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SYNOPSIS FOR DISSERTATION



"LYMPHOCYTE/MONOCYTE RATIO COMPARED WITH MELD-SODIUM AND CHILD PUGH SCORING SYSTEMS IN LIVER CIRRHOSIS PATIENTS"



BY

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"LYMPHOCYTE/MONOCYTE RATIO COMPARED WITH MELD-SODIUM AND CHILD PUGH SCORING SYSTEMS IN LIVER CIRRHOSIS PATIENTS"
"ABSTRACT" 'Introduction': Cirrhosis is a common liver disease causing abnormal tissue replacement, leading to advanced hepatic injury and potential end-stage liver failure or cancer. Hepatologists struggle to develop reliable prognostic scores for patients with liver cirrhosis. This study aims to assess the 'lymphocyte-to-monocyte ratio (LMR)' in comparison to 'MELD' and 'Child- Pugh scores'. 'Materials and Methods':

The present 'prospective observational study' was conducted on 136 patients suffering from liver cirrhosis in 'Sri devraj URS academy of higher education and research' for a period of 24 months from June 2022 to September 2024. Prior to the initiation of the study, Ethical and Research Committee clearance was obtained from Institutional Ethical Committee. 'Results and observations': The study involved 136 male subjects aged 41-50 years, most with alcoholism. The majority had hepatitis virus and steatosis, diagnosed within 0-5 years. The subjects had symptoms like

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined unanimously approved the synopsis entitled "Lymphocyte/Monocyte ratio compared with meld sodium and child pugh scoring system in liver cirrhosis patients" being investigated by Dr.S.A.Gagan Dr.B.N.Ragavendra Prasad in the Department of General Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. Permission is granted by the Ethics Committee to start the study.

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Place: Kolar DR. S A GAGAN

LYMPHOCYTE/MONOCYTE RATIO COMPARED WITH MELD-SODIUM AND CHILD PUGH SCORING SYSTEMS IN LIVER CIRRHOSIS PATIENTS

ABSTRACT

Introduction: Cirrhosis is a common liver disease causing abnormal tissue replacement, leading to advanced hepatic injury and potential end-stage liver failure or cancer. Hepatologists struggle to develop reliable prognostic scores for patients with liver cirrhosis. This study aims to assess the 'lymphocyte-to-monocyte ratio (LMR)' in comparison to 'MELD' and 'Child-Pugh scores'.

Materials and Methods: The present 'prospective observational study' was conducted on 136 patients suffering from liver cirrhosis in 'Sri devraj URS academy of higher education and research' for a period of 24 months from June 2022 to September 2024. Prior to the initiation of the study, Ethical and Research Committee clearance was obtained from Institutional Ethical Committee.

Results and observations: The study involved 136 male subjects aged 41-50 years, most with alcoholism. The majority had hepatitis virus and steatosis, diagnosed within 0-5 years. The subjects had symptoms like fatigue, anorexia, jaundice, insomnia, leg edema, and more. Most belonged to Group C, with the highest lymphocyte to monocyte ratio in Group A.

Conclusion: The study found that 'lymphocyte/monocyte ratios (LMR)' could be a useful bio-marker for measuring liver cirrhosis severity, potentially complementing or surpassing established grading systems like 'MELD-Na' and 'Child-Pugh'. However, further research is needed to validate these findings and investigate the clinical consequences of introducing 'LMR' into standard diagnostic processes.

'Key words': 'Cirrhosis, fatigue, LMR, MELD, Child Pugh score.'

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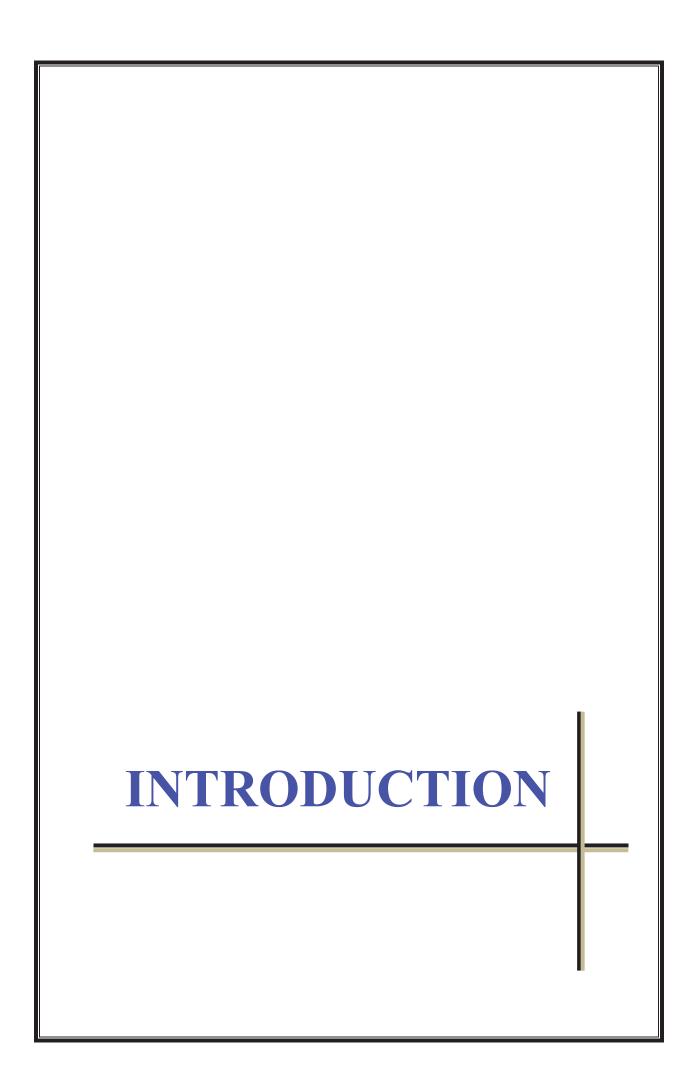
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ABBREVIATIONS

