total	102	100.00

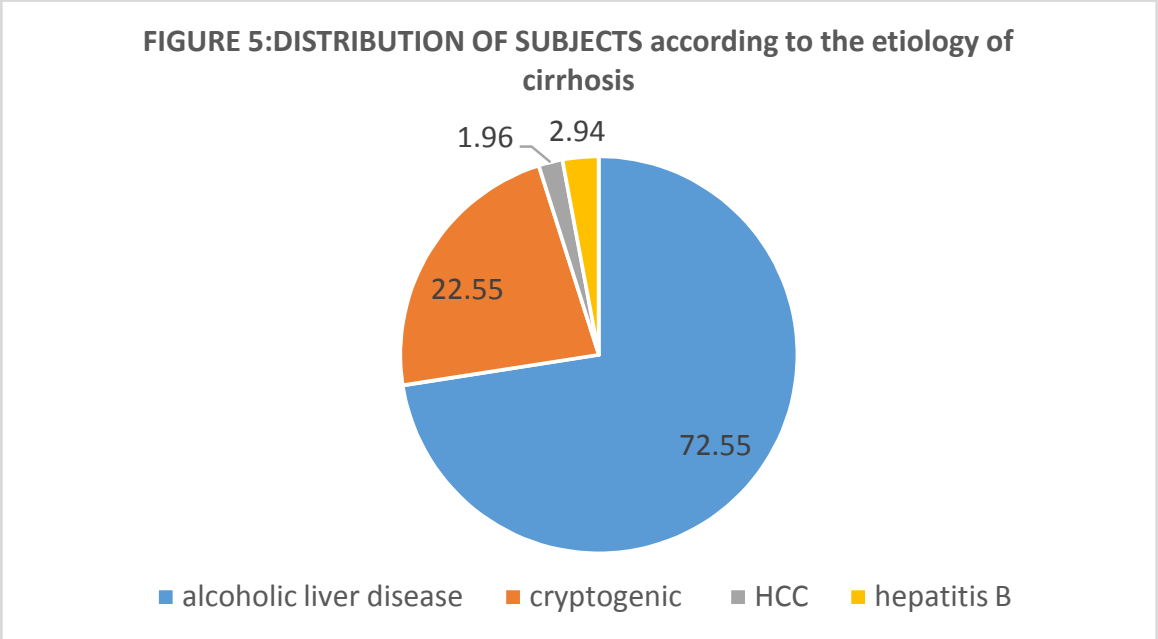
Figure 4: Alcohol habit wise distribution of patients

From this diagram, we observe that, 74(72.55%) are alcoholics and 28(27.45%) are non-alcoholics.

Table 5 : Distribution of subjects according to the etiology of cirrhosis

DIAGNOSIS	No of patients (N=102)	% of patients
Alcoholic liver disease	74	72.55
Cryptogenic	23	22.55
HCC	2	1.96
Hepatitis B	3	2.94
Total	102	100.00

ABBREVIATION: HCC- hepatocellular carcinoma

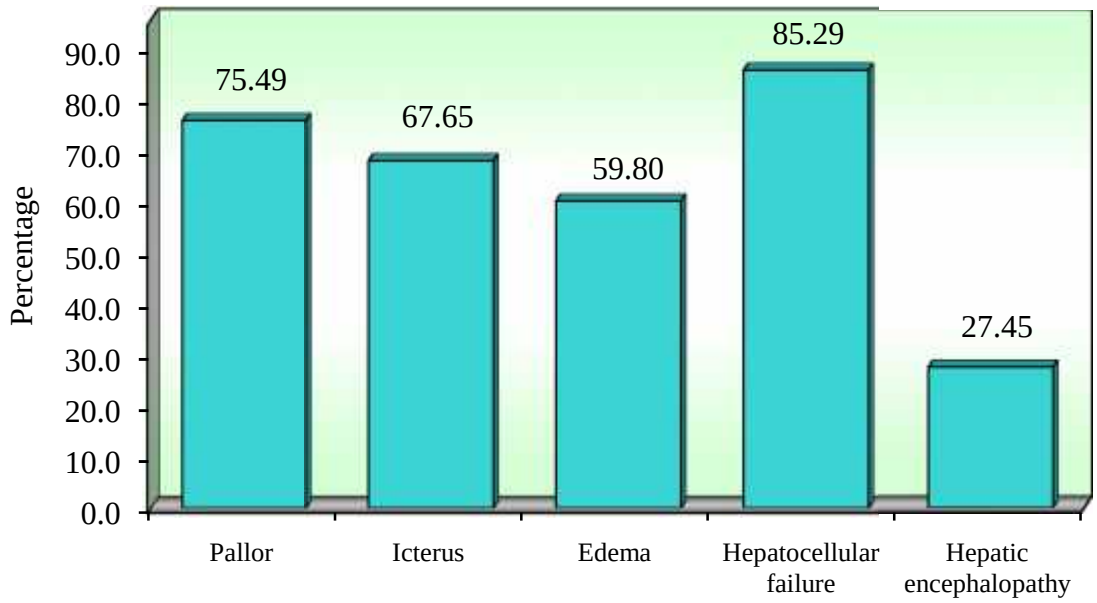


From the table 5, we observe that, 74(72.55%) of subjects in the sample were diagnosed with Alcohol liver disease, followed by Cryptogenic cirrhosis 23(22.55%), Hepatitis B in 3(2.94%), and the least number of subjects are diagnosed with hepatocellular carcinoma i.e 2(1.96%).

Table 6: Signs wise distribution of patients

Signs	No of patients	% of patients
Pallor (Anaemia)	77	75.49
Icterus (Hyperbilirubinemia)	69	67.65
Edema	61	59.80
Hepatocellular failure	87	85.29
Hepatic encephalopathy	28	27.45

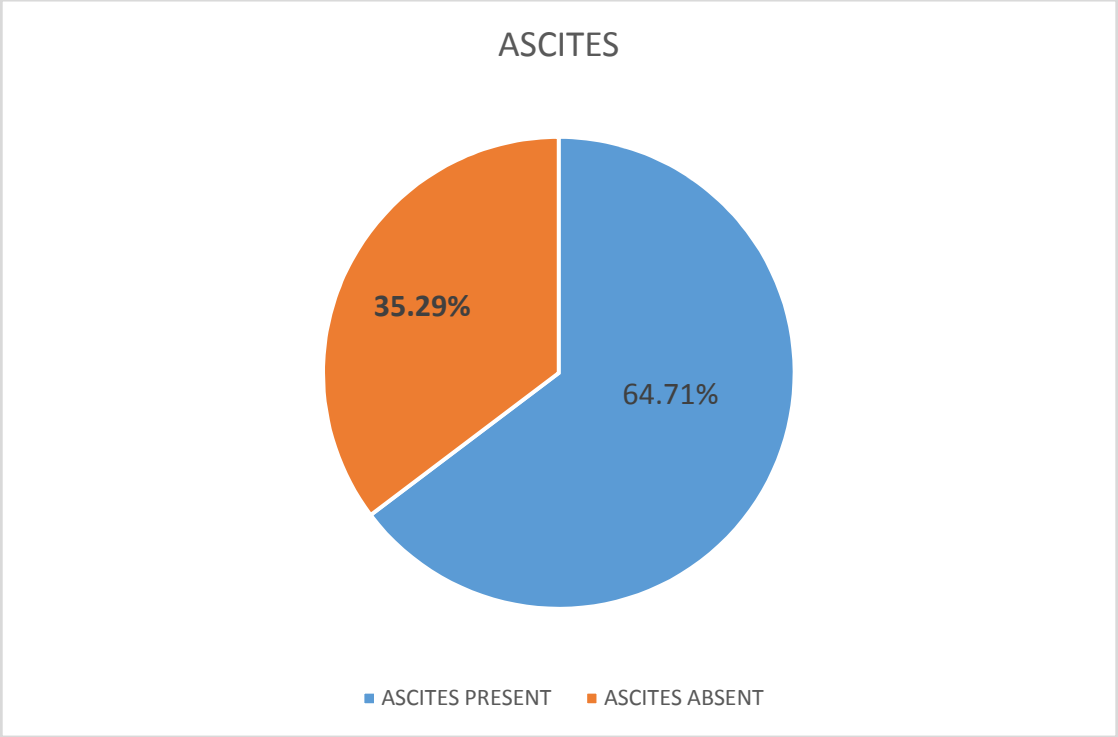
Figure 6:Presence of Signs wise distribution of patients



From Table 6 and above bar diagram, we conclude that, 87(85.29%) had signs of hepatocellular failure followed by pallor in 77(75.49%), icterus in 69(67.65%), edema in 61(59.80%) and signs of hepatic encephalopathy in 28(27.45%) subjects.

Table 6 :(continued): Signs wise distribution of patients

Ascites	No. of Patients	% of Patients
Present	66	64.71
Absent	36	35.29
Total	102	100

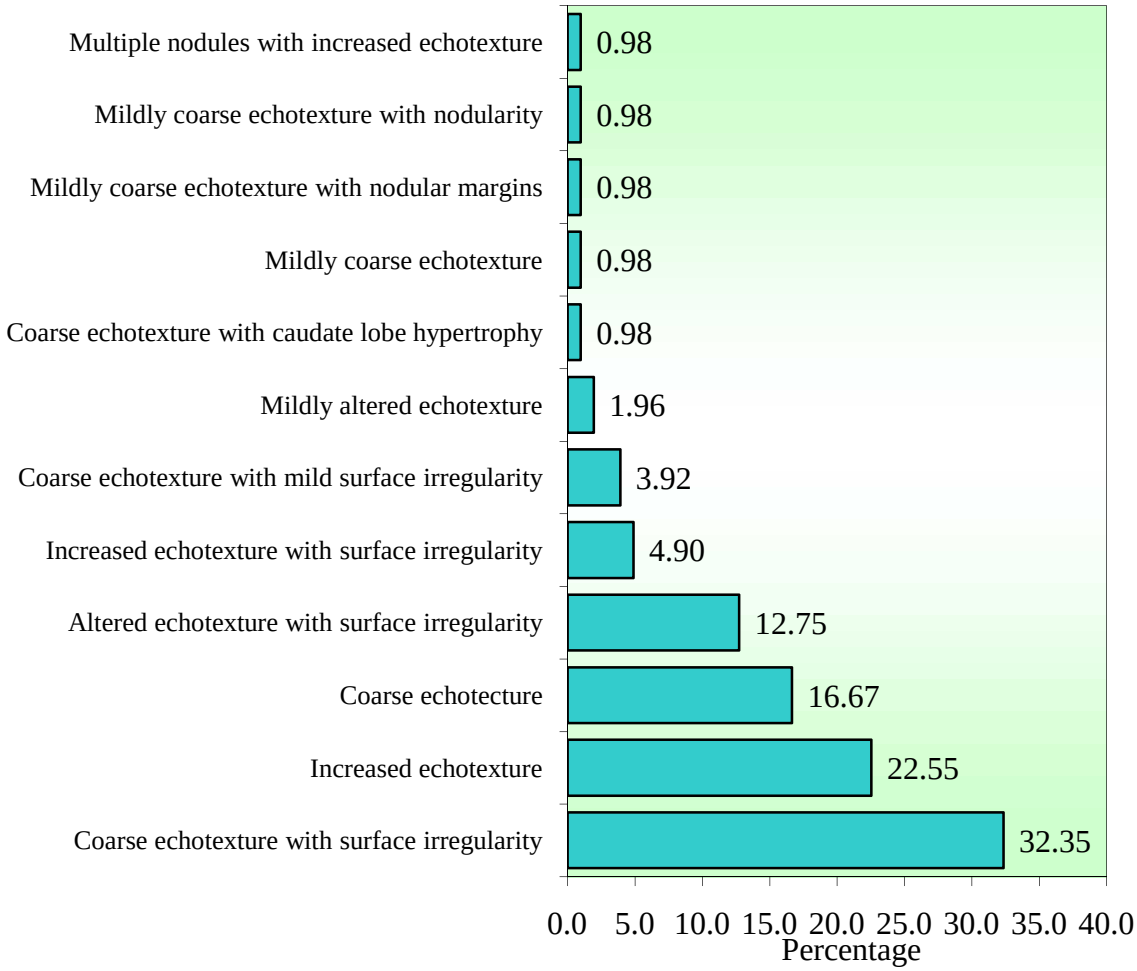


From the above pie diagram, we observed that, 66(64.71%) subjects had ascites and 36(35.29%) did not have.

Table 7: USG Abdomen liver echotexture wise distribution of patients.

USG abdomen liver echotexture	No of patients	% of patients
Altered echotexture with surface irregularity	13	12.75
Coarse echotecture	17	16.67
Coarse echotexture with caudate lobe hypertrophy	1	0.98
Coarse echotexture with mild surface irregularity	4	3.92
Coarse echotexture with surface irregularity	33	32.35
Increased echotexture	23	22.55
Increased echotexture with surface irregularity	5	4.90
Mildly altered echotexture	2	1.96
Mildly coarse echotexture	1	0.98
Mildly coarse echotexture with nodular margins	1	0.98
Mildly coarse echotexture with nodularity	1	0.98
Multiple nodules with increased echotexture	1	0.98
Total	102	100.00

Figure 7: USG Abdomen liver echotexture wise distribution of patients



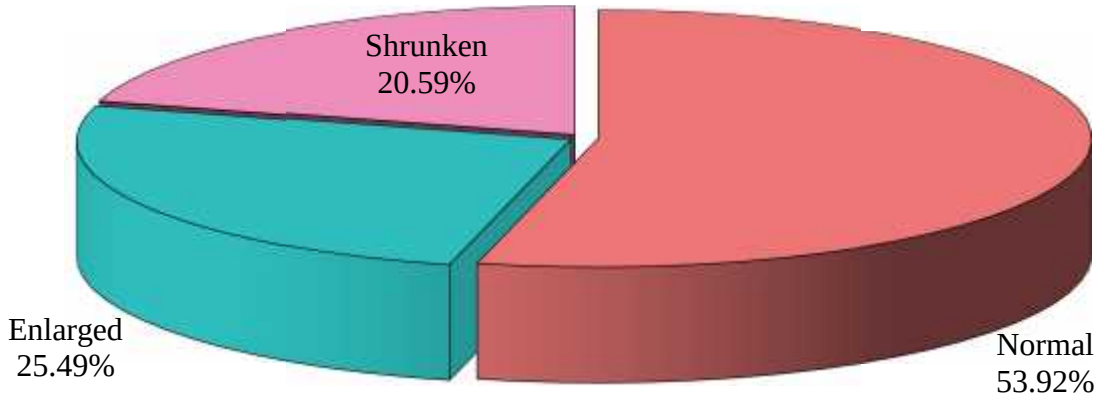
From Table and Fig 7, we observed that, coarse echotexture with surface irregularity was seen in 33(32.35%) subjects, followed by increased echotexture of liver in 23(22.55%), coarse echotexture in 17(16.67%), altered echotexture with surface irregularity in 13(12.75%), Increased echotexture with surface irregularity in 5(4.90%), Coarse echotexture with mild surface irregularity in 4(3.92%), Mildly altered echotexture in 2(1.96%).

Coarse echotexture with caudate lobe hypertrophy, Mildly coarse echotexture, Mildly coarse echotexture with nodular margins, Mildly coarse echotexture with nodularity, Multiple nodules with increased echotexture each of them seen in 1(0.98%) subject.

Table 8: USG abdomen- liver size wise distribution of patients

USG abdomen- liver size	No of patients	% of patients
Normal	55	53.92
Enlarged	26	25.49
Shrunken	21	20.59
Total	102	100.00

Figure 8: USG abdomen- liver size wise distribution of patients

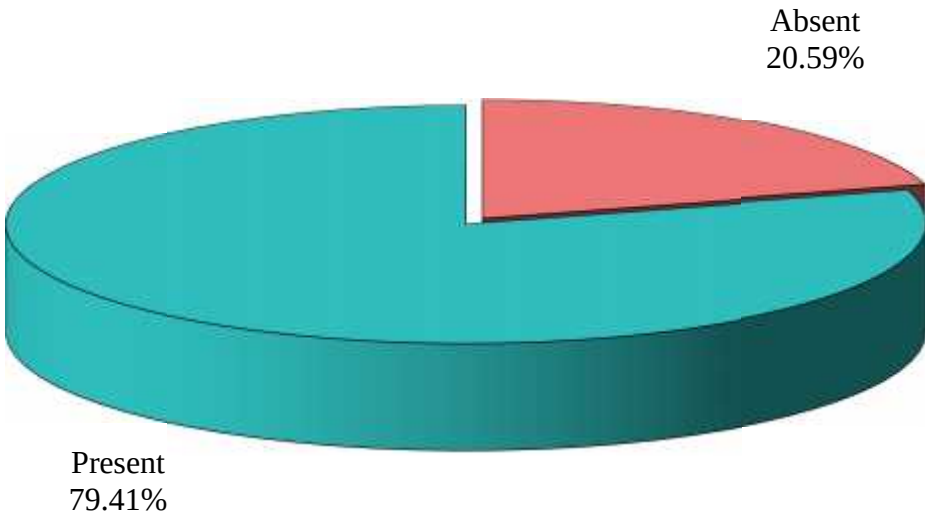


From Table and figure 8, we observed that, liver size was normal in 55(53.92%) subjects, enlarged in 26(25.49%) and shrunken in 21(20.59%).

Table 9: USG abdomen- splenic enlargement wise distribution of patients

USG abdomen- splenic enlargement	No of patients	% of patients
Absent	21	20.59
Present	81	79.41
Total	102	100.00

Figure 9: USG abdomen- splenic enlargement wise distribution of patients

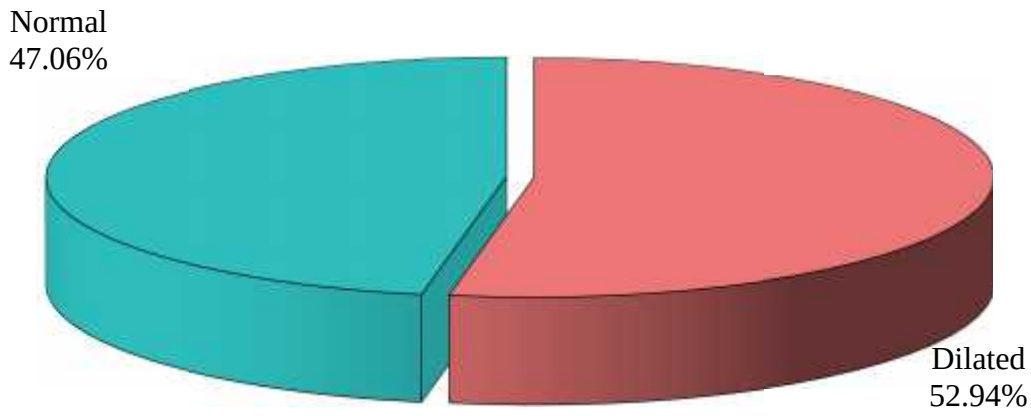


From Table and Figure no 9, we observed that, splenomegaly was present in 81(79.41%) and absent in 21(20.59%).

Table 10: Portal vein diameter on USG wise distribution of patients.

Portal vein diameter on USG	No of patients	% of patients
Dilated	54	52.94
Normal	48	47.06
Total	102	100.00

Figure 10: Portal vein diameter on USG wise distribution of patients



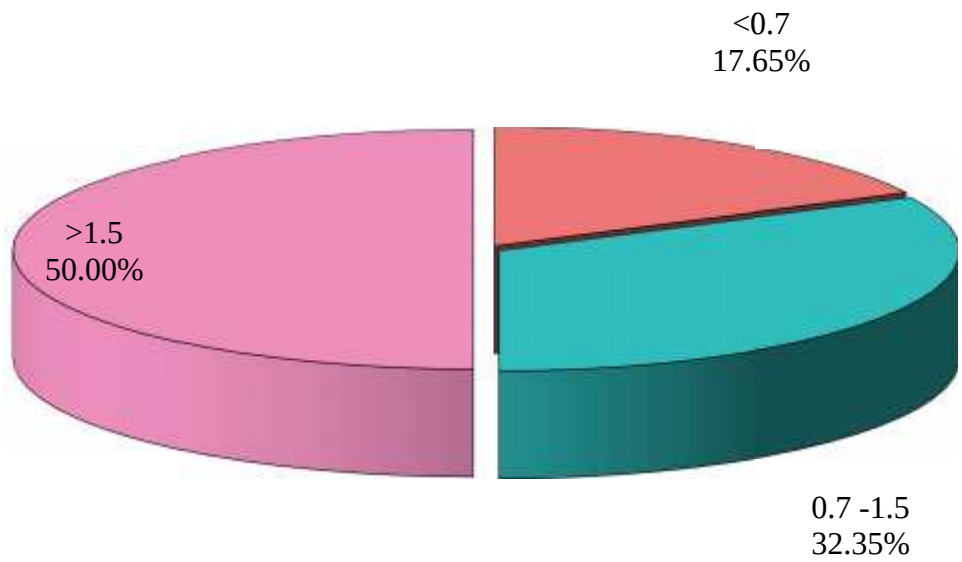
From Table and Figure no 10, we observed that, portal vein was dilated in 54(52.94%) subjects and normal in 48(47.06%) subjects.

Table 11: APRI criteria wise distribution of patients.

APRI criteria	No of patients	% of patients
<0.7	18	17.65
0.7 -1.5	33	32.35
>1.5	51	50.00
Total	102	100.00

ABBREVIATION: APRI- Aspartate aminotransferase to platelet ratio index.

Figure 11: APRI criteria wise distribution of patients

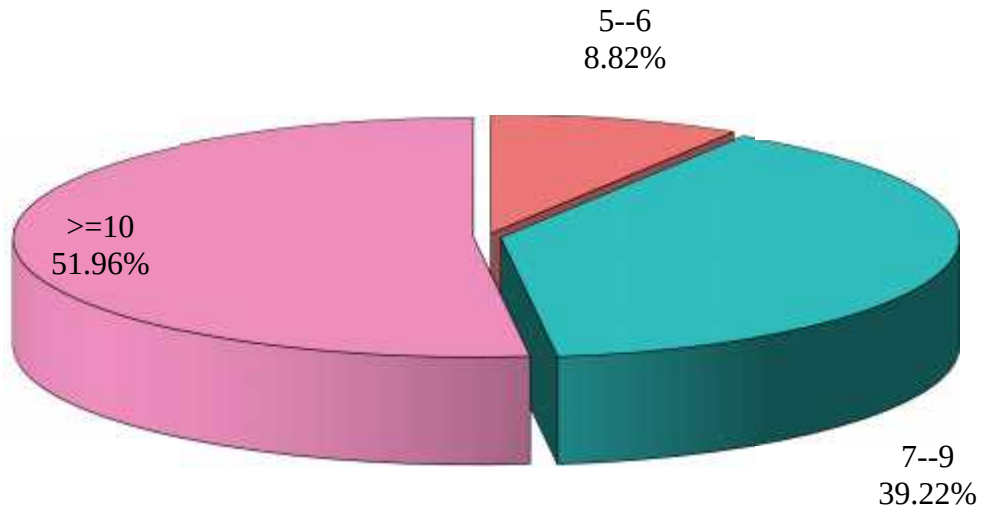


From Table and Figure no 11, we observed that, 51subjects(50%) had APRI >1.5; 33subjects(32.35%) had APRI 0.7-1.5 and 18(17.65%) had APRI <0.7.

Table 12: Child PUGH criteria wise distribution of patients.

Child PUGH criteria	No of patients	% of patients
5—6 (A)	9	8.82
7—9 (B)	40	39.22
>=10 (C)	53	51.96
Total	102	100.00

Figure 12: Child PUGH criteria wise distribution of patients

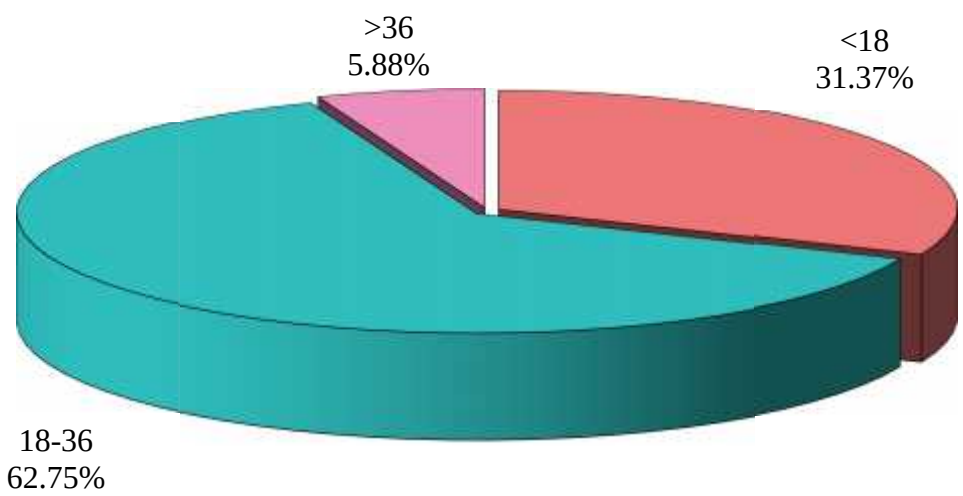


From Table and Figure no 12, we observed that, Child-Pugh C seen in 53(51.96%) subjects; Child-Pugh B seen in 40(39.22%) and Child-Pugh A was in 9(8.82%).

Table 13: MELD criteria wise distribution of patients.

MELD criteria	No of patients	% of patients
<18	32	31.37
18-36	64	62.75
>36	6	5.88
Total	102	100.00

Figure 13: MELD criteria wise distribution of patients



From Table and figure no 13, we observed that, maximum subjects 64(62.75%) had MELD score between 18-36; followed by 32(31.37%) had MELD <18; and least subjects 6(5.88%) had MELD >36.

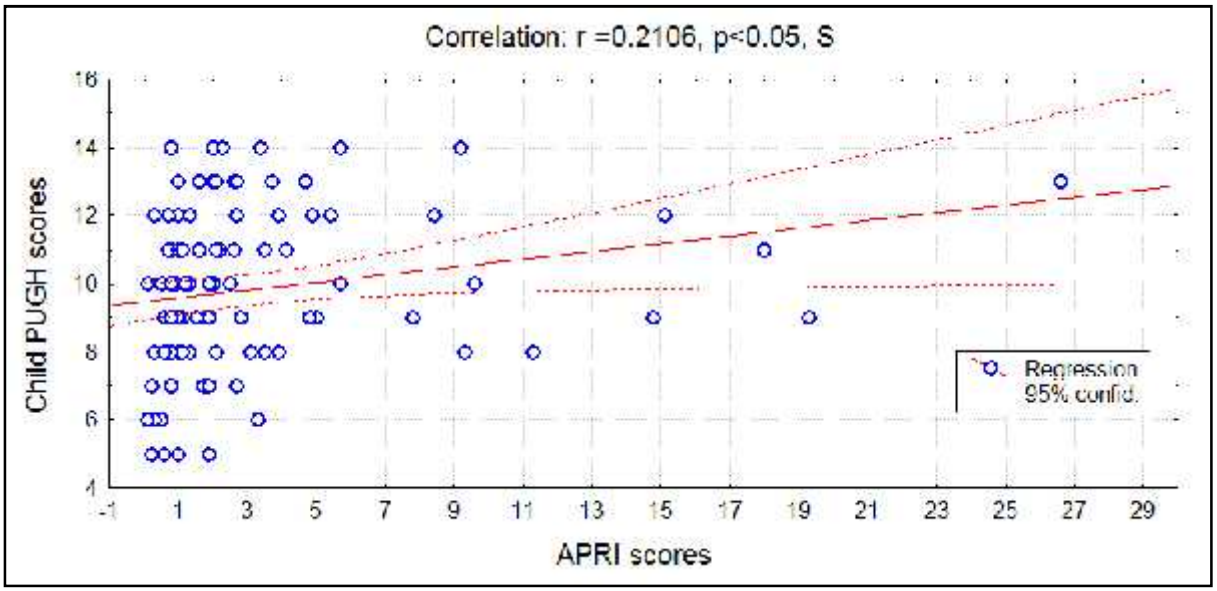
Table 14: Correlation between APRI and Child Pugh scores by Karl Pearson's correlation coefficient.

Variables	r-value	t-value	p-value	Significance
APRI and Child PUGH scores	0.2106	2.1543	0.0336	S

ABBREVIATION- S= significant

From above Table 14, correlation between APRI and Child-Pugh score was studied; it showed that p-value was 0.0336 (significant), r-value was 0.2106.

Figure 14: Scatter diagram showing the correlation between APRI and Child PUGH scores.



The above scatter diagram showed correlation between APRI and Child-Pugh Score. This study showed positive correlation between APRI and Child-Pugh Score.

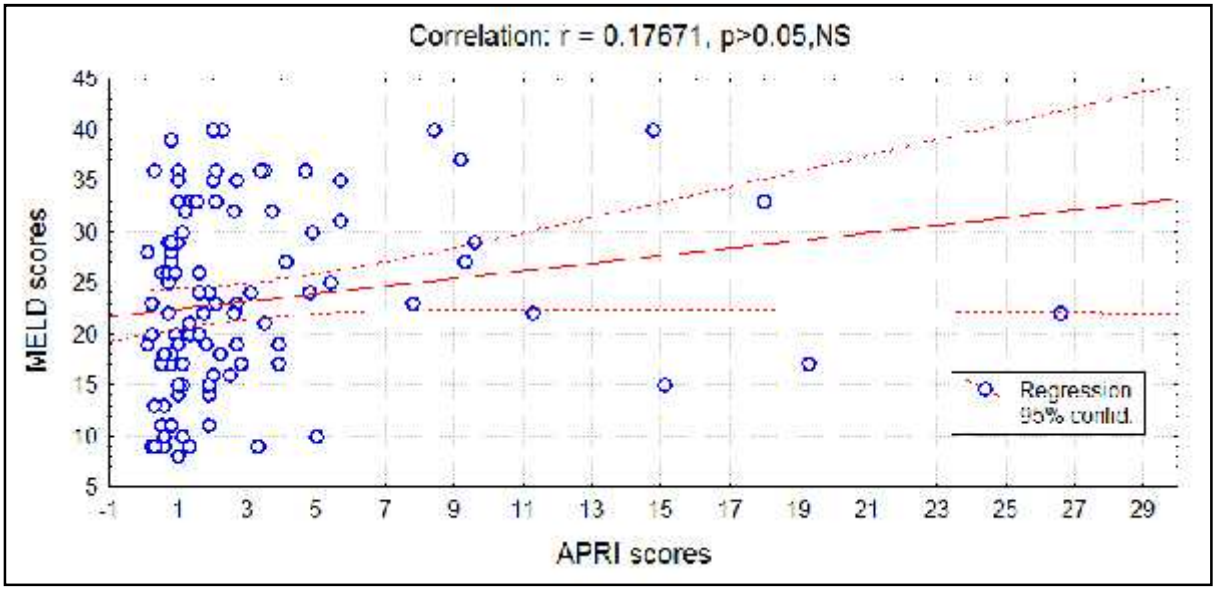
Table 15: Correlation between APRI and MELD scores by Karl Pearson's correlation coefficient.

Variables	r-value	t-value	p-value	Significance
APRI and MELD scores	0.1767	1.7953	0.0756	NS

ABBREVIATION- NS= not significant

From above Table 15, correlation between APRI and MELD score was studied; it showed that p-value was 0.0756 (not significant), r-value was 0.1767.

Figure 15: Scatter diagram showing the correlation between APRI and MELD scores.



From the above scatter diagram we observed that, there was no significant correlation between APRI and MELD score among the 102 subjects studied. P value was not significant.

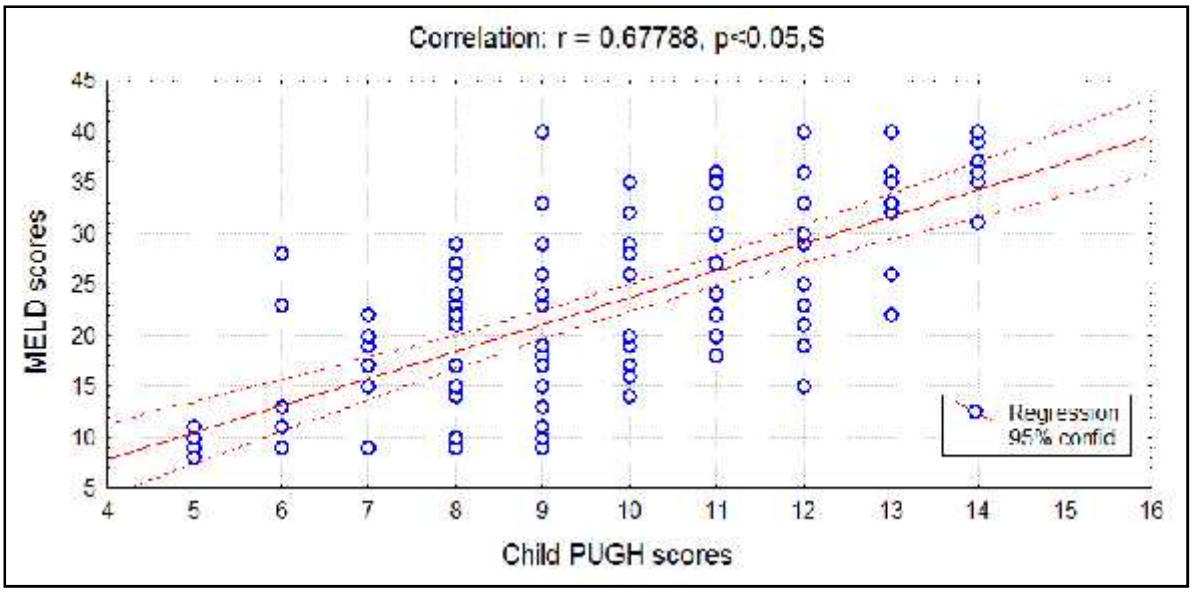
Table 16: Correlation between Child PUGH and MELD scores by Karl Pearson's correlation coefficient.

Variables	r-value	t-value	p-value	Significance
Child PUGH and MELD scores	0.6779	9.2206	0.0001	S

ABBREVIATION- S= significance

From above Table 16, correlation between Child-Pugh and MELD score was studied; it showed that p-value was 0.0001(significant), r-value was 0.6779.

Figure 16: Scatter diagram showing the correlation between Child Pugh and MELD scores.



From the above scatter diagram we observed that, there was positive correlation between Child-Pugh and MELD score. P value was significant.

Table 17: Comparison between APRI and Child PUGH criteria's.

APRI criteria	Child PUGH criteria				
	5—6 (class-A)	7—9 (class-B)	>=10 (class-C)	Total	%
<0.7	6	8	4	18	17.65
0.7 -1.5	1	15	17	33	32.35
>1.5	2	17	32	51	50.00
Total	9	40	53	102	100.00
%	8.82	39.22	51.96	100.00	
Agreement		Kappa	Std. Err.	Z-value	p-value
51.96%		0.1963	0.0749	2.6200	0.0044, S

From table 17 we observed that,

Out of the 53 patients belonging to the CP class C, 4 had APRI <0.7, 17 had APRI between 0.7-1.5 and 32 had APRI greater than 1.5.

Out of the 40 patients belonging to CP class B, 8 had APRI <0.7, 15 had APRI between 0.7-1.5, 17 patients had APRI above 1.5.

Out of the 9 patients belonging to CP class A, 6 had APRI <0.7, 1 had APRI between 0.7-1.5, 2 had APRI >1.5.

The kappa agreement between APRI and Child-Pugh score was 51.96%

Table 18: Comparison between APRI and MELD criteria's.

APRI criteria	MELD criteria				
	<18	18-36	>36	Total	%
<0.7	11	7	0	18	17.65
0.7 -1.5	10	22	1	33	32.35
>1.5	11	35	5	51	50.00
Total	32	64	6	102	100.00
%	31.37	62.75	5.88	100.00	
Agreement		Kappa	Std. Err.	Z-value	p-value
37.25%		0.1190	0.0509	2.3400	0.0097,S

From table 18 we observed that,

Out of the 6 patients belonging to the MELD score >36, 1 had APRI between 0.7-1.5 and 5 had APRI greater than 1.5.