CLD	Chronic Liver Disease
NASH	Nonalcoholic steatohepatitis
CPS	Child-Pugh score
MELD	Model for End-Stage Liver Disease
TIPS	Transjugular Intrahepatic Portosystemic Shunts
INR	International Normalized Ratio
PT	Prothrombin Time
LMR	Lymphocyte to Monocyte Ratio
СР	Child-Pugh
MELD-Na	Model for End-Stage Liver Disease Sodium
HCV	Hepatic C Virus
CTL	Cytotoxic T Lymphocytes
HBV	Hepatic B Virus
APACHE	Acute Physiology and Chronic Health
	Evaluation System
UNOS	United Network for Organ Sharing
PELD	Pediatric End-stage Liver Disease
HCC	Hepatocellular Carcinoma
CLIF-SOFA	Chronic Liver Failure-SOFA
TLC	Total Leukocyte Count



INTRODUCTION

Cirrhosis is a common liver disease that impairs liver function due to the replacement of normal hepatic tissue with abnormal structures. It is an progresse stage of liver injury due to chronic liver diseases (CLD) like alcoholic liver disease and hepatitis, and may evolve into end-stage liver failure or hepatic carcinoma. Cirrhosis mortality rates dropped in the United States in the early 1920s due to the national Prohibition act, but increased again in the mid-70s due to alcohol abuse awareness and participation in Alcoholics Anonymous meetings. Similarities in trends were seen in countries of europe, except United Kingdom and few Nordic countries. Scotland's high cirrhosis associated rates of mortality, attributed to doubled alcoholism over the past forty years, remains alarming. Without effective treatment, hepatic cirrhosis might become a worldwide health issue, resulting in an estimated 800,000 deaths annually.

In 2017, nearly four million Americans had liver cirrhosis, that is 1.8% of their adult population. Cirrhosis is the 11th commonest cause of death worldwide, causing approximately 20 lakh deaths every year. In 2017, it killed "41,473 people (12.8 per 100,000)". Cirrhosis has many etiologies, with alcohol, NASH, and viruses being most common. It has a prevalence of 4.5% to 9%.

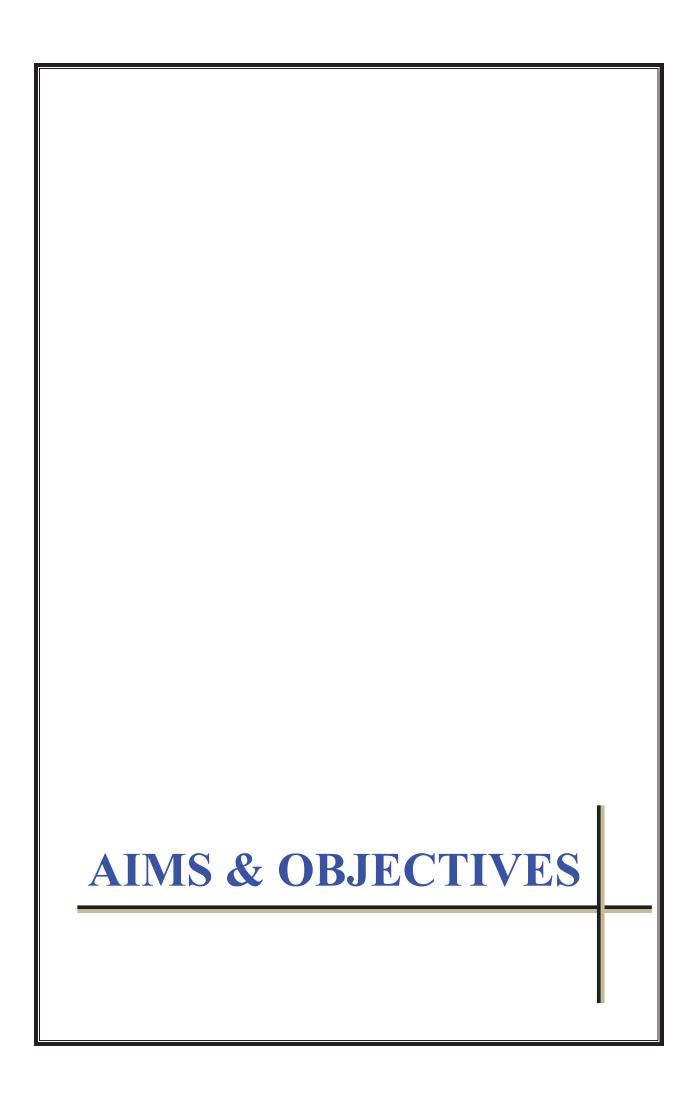
"The Child-Pugh score (CPS)" is the gold standard in determination of prognosis in cirrhosis for almost 3 decades. It was previously used for predicting mortality associated with surgery and to determine requirement of transplantation of liver.