Out of the 64 patients belonging to MELD score 18-36, 7 had APRI <0.7, 22 had APRI between 0.7-1.5, 35 patients had APRI above 1.5.

Out of the 32 patients belonging to MELD <18, 11 had APRI <0.7, 10 had APRI between 0.7-1.5, 11 had APRI >1.5.

The kappa agreement between APRI and MELD score was 37.25%

Table 19: Comparison between Child PUGH and MELD criteria's

Child PUGH criteria	MELD criteria				
	<18	18-36	>36	Total	%
5—6 (class A)	7	2	0	9	8.82
7—9 (class B)	20	19	1	40	39.22
>=10 (class C)	5	43	5	53	51.96
Total	32	64	6	102	100.00
%	31.37	62.75	5.88	100.00	
Agreement		Kappa	Std. Err.	Z-value	p-value
30.39%		-0.0006	0.0487	-0.0100	0.5045

From the above table no 19, we observed that,

Out of 32 patients belonging to MELD <18, 7 had CP score A, 20 had CP score B and 5 had CP score C.

Out of 64 patients belonging to MELD 18-36, 2 had CP score A, 19 had CPscore B and 43 had CP score C.

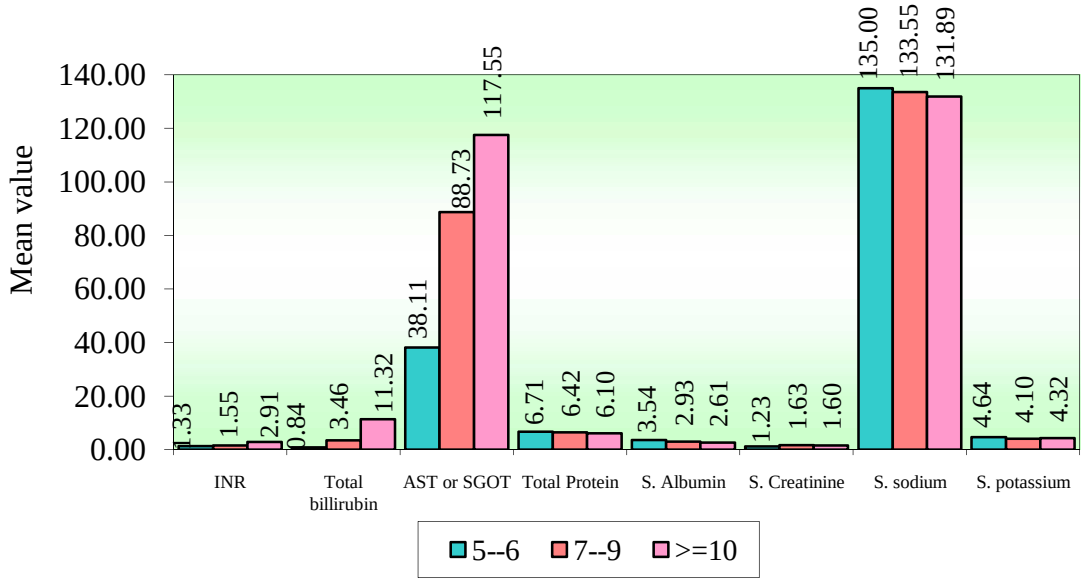
Out of 6 patients belonging to MELD >36, 1 had CP score B and 5 had CP score C.

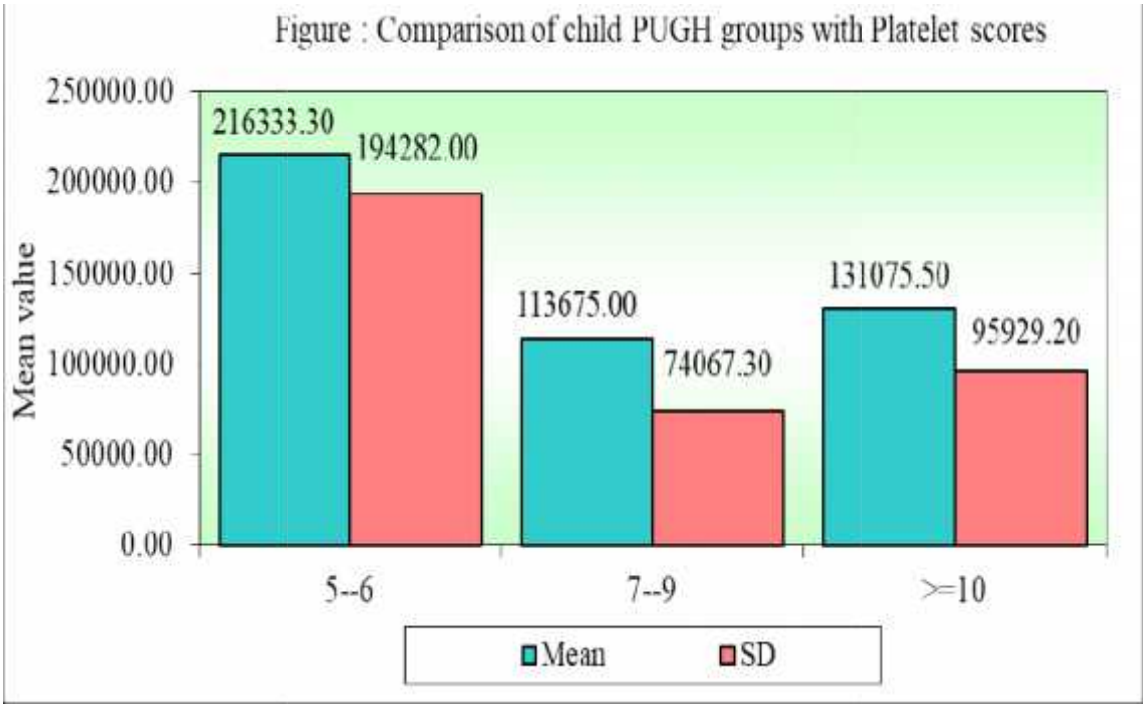
The kappa agreement between CP score and MELD score is 30.39%.

Table 20: Correlation between Child Pugh scores with clinical parameters by Karl Pearsons correlation coefficient method.

Clinical parameters	Correlation between child PUGH scores with			
	r-value	t-value	p-value	Signi.
Platelet	-0.1107	-1.1142	0.2679	NS
International nationalized ratio (INR)	0.3359	3.5657	0.0006	S
Total billirubin	0.5357	6.3444	0.0001	S
AST or SGOT	0.2504	2.5861	0.0111	S
Total Protein	-0.2225	-2.2817	0.0246	S
S. Albumin	-0.4496	-5.0341	0.0001	S
S. Creatinine	0.0806	0.8084	0.4208	NS
S. sodium	-0.1860	-1.8931	0.0612	NS
S. potassium	0.0988	0.9931	0.3231	NS

Figure 17 : Comparison of child PUGH groups with clinical parameters





From table no 20 and Fig 17, we observed that, there was significant positive correlation between Child-Pugh score and INR, Total Bilirubin, AST/SGOT.

Also there was significant negative correlation between Child-Pugh score and Total Protein and serum albumin.

DISCUSSION

A total of 102 patients with cirrhosis of the liver, admitted in the Dept. of Medicine in "KLES Dr. Prabhakar Kore Hospital and Medical Research Centre" were enrolled in the study during the period January 2019 to December 2019. The objective of the study was"To calculate APRI INDEX, Child Pugh score and MELD Score in cirrhosis of liver patients and to find a correlation between APRI Index, Child Pugh Score and MELD score in cirrhosis of liver patients."

AGE DISTRIBUTION

The mean age in our study population was 47.32 ± 12.91 years. Maximumage was observed in the age group of 41-50 years(34.31%). This was in concordance with a study conducted by B C Prakash et al.,[1] in Bangalore where the mean age was 46.14 years. Similarly, in another study done in New Delhi by Pravin Prabhu et al., the mean age was found to be 43.08 years.

GENDER DISTRIBUTION

In the present study, 86.27% of the patients with cirrhosis were males which is similar to a study done by B C Prakash et al[1]., in which 93 % of the subjects were males. In a study done by Ajay Pratap Singh et al.[5]76% were males.

ETIOLOGY OF CIRRHOSIS

Alcohol was the most common etiology for cirrhosis in our study, i.e. 72.55 %. This is in concordance with the study done by B C Prakash et al.[1],in which majority of the cases (75%) were alcoholic liver disease. Similarly, Princi Jain et al[6] conducted a study in 2015, 49 % of patients had alcoholic liver disease patients and 51 % were patients with viral hepatitis.

CLINICAL SIGNS

In our study, 87(85.29%) subjects showed signs of hepatocellular failure; anaemia was present in 77(75.49%) ; 69(67.65%) had hyperbilirubinemia; 66(64.74%) presented with ascites ; 61(59.80%) had edema; and hepatic encephalopathy features were seen in 28(27.48%) subjects. This was in concordance with study conducted by B C Prakash et al[1] which showed ascites in 82% and hepatic encephalopathy in 41%.

DIAGNOSIS-

a) Echotexture of liver-

In our study, we used ultrasound graded echotexture of liver for the diagnosis of chronic liver disease. Our study showed that maximum 33(32.35%) subjects had coarse echotexture with surface irregularity followed by increased echotexture of liver in 23(22.55%), coarse echotexture in 17(16.67%), altered echotexture with surface irregularity in 13(12.75%),Increased echotexture with surface irregularity in 5(4.90%), Coarse echotexture with mild surface irregularity in 4(3.92%), Mildly altered echotexture in 2(1.96%).

Coarse echotexture with caudate lobe hypertrophy, Mildly coarse echotexture, Mildly coarse echotexture with nodular margins, Mildly coarse echotexture with nodularity, Multiple nodules with increased echotexture was observed in 1subject(0.98%) each. This was in concordance with study conducted by J F Gerstenmer et al[60] which studied the ultrasound in chronic liver disease.

b) Size of liver on Ultrasound-

In our study, 55(53.92%) had normal size of liver; 26(25.49%) showed enlarged liver and 21(20.59%) showed shrunken liver. This was in concordance with study conducted by Sadisu Mohammad Maaji et al,[61] in 2013, which showed

enlarged liver in 31% and shrunken liver in 25% subjects with cirrhosis of liver disease.

c) Spleen size on ultrasound-

In our study, splenomegaly was seen in 81(79.41%) subjects. A study conducted by Sadisu Mohammad Maaji et al,[61] in 2013, showed splenic enlargement in 97% subjects.

d) Portal vein diameter on ultrasound-

In our study, dilated portal vein was seen in 54% subjects which was an indicator of portal hypertension in cirrhosis of liver. A study conducted by Sadisu Mohammad Maaji et al,[61] in 2013, showed dilated portal vein in 41%.

APRI INDEX

Our study evaluated the accuracy of APRI as a non-invasive marker for cirrhosis in Indians and our study showed that APRI is a fair and accurate marker for cirrhosis. The low cost and easy availability of its two variables (AST and platelets) makes APRI a useful and simple test.We observed that, maximum 51subjects(50%) had APRI >1.5 which indicates advanced liver fibrosis/cirrhosis; 33subjects(32.35%) had APRI between 0.7-1.5 and 18(17.65%) had APRI <0.7. The APRI mean value found in our study was 3.03 with standard deviation of 4.33. This was in concordance with study conducted by Dr Jithin George et al,[4] in 2018**.**Ajay Pratap Singh et al,[5] observed a mean APRI of 2.47 in their study.B C Prakash et al,[1] observed in their study that APRI index had a mean value of 3.4. Han Deng et al,[7] observed APRI mean of 4.05 in their study.

CHILD- PUGH SCORE

In our study, we observed that majority of subjects belonged to CP class C seen in 53(51.96%) subjects followed by CP class B seen in 40(39.22%) and CP class A was in 9(8.82%). Dr Jithin George et al, observed that 33 of his subjects belonged to CP class C, 15 to CP class B and 2 to CP class A. The mean CP score was 9.81 with standard deviation of 2.34 in our study. CP had a mean score of 13.2 in the study conducted by B C Prakash et al[1]. Han Deng et al,[7] observed in their study that CP had a mean score of 6.60.

COMPARISON BETWEEN CP SCORE AND CLINICAL PARAMETERS-

We found that there was significant positive correlation between CP score and INR with p value- 0.0006, CP score and total bilirubin with p value-0.0001, CP score and AST with p value-0.0111.

Also there was significant negative correlation between CP score and Total Protein with p value-0.0246, CP score and serum albumin with p value 0.0001. Our study could not find any correlation between CP score and platelet count , serum creatinine, serum sodium and serum potassium.

MELD SCORE

It was observed in our study that maximum subjects 64(62.75%) had MELD score between 18-36; followed by 32(31.37%) had MELD score of<18; and least subjects 6(5.88%) had MELD scoreof >36. Mean MELD score was 23.19 with standard deviation of 9.11 in this study. MELD mean score of 36.08 was seen in the study conducted by B C Prakash et al[1]. Han Deng et al,[7] observed in their study that MELD mean score was 5.07.

CORRELATION BETWEEN APRI, CP AND MELD SCORE

a) CORRELATION BETWEEN APRI AND CP-

In this study, it was found that, the majority of liver cirrhosis patients were in the APRI scoregroup > 1.5 categoryand inCPclass C. The analytical charts found a strong positive correlation between APRI scores and CP scores(r = 0.2106) and is significant, as indicated by a 'p' value of0.0336. The value of α is 0.05. This indicates that there is significant relationship between APRIscores and Child Pugh score.George et al,[4] stated that there was positive correlation between APRI and CP score with a significant 'p' value of <0.001. Another study done Adelia Muhlifa Saputriet al,[3] concluded that there was a weak correlation between APRIscores and CP scores as the 'p' value obtained in their study was 0.868 which was insignificant. This was not in concordance with our study.

b) CORRELATION BETWEEN APRI AND MELD-

In this study, it was found that, the majority of patients with cirrhosis of liver were in the APRI scoregroup > 1.5 category andin MELD score of 18-36. The statisticsamong APRI scores and MELD scores obtained a weak correlation (r = 0.1767) and is not significant as indicated by'p' value of0.0756. The value of α is 0.05. This was different from the study done by Dr Jithin George et al,[4] which showed a positive correlation between the two and obtained a significant 'p' value of <0.001. Also study done by B C Prakash et al,[1] showed positive correlation between the two variables and had significant 'p' value.

c) **CORRELATION BETWEEN CP SCORE AND MELD-**

In this study, it was found that, the majority of patients with cirrhosis of liverwere in the CP class C and with MELD scores 18-36. The results of the analysis of the relationship between CP scores and MELD scores obtained a strong positive correlation (r = 0.6779) and is significant, as indicated by a 'p' value of0.0001. The value of α is 0.05.

COMPARISON BETWEEN APRI, CP SCORE AND MELD SCORE-

In this study, it was found that, the majority of patients with cirrhosis of liver were in the APRI scoregroup > 1.5 category, in the CP ClassC and in MELD score 18-36. It was found that 6 patients with APRI score of <0.7 belonged to Child Pugh Class A, 15 patients of APRI score of 0.7-1.5 belonged to Child Pugh Class B and 32 patients with APRI score of >1.5 belonged to Child Pugh Class C.

11 patients with APRI score of <0.7 belonged to MELD score of <18, 22 patients with APRI score of 0.7-1.5 belonged to MELD score of 18-36 and 5 patients with APRI score of >1.5 belonged to MELD score of >36.

7 patients of Child Pugh Class A belonged to MELD score of <18, 19 patients of Child Pugh Class B belonged to MELD score of 18-36 and 5 patients of Child Pugh Class C belonged to MELD score of >36.

Thus, using kappa agreement it showed that APRI and CP score had agreement(similarity) percentage of 51.96%, APRI and MELD score had agreement(similarity) percentage of 37.25% and also CP score and MELD score had agreement (similarity) percentage of 30.39%. From these percentage values we observed that there is moderate to fair strength of agreement / similarity among the

three scores for being used interchangeably in assessing the severity of cirrhosis of liver.

Study conducted by B C Prakash et al,[1]found that prognostic performance of all 3 was comparable and all showed positive correlation with significant p value, also as the complications started setting in cirrhosis all the three scores increased. Study done by Dr Jithin George et al,[4] found a significant relationship of MELD Score and CP Score, and MELD Score and APRI index with significant p value. The study also showed significant 'p' value between APRI Index and CP Score.

Study done by Weilin Mao et al,[1] on 193 chronic Hepatitis B infected patients and concluded that, APRI is a simple and non-invasive scoring system that predicted risk for the development of liver-related complications and mortality in patients with Hepatitis B-related cirrhosis. The study also showed that APRI can be used as an additional marker for managing HBV-related cirrhosis patients.

Study conducted Wong et al,[6] on 3619 patients on Hepatitis B and Hepatitis C infected patients concluded that, APRI score is a good marker and can be used as alternate non-invasive marker in predicting fibrosis of liver.

In a study by Verma et al,[8] concluded that there issignificant correlation between APRI and hepatic venous pressure gradient (HVPG) with the median APRI score of 1.19 (range 0.17–7.92). The APRI of ≥1.09 was 66% sensitive and 73% specific with positive and negative predictive value of 85% and 47% respectively.

Ucar et al[6], suggested that APRI has better diagnostic value in patients with significantfibrosis as compared to other serum markers.

CONCLUSION

102 students were enrolled in our study based on clinical, laboratory and sonography features of cirrhosis of liver. The study was done to to calculate APRI index, CP score and MELD Score in cirrhosis of liver patients and to find a correlation between APRI index, CP Score and MELD score in cirrhosis of liver patients.Our findings revealed that there is a significant positive correlation between APRI Index, CP Score and MELD scores in subjects with cirrhosis of liver.

The mean age in our study population was 47.32 ± 12.91 years, with maximum distributionin the age group of 41-50 years(34.31%). In the present study, 86.27% of the patients with cirrhosis were males.Alcohol was the most common etiology for cirrhosis in our study, i.e. 72.55 %.