"The Model for End-Stage Liver Disease (MELD) score" is a scoring system used for cirrhosis. It was initially used to predict mortality in patients who underwent "transjugular intrahepatic portosystemic shunts (TIPS)" procedures & to determine prognosis and prioritize liver transplants. "MELD score" is a good score for predicting mortality of patients having

"end-stage liver disease" and shifts the policy of organ allocation to prioritize the sickest patient. The score includes 3 objective variables: sr. bilirubin, "international normalized ratio" for "prothrombin time", and sr. creatinine. A study in europe found that "MELD score" is a very good predictor of medium and short-term survival, and th score is inversely proportional to residual liver function. However, the score has some drawbacks, like inaccurate prediction of survival in 15%-20% cases.

Oxidative stress, a balance between prooxidants and antioxidants, is a significant factor in the chronic liver disease progression, leading to sudden deterioration death in cirrhosis patients. immune deficiency and Inflammation account for 30% of mortality in patients of cirrhosis. The Monocytes are crucial in liver fibrosis pathogenesis, with B lymphocyte dysfunction in memory cells and reduced helper and cytotoxic T-lymphocytes. Research has focused on hematological markers of inflammation, such as the lymphocyte to monocyte ratio (LMR), to predict liver cirrhosis outcomes.

The purpose of this study was to evaluate the lymphocyte-to-monocyte ratio (LMR) in patients with liver cirrhosis by comparing it to the "Child-Pugh (CP)" and "Model for End-Stage Liver Disease (MELD) scores".



"AIMS AND OBJECTIVES"

"AIM":

current study aims "to compare the lymphocyte/monocyte ratio with the Model for End-Stage Liver Disease Sodium (MELD-Na) and Child-Pugh (CP) scores in liver cirrhosis patients."

OBJECTIVES:

The following were the objectives of present study:

- To calculate "the lymphocyte-to-monocyte ratio (LMR)"
- To estimate "Model for End-Stage Liver Disease Sodium (MELD-Na) score."
- To estimate "Child-Pugh (CP) score"
- To compare calculated values of the LMR, MELD-Na, CP score.



REVIEW OF LITERATURE

Liver, or Hepar in Greek, is the biggest parenchymal organ. It weighs between 1.2 and 1.5 kg at rest, and it produces nearly 25% of the heart's entire output, demonstrating the liver's vital function in metabolism and its requirement for maintaining. The liver is responsible for performing numerous metabolic and detoxification processes.

The liver plays a role in controlling blood sugar levels since it is the primary location of glycogenolysis and gluconeogenesis. The creation of triglycerides and cholesterol as well as the disintegration of lipids and proteins are examples of other metabolic processes.

Primary function of liver related to detoxification is bio-transforming lipophilic compounds (food additives, medications, steroid hormones, etc.) to improve their water solubility and potential excretion.² Every day, the liver produces roughly 500 millilitres of bile, which will either flow straight into the duodenum or will briefly be held in the gallbladder. The liver depends on three mass transport systems, liver cells, and the metabolic workload to remove poisons and medicines from the blood. The biliary system, vascular system and lymphatic system are the three mass transport systems.³

LIVER FUNCTIONS:

- Metabolic funtion fat, proteins & carbohydrates
- Secretory function bile, Bile salts, pigments & acids
- Excretory function drugs, bilirubin, toxic substances
- Synthetic function coagulative factors and albumin
- Storage function—carbohydrates, vitamins etc.
- Detoxifying function ammonia, toxins, etc.

"STAGES OF LIVER DISEASE":

- inflammation
- fibrosis
- cirrhosis
- hepatocellular carcinoma/End-stage liver disease

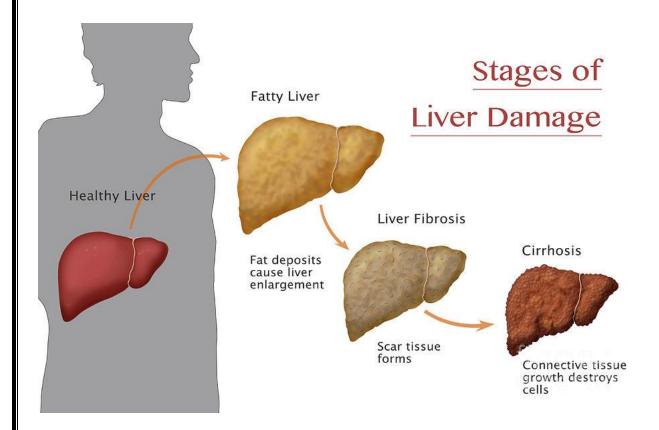


Figure 1: Stages of Liver damage

FATTY LIVER:

Classified as:

Grade	finding	steatosis
Grade-1	normal echogenicity	absent
Grade-2	echogenicity is slightly increased compared to the renal cortex	mild
Grade-3	echogenicity of portal vessels is impaired	moderate
Grade-4	visualization of posterior parenchyma is impaired	severe

Based on the involvement of parenchyma:

Grade	Involvement of parenchyma
Grade-0	less than 5%
Grade-1	5% to 33%
Grade-2	> 33%–66%
Grade-3	> 66%

PORTAL INFLAMMATION:

It is graded as:

- i. 0 (none)
- ii. 1 (mild), and
- iii. 2 (moderate till severe.)

FIBROSIS:

Fibrosis, recognized by using the Massons-trichrome stain, is caused by the inducement of the stellate-cells in the Space of Disse, causing scar formation, collagen matrix destruction, and fibrogenesis through immune mediators.

Classified as

Grades	findings
Grade 0	none
Grade 1	Peri-sinusoidal fibrosis
Grade 2	Peri-sinusoidal and portal/peri-portal fibrosis
Grade 3	bridging fibrosis
Grade 4	cirrhosis

CIRRHOSIS:

The liver's remarkable ability to rebuild lost tissue following injury makes it an organ that can tolerate some level of harm. On the other hand, in cases of cirrhosis, recurrent injuries or persistent liver disease cause diffuse damage to the liver cells throughout the organ, resulting in inflammatory alterations that lead to necrosis of the cells. Fibrogenesis is triggered in response to the destructive process, aiding in the healing of the wound. Furthermore, the remaining cells eventually undergo hyperplasia, which results in the development of hepatocellular nodules. Hepatic impairment results from progressive liver scarring and deformation of the liver's architecture as harm to the liver cells progresses. With time, cirrhosis usually worsens and can possibly become fatal. There has been multiple attempts to define cirrhosis, but none of them fully captures the disease's characteristics. 5.6

"Cirrhosis is a diffuse process characterized by fibrosis and the conversion of normal architecture into structurally abnormal (regenerative) nodules."

Regardless of the etiology, connective tissue septa and regenerating nodules are two distinct features that together explain the primary pathophysiologic chronic condition of cirrhosis. It is usually assumed that parenchymal necrosis accounts for a significant portion of the fibrosis synthesis, even though this condition is not included in the morphological definition. Therefore, necrosis is a necessary characteristic since it includes not only the early death of cells but also the responses of the surrounding environment to the dead cells and their absence. Thus, the term "cirrhosis" denotes a change in the hepatic circulation, which is commonly understood to be irreversible. On the other hand, a new study suggests that antifibrotic medications combined with effective therapy of the underlying liver disease can result in cirrhosis regression or even reversal.

Etiology:

Widespread fibrosis and nodule development are characteristics of cirrhosis. Hepatic fibrosis without nodules is known as congenital fibrosis. There are nodules without fibrosis in a partial nodular transformation. A number of factors can contribute to the dynamic and complex development of cirrhosis, including poor nutrition, exposure to chemicals, low oxygen levels, bacterial and viral infections, and disruptions in metabolism. Fatty liver disease, chronic hepatitis and chronic biliary disease are the most prevalent conditions that cause cirrhosis.⁹

In Western countries, alcoholism and HCV are the commonest causes of cirrhosis, while the hepatitis B-induced cirrhosis is becoming more and more common in underdeveloped nations. Since identification of the non-alcoholic steato-hepatitis and hepatitis C virus, cystogenic cirrhosis—cirrhosis for which the aetiology is unknown—occurs very infrequently.⁴ Co-factors, such as age, sex, alcoholism, obesity, and genetics, frequently interact with the primary cause. For example, drinking alcohol may significantly raise the risk that hepatitis B or C patients' condition may advance.¹⁰

The various causes of Cirrhosis are Alcohol, NASH, hepatitis (viral i.e G, D, C, B and A), Metabolism associated overload of iron i.e HFE haemochromatosis, overload of copper i.e "Wilson's disease", "Alpha 1 - antitrypsin deficiency", "Type 4 storage diseases of glycogen", "Galactosaemia, Tyrosinaemia, Primary biliary cirrhosis, Primary sclerosing cholangitis", outflow block of hepatic vein i.e "Budd – Chiari syndrome, Heart failure, Autoimmune hepatitis, drugs e.g. methotrexate and amiodarone, and toxins".

Most of the diseases of liver commonly have a major factor of initiation along with multiple co-factors which contribute to cirrhosis development.

"TYPES OF CIRRHOSIS":

• Alcoholic / Laennecs, Biliary, Cardiac and Post necrotic cirrhosis.