In our study, 87(85.29%) subjects showed signs of hepatocellular failure; anaemia was present in 77(75.49%); 69(67.65%) had hyperbilirubinemia; 66(64.74%) presented with ascites; 61(59.80%) had edema; and hepatic encephalopathy features was seen in 28(27.48%) subjects.

In our study, we used ultrasound graded echotexture of liver in diagnosing chronic liver disease. Maximum 33(32.35%) subjects had coarse echotexture with surface irregularity followed by increased echotexture of liver in 23(22.55%).

In our study, 55 subjects(53.92%) had normal size of liver; 26(25.49%) showed enlarged liver and 21(20.59%) showed shrunken liver, splenomegaly was seen in 81(79.41%) and dilated portal vein was seen in 54% subjects, which was an indicator of portal hypertension in cirrhosis of liver.

Our study appraised the preciseness of APRI as a non-invasive tool for prognosticating cirrhosis in Indians. Our studyforetold that, APRIcan be used as a marker for cirrhosis. We observed that, maximum 51subjects(50%) had APRI >1.5 which indicates advanced liver fibrosis/cirrhosis; 33 subjects (32.35%) had APRI between 0.7-1.5 and 18(17.65%) had APRI <0.7. The APRI mean value found in our study was 3.03 with standard deviation of 4.33.

In our study, we observed that majority of subjects belonged to Child Pugh C seen in 53(51.96%), followed by Child Pugh B seen in 40(39.22%) and Child Pugh A was in 9(8.82%). The mean Child Pugh score was 9.8, with standard deviation of 2.34 in our study.

We found that, there was significant positive correlation between CP score and INR with ‘p’ value 0.0006, CP score and total bilirubin with ‘p’ value-0.0001, CP score and AST with ‘p’ value-0.0111.

Also there was significant negative correlation between CP score and Total Protein with ‘p’ value-0.0246, CP score and serum albumin with ‘p’ value 0.0001. Our study could not find any correlation between CP score and platelet count, serum creatinine, serum sodium and serum potassium.

It was observed in our study, that maximum subjects 64(62.75%) had MELD score between 18-36; followed by 32(31.37%) had MELD score of <18; and 6 subjects (5.88%) had MELD score of >36. Mean MELD score was 23.19 with standard deviation of 9.11 in this study.

In this study, it was found that the analytical charts of the relationship between APRI scores and CP scores obtained a strongpositive correlation (r = 0.2106) and was

significant, as indicated by a ‘p’ value of0.0336. The value of α is 0.05. This indicates that there is significant relationship between APRIscores and CP score. Thus, showing that, higher the APRI index, higher is the CP score and more severe is the cirrhosis/fibrosis of liver.

In this study, it was seen that analysis of the relationship between APRI scores and MELD scores obtained a weak correlation (r = 0.1767). It was notsignificant, as indicated by a ‘p’ value of0.0756. The value of α is 0.05.

In this study, it was found that, the statistical analysis of the relationship between CP scores and MELD scores obtained a strong correlation (r = 0.6779) andissignificant as indicated by a ‘p’ value of0.0001.

In this study, it was found that 6 patients with APRI score of <0.7 belonged to Child Pugh Class A, 15 patients of APRI score of 0.7-1.5 belonged to Child Pugh Class B and 32 patients with APRI score of >1.5 belonged to Child Pugh Class C, indicating that maximum patients with higher APRI score belonged to Child Pugh Class C.Thus, showing that, the kappa agreement (similarity) between APRI and Child Pugh Score was 51.96% which suggests moderate strength of agreement/reliability among the two variables. From this observation, we conclude that, APRI index can be used interchangeably with Child Pugh score for prognosticating patients with cirrhosis of liver.

In this study, it was found that, 11 patients with APRI score of <0.7 belonged to MELD score of <18, 22 patients with APRI score of 0.7-1.5 belonged to MELD score of 18-36 and 5 patients with APRI score of >1.5 belonged to MELD score of >36. Thus, showing that, the kappa agreement (similarity) between APRI and MELD

score in cirrhosis of liver was 37.25%. This suggests fair strength of agreement/reliability among the two variables.

In this study it was found that, 7 patients of Child Pugh Class A belonged to MELD score of <18, 19 patients of Child Pugh Class B belonged to MELD score of 18-36 and 5 patients of Child Pugh Class C belonged to MELD score of >36. Thus showing that the kappa agreement (similarity) between Child Pugh and MELD score in cirrhosis of liver patients was 30.39% which suggests fair strength of agreement/reliability among the two variables.

Thus, this study concludes that, there was significant correlation between APRI, CP score and MELD score in cirrhosis of liver. We also found that, there was moderate strength of agreement/reliability among APRI and CP score as indicated by association of higher APRI index with CP Class C in patients with cirrhosis of liver. Our study, also found that there was fair strength of agreement/reliability between APRI-MELD score and CP score- MELD scores. Currently, the CP score is being used for severity assessment in patients with cirrhosis of liver.

Hence, a simple, non-invasive laboratory test such as APRI, which has a significant association with the severity of cirrhosis of liver, can be considered for use of prognostication of cirrhosis of liver in future clinical practice.

LIMITATIONS OF THE STUDY

This study has a few limitations-

1) The patients of cirrhosis were taken using the ultrasonography technique instead of liver biopsy, which is the gold standard for the diagnosis of cirrhosis.
2) The patients who had thrombocytopenia were not worked up for other causes and hence it could be a confounding factor as platelet count is one of the parameter used to calculate the APRI index.
3) Other causes for raised AST levels were not ruled out and hence could again be a confounding factor in calcution of APRI index.
4) Our study had limited sample size. A larger number of study population could have established a stronger correlation between APRI, Child-Pugh score and MELD score.

STRENGTH OF THE STUDY

In our study done on 102 patients, we could conclude by saying that there was positive correlation between APRI index and Child Pugh score.

We also observed that, higher APRI index scores were associated with increasing Child Pugh and MELD scores.

The study also showed that, there was moderate strength of agreement between APRI and Child Pugh score and hence both the scores could be used interchangeably for liver cirrhosis prognostication.

Hence, a simple, non-invasive laboratory test such as APRI, which has a significant association with the severity of cirrhosis of liver, can be considered for use of prognostication of cirrhosis of liver in future clinical practice.

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ANNEXURE I. ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office: 2472550
Principal 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 46 **Date: 24/11/2018**

To,
REG.NO.BG0118010
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CORRELATION BETWEEN APRI INDEX, MELD SCORE AND CHILD FUGH SCORE IN CIRRHOSIS OF LIVER – A ONE YEAR CROSS SECTIONAL STUDY IN KLE'S PRABHAKAR KORE HOSPITAL AND MEDICAL RESEARCH CENTRE, BELAGAVI",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE -II

INFORMED CONSENT

Dear Mr./Mrs./Dr._____,you are kindly requested to enroll yourself in a research study tilted, "CORRELATION BETWEEN APRI INDEX, MELD SCORE AND CHILD PUGH SCORE IN CIRRHOSIS OF LIVER" being conducted by **REG.NO.BG0118010,**a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of Dr._____ , Professor, Department of General Medicine, JawaharlalNehruMedicalCollege,Belgaum.Youhavebeenrequestedtoparticipateinthisa syoufitintothelaidoutcriteria forastudy'subject'/participant.Yourparticipationinstudyisvoluntary.Duringthestudyyou willbeaskedsome questionsandyouaresupposedtoanswertothebestofyourknowledge.Your decisionwhetherornottoparticipateinthestudywillnotaffectyourtreatment inanyform.Ifyoudecidetoparticipateyouarefreetowithdrawatanytime.

TITLEOFTHESTUDY:

"CORRELATIONBETWEENAPRIINDEX,MELDSCOREANDCHILDPUGHSCOR EINCIRRHOSISOFLIVER"

PURPOSEOFTHESTUDY:

TocomparethecorrelationofCHILDPUGHSCORE,MELDSCOREandAPRIIN DEXfortheassessmentofprognosisincirrhosisofliverpatients.

PROCEDURESINVOLVED:

Ifyouagreetoenrollyourselfinmystudy,youwillbe interviewedregardingyourpresent,pastandfamilyhistorythenyouwillbeclinicallyexamin edindetailandinvestigatedaccordingly.Thenyouwillbesubjectedtoafewbloodinvestigati ons,namelyLiverfunctiontests,Plateletcount,Serum Bloodureanitrogen,Serum Creatinine,PT/INR.

RISKSANDBENEFITS:

Therearenopotentialrisksinvolvedinthisstudy.Benefitsoftakingpartinthisresearc h:Bytakingpartinthisstudy,prognosis andriskofdevelopmentofcirrhosisoflivercanbedetectedwiththehelpofnon-invasivemarkerslikeplateletandaspartateaminotransferase.

VOLUNTARYPARTICIPATION/WITHDRAWALFROM THESTUDY:

Takingpartinthestudyisvoluntary.Youmaychoosenottoenrollyourselfinthisstudy andmaychoosetoleavethestudyanytimeinbetween.

ALTERNATIVES:

Yourdecisionregardingparticipationinstudywillnotchange presentorfuturehealthcareservicesofferedtoyouatKLESDr.PrabhakarKore HospitalandMedicalResearchCentre,Belgaum.Youwouldsimplybeexcluded from thestudyifyouwishto,andallyourdetailsshallbekeptconfidentialandyouwillgettheroutine lineofmanagement.

PRIVACYANDCONFIDENTIALITY:

Alldatacollectedordisclosedbyyouduringthecourseofparticipationofstudy,willbekeptfullyconfidential.Ifhowever duringthecourseitbecomesnecessaryfortheprogressofthecoursetodisclose theidentity,itwouldbedonesoonlyafteryourinformed&writtenconsent.

Theonlypeopletoknowthatyouarea researchsubjectaremembersoftheresearchteam.Noinformationaboutyouwillbedisclosed withoutyourwrittenpermissionexcept:Inemergencytoprotectyourrights ANDwelfare ifrequiredbylaw.

AUTHORIZATIONTOPUBLISHRESULT:

Theresultsofthestudymaybeusedto publishanarticle.Whentheresultsofresearchpublishedordiscussed,inaconference,noinformationwillbedisplayedthatwoulddiscloseyouridentity.

Anyinformationobtainedinconnectionwiththisstudyandthatcanbeidentified withyouwillremainconfidential.

FINANCIALINCENTIVESFORPARTICIPATION:

Noadditionalcostsshallbeincurreduponyouforthepurposeofthisstudy.Itispurelybeingdonewiththeideaofresearchandallthecostofstudywillbebornebytheinvestigator.

COMPENSATION:

Inthe event thatyoubecomeinjuredasaresultoftakingpartinthisstudy,treatmentwillbeofferedtoyouat KLESDr.PrabhakarKoreHospitalandMedicalResearchCentre,Belgaum,oryouwillbegiveninformationaboutwheretoreceivemedicalcare.However,noreimbursement,compensationorfreemedicalcarewillbegiven.

QUESTIONS / CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

PRINCIPAL INVESTIGATOR:

REG.NO.BG0118010

MD (Post Graduate Student),

Department of General Medicine,,

Jawaharlal Nehru Medical College, Nehru Nagar,

KLE Hospital, Belagavi 590010,

GUIDE:

Dr._____,

MD (GEN.MEDICINE), FICP

Professor, Head of Department of General Medicine,

Jawaharlal Nehru Medical College, Nehru Nagar,

KLE Hospital Road,

Belagavi 590010

Dr. Roopa M Bellad, M.D. D.C.H (PROFESSOR OF PAEDIATRICS)

Chairman,

J.N.M.C Ethical Committee for Human Research, 9448113403

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I maywithdraw at anytime. I am not giving up any of my legal rights by signingthis form. My signature below indicates that I have read this consent form,or it has been read to me, this consent form and have had all the questionsanswered.

Signature/Left Thumb print of the Participant or legally authorized representative

Participant's name:……...

Signature/Left thumb impression of the participant: ……

Name of the legally authorized representative/guardian:……

Signature/Left thumb impression:……...

Witness' name:……...

Signature/Left thumb impression:……...

Investigator's name and signature:……...

Date:

Place:

ತಿಳಿವಳಿಕೆ

ಆತ್ಮೀಯ ಶ್ರೀ / ಶ್ರೀಮತಿ / ಡಾ. _____, ನೀವು ಎಂಡಿ ಜನರಲ್ ಮೆಡಿಸಿನ್ ನಲ್ಲಿ ಪೋಸ್ಟ್ ಪದವೀಧರ ವಿದ್ಯಾರ್ಥಿ ಡಾ. ನಡೆಸಿದ "ಯಕೃತ್ತಿನ ರೋಗಿಗಳ ಸಿರೋಸಿಸ್ನಲ್ಲಿನ ಮುನ್ನರಿವಿನ ಮೌಲ್ಯಮಾಪನಕ್ಕಾಗಿ CHILD PUGH SCORE, MELD SCORE ಮತ್ತು APRI INDEX ನ ಪರಸ್ಪರ ಸಂಬಂಧ", ಬಾಗಿರುವ ಸಂಶೋಧನಾ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮನ್ನು ತೊಡಗಿಸಿಕೊಳ್ಳಲು ವಿನಂತಿಸಲಾಗಿದೆ. ಬೆಳಗಾವಿ ಜವಾಹರಲಾಲ್ ನೆಹರು ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ನ ಪ್ರೊಫೆಸರ್, ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಪ್ರೊಫೆಸರ್ * ಅವರ ನೇರ ಮೇಲ್ವಿಚಾರಣೆ ಮತ್ತು ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನವನ್ನು ಕೈಗೊಳ್ಳಲಾಗುವುದು.

'ವಿಷಯ' / ಪಾಲ್ಗೊಂಡಿರುವ ಅಧ್ಯಯನಕ್ಕೆ ನೀವು ಸಿದ್ಧಪಡಿಸಿದ ಮಾನದಂಡಕ್ಕೆ ಸರಿಹೊಂದುವಂತೆ ಇದನ್ನು ಭಾಗವಹಿಸಲು ನಿಮ್ಮನ್ನು ವಿನಂತಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ. ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮಗೆ ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ನಿಮ್ಮ ಜ್ಞಾನದ ಅತ್ಯುತ್ತಮವಾದ ಉತ್ತರಕ್ಕೆ ನೀವು ಉತ್ತರಿಸಬೇಕು. ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಬೇಕೇ ಅಥವಾ ಇಲ್ಲವೇ ಎಂಬ ನಿಮ್ಮ ನಿರ್ಧಾರವು ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯನ್ನು ಯಾವುದೇ ರೂಪದಲ್ಲಿ ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂಪಡೆಯಲು ಸ್ವತಂತ್ರರಾಗಿರುತ್ತಾರೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ:
ಯಕೃತ್ತಿನ ರೋಗಿಗಳ ಸಿರೋಸಿಸ್ನಲ್ಲಿನ ಮುನ್ನರಿವಿನ ಮೌಲ್ಯಮಾಪನಕ್ಕಾಗಿ CHILD PUGH SCORE, MELD SCORE ಮತ್ತು APRI INDEX ನ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಹೋಲಿಸಲು.

ಅಧ್ಯಯನದ ಉದ್ದೇಶ: ಯಕೃತ್ತಿನ ರೋಗಿಗಳ ಸಿರೋಸಿಸ್ನಲ್ಲಿನ ಮುನ್ನರಿವಿನ ಮೌಲ್ಯಮಾಪನಕ್ಕಾಗಿ CHILD PUGH SCORE, MELD SCORE ಮತ್ತು APRI INDEX ನ ಪರಸ್ಪರ ಸಂಬಂಧವನ್ನು ಹೋಲಿಸಲು.
ಕಾರ್ಯವಿಧಾನಗಳು ಸೇರಿಕೊಂಡವು: ನನ್ನ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮನ್ನು ತೊಡಗಿಸಿಕೊಳ್ಳಲು ನೀವು ಒಪ್ಪಿಕೊಂಡರೆ, ನಿಮ್ಮ ಪ್ರಸ್ತುತ, ಹಿಂದಿನ ಮತ್ತು ಕುಟುಂಬದ ಇತಿಹಾಸದ ಬಗ್ಗೆ, ಸಂದರ್ಶನ ಮಾಡಲಾಗುವುದು, ನಂತರ ನೀವು ಪ್ರಾಯೋಗಿಕವಾಗಿ ವಿವರವಾಗಿ ಪರಿಶೀಲಿಸಲಾಗುವುದು ಮತ್ತು ಅದಕ್ಕೆ ಅನುಗುಣವಾಗಿ ತನಿಖೆ ಮಾಡಲಾಗುತ್ತದೆ.

ನಂತರ ನೀವು ಕೆಲವು ರಕ್ತ ಪರೀಕ್ಷೆಗಳಿಗೆ ಒಳಪಡುತ್ತಾರೆ, ಅವುಗಳೆಂದರೆ ಲಿವರ್ ಫಂಕ್ಷನ್ ಪರೀಕ್ಷೆಗಳು, ಪ್ಲೇಟ್ಲೆಟ್ ಎಣಿಕೆ, ಸೀರಮ್ ಬ್ಲಡ್ ಯೂರಿಯಾ ನೈಟ್ರೋಜನ್, ಸೀರಮ್ ಕ್ರಿಯೇಟಿನೈನ್, ಪಿಟಿ / ಐಎನ್ಆರ್.

ಅಪಾಯಗಳು ಮತ್ತು ಲಾಭಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಯಾವುದೇ ಸಂಭವನೀಯ ಅಪಾಯಗಳಿಲ್ಲ.
ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಪ್ರಯೋಜನಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೂಲಕ, ಪಿತ್ತಜನಕಾಂಗದ ಸಿರೋಸಿಸ್ ಬೆಳವಣಿಗೆಯ ಅಪಾಯ ಮತ್ತು ಅಪಾಯದ ಅಪಾಯವನ್ನು ಪ್ಲೇಟ್ಲೆಟ್ ಮತ್ತು ಆಸ್ಪರ್ಟೇಟ್ ಅಮಿನೊಟ್ರಾನ್ಸ್ಫೆರೇಸ್ನಂತಹ ಆಕ್ರಮಣಶೀಲ ಗುರುತುಗಳ ಸಹಾಯದಿಂದ ಕಂಡುಹಿಡಿಯಬಹುದು.