Alcoholism associated liver Cirrhosis:

Alcohol use is the cause of micro nodular or portal cirrhosis. First alteration in liver is the fat buildup; simple fatty alterations in liver can be reversed if alcohol use is reduced. If alcohol is consumed over time, the liver develops large scars that upset the architecture of the organ.¹¹

Biliary cirrhosis:

It results from persistent infection and biliary blockage. There is jaundice and widespread liver fibrosis. 12

Cardiac cirrhosis:

Recurrent and prolonged right side heart failure with cor-pulmonale, tricuspid regurgitation, constrictive pericarditis is the cause of chronic liver disease and cirrhosis.¹³

Post necrotic cirrhosis:

The type of cirrhosis is macro nodular. It is the most prevalent type of cirrhosis worldwide. It is brought on by an idiopathic, autoimmune, toxic, or viral form of hepatitis. 14

CAUSES OF CIRRHOSIS

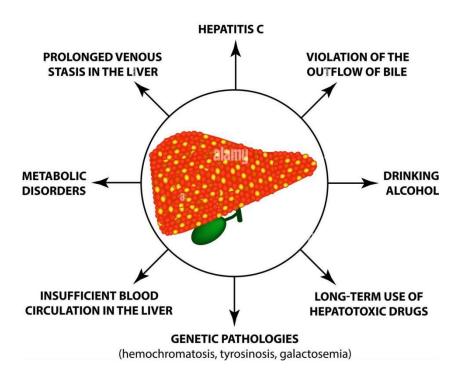


Figure 2: Causes of cirrhosis

Individuals who suffer from immunological suppression, people with diabetes mellitus, or insulin resistance are more vulnerable to developing cirrhosis of various causes. Thus, cirrhosis can often be caused by a major component as well as interacting co-factors in many cases. From patient to patient, the relative relevance of various co-factors may differ. Hepatitis B, C, and autoimmune hepatitis are common types of chronic hepatitis. Periportal inflammation and parenchymal cell necrosis are two pathological characteristics of chronic viral hepatitis.

It is quite likely that hepatitis-C virus i.e HCV has evolved a method of obstructing immunologic signalling pathways hence preventing the effects of antiviral medications. Thus, within infected hepatocytes, virus facilitates dodging of host's innate & adaptive immunologic response. Furthermore, natural killer cells' cytotoxic function against liver cells infected with HIV is downregulated. Therefore, once chronicity has been established, the

virus is thought to be resistant to antiviral cytokines and can persist even if cytotoxic T lymphocytes (CTL) are present. Nevertheless, inflammatory and contaminated regions continue to trigger fibrogenesis by stimulating stellate cells of liver by cytokines & other signalling molecules. The fibrosis then slowly spreads out to the lobules and in the direction of central veins, with the afflicted regions mostly being the areas around the portal tracts. ¹⁰

The complex connections between the virus and the human immune system are what drive the pathophysiology of liver damage caused by the hepatic B virus (HBV). Antibodies against hepatitis B are present in patients with chronic hepatitis B; nonetheless, they are insufficient to combat the virus. Through endocytosis, the virus is able to enter hepatocytes. Adaptive immunologic response, particularly virus - specific CTLs, reacts to the virus's ongoing reproduction within hepatocytes in an effort to remove HBV from impacted hepatocytes. The majority of hepatocellular necrosis is caused by the death of infected but still functioning liver cells. ^{10,15}

When the hepatic cells are invaded by the human immune system, autoimmune hepatitis results. The underlying cause could be an acute liver infection or a genetic susceptibility.

Liver inflammation and necrosis of the cells are caused by the aberrant immune response. Unlike hepatitis C, autoimmune hepatitis and hepatitis B are classified as high grade necro-inflammatory illnesses. As so, the former frequently results in extensive zones of parenchymal extinction.¹⁰

Cholestasis either intra- or extra-hepatic is a characteristic of chronic biliary disorders. While the inflammatory deterioration of bile ducts is typically caused by protracted biliary blockage, metabolic abnormalities (such as genetic disorders and drugs) are also thought to be contributing factors. Bile acid drainage is hampered by the ducts' increasing degradation. As biliary products build up in the liver, they might seep out and harm healthy tissue. In

biliary cirrhosis, larger portal triads are frequently connected by a network of fibrotic septa.

Anastomoses and regenerative nodules are two less common and less developed cirrhotic characteristics. 9,16

RISK FACTORS:

These include Alcoholism, Obesity, Unsafe sex and prostitutes, intravenous drug abuse, Hepato-toxic drugs, Blood transfusion, low socio-economic status, Tattoo culture, workers in chemical industries and health care..

CLINICAL FEATURES:

The physician typically notices growing belly circumference when they initially suspect ascites as the first symptom. A more noticeable buildup of fluid might raise the diaphragm, which can result in dyspnea. Physical examination may reveal ascites when there are bulging flanks, a fluid thrill or changing dullness if peritoneal or ascitic fluid quantity exceeds 500 mL. Lesser quantities of ascitic fluid can be found using a USG, ideally in conjunction with a doppler investigation.

The development of extensive portal venous connections is necessary to reduce pressure in portal venous system with high pressure. An increase in splanchnic blood flow that occurs following the formation of collateral is thought to be responsible for the maintenance of portal hypertension. Oesophageal and stomach varices (joining the diaphragmatic, oesophageal, azygos, and intercostal veins of the caval system at the junction of the left and short gastric veins).

Haemorrhoids (from the caval system's middle and inferior hemorrhoidal veins to the portal system's superior hemorrhoidal vein).

Caput medusa (A big paraumbilical vein may arise from remnants of foetus's umbilical circulation present in the falciform ligament).

Additional locations for anastomoses include the omental veins, lumbar vein, retroperitoneal vein and other veins covering the exposed liver tissue.

COMPLICATIONS:

Cirrhosis is classified as compensated and decompensated based on clinical results. Decompensation denotes the appearance of clinically noticeable side effects (e.g., jaundice) brought on by either portal hypertension or liver failure. However, because the liver can heal itself after suffering liver injury, compensated cirrhosis does not show any symptoms. It may, nevertheless, advance to decompensation.¹⁷

Table 1: Complications of Cirrhosis.

Portal hypertension	Coagulopathy
Gastroesophageal varices	Factor deficiency
Portal hypertensive gastropathy	Fibrinolysis
Splenomegaly, hypersplenism	Thrombocytopenia
Ascites	Bone disease
Spontaneous bacterial peritonitis	Osteopenia
Hepatorenal syndrome	Osteoporosis
Type 1	Osteomalacia
Type 2	Hematologic abnormalities
Hepatic encephalopathy	Anemia
Hepatopulmonary syndrome	Hemolysis
Portopulmonary hypertension	Thrombocytopenia
Malnutrition	Neutropenia

Blockage of portal flow of blood is the main cause for portal hypertension, which is seen in all kinds of cirrhosis. Part of the blood from the portal vein is redirected into collateral channels, avoiding the hepatocytes and going straight into venous radicles of liver within the fibrous septa.

These preexisting sinusoids that are occluded in the septa give rise to these portohepatic anastomoses. The cirrhotic liver has even greater portohepatic venous anastomoses. Via these pathways, about 1/3rd of total blood flow supplying the liver that has cirrhosis may avoid the sinusoids & subsequently the working tissues of liver. Hepatic venous radicle compression caused by nodules is a contributing factor in the restriction of portal flow. A postsinusoidal portal hypertension would result from this. The main portal pressure and the wedged hepatic venous (sinusoidal) pressure in cirrhosis, however, are nearly equal, and this stasis needs to reach the inflow vessels of portal system. Most likely, Sinusoids offer highest flow resistance. Sinusoidal narrowing is caused by changes in the Disse space, including collagenization, and this may be especially significant in alcoholics. Additionally, the alcoholic's hepatocyte enlargement may reduce sinusoidal flow.

Increased intrahepatic vascular resistance (fibrous tissue and regenerating nodules) combined with increased blood flow in portal system (hyperdynamic circulation) can cause portal hypertension in cirrhotic livers. The deformed angio-architecture and aberrant hemodynamics cause the blood pressure of the portal venous system, which typically ranges from 5 to 10 mmHg, to surpass this threshold.^{17,18} The majority of the clinical problems associated with cirrhosis can be traced back to hypertension.

"The buildup of too much fluid in the peritoneal cavity is known as ascites". Although many different causes are attributed to exudative and transudative ascites, persons with cirrhosis and other serious liver diseases are most likely to experience it.

The extrahepatic blood flow's excess nitric oxide generation, a vasodilator molecule, is most likely what's causing the hyperdynamic circulation. The enhanced intestinal permeability of endotoxins of bacteriae encourages the overproduction of NO. The NO molecule causes a significant reduction in vascular resistance in the systemic and splanchnic circulations, which increases cardiac output and blood flow to splanchnic vessels (including portal vessels).

SEVERITY & GRADING IN CIRRHOSIS:

Prognostic models can be important medical decision-making tools for directing patient care since they can be used to estimate disease severity and survival. These models are created by statistical techniques that entail figuring out how certain variables (such laboratory values, clinical data, and demographics) affect particular outcomes, like death.

These predictive models are being derived more and more by machine learning. In healthcare settings, there are several prognostic models in use. "Acute Physiology and Chronic Health Evaluation System (APACHE III)" is one that focuses on overall health status, whereas other tools are disease-specific. "The Model for End-stage Liver Disease (MELD) score", "the MELD-Sodium (MELD-Na) score", and "the Child-Pugh score" are frequently used for treatment of patients suffering from cirrhosis.

MELD Na SCORE:

Original "MELD score" is a prospectively established and accepted system of rating severity of chronic liver disease. It predicts three-month survival by utilising test results for blood bilirubin, sr. creatinine, & "international normalised ratio (INR)" for "prothrombin time". An rising "MELD score" is linked to a greater risk of three-month death and a worsening of hepatic dysfunction in cirrhosis patients.