ಅಧ್ಯಯನದಿಂದ ಸಂಭಾವ್ಯ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆ / ವಿರೋಧಿ: ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮನ್ನು ಸೇರಿಸಿಕೊಳ್ಳದಿರಲು ನೀವು ಆಯ್ಕೆ ಮಾಡಬಹುದು ಮತ್ತು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಯಾವ ಸಮಯದಲ್ಲಾದರೂ ಅಧ್ಯಯನವನ್ನು ಬಿಡಲು ಆಯ್ಕೆ ಮಾಡಬಹುದು.

ಪರ್ಯಾಯಗಳು: ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯ ಬಗ್ಗೆ ನಿಮ್ಮ ನಿರ್ಧಾರವು ನಿಮಗೆ KLES ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಬೆಳಗಾಮ್ನಲ್ಲಿ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೋಗ್ಯ ಸೇವೆಗಳನ್ನು ಬದಲಿಸುವುದಿಲ್ಲ. ನೀವು ಬಯಸಿದಲ್ಲಿ ನೀವು ಕೇವಲ ಅಧ್ಯಯನದಿಂದ ಹೊರಗಿಡಬೇಕು, ಮತ್ತು ನಿಮ್ಮ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಗೌಪ್ಯವಾಗಿಡಬೇಕು ಮತ್ತು ನೀವು ನಿರ್ಗಹಣೆಯ ನಿಯಮಿತ ಮಾರ್ಗವನ್ನು ಪಡೆಯುತ್ತೀರಿ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ: ಅಧ್ಯಯನದ ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಯ ಸಮಯದಲ್ಲಿ ನೀವು ಸಂಗ್ರಹಿಸಿದ ಅಥವಾ ಬಹಿರಂಗಪಡಿಸಿದ ಎಲ್ಲ ಡೇಟಾವನ್ನು ಸಂಪೂರ್ಣವಾಗಿ ಗೌಪ್ಯವಾಗಿರಿಸಲಾಗುತ್ತದೆ. ಆದರೆ ಸಹಜವಾಗಿ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲು ಕೋರ್ಸ್ ಪ್ರಗತಿಗೆ ಅಗತ್ಯವಾದರೆ, ನಿಮ್ಮ ಮಾಹಿತಿಯ ನಂತರ ಮಾತ್ರ ಅದನ್ನು ಮಾಡಲಾಗುವುದು.

ತುರ್ತುಸ್ಥಿತಿಯಲ್ಲಿ, ನಿಮ್ಮ ಹಕ್ಕುಗಳನ್ನು ಮತ್ತು ಕಲ್ಯಾಣವನ್ನು ರಕ್ಷಿಸಲು.
ಕಾನೂನಿನಿಂದ ಅಗತ್ಯವಿದ್ದರೆ.
ಪ್ರಕಟಣೆಯ ಪ್ರಕಟಣೆ: ಲೇಖನವನ್ನು ಪ್ರಕಟಿಸಲು ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಬಳಸಬಹುದು. ಸಂಶೋಧನೆಯ ಫಲಿತಾಂಶಗಳು ಪ್ರಕಟವಾದಾಗ ಅಥವಾ ಚರ್ಚಿಸಿದಾಗ, ಒಂದು ಸಮ್ಮೇಳನದಲ್ಲಿ, ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವ ಯಾವುದೇ ಮಾಹಿತಿಯನ್ನು ಪ್ರದರ್ಶಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಯಾವುದೇ ಮಾಹಿತಿ ಮತ್ತು ನಿಮ್ಮೊಂದಿಗೆ ಗುರುತಿಸಬಹುದಾದ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಉಳಿಯುತ್ತದೆ.

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಹಣಕಾಸಿನ ಒಳನೋಟಗಳು: ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚಗಳನ್ನು ನಿಮಗೆ ನೀಡಲಾಗುವುದಿಲ್ಲ. ಇದು ಕೇವಲ ಸಂಶೋಧನೆಯ ಪರಿಕಲ್ಪನೆಯಿಂದ ಮಾಡಲ್ಪಟ್ಟಿದೆ ಮತ್ತು ಅಧ್ಯಯನದ ಎಲ್ಲಾ ವೆಚ್ಚವನ್ನು ತನಿಖೆದಾರರು ಹೊಂದುತ್ತಾರೆ.

ಪರಿಹಾರ: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಪರಿಣಾಮವಾಗಿ ನೀವು ಗಾಯಗೊಂಡರೆ, ಕೆಎಲ್ಎಸ್ ಡಾ. ಪ್ರಭಾಕರ್ ಕೋರೆ ಆಸ್ಪತ್ರೆ ಮತ್ತು ವೈದ್ಯಕೀಯ ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಬೆಳಗಾವಿನಲ್ಲಿ ನಿಮಗೆ ಚಿಕಿತ್ಸೆಯನ್ನು ನೀಡಲಾಗುವುದು ಅಥವಾ ವೈದ್ಯಕೀಯವನ್ನು ಎಲ್ಲಿ ಪಡೆಯಬೇಕು ಎಂಬುದರ ಬಗ್ಗೆ ನಿಮಗೆ ಮಾಹಿತಿ ನೀಡಲಾಗುವುದು. ಆದ್ಯಕೆ. ಹೇಗಾದರೂ, ಯಾವುದೇ ಮರುಪಾವತಿ, ಪರಿಹಾರ ಅಥವಾ ಉಚಿತ ವೈದ್ಯಕೀಯ ಆರೈಕೆ ನೀಡಲಾಗುವುದು.

ಪ್ರಶ್ನೆಗಳು / ಸಂಪರ್ಕ ವಿವರಗಳು: ಕೆಳಗಿನ ಹೆಸರನ್ನು ನೀವು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತವಾಗಿರಬೇಕು

ಪ್ರಿನ್ಸಿಪಲ್ ಇನ್ವೆಸ್ಟಿಗಾರ್ಟರ್:
ಡಾ. _ (ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ), ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜವಾಹರಲಾಲ್ ನೆಹರು ಮೆಡಿಕಲ್ ಕಾಲೇಜು, ನೆಹರು ನಗರ, ಕೆಎಲ್ಇ ಆಸ್ಪತ್ರೆ ರಸ್ತೆ, ಬೆಲಾಗವಿ 590010,

ಗೈಡ್:
ಡಾ. (GEN ಮೆಡಿಕೈನ್), FICP
ಪ್ರೊಫೆಸರ್, ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗ, ಜವಾಹರಲಾಲ್ ನೆಹರೂ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು,
ನೆಹರು ನಗರ, ಕೆಎಲ್ ಆಸ್ಪತ್ರೆ ರಸ್ತೆ, ಬೆಳಗಾವಿ 590010,

ಡಾ. ರೂಪಾ ಎಂ ಬೆಲ್ಲಾಡ್, ಎಮ್.ಡಿ. ಡಿ.ಸಿ.ಎಚ್ (ಪ್ರೊಫೆಸರ್ ಆಫ್ ಪೆಡಿಯಾಟ್ರಿಕ್ಸ್ ಅಧ್ಯಕ್ಷರು,
ಜೆ.ಎನ್.ಎಂ.ಸಿ. ಮಾನವ ಸಂಶೋಧನೆಗಾಗಿ ಎಥಿಕಲ್ ಕಮಿಟಿ, 9448113403

ಒಪ್ಪಿಗೆ ಪತ್ರ
ನಾನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂಪಡೆಯಬಹುದು. ಈ ಫಾರ್ಮ್ ಅನ್ನು ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ನಾನು ಬಿಟ್ಟುಕೊಡುವುದಿಲ್ಲ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನಾನು ಈ ಸಮ್ಮತಿಯ ನಮೂನೆಯನ್ನು ಓದಿದ್ದೇನೆ ಎಂದು ಸೂಚಿಸುತ್ತದೆ, ಅಥವಾ ನನಗೆ ಈ ಸಮ್ಮತಿ ರೂಪವನ್ನು ಓದಿದೆ ಮತ್ತು ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿದ.

ಸಹಿ / ಪಾಲ್ಗೊಳ್ಳುವವರ ಅಥವಾ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿಗಳ ಎಡ ತಮ್ ಮುದ್ರಣ

ಭಾಗವಹಿಸುವವರ ಹೆಸರು

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಗುರುತು ಭಾಗವಹಿಸುವವರ

ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕಾರ ಪಡೆದ ಹೆಸರು: .....
ಪ್ರತಿನಿಧಿ / ರಕ್ಷಕ

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಗುರುತು: .....

ವಿಟ್ನೆಸ್ 'ಹೆಸರು : .....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಗುರುತು: .....

ತನಿಖಾಧಿಕಾರಿ ಹೆಸರು ಮತ್ತು ಸಹಿ: .....

ದಿನಾಂಕ:

ಸ್ಥಳ:

जानकारी मिलने के बाद दी गई सम्मती

माननीय श्री/ श्रीमती/डा. _____ इस संशोधन अभ्यासमें "एपीआरआय इन्डेक्स, एमीएलडी स्कोअर और चाईल्ड पण स्कोअर इनका यकृतके कार्य में खराबी आने का क्या संबंध है।" यह है और यहह अभ्यासक्रम डा. ; पोस्ट ग्रेज्युएट विद्यार्थीनी जे.एन.एम.सी. कालेज बेळगावी 7996185618 जो जनरल मेडीसीनमें एम डी पदवी प्राप्त करने हेतु कर रही है। यह अभ्यासक्रम व्ह डा. प्रोफेसर सामान्य औषधी विभाग इनके मार्गदर्शन मुत्ताबिक जवाहरलाल नेहरु मेडीकल कालेज बेळगावी, इस संस्थामें कर रही है।

आप इस अभ्यास करने हेतु योग्य होने के कारण आपको इसमें आमंत्रित किया जा रहा है और आपको यह उचित लगता है आप इस अभ्यासक्रम से सम्मिलित हो सकते हो।

आपकी सम्मति इस अभ्यासक्रमके लिए आपकी इच्छा हो तो दे सकते हो। इस अभ्यास संशोधनमें आपको कई सवाल पुछे जायेंगे और आपको इसके जवाब में आपके बुध्दी के अनुरुप देने होंगे । इस अभ्यासक्रममें आपने हिस्सा लिया या न लिया तो भी आपको यहांपर मिलनेवाली वैद्यकिय सुविधाओपर कई असर नही होगा। आप इस अभ्यासक्रममें शामिल होने के बाद आप किसी भी वक्त / समयपर आपका नाम इस अभ्यास संधोधन से कम कर सकते हो ।

- **अभ्यासाचा विषय** – "एपीआरआय इन्डेक्स, एमीएलडी स्कोअर और चाइल्ड पग स्कोअर इनका यकृतके कार्य में खराबी आने का क्या संबंध है" ऐसा है।
- **अभ्यास का हेतु** : चाईल्ड पग स्कोअर, एमीएलडी स्कोअर और एपीआरआई इंडेक्स इनका आपसमें क्या संबंध है, इसकी तुलनात्मक जानकारी यकृत मे खराबी होने पर हासील करना है ।
- **पध्दती** : यदी आप इस संशोधन अभ्यास में सम्मिलित होना चाहते हो तो आपको आपकी वर्तमान और भूतकाल की जानकारी और आपकी कुटुंब का इतिहास पूछा जायेगा । इसके बाद आपकी वैद्यकीय जांच की जायेगी और आपके रक्त का नमूना लेकर यकृत किस प्रकार कार्य कर रहा है, यह पेटलेट के छोटे कणोंकी संख्या, रक्त का सिरम, मुत्रमें युरिया नैट्रोजन, और सिरम क्रियाटीनीन, पी टी/ आय एन आर इनकी जानकारी, रक्त की जांच करके कि जायेगी।
- **धोके और मुनाफा** :

इस अभ्यास क्रममें कोई धोका नही है ।

- संशोधन अभ्यासक्रमसे मिलनेवाले **फायदे :**
  इस अभ्यासक्रम में हिस्सा लेने से रोगी में यकृत का संतुलन खराब होने से किसप्रकार रोगोंका संक्रमण होता है और उसका पूर्व निदान का अनुमान लगाय जा सकता है और रोगपर किस प्रकार से काबू किया जाय तथा रोका जाय इसका पेटलेट और एस्परेट अमिनोट्रान्सपसी के उपयोग करके समझना है।
- **अभ्यास क्रम में सम्मिलिन होना या उसमें से अपना नाम कम करना :**
  आप इस अभ्यासक्रममें स्वयंभू सम्मिलित हो सकते हो। आप इस अभ्यासक्रम में हिस्सा लेना या न लेना यह तय कर सकते हो और आपका नाम इस अभ्यासक्रम से कभी भी, किसी भी समय कम कर सकते हो।
- **पर्याय :** आप के इस निर्णय से आपको भविष्यमें के.एल.ई.एस. डा. प्रभाकर कोरे हास्पीटल और संशोधन केंद्र बेळगावी से मिलनेवाली वैद्यकिय सेवाओंपर कोई भी असर नही होगा । और आपसे प्राप्त हुई जानकारी गुप्त रखी जाएगी और आपको योग्य सेवाएं प्रदान कि जाएगी।
- **गोपनियता :** इस संशोधन में आपके द्वारा प्राप्त की हुई जानकारी पूर्णत : गोपनिय रखी जाएगी। यदी संशोधन के दौरान इसकी जरुरत पडी तो, उसको उपयोगता में लाया जाएगा और यह कारवाई करने के लिए आपसे लेखी अनुमती प्राप्त की जाएगी।
  सिर्फ संशोधन करनेवाले समुदाय को आप इस संशोधन का एक हिस्सा है इसकी जानकारी होगी। आपके द्वारा दी गई कोई भी जानकारी किसीको भी आपकी लेखी सहमती लेकर ही दी जाएगी। लेकिन इसके लिए निम्नलिखित अपवाद यह है।
  --- अपादकाल में इसका उपयोग आप के अधिकार्व कल्याण को ध्यान में रखने हुए किया जाएगा।
  --- कानून के मुताबिक इसको उपयोग में लाया जाएगा।
- **अभ्यास के निष्कर्ष प्रसिध्द करने के अधिकार :**
  अभ्यास के निष्कर्ष लेखाद्वारा प्रसिध्द किये जाएंगे । इस संशोधन के नजीजोंपर सभा में चर्चा की जाएगी । लेकिन इससे आपके बारे में किसी भी प्रकार की जानकारी नही दी जाएगी । आप के द्वारा मिली हुई पुरी जानकारी फ़ोपनिय रखी जाएगी ।
- सम्मिलित होनेवालोंको मिलनेवाली आर्थिक सहायता :

- इस अभ्यासक्रम में आप को पैसा देनेकी जरुरत नही है। यह सब संशोधन के हेतु किया जा रहा है। और इसका पुरा खर्चा संशोधक ही करेंगे ।
- नुकसानभरपाई :
  इस संशोधन अभ्यास को करते समय आपको कोई जख्म होगी तो आपको के.एल.ई.एस. डा. प्रभाकर कोरे और मेडीकल संशोधन केंद्र द्वारा इलाज किया जाएगा या आपको आगे कहांसे वैद्यकीय सेवा प्राप्त होगी यह बताया जाएगा। लेकिन किसी प्रकार की, नुकसानभरपई नही दी जाएगी ।
- **निराकरण और संपर्क :**
  आप निम्नलिखित व्यक्ती जिनका पता नमुद किया है उनसे इस संशोधन हेतु जानकारी प्राप्त कर सकते हो या किसी भी प्रकार की मदद मांग सकते हो ।

प्रमुख संशोधक : डा.

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सम्मती पत्र

मै निचे सही करनेवाला स्वइच्छेसे इस अभ्यासमें भाग लेने के लिए मान्यता देता हू । मै अपना नाम किसी भी वक्त इसमेसे वापस ले सकता हू और इस सम्मती के कारण मै मेरे कोई भी कानुनी हक्क नही छोड रहा हू । यह सब उपर के विषय के बारे में स्वयं पढकर या पढने के सूनने के बाद मैं इस सम्मती पत्र पर अपने हस्ताक्षर कर के सभी प्रश्नो का उत्तर दिया हू ।

हिस्सा लेने वाले का नाम : .....सही/अंगठा

साक्षीदार का नाम: .....सही/अंगठा

परीक्षण करने वाले का नाम:.....सही

तारीख: स्थळ:

माहिती मिळाल्यानंतर दिलेली सहमती

माननीय श्री/ श्रीमती/डाक्टर. _____ हा संशोधन अभ्यासक्रम "एपीआरआय इन्डेक्स, एमीएलडी स्कोअर आणि चाइल्ड पग स्कोअर यांच्यामध्ये यकृतामध्ये बिघाड झाल्यानंतर काय संबंध आहे." याचा असून हा अभ्यासक्रम डा.

पोस्ट ग्रेज्युएट विद्यार्थीनी जे.एन.एम.सी. कालेज बेळगावी,

मेडीसीन अभ्यासक्रमात एम डी पदवी प्राप्त करण्यासाठी करत आहेत. सदरचा अभ्यासक्रम हा त्या डा. आरथी दर्शन प्रोफेसर सामान्य औषधी विभग यांच्या मार्गदर्षनखली जवाहरलाल नेहरु मेडीकल कालेज, बेळगावि येथे करत आहेत.

तुम्ही हा अभ्यासासाठी योग्य असल्यामुळे आम्ही आमच्या अभ्यासासाठी तुम्हास आमंत्रित केले आहे आणि तुम्हास योग्य वाटत असेल तर तुम्ही या अभ्यासक्रमात सहभागी होऊ शकत्ता.

तुमचा सहभाग हा या अभ्यासक्रमात तुमच्या इच्छेनुसार असेल. या अभ्यासक्रमामध्ये तुम्हास कांही प्रश्न विचारले जातील व त्याची तुम्ही योग्य त्ती उत्तरे तुमच्या विवेकाबुध्दीनुसार घ्यावीत. या अभ्यासक्रमात तुम्ही भाग घेतला किंवा घेतला नाही तरी सुध्दा तुम्न्हास मिळण्याच्या वैद्यकीय सेवेवर याचा परिणाम होणार नाही. तुम्ही या अभ्यासक्रमात भाग घेतल्यानंतर सुध्दा तुम्ही तुमचे नांव या अभ्यासक्रमातून कोणत्याही क्षणी काढून घेऊ शकता.

अभ्यासाचा विषय – "एपीआरआय इन्डेक्स, एमीएलडी स्कोअर आणि चाइल्ड पग स्कोअर यांच्यामध्ये यकृतमध्ये बिघाड झाल्यानंतर काय संबंध आहे." असा आहे.

- **अभ्यासाचा उद्देश :** चाइल्ड पग स्कोअर, एमीएलडी स्कोअर आणि एपीआरआई इंडेक्स यांचा यकृतामध्ये बिघाड झाल्यानंतर एकमेकांशी काय संबंध आहे. याचा तुलनात्मक अभ्यास करणे असा आहे.
- पध्दती : जर का तुम्ही संधोधन अभ्यासात सहभगी होत असाल तर तुमच्या विषयीची सध्याची, पूर्वीची माहिती व तुमचा कौटुंबिक इतिहास विचारला जाईल. यानंतर वैद्यकीय तपासणी केली जाईल आणि तुमच्या रक्ताच्या काही चाचण्या केल्या जातील. त्यामुळे यकृत कसे कार्य करत्ते आहे, हे मुत्राम्ध्ये किती युरीया नैट्रोजन आहे तसेच सिरम क्रिअटीनीन, पीटी / आय एन आर. किती आहे याची तपासणी केली जाईल.
- धोके आणि फयदे :
  या अभ्यासाम्ध्ये कोणते धोके नाहीत.

- संशोधन अभ्यासामुळे मिळालेले फयदे:

  या अभ्यासक्रमात भाग घेतल्यामुळे यकृतात बिघाड कशाप्रकारे सुरु गोतो व तो कशा प्रकारे वाढतो याचे पूर्व निदान करता येईल आणि रोगाचे कशाप्रकारे नियंत्रण करता येईल हे पेटलेट आणि अस्परेट आमिनोट्रान्सफरन्सी चा उपयोग करुन समजून घेतले जाईल.

  स्वतःगून दिलेली सहमती किंवा सहमती न होण्याची क्रिया :

  तुमच्या या अभ्यासामध्ये सहभाग हा स्वयंभू असेल आणि या अभ्यास संशोधनातून तुम्ही आपले नंव कोणत्याही क्षणी काढून घेऊ शकता किंवा तुम्ही या अभ्यासक्रमात तुमच्या इच्छेनुसार भाग न घेऊ शकता.

- पर्याय : या अभ्यासक्रमात तुम्ही भाग न घेतल्यामुळे तुम्हास के.एल.ई.एस. डा. प्रभाकर कोरे हास्पीटल व संशोधन केंद्र बेळगावी मधून मिळण्याच्या वैद्यकीय सेवेवर कोणताही परिणाम होणर नाही. तुमची इच्छा असेल तर तुमचे नांव या अभ्यासक्रमातून कमी केले जाईल व तुमच्या विषयीची सर्व माहिती ही गुप्त ठेवली जाईल आणि तुम्हास नेहमीप्रमाणे भविष्यात वैद्यकीय सेवा प्राप्त होईल.
- गुप्तता : या अभ्यास संशोधनामध्ये तुमच्या संदर्भात मिळवलेली माहिती ही गुप्त ठेवण्यात येईल. जर ही माहिती जरुरी आहे असे वाट्तल्यास, ती तुमची सहमती घेतल्यानंतर आणि लेखी परवानगी त्यासाठी प्राप्त झाल्यानंतर उघडकीस करण्यात येईल.

  तुम्ही या संशोधनामध्ये भाग घेतला आहे हे फक्त संशोधन करण्याच्या गट समूहास माहित असेल. तुमच्या विषयीची ही माहित्ती तुमच्या लेखी परवानगी शिवाय कोणासही प्राप्त होवू शकणार नाही. परंतु यास खालील अपवाद आहेत.

  - आणिबणि प्रसंगी तुमचे अधिकार आणि कल्याण करण्यासाठी आणि
  - कायद्यानुसार जरुरी असेल त्तर याचा उपयोग केला जाईल
- अभ्यासाचे निष्कर्ष प्रसिध्द करण्याचे अधिकार :

  या अभ्यासक्रमामध्ये निष्कर्ष हे एका लेखाद्वारे प्रसिध्द करण्यात येतील. या अभ्यासक्रमाचे निष्कर्ष हे प्रसिध्द करतेवेळी किंवा सभेमधे विचार विनियम करते वेळी तुमची ओळख ही उघड केली जाणार नाही. या अभ्यासक्रमची सर्व माहिती ही तुम्हास सांगितल्याप्रमाणे गुप्त राखण्यात येईल.

- सहभागी होण्याचा खर्च : या अभ्यासामध्ये भाग घेण्यासाठी तुम्हास कोणताही खर्च करावा लागणर नाही. हा अभ्यास संशोधनासाठी केल्या असल्यामुळे या अभ्यासासाठी होणरा खर्च हा संशोधन करतील.
- नुकसान भरपाई : या अभ्यासक्रमामध्ये तुम्ही भाग घेतल्यामुळे तुम्हास कोणतीही इच्छा झालीतर के.एल.ई.एस. डा. प्रभाकर कोरे हास्पीटल व संशोधन केंद्र बेळगावी हे योग्य ते उपचार तुमच्यावर करतील किंवा या संबंधी पुढे कोठे उपचार करावे हे सांगतील तथापी, तुम्हास कोणतीही नुकसान भरपाई मिळणार नाही. परंतु तुम्हास मोफत्त वैद्यकीय सेवा देण्यात येईल.
- **शंका किंवा विचारणा कोणाकडे करावी :**
  या संबंधी तुम्ही खालील व्यक्तींशी कोणत्याही वेळी संबंध प्रस्थापित करुन तुमचे शंकासमाधान करु शकता किंवा या संबंधी तुम्हास योग्य ती मदत त्याच्याद्वारे जाईल.

प्रमुख संशोधक :
एम. डी (पोस्ट ग्रेज्युएट विद्यार्थिनी)
सामान्य औषधी विभाह, जवाहरलाल नेहरु मेडीकल कालेज
नेहरु नगर, के.एल.ई. हास्पीटल रोड,

मार्गदर्षक :
एम. डी. (जनरल मेडीसीन) ऍफ. आय सी. पी
प्रोफेसर
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नेहरु नगर, के.एल.ई. हास्पीटल रोड,
बेळगावी – ५९०००१०

डा. रुपा एम. बेल्लद एम. डी. सी. एच. (बालरोग तद्न्य)
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चेअरमन
जे.एन.एम.सी. इथिक्स कमिटी, आणि मानव संशोधन केंद्र
बेळगांवी मोबा नं. 9448113403

सम्मती पत्र

मै निचे सही करनेवाला स्वइच्छेसे इस अभ्यासमें भाग लेने के लिए मान्यता देता हू । मै अपना नाम किसी भी वक्त इसमेसे वापस ले सकता हू और इस सम्मती के कारण मै मेरे कोई भी कानुनी हक्क नही छोड रहा हू । यह सब उपर के विषय के बारे में स्वयं पढकर या पढने के सूनने के बाद मैं इस सम्मती पत्र पर अपने हस्ताक्षर कर के सभी प्रश्नो का उतर दिया हू ।

हिस्सा लेने वाले का नाम : .....सही/अंगठा

साक्षीदार का नाम: .....सही/अंगठा

परीक्षण करने वाले का नाम:.....सही

तारीख: स्थळ:

ANNEXURE - III

PROFORMA

CORRELATION BETWEEN APRI INDEX, MELD SCORE AND CHILD-PUGH SCORE IN CIRRHOSIS OF LIVER – A One Year Cross Sectional Study in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

PROFORMA

CASENO:

NAME:

AGE/SEX:

IPNO.:

ADDRESS:

OCCUPATION:

COMPLAINTSATPRESENTATION:

Past history:

Family history

Personal history

Treatment history

PHYSICAL EXAMINATION:

GENERAL CONDITION:

PALLOR- YES/NO

ICTERUS- YES/NO

LYMPHADENOPATHY- YES/NO

CYANOSIS- YES/NO

CLUBBING- YES/NO

EDEMA- YES/NO

VITALS:

TEMPERATURE:

PULSE:

RESPIRATORY RATE:

BLOOD PRESSURE:

SIGNS OF HEPATOCELLULAR FAILURE:

Loss of axillary hair

Leuconychia

Dupyutren's contracture

Gynecomastia

Spider Naevi

Ascitis

Testicular atrophy

SIGNS OF HEPATIC ENCEPHALOPATHY:

Asterixis

Constructional Apraxia

SYSTEMIC EXAMINATION:

R.S.:

C.V.S.:

C.N.S.:

P.A.:

INVESTIGATIONS

CBC

LFT, including INR

RFT

Ascitic fluid analysis for glucose, proteins, cytology

USG abdomen including echotexture of liver, size of liver, splenic enlargement and portal vein diameter.

APRI score-

MELD score-

Child Pugh score-

ANNEXURE - IV

KEY TO MASTERCHART

S. No - Serial number

IP No - Inpatient Number

Gender - M=Male; F=Female

Past History- NAD= No appreciable disease;

T2DM= Type 2 Diabetes Mellitus

Habits- Alcohol / No Habits

Pallor- Yes / No

Icterus- Yes / No

Edema- Yes / No

Signs of hepatocellular Failure – Yes / No

Signs of Hepatic Encephalopathy – Yes(asterixis) / No

O/E – P/A= On examination – Per Abdomen= Fluid Thrill

Shifting Dullness

Hepatosplenomegaly

No fluid

CNS- Central Nervous System= Conscious, No Flaps

Conscious, Flaps+

Drowsy, Flaps+

Stuporous

Hemoglobin

Platelet

Total count

INR- International Normalized Ratio

Total Bilirubin

Direct Bilirubin

AST – Aspartate aminotransferase

ALT – Alanine aminotransferase

ALP- Alkaline Phosphatase

Total Protein

S. Albumin – Serum Albumin

S. Creatinine- Serum Creatinine

S. Potassium- Serum Potassium

Ascitic Fluid Count

Fluid Glucose

Fluid Protein

USG Abdomen liver echotexture- Ultrasonography Abdomen Liver Echotexture

USG Abdomen- Liver Size= Ultrasonography Abdomen Liver Size- Enlarged / Shrunken / Normal

USG Abdomen- Splenic enlargement = Ultrasonography Abdomen Splenic enlargement – Present / Absent

Portal Vein Diameter On USG – Normal / Dilated

Ascitis – Present / Absent

APRI- Aspartate Aminotransferase : Platelet Ratio Index

APRI = A-<0.7

B-0.7 TO 1.5

C->1.5

CHILD PUGH SCORE- Child Pugh class A (score of 5-6)

Child Pugh class B(score of 7-9)

Child Pugh class C(score of >/=10)

MELD SCORE – Model for End Stage Liver Disease MELD 1-<18

2-18-36

3->36

S.No	IP No.	Name	Age	Sex	Complaints At Presentation	Past History	Habits- alcohol	YEARS OF ALCOHOL CONSUMPTION	QUANTITY OF ALCOHOL(ML/DAY)	Pallor	Icterus	Edema	Signs of hepatocellular failure	Signs of hepatic encephalopathy	O/E- P/A	CNS	Hemoglobin
1	1009484	MANOJ PATIL	46	M	PAIN ABDOMEN AND ABDOMINAL DISTENSION SINCE 1 & 1/2 MONTH	NAD	ALCOHOL	15	90	NO	YES	NO	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	11.5
2	1009632	BHARAT	32	M	ALTERED SENSORIUM, JAUNDICE	NAD	ALCOHOL	15	90	YES	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	7
3	941692	KHATUNBI	60	F	ABDOMINAL DISTENSION, PEDAL EDEMA	NAD	NO HABITS			YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	7.7
4	941275	MALLIKARJUN N	45	M	FEVER, LOOSE STOOLS, JAUNDICE	NAD	ALCOHOL	15	45	NO	YES	NO	YES	YES(ASTERIXIS+)	SHIFTING DULLNESS+	DROWSY, FLAPS+	11.6
5	941669	BASAVARAJ BANGARAPPA	35	M	ABDOMINAL DISTENSION, PEDAL EDEMA	NAD	ALCOHOL	10	90	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	8.5
6	941427	UMESH CHAVAN	30	M	YELLOWISH DISCOLOURATION OF SCLERA, ABDOMINAL PAIN, FEVER	NAD	ALCOHOL	10	90	NO	YES	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	11.2
7	943368	SUBHASH NAIK	42	M	YELLOWISH DISCOLOURATION OF SCLERA, ABDOMINAL DISTENSION AND PAIN	NAD	NO HABITS			YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	10.4
8	951680	MALIYAPPA KATTI	32	M	FEVER, ABDOMINAL PAIN AND DISTENSION	NAD	ALCOHOL	15	180	YES	NO	NO	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	10.2
9	948577	SAGAR RANE	36	M	ABDOMINAL PAIN, DISTENSION AND PEDAL EDEMA	NAD	ALCOHOL	16	90	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	10.1
10	953536	SOMANING DESHANOOR	40	M	YELLOWISH DISCOLOURATION OF SCLERA	NAD	ALCOHOL	20	90	NO	YES	YES	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	13.8
11	954657	SANJAY NAIK	41	M	YELLOWISH DISCOLOURATION OF SCLERA	NAD	ALCOHOL	10	90	NO	YES	NO	NO	NO	HEPATOSPLENOMEGALY+	CONSCIOUS, NO FLAPS	11.3
12	950381	SIDDAPPA	53	M	JAUNDICE, PAIN ABDOMEN, FEVER	NAD	NO HABITS		180	NO	YES	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	11.8
13	954451	MANESH BULLAPUR	45	M	NAUSEA, PAIN ABDOMEN, FEVER	T2DM+	ALCOHOL	8	45	YES	YES	NO	NO	NO	HEPATOSPLENOMEGALY+	CONSCIOUS, NO FLAPS	8
14	957939	SHANKAR IRAGAR	49	M	PAIN ABDOMEN FEVER JAUNDICE	NAD	ALCOHOL	20	45	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	9.6
15	958455	SURESH DASAR	35	M	ALTERED SLEEP CYCLE, JAUNDICE	NAD	ALCOHOL	10	90	YES	YES	NO	YES	YES(ASTERIXIS+)	NO FLUID	CONSCIOUS, FLAPS +	7.3
16	957252	RAJASHEKHAR	55	M	JAUNDICE, PAIN ABDOMEN	NAD	ALCOHOL	30	180	NO	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	CONSCIOUS, FLAPS +	11.7
17	959140	LAXMIBAI CHAVAN	71	F	PAIN ABDOMEN, PEDAL EDEMA	T2DM+	NO HABITS			YES	NO	YES	YES	NO	NO FLUID	CONSCIUOS, NO FLAPS	7.2
18	959048	DATTATREY KULKARNI	58	M	ABDOMINAL PAIN, DISTENSION AND FEVER	NAD	NO HABITS			YES	YES	NO	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	10.6
19	960214	VITTHAL KURABAR	42	M	ABDOMINAL DISTENSION,PAIN ABDOMEN, JAUNDICE	NAD	ALCOHOL	12	45	NO	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	12.1
20	935980	BABAN SHREYAKAR	55	M	HEMATEMESIS, MELENA	NAD	ALCOHOL	20	90	NO	NO	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	11.6
21	935934	SUJATA KURBAGI	40	F	ABDOMINAL PAIN, DISTENSION AND PEDAL EDEMA	NAD	NO HABITS			YES	NO	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	10.1
22	910015	HANAMANTH MORE	40	M	ALTERED SENSORIUM, DECREASED URINE OUTPUT	NAD	ALCOHOL	25	180	YES	YES	NO	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	8.8
23	923716	ARJUN PALEKAR	53	M	ABDOMINAL DISTENSION, PEDAL EDEMA, JAUNDICE, FEVER	NAD	ALCOHOL	25	90	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	6.3
24	920757	GAJANAN GANIGER	36	M	ABDOMINAL DISTENSION, PEDAL EDEMA,DECREASED URINE OUTPUT	NAD	ALCOHOL	15	90	YES	YES	NO	YES	NO	SHIFTING DULLNESS+	CONSCIUOS, NO FLAPS	7.3
25	912724	KARABASAPPA VANAKYAL	41	M	ABDOMINAL DISTENSION, PEDAL EDEMA, FEVER	NAD	ALCOHOL	10	90	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	7.7
26	979544	RESHMA PATIL	32	F	ABDOMINAL DISTENSION, PEDAL EDEMA, BREATHLESSNESS	NAD	NO HABITS			NO	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	11.5
27	928082	CHANDRASHEKHAR	45	M	MELENA	NAD	NO HABITS			YES	NO	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	5
28	946928	LAXMAN Y	55	M	HEMATEMESIS, MELENA	NAD	NO HABITS			YES	NO	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	6.5
29	981556	DAYANAND	36	M	ABDOMINAL PAIN, PEDAL EDEMA	NAD	ALCOHOL	10	90	YES	YES	NO	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	7.9
30	979793	SANGAMESH BALIKAI	24	M	ABDOMINAL PAIN	AUTOIMMUNE HEPATITIS	NO HABITS			YES	YES	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	9.9
31	950656	SAVITA PATIL	45	F	ABDOMINAL DISTENSION, PEDAL EDEMA, DECREASED URINE OUTPUT	NAD	NO HABITS			YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	6.2
32	**985232**	**PRATAP RAJPUT**	**48**	**M**	**ABDOMINAL DISTENSION, PEDAL EDEMA JAUNDICE**	**NAD**	**ALCOHOL**	**15**	**90**	**YES**	**NO**	**YES**	**YES**	**NO**	**FLUID THRILL+**	**CONSCIOUS, NO FLAPS**	**7.9**
33	943916	SIDDAWWA MURAGOD	35	F	ALTERES SENSORIUM, ABDOMINAL DISTENSION,LOSS OF APPETITE	NAD	NO HABITS			NO	YES	YES	YES	YES(ASTERIXIS+)	SHIFTING DULLNESS+	CONSCIOUS, FLAPS +	12.8
34	986555	VINAYAK BANDAGE	37	M	ABDOMINAL PAIN, PEDAL EDEMA, LOSS OF APPETITE	NAD	ALCOHOL	10	180	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	7.1
35	936032	VEERENDRA REDDY	46	M	ABDOMINAL DISTENSION, FEVER, PEDAL EDEMA	NAD	ALCOHOL	20	180	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	8.3
36	955078	DUNDAPPA JADENNAVAR	30	M	ALTERED SENSORIUM, MELENA	NAD	ALCOHOL	10	180	YES	YES	YES	YES	YES(ASTERIXIS+)	SHIFTING DULLNESS+	DROWSY, FLAPS+	9.1
37	955745	SHEKHAR PARAPPANAVAR	57	M	PEDAL EDEMA, ABDOMINAL DISTENSION	NAD	NO HABITS			YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	9.1
38	1009943	SHANTADEVI K	87	F	BREATHLESSNESS, PEDAL EDEMA, ABDOMINAL DISTENSION	T2DM+	NO HABITS			YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIUOS, NO FLAPS	10.3
39	950045	SUDESH GURAV	28	M	ABDOMINAL DISTENSION, PEDAL EDEMA, PAIN ABDOMEN	NAD	ALCOHOL	9	90	NO	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	11.8
40	945092	MAHADEVI SOMAPUR	57	F	FEVER, ALTERED SENSORIUM	T2DM+; H/O CLD+	NO HABITS			YES	YES	YES	NO	YES(ASTERIXIS+)	NO FLUID	DROWSY, FLAPS+	10.9
41	926400	RANGANATH	52	M	ALTERED BEHAVIOUR	T2DM+	ALCOHOL	25	90	YES	NO	YES	YES	YES(ASTERIXIS+)	NO FLUID	CONSCIOUS, FLAPS +	9.3
42	940138	MALA	34	F	ABDOMINAL DISTENSION, REDUCED APPETITE	NAD	NO HABITS			YES	NO	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	6
43	942064	VIJAYKUMAR D MANTUR	38	M	ABDOMINAL DISTENSION,PEDAL EDEMA, JAUNDICE, FEVER	NAD	ALCOHOL	20	90	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	10.5
44	957019	SHAKUNTALA NAVANYALE	58	F	ALTERED BEHAVIOUR	H/O CLD+	NO HABITS			YES	YES	NO	NO	YES(ASTERIXIS+)	NO FLUID	CONSCIOUS, FLAPS +	9.7
45	943751	HANUMANTH MADIWAL	53	M	ABDOMINAL PAIN, FEVRE, LOOSE STOOLS	NAD	NO HABITS			YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	9
46	942407	MAHANTESH NALATAWAD	46	M	FEVER, JAUNDICE, ABDOMINAL PAIN	NAD	ALCOHOL	30	90	NO	YES	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	14