In order to prioritise patients awaiting transplantation of liver in the United States, "the United Network for Organ Sharing (UNOS)" first adopted MELD in 2002 due to its efficiency in prediction of short-term survival in patients of liver cirrhosis. 11,19,20

CALCULATION OF "MELD SCORE":

MELD formula used by UNOS in year 2002 is as follows:

"MELD = 3.8*loge(serum bilirubin [mg/dL]) + 11.2*loge(INR) + 9.6*loge(serum creatinine [mg/dL]) + 6.4"

- Sodium values below 125 mmol/L is made 125, and above 137 mmol/L is made 137.
- any patient dialyzed twice in previous week then creatinine value used should be
 4.
- Any value < 1 is made 1 to prevent a negative value because log of 1 becomes 0
 and value < 1 gives negative result.

Scores in this model can range from negative numbers till infinity. However, "UNOS" changed "MELD score" and removed negative scores by replacing any of the laboratory results which were less than 1 with 1. This was done to prevent confusion. Patients will therefore obtain a minimum score of 6 MELD points if they have INR \leq 1, sr. creatinine \leq 1 mg/decilitre and total bilirubin of \leq 1 mg/decilitre combined. Furthermore, UNOS established a 40-point maximum MELD score.

MELD- Na score:

Updates to the Organ Transplantation Network now factor sr. sodium into "MELD score" computation. UNOS allocates livers from dead donors based on the "MELD na score". One can compute the "MELD-Na Score" online.

Patients with cirrhosis frequently experience hyponatremia, and level of hypo-natremia is good indicator of severity in cirrhosis.

"MELD-Na = MELD + 1.32 * (137-Na) - [0.033*MELD * (137-Na)]"

SCORE	MORTALITY(%)
MELD/Na score >40	71.3
MELD/Na score 30-39	52.6
MELD/Na score 20-29	19.6
MELD/Na score 10-19	6
MELD/Na score <9	1.9

Table2: MELD- Na score.

Independent of the "MELD score", serum sodium predicts waitlist mortality and represents the vasodilatory condition in cirrhosis. ²¹ Between 125 and 140 milli-mol/L, there appears to be an increased mortality linearly by 5% with every milli-mol drop in sr. sodium. many studies show that in patients suffering from hyponatremia with lower "MELD scores" who are waiting for hepatic transplantation, the usage of the concentration of sr. sodium increases the predictive power of "MELD score". In roughly 12 % the patients on the list, the transplant priority is increased when serum sodium is included in the MELD model.

One drawback of the MELD-Na score is that intravenous fluid treatment and the use of diuretics may change serum sodium levels.

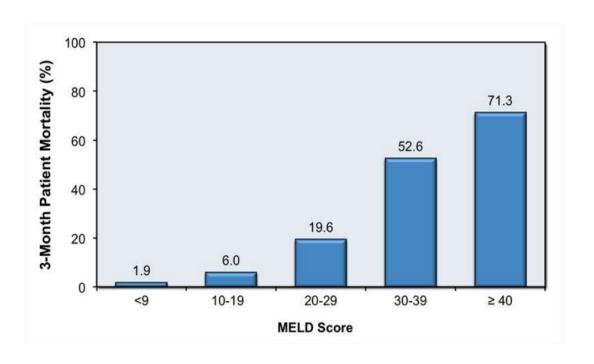


Figure 3: Mortality based on MELD score

"LIMITATIONS OF THE MELD SCORE":

"MELD score" is susceptible to changes among the measurements made in a lab. For instance, even after being adjusted for thromboplastin's sensitivity, the international normalised ratio (INR) can differ between labs if recombinant thromboplastin is utilised instead of thromboplastin derived from rabbit brain. This could potentially result in significant variations in patient prioritisation based on MELD.²²The technique used to quantify serum creatinine may potentially have an impact on the MELD score. When there is an increased serum bilirubin concentration, variability in the measurement of serum creatinine using different assays can be especially problematic. However, this can be avoided by measuring serum creatinine using an enzymatic method, especially when the total bilirubin level is greater than 25 mg/Dl.Furthermore, the administration of free water or the use of diuretics may cause variations in serum sodium levels.²³

Child Turcotte Pugh score:²⁴

It was created by Turcotte and child in year 1964 to regulate picking of patients that would profit from a portal decompression surgery. Originally intended for prediction of prognosis of the patients scheduled for liver-disease associated portosystemic shunt surgery.

Additionally provides data on the occurrence of post-operative problems such as worsening liver functions, intractable ascites, renal failure, gastrointestinal bleeding, and hepatic encephalopathy.

It is classified as:

Class	Liver function
A	good
В	moderately impaired
С	Advanced hepatic dysfunction.