47	945047	BALKRISHNA TORGAL	87	M	ABDOMINAL DISTENSION, PAIN, PEDAL EDEMA, FEVER	NAD	NO HABITS			NO	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	11.5
48	924406	ARJUN S HUGAR	39	M	ABDOMINAL DISTENSION, PEDAL EDEMA	NAD	ALCOHOL	10	90	YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	5.9
49	959703	SADANAND BAILHONGAL	54	M	ABDOMINAL DISTENSION, ALTERED SENSORIUM	T2DM+	ALCOHOL	25	90	YES	NO	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	5.7
50	961119	YALLANGOUDA PATIL	46	M	ABDOMINAL DISTENSION, HEMATEMESIS	NAD	ALCOHOL	25	90	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIUOS, NO FLAPS	9.6
51	961710	MULLAM SHAIKH	64	M	PAIN ABDOMEN, ABDOMINAL DISTENSION, ALTERED SENSORIUM	NAD	ALCOHOL	30	180	YES	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	10.9
52	964420	BASAPPA	45	M	HEMATEMESIS, ALTERED SENSORIUM, ABDOMINAL DISTENSION	NAD	ALCOHOL	20	90	YES	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	CONSCIOUS, FLAPS +	9.1
53	962037	BALASAHEB SATALE	68	M	PAIN ABDOMEN, HEMATEMESIS	T2DM+	ALCOHOL	30	45	YES	YES	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	9.9
54	964883	VIJAYA	60	F	ABDOMINAL DISTENSION, MELENA	NAD	NO HABITS			YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIUOS, NO FLAPS	9.7
55	965010	HANMANTGOUDA PATIL	45	M	MELENA, JAUNDICE	NAD	ALCOHOL	20	360	YES	YES	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	9
56	964420	BASAPPA HADIMANI	45	M	PAIN ABDOMEN, ABDOMINAL DISTENSION, ALTERED SENSORIUM	HTN	ALCOHOL	10	180	YES	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	CONSCIOUS, FLAPS +	6.7
57	964039	PRADEEP MADABHAVI	27	M	JAUNDICE, ABDOMINAL DISTENSION PAIN	NAD	ALCOHOL	10	180	NO	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	CONSCIOUS, FLAPS +	13.1
58	967885	MAHESH HOSAGOUDAR	38	M	FEVER, PAIN ABDOMEN, JAUNDICE	NAD	ALCOHOL	15	90	YES	YES	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	12.5
59	968442	YAMNAPPA	61	M	ABDOMINAL DISTENSION, ALTERED SENSORIUM	T2DM+	ALCOHOL	34	90	YES	NO	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	11.2
60	968410	BASAVARAJ ATHANI	33	M	PAIN ABDOMEN, FEVER	NAD	ALCOHOL	10	90	NO	NO	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	17.5
61	969581	VIJAY MANTUR	38	M	WEAKNESS, JAUNDICE	NAD	ALCOHOL	10	180	YES	YES	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	7.9
62	971208	LAXMAN MALYAGOL	29	M	JAUNDICE	NAD	ALCOHOL	10	90	YES	YES	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	7.7
63	972048	MARUTI DESAI	54	M	FEVER, PAIN ABDOMEN, JAUNDICE	T2DM	ALCOHOL	20	45	YES	YES	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	8.2
64	973170	ASHOK HATTARKI	41	M	ALTERED SLEEP CYCLE, JAUNDICE, ABDOMINAL DISTENSION	NAD	ALCOHOL	20	90	NO	YES	NO	YES	YES(ASTERIXIS+)	SHIFTING DULLNESS+	CONSCIOUS, FLAPS +	11.5
65	974017	SARANG SAWANT	47	M	JAUNDICE, PAIN ABDOMEN	NAD	ALCOHOL	20	90	YES	YES	YES	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	11
66	975577	MARASIDDAPPA PUJAR	59	M	JAUNDICE, ABDOMINAL DISTENSION	NAD	ALCOHOL	25	90	NO	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	12.2
67	976479	FIROZKHAN MULLA	34	M	ALTERED SENSORIUM, HEMATEMESIS, PAIN ABDOMEN	NAD	ALCOHOL	15	90	YES	YES	YES	YES	YES(ASTERIXIS+)	NO FLUID	CONSCIOUS, FLAPS +	7.9
68	988069	SOMAPPA	80	M	PAIN IN RIGHT HYPOCHONDRIUM, FEVER	T2DM+	NO HABITS			YES	NO	NO	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	9.1
69	988531	PARSHURAM BHAJANTRI	45	M	JAUNDICE, ABDOMINAL DISTENSION, PEDAL EDEMA	NAD	ALCOHOL	20	90	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	7.9
70	988151	SHIDDAPPA GOLABHAVI	42	M	HEMATEMESIS, ALTERED SENSNSORIUM	NAD	ALCOHOL	20	180	YES	NO	NO	YES	YES(ASTERIXIS+)	NO FLUID	DROWSY, FLAPS+	9.3
71	988335	VIJAYA KULKARNI	65	F	ALTERED SENSORIUM, JAUNDICE	T2DM+	NO HABITS			YES	YES	NO	YES	YES(ASTERIXIS+)	NO FLUID	DROWSY, FLAPS+	10.3
72	988615	BASAVANT PARUSCHE	58	M	HEMATEMESIS	NAD	ALCOHOL	20	90	YES	NO	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	6.6
73	982991	UMESH KULKARNI	46	M	PEDAL EDEMA, ABDOMINAL DISTENSION, JAUNDICE	NAD	ALCOHOL	20	90	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	9.1
74	990600	BASAVANNI IRANATTI	55	M	ALTERED SENSORIUM, JAUNDICE	T2DM+	ALCOHOL	25	90	YES	YES	YES	YES	YES(ASTERIXIS+)	SHIFTING DULLNESS+	CONSCIOUS, FLAPS +	10.2
75	992175	APPASAHEB ZOND	68	M	JAUNDICE, ALTERED SENSORIUM. ABDOMINAL DISTENSION	T2DM_	ALCOHOL	30	90	NO	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	12.3
76	994977	RAVNDRA PATIL	44	M	HEMATEMESIS, MELENA	NAD	ALCOHOL	20	180	YES	NO	NO	YES	YES(ASTERIXIS+)	SHIFTING DULLNESS+	CONSCIOUS, FLAPS +	3.8
77	995398	PRALHAD GADKARI	45	M	BREATHLESSNESS, JAUNDICE, DECREASED URINE OUTPUT	NAD	ALCOHOL	15	90	NO	YES	YES	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	16
78	995193	RANGRAO PATIL	66	M	ABDOMINAL DISTENSION, DECREASED URINE OUTPUT, MELENA	T2DM+	NO HABITS			YES	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	CONSCIOUS, FLAPS +	7.3
79	941470	SHRIMANT SANKANNAVAR	39	M	ABDOMINAL PAIN, DISTENSION, PEDAL EDEMA	NAD	ALCOHOL	15	180	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	10.4
80	997383	VINAYAK BANDIGE	37	M	VOMITING, PEDAL EDEMA, PAIN ABDOMEN	NAD	ALCOHOL	12	90	YES	NO	YES	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	9.9
81	996236	NINGAPPA BOMANNAVAR	48	M	ABDOMINAL DISTENSION, EDEMA, PAIN ABDOMEN	NAD	ALCOHOL	20	360	YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	9.6
82	999939	UMADEVI NAVADGI	66	F	ABDOMINAL DISTENSION, JAUNDICE, PEDAL EDEMA	T2DM+	NO HABITS			YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS,NO FLAPS	10
83	1001528	SUBHASH PATIL	56	M	PEDAL EDEMA, DECREASED URINE OUTPUT	T2DM	ALCOHOL	25	45	NO	NO	YES	NO	NO	NO FLUID	CONSCIOUS, NO FLAPS	11
84	1002581	BASAPPA KUMBAR	46	M	JAUNDICE, DECREASED APPETITE, PAIN ABDOMEN	NAD	ALCOHOL	15	90	NO	YES	NO	NO	NO	NO FLUID	CONSCIOUS,NO FLAPS	12.2
85	1002866	MALIK NADAF	33	M	JAUNDICE, INCREASED DAYTIME SLEEPINESS	NAD	ALCOHOL	10	180	YES	YES	NO	YES	YES(ASTERIXIS+)	NO FLUID	CONSCIOUS, FLAPS +	10.8
86		MALLAPPA K	68	M	ABDOMINAL DISTENSION, PEDAL EDEMA	T2DM+	ALCOHOL	30	90	YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	8.8
87	1010100	ASHOK MALED	45	M	SEIZURES, ALTERED SENSORIUM, DECREASED URINE OUTPUT	NAD	ALCOHOL	20	90	YES	NO	NO	YES	NOT ASSESSED	NO FLUID	STUPOROUS	7.9
88	1010250	SHIVAPPA KUMBAR	42	M	ABDOMINAL DISTENSION, JAUNDICE, ALTERED SENSORIUM	NAD	ALCOHOL	20	90	YES	YES	YES	YES	YES(ASTERIXIS+)	FLUID THRILL+	DROWSY, FLAPS+	8.4
89	1010159	MAHANTESH MATTIKALI	48	M	JAUNDICE, DECREASED URINE OUTPUT, ALTERED SENSORIUM	NAD	ALCOHOL	20	90	NO	YES	YES	YES	YES(ASTERIXIS+)	NO FLUID	DROWSY, FLAPS+	12.3
90	1010592	HANMANTHAPPA KAMBAR	39	M	ABDOMINAL DISTENSION, PEDAL EDEMA, MELENA	NAD	ALCOHOL	15	90	YES	YES	NO	YES	N0	FLUID THRILL+	CONSCIOUS, NO FLAPS	7.8
91	1011258	UMESH KULKARNI	46	M	ABDOMINAL DISTENSION, PEDAL EDEMA	NAD	ALCOHOL	10	90	YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	10.1
92	1011129	BASANGOUDA HADIMANI	67	M	ABDOMINAL DISTENSION, SCROTAL SWELLING, PEDAL EDEMA	T2DM+	NO HABITS			YES	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	9.8
93	1011156	SHANKAR MODAGE	45	M	ALTERED BEHAVIOUR	NAD	ALCOHOL	12	180	NO	NO	NO	YES	N0	NO FLUID	CONSCIOUS, RESTLESS	13.4
94	1011288	CHANDRAYYA HIREMATH	65	M	PAIN ABDOMEN, ABDOMINAL DISTENSION,PEDAL EDEMA	NAD	ALCOHOL	25	90	YES	YES	YES	YES	NO	FLUID THRILL+	CONSCIOUS, NO FLAPS	8.9
95	988529	RATNAKAR ASNOTKAR	75	M	ABDOMINAL DISTENSION, PEDAL EDEMA	NAD	ALCOHOL	30	90	NO	NO	YES	YES	NO	FLUID THRILL+	CONSCIOUS,NO FLAPS	11.9
96	938581	VIJAY SHETKAR	44	M	ALTERED BEHAVIOUR	NAD	ALCOHOL	20	90	YES	YES	NO	YES	YES(ASTERIXIS+)	NO FLUID	CONSCIOUS, FLAPS +	10.6
97	935049	ASHWINI VAJJARAMATTI	30	F	ABDOMINAL PAIN, FEVER	NAD	NO HABITS			YES	NO	NO	YES	NO	NO FLUID	CONSCIOUS,NO FLAPS	8.4
98	933148	SHIVAKUMAR BANDI	42	M	ABDOMINAL PAIN, DISTENSION, JAUNDICE	NAD	ALCOHOL	10	90	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	11
99	924331	VIGNESHWAR PUJARI	36	M	ABDOMINAL DISTENSION, SCROTAL SWELLING, PEDAL EDEMA	NAD	ALCOHOL	15	45	YES	NO	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS,NO FLAPS	8.60
100	929115	SHAHID HAMID	41	M	ABDOMINAL PAIN, FEVER	T2DM+	NO HABITS			YES	NO	NO	YES	NO	NO FLUID	CONSCIOUS, NO FLAPS	10
101	926930	BHIMU GOLSANGI	47	M	ABDOMINAL PAIN, PEDAL EDEMA,ABDOMINAL DISTENSION	T2DM+	ALCOHOL	10	360	YES	YES	YES	YES	NO	SHIFTING DULLNESS+	CONSCIOUS, NO FLAPS	7.3

Platelet	Total count	INR	total bilirubin	direct bilirubin	AST or SGOT	ALT or SGPT	ALP	Total Protein	S. Albumin	S. Creatinine	S. sodium	S. potassium	Ascitic fluid cell count	fluid glucose	fluid protein	USG Abdomen liver echotexture	USG abdomen- liver size	USG abdomen- splenic enlargement	Portal vein diameter on USG	Ascitis	APRI	CHILD PUGH SCORE	MELD SCORE		
329000	17100	1.34	2	0.59	16	10	163	6.7	2.5	0.69	129	4.56	151	137	4	INCREASED	ENLARGED	PRESENT	NORMAL	PRESENT	0.1	10	19	AGE(AN2:AN103)"	
96000	18700	2.78	21.61	19.94	32	50	118	4.2	2.1	2.62	126	3.77	772	122	0.6	COARSE ECHOTEXTURE	ENLARGED	PRESENT	1.5CMS(DILATED)	PRESENT	0.8	14	39		
204000	8600	1.48	0.5	0.2	73	15	234	6.8	3	1.5	142	2				COARSE ECHOTEXTURE	SHRUNKEN	ABSENT	NORMAL	PRESENT	0.9	10	20		
26000	10700	3.19	15.86	9.27	49	30	164	5.6	2.5	1.29	137	4.7				COARSE ECHOTEXTURE	NORMAL	ABSENT	1.3CM(DILATED)	PRESENT	4.7	13	36		
68000	3920	1.65	3.2	0.8	55	15	482	6.7	2.3	0.7	138	3.5				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	2	10	16		
67000	11740	2.74	17	9.4	396	106	206	7.1	3.9	4.2	131	2.7				INCREASED	ENLARGED	ABSENT	NORMAL	ABSENT	14.8	9	40		
148000	5130	1.27	4	2	114	42	835	6.7	2.7	1	137	3.5				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.3CM(DILATED)	PRESENT	1.9	10	14		
307000	6500	1.32	0.82	0.37	57	13	120	6.5	2.6	1.08	130	4.74				COARSE ECHOTEXTURE WITH CAUDATE LOBE HYPERTROPHY	NORMAL	ABSENT	NORMAL	PRESENT	5	9	10		
62000	12300	3.2	2.8	0.6	102	110			2.8	0.56	130	3.51	300	84	4.6	INCREASED WITH SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	PRESENT	4.1	11	27		
206000	28100	2.51	22.86	18.55	290	80	94	6.8	2.7	0.76	130	4.38				INCREASED WITH MINIMAL SURFACE IRREGULARITY	ENLARGED	PRESENT	NORMAL	ABSENT	3.5	11	36		
200000	8100	1.55	21.95	16.58	170	57	97	6.7	3.4	0.55	138	4.32				MILDLY COARSE ECHOTEXTURE	ENLARGED	PRESENT	NORMAL	ABSENT	2.1	8	23		
129000	8100	1.35	4.68	3.06	51	24	141	10.6	3.1	1.35	110	6.29				MILDLY COARSE ECHOTEXTURE WITH NODULAR MARGINS	NORMAL	ABSENT	NORMAL	ABSENT	1	9	29		
78000	12000	3.28	10.2	8.3	500	488	140	7.3	2.5	1.1	124	3.5				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.1CM(DILATED)	ABSENT	18	11	33		
95000	8200	2.95	8.65	6.44	147	38	130	6.9	2.3	0.89	142	3.6				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.4CMS(DILATED)	PRESENT	3.9	12	19		
91000	16100	1.61	2.48	1.85	102	44	124	5.8	2.4	1.19	140	3.89				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	NORMAL	ABSENT	2.8	9	17		
76000	7200	1.99	5.04	4	165	80	234	5.4	2.8	1.2	132	3.84				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	5.4	12	25		
71000	3200	1.05	0.48	0.21	48	16	71	6.8	2.4	6	134	2.84				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	ABSENT	1.7	7	22		
112000	16600	2.1	25.3	19.7	43	41	112	5.4	1.8	2.1	132	4.6				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.2CM(DILATED)	PRESENT	1	11	35		
362000	23700	2.68	29.42	22.03	150	29	130	6.6	2.9	1.85	137	4.2	40		0.7	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	NORMAL	PRESENT	1	12	36		
110000	6200	1.2	0.4	1.6	11	10	83	6.8	4.2	1.09	132	4.12				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	PRESENT	1.4CMS(DILATED)	ABSENT	0.2	5	9		
201000	6700	1.21	0.68	0.29	14	54	124	5.6	3.2	0.54	139	3.46	344		1.9	INCREASED	SHRUNKEN	PRESENT	1.4CMS(DILATED)	PRESENT	0.2	7	9		
96000	7500	1.63	4.74	4.57	51	25	192	7.5	2.5	4.8	130	6.1				COARSE ECHOTEXTURE	NORMAL	PRESENT	1.1CM(DILATED)	PRESENT	1.3	12	33		
85000	15300	2.3	10.85	8.73	33	18	123	6.1	2.6	1.67	128	4.21	840		1.6	INCREASED	NORMAL	PRESENT	NORMAL	PRESENT	1	13	33		
101000	6200	2.09	2.78	1.83	51	37	146	6.5	2.5	1.04	135	4.38	18		0.2	COARSE ECHOTEXTURE	SHRUNKEN	PRESENT	NORMAL	PRESENT	1.3	10	20		
159000	9800	7.72	6.27	2.43	62	33	113	4.9	1.4	1.19	124	4.95	5703		0.7	COARSE ECHOTECTURE	SHRUNKEN	PRESENT	NORMAL	PRESENT	2.6	13	32	HbSAg+	
88000	4600	6.61	1.66	1.76	55	25	148	6.3	3	0.51	140	3.8				COARSE ECHOTEXTURE	NORMAL	PRESENT	1.2CM(DILATED)	PRESENT	1.6	11	20	TB	
100000	5800	1.26	0.89	1.61	31	27	56	4.9	2.8	1.04	133	3.67				MILDLY ALTERED ECHOTEXTURE	NORMAL	PRESENT	NORMAL	ABSENT	0.8	7	17		
114000	5200	1.52	0.2	0.08	23	147	112	4.9	3.1	0.68	143	5.01				COARSE ECHOTEXTURE	SHRUNKEN	PRESENT	1.3CM(DILATED)	ABSENT	0.5	6	11		
264000	1900	17.93	12.54	2.02	52	20	134	5.3	3.4	0.46	134	4.28				INCREASED ECHOTEXTURE	ENLARGED	PRESENT	1.3CM(DILATED)	PRESENT	0.5	10	26		
57000	1800	3.69	2.79	2.26	177	138	239	6.3	3.7	0.42	133	3.67				COARSE ECHOTEXTURE	NORMAL	PRESENT	1.1CM(DILATED)	ABSENT	7.8	9	23		
49000	2100	1.53	1.68	0.6	20	11	77	6.3	2.9	1.04	141	3.88	100		1.2	INCREASED ECHOTEXTURE	SHRUNKEN	PRESENT	1.1CM(DILATED)	PRESENT	1	8	14		
150000	**6000**	**1.21**	**1.28**	**0.79**	**67**	**33**	**128**	**5.6**	**2.7**	**1.45**	**135**	**3.72**				**INCREASED ECHOTEXTURE**	**SHRUNKEN**	**PRESENT**	**1.3CM(DILATED)**	**PRESENT**	**1.1**	**9**	**15**	**HbSAg+**	
50000	4200	1.16	1.18	0.46	26	25	70	5.1	2.8	0.71	135	3.68	100		0.5	INCREASE ECHOTEXTURE	NORMAL	PRESENT	1.4CMS(DILATED)	PRESENT	1.3	8	9		
90000	3300	1.97	1.82	1.31	171	49	256	8	2.1	0.71	127	3.18				INCREASED ECHOTEXTURE	NORMAL	PRESENT	1.1CM(DILATED)	PRESENT	4.8	9	24		
260000	11700	2.27	4.93	3.7	190	32	21	7.8	2.2	3.5	119	7.41	2030		3.7	COARSE ECHOTEXTURE	SHRUNKEN	PRESENT	1.2CM(DILATED)	PRESENT	0.3	12	36		
141000	9100	4.07	12.04	7.76	206	81	110	5.4	2.3	0.8	136	3.95				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	3.7	13	32		
69000	5100	1.55	3.46	1.66	61	38	74	6.4	2.5	1.01	134	4.01	170		1.2	COARSE ECHOTEXTURE	SHRUNKEN	PRESENT	1.3CM(DILATED)	PRESENT	2.2	11	18		
112000	6200	1.29	1.03	0.58	26	15	162	5.4	2.5	0.88	136	3.6				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	0.6	9	9		
74000	7400	1.76	1.93	1.19	24	13	119	7.4	3.6	0.78	135	3.87	400		2.9	COARSE ECHOTEXTURE	SHRUNKEN	PRESENT	1.5CMS(DILATED)	PRESENT	0.8	8	17		
66000	5500	1.56	5.69	3.6	47	21	189	6.7	3.1	0.56	135	4.29				COARSE ECHOTEXTURE	SHRUNKEN	PRESENT	1.5CMS(DILATED)	ABSENT	1.8	9	19		
101000	5500	2.23	9.4	6.27	31	19	149	6.7	3.2	0.8	132	4.15				COARSE ECHOTEXTURE	NORMAL	PRESENT	NORMAL	ABSENT	0.8	11	27		
23000	9200	1.3	0.67	0.28	30	24	81	6.8	3.6	0.56	136	3.79				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	PRESENT	1.4CMS(DILATED)	PRESENT	3.3	6	9		
140000	9600	2.03	2.32	1.42	47	41	186	6	2.7	0.74	142	4.74	1503		1	COARSE ECHOTEXTURE	NORMAL	PRESENT	1.2CM(DILATED)	PRESENT	0.8	11	18		
41000	2700	1.46	1.64	0.9	44	19	140	6.3	3.2	1.35	132	3.45				INCREASED ECHOTEXTURE	NORMAL	PRESENT	1.5CMS(DILATED)	ABSENT	2.7	7	19		
316000	14900	1.93	5.25	4.42	85	34	214	6.5	2.4	0.61	129	5.36	172		1.7	COARSE ECHOTEXTURE	NORMAL	PRESENT	1.3CM(DILATED)	PRESENT	0.7	12	25	HbSAg+	
102000	12300	1.7	4.04	1.52	78	32	198	6.5	2.9	1.35	132	4.85				INCREASED ECHOTEXTURE	NORMAL	PRESENT	1.2CM(DILATED)	ABSENT	1.9	9	24		

51000	3800	1.56	1.65	0.6	21	15	61	5.4	2.8	1.18	139	3.89	1264		1.1	INCREASED ECHOTEXTURE	NORMAL	PRESENT	1.2CM(DILATED)	PRESENT	1	8	15		
45000	12700	1.33	0.65	0.53	19	14	308	6.6	3.1	0.81	135	4.35	100		0.7	INCREASED ECHOTEXTURE	NORMAL	PRESENT	1.3CM(DILATED)	PRESENT	1.1	8	10		
180000	8300	1.65	0.87	0.47	38	25	109	6.4	2.4	0.92	132	1.85	40		0.6	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	1.4CMS(DILATED)	PRESENT	0.5	10	17		
30000	3500	2.35	5.36	4	319	71	159	7.2	1.2	0.73	140	4.68	125	11	0.6	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.3CM(DILATED)	PRESENT	26.6	13	22		
80000	10700	4.47	19.74	15.3	265	129	525	6.4	2.7	1.1	105	6.04				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	ABSENT	NORMAL	PRESENT	9.2	14	37		
65000	14100	3	6.29	3.79	53	35	128	4.7	1.7	2.74	134	5.54	71		0.8	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	2	14	35		
98000	4100	2	1.82	1.63	137	39	143	6.1	2.6	1.53	141	3.25				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.1CM(DILATED)	ABSENT	3.5	8	21		
109000	10300	1.38	4.51	2.91	29	12	105	6.7	2.2	1.74	136	2.52	110		0.7	COARSE ECHOTEXTURE WITH MILD SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	PRESENT	0.7	11	22		
68000	11800	1.21	4.67	3.39	105	31		6.1	3.3	0.44	134	3.93				COARSE ECHOTEXTURE WITH MILD SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	ABSENT	3.9	8	17		
35000	9300	3.24	8.87	6.21	47	27	77	7.7	2.3	2.49	135	5.7				COARSE ECHOTEXTURE WITH MILD SURFACE IRREGULARITY	NORMAL	PRESENT	1.1CM(DILATED)	ABSENT	3.4	14	36		
89000	22600	2.31	32.56	28.27	193	183	225	5.3	2.4	0.65	131	3.98	170			COARSE ECHOTEXTURE WITH MILD SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	5.7	14	31		
23000	3500	2.87	1.62	0.89	86	36	202	5.8	2.8	0.97	127	4.59				MILDLY ALTERED ECHOTEXTURE	NORMAL	PRESENT	NORMAL	ABSENT	9.3	8	27		
127000	4600	1.43	1.92	0.83	30	12	89	7.6	3.2	0.84	137	3.71	43		4.2	COARSE ECHOTEXTURE	ENLARGED	PRESENT	1.1CM(DILATED)	PRESENT	0.6	9	13		
213000	11500	1.16	0.96	0.34	87	57	88	7.5	4.4	0.92	133	4.51				INCREASED ECHOTEXTURE	ENLARGED	PRESENT	NORMAL	ABSENT	1	5	8		
141000	10200	1.82	1.73	1.35	39	23	101	4.3	2.1	2.87	135	5.21				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	ABSENT	0.7	8	26		
134000	4600	1.04	11.3	10.03	604	64	298	6.1	2.8	0.59	130	2.43				INCREASED ECHOTEXTURE	ENLARGED	ABSENT	NORMAL	ABSENT	11.3	8	22		
135000	6000	2	2.14	1.8	48	14	161	5.1	2.7	1.79	132	3.4				INCREASED ECHOTEXTURE	ENLARGED	PRESENT	NORMAL	ABSENT	0.9	9	26		
180000	19900	2.46	26.26	20.03	168	47	187	4.2	2.3	5.75	128	4.12				MILDLY COARSE ECHOTEXTURE WITH NODULARITY	NORMAL	PRESENT	NORMAL	PRESENT	2.3	14	40		
56000	22800	2.8	10.52	5.69	128	77	143	7.7	3	1.56	104	5.08				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	1.6CM(DILATED)	ABSENT	5.7	10	35		
374000	8200	1.43	5.28	4.69	228	115	163	6.7	3.7	3.14	115	3.93	54		2.2	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1CM	PRESENT	1.5	9	33		
471000	15100	2.02	13.72	11.83	133	56	119	6.1	2.6	0.65	127	4.23				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	NORMAL	PRESENT	0.7	12	29		
546000	11100	1.23	0.6	0.37	49	49	216	5.5	2.8	2.71	130	5.78				MULTIPLE NODULES WITH INCREASED ECHOTEXTURE	ENLARGED	ABSENT	NORMAL	ABSENT	0.2	6	23	HCC	
54000	9600	8.82	10.63	7.67	43	17	136	6.4	2.4	2.64	134	4.69				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.1CM(DILATED)	PRESENT	2	13	40		
113000	8800	1.19	1.32	0.88	13	11	377	5.2	2.5	0.89	120	4.35				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	ABSENT	0.3	8	9		
52000	14500	1.57	3.5	1.42	53	22	56	6.2	2.6	0.78	137	3.98				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	PRESENT	NORMAL	ABSENT	2.5	10	16		
261000	3300	1.16	1.4	0.69	29	19	98	5.9	3.2	1.39	138	3.94				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	ABSENT	0.3	6	13		
140000	9700	2.39	27.06	19.63	118	35	78	5.8	2.6	0.53	125	4.4	30		2.5	INCREASED ECHOTEXTURE	SHRUNKEN	PRESENT	1.4CMS(DILATED)	PRESENT	2.1	13	33		
38000	7300	1.29	4.92	4.07	230	103	171	5.4	2.1	0.71	140	3.59				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	NORMAL	PRESENT	15.1	12	15		
66000	20000	1.46	7.85	5.08	130	53	123	7	2.7	3.03	134	3.89				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1CM	PRESENT	4.9	12	30		
70000	12000	4.37	1.32	0.94	45	17	56	4.2	1.8	0.93	138	5.26				COARSE ECHOTEXTURE WITH SURFACE NODULARITY	NORMAL	PRESENT	1.4CMS(DILATED)	PRESENT	1.6	11	24		
196000	16400	1.19	3.3	1.94	246	50	102	6.7	4.5	1.71	129	6.58				INCREASED ECHOTEXTURE	NORMAL	ABSENT	NORMAL	PRESENT	3.1	8	24		
47000	6400	1.7	12.46	10.76	180	34	100	6	3.6	1.32	128	4.32				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	9.6	10	29		
147000	12600	1.75	29.4	28.18	124	25	129	5.9	3	2.65	129	3.85				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	PRESENT	NORMAL	PRESENT	2.1	11	36		
119000	3200	1.71	1.67	1.1	19	46	126	8.8	3.5	0.79	136	5.11				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NOT VISUALISED	ABSENT	1.9	7	15		
111000	3200	1.67	0.59	0.42	25	12	98	8.8	2.3	0.69	130	3.18	69			COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.1CM(DILATED)	PRESENT	0.6	9	18		
114000	7200	3.09	5.15	3.34	75	29	112	6.2	2.3	0.85	134	3.71		135	1.2	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	ABSENT	1.2CM(DILATED)	PRESENT	1.6	13	26		
522000	19200	1.57	1.63	0.74	23	36	177	7.1	2.9	2.69	128	5.36				INCREASED ECHOTEXTURE	NORMAL	ABSENT	NORMAL	ABSENT	0.1	6	28		
21000	14400	0.87	15.06	13.59	162	110	316	4.5	2.6	1.06	142	3.81				INCREASED ECHOTEXTURE	ENLARGED	PRESENT	1.2CM(DILATED)	ABSENT	19.3	9	17		
172000	10800	3.69	39.35	31.44	577	143	175	5.6	2.8	4.8	136	6.29				INCREASED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.0CM	ABSENT	8.4	12	40		
92000	6200	1.38	1.83	1.36	36	64	80	5.9	2.6	1.5	135	3.91				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.3CM(DILATED)	PRESENT	1	9	19		
118000	26500	2.14	1.71	1.22	58	15	223	6.9	2.4	3.53	128	4.94				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.3CM(DILATED)	ABSENT	1.2	10	32		
127000	11100	2.36	33.35	26.53	136	49	75	5.6	3.4	1.82	138	3.82	70		1.6	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.2CM(DILATED)	PRESENT	2.7	13	35		
379000	19100	1.68	40.39	33.34	157	73	251	4.3	2.6	2.2	130	3.63				ALTERED ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	1.3CM(DILATED)	PRESENT	1	11	35		
95000	7000	2.29	2.68	1.52	51	9	87	6.4	2.6	0.62	134	4.3				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.2CM(DILATED)	PRESENT	1.3	12	21		
111000	5900	1.45	1.75	1.16	51	24	105	6.4	3.1	0.9	133	4.71	46		1.4	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	PRESENT	1.1CM(DILATED)	PRESENT	1.1	8	17		
106000	7100	1.35	0.96	0.42	26	16	125	6.3	2.7	1.59	144	4.71	79	242	1.4	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	PRESENT	0.6	9	10	HCC	
276000	10600	0.95	1.17	0.43	27	31	98	6.7	1.9	3.72	142	3.72				INCREASED ECHOTEXTURE	NORMAL	ABSENT	NORMAL	ABSENT	0.2	7	20		
100000	7000	1.4	8.27	7.08	46	13	191	5	2.6	3.02	134	4.68	90	47	0.8	COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	ENLARGED	PRESENT	NORMAL	PRESENT	1.1	11	30		
91000	6600	1.31	1.36	0.58	30	17	84	6.8	2.3	0.96	139	4.08				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	ABSENT	NORMAL	PRESENT	0.8	9	11		
123000	6500	1.26	5.39	2.46	40	21	139	6.6	3.5	2.07	127	4.85				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.3CM(DILATED)	PRESENT	0.8	10	28		
57000	6300	1.41	0.51	0.1	13	11	78	7.6	4.1	0.51	135	4.93				INCREASED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	PRESENT	0.6	5	10		
56000	4000	2.41	5.64	3.37	60	33	190	7.6	2.3	0.9	137	3.9				INCREASED ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	NORMAL	ABSENT	2.7	12	23		
72000	3700	2.25	4.7	2.28	74	28	105	3.3	6.6	0.71	136	4.33				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	NORMAL	PRESENT	1.2CM(DILATED)	PRESENT	2.6	11	22		
101000	3800	1.39	1.21	0.13	78	45		8.3	3.6	0.52	140	4.28				COARSE ECHOTEXTURE WITH SURFACE IRREGULARITY	SHRUNKEN	PRESENT	1.3CM(DILATED)	ABSENT	1.9	5	11		
80000	4300	1.47	3.5	3.26	29	18	75	6.7	3.8	3.94	136	6.75				INCREASED ECHOTEXTURE	SHRUNKEN	PRESENT	1.2CM(DILATED)	PRESENT	0.8	8	29		