This system of scoring originally used 5 criteria of laboratory and clinical findings to classify patients: sr. albumin, sr. bilirubin, neurological disorder, ascitic fluid and status of nutrition. This system was later modified by "Pugh" by replacing the "prothrombin time" with clinical status of nutrition.

class in CP score	points	1 year survival	2 year survival
Class A	5-6	100%	65%
Class B	7-9	80%	60%
Class C	10-15	45%	35%

Factor	1 point	2 points	3 points
Total bilirubin (μmol/L)	<34	34-50	>50
Serum albumin (g/L)	>35	28-35	<28
PT INR	<1.7	1.71-2.30	>2.30
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

	Class A	Class B	Class C
Total points	5-6	7-9	10-15
1-year survival	100%	80%	45%

Table 3: Child Pugh Score.

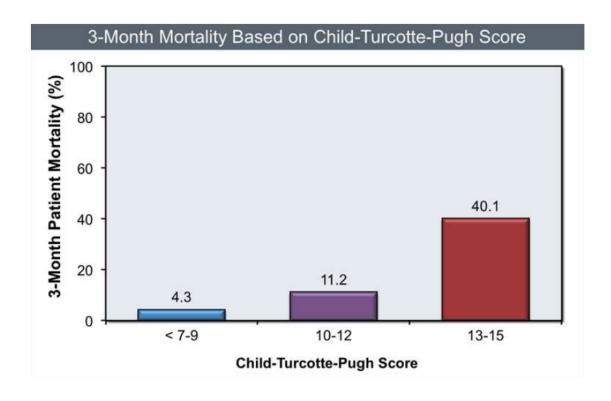


Figure 4: Mortality based on Child-Pugh score

"Child Pugh classes and their surgical outcomes: 25,26

Class A – well compensated cirrhosis:

• Moderate increase in surgical risk

Class B and C – decompensated cirrhosis:²⁷

- Substantial increase in surgical risk.
- Complications should be treated before elective surgery.
- In Child class C surgery must be done only in life threatening conditions eg. incarcerated hernia.
- Meticulous care is essential in patients undergoing surgery such as 28,29
 - > Improvement in general nutritional status
 - ➤ Maintenance of hemodynamic stability
 - > Broad spectrum antibiotics
 - Correction of any Coagulopathies
 - > Avoid nephro-toxins
 - ➤ Sedatives should be avoided precipitate hepatic encephalopathy"

The "lymphocyte/monocyte ratio (LMR)" which is the ratio of lymphocyte count to monocyte count, is an biomarker of inflammation and indicative of balance between the tumor microenvironment and host immune system. ³⁰The inflammatory marker "lymphocyte-to-monocyte ratio (LMR)" has been shown to determine the patient's survival with various ailments like colorectal carcinoma, gastrointestinal diseases eg. Crohn disease and cardiovascular diseases. ³¹⁻³⁴ in most studies. LMR is largely researched due to its low cost and simplicity of calculation and interpretation. Few researchers have studied its role in determination of "chronic hepatitis B-related liver cirrhosis patient" outcomes. ³⁰

PUBLISHED PAPERS

Jamil Z et al³⁵ conducted a study on 182 cirrhosis patients found that LMR and "MELD" and "CP scores" correlated positively, while LMR correlated negatively. The study also found that patients were devided into groups of low and high LMR, with low LMR scores notably higher than high "LMR" scores. Patients in the low "LMR" group showed lesser survival, while non-survival group had low LMR and high "MELD" and "CP scores". Logistic regression models showed MELD, CP score, 1/LMR, ALT level, and international normalized ratio as predictors of patient outcomes. The study concluded that LMR can be used to determine patient outcomes during hospital stays, as it is easily calculated and efficaciously interpreted similar to MELD and CP scores.

Nguyen DT et al³⁶ conducted a study that examined the cirrhotic patients, focusing on the interrelationship between "lymphocyte-to-monocyte ratio" and "Child-Pugh" and "MELD/MELDNa scores". The analysis of 153 patients at "Can Tho Central General Hospital" revealed that the majority were male i.e 66.7% and over 60 years old i.e 51.6%. Cirrhosis was primarily caused by alcoholism and hepatitis B.³⁷ Laboratory and clinical characteristics resembled previous studies. The mean "CP score" was 9.3 ± 2.1 , with Child A and B scores of 44.4% and 45.8%, respectively. The mean "MELDNa" and "MELD" scores were 19.4 ± 8.1 and 16.9 ± 7.1 , respectively. The "lymphocyte-to-monocyte ratio (LMR)" was found to be negatively correlated with other scores, with patients with LMR less than 3.31 more incline towards classification under Child-Pugh classes B and C.³⁸

Salehi A et al³⁹ conducted a study that concluded "LMR" has a strong negative relation with "PELD/MELD" and "Child-Pugh scores", with the maximum area under curve i.e AUC for "LMR" (0.861). The values of "AUC" for "LMR" in patients above an under 6 years age were 0.926 and 0.675, respectively. "PELD/MELD scores" were notably higher in

the low LMR group in contrast to the high "LMR" group. 40 LMR could be utilized to establish the outcome of children above 6 years of age and suffering from cirrhosis during hospital stays, as it is easily calculated and has comparable efficacy to "PELD/MELD scores".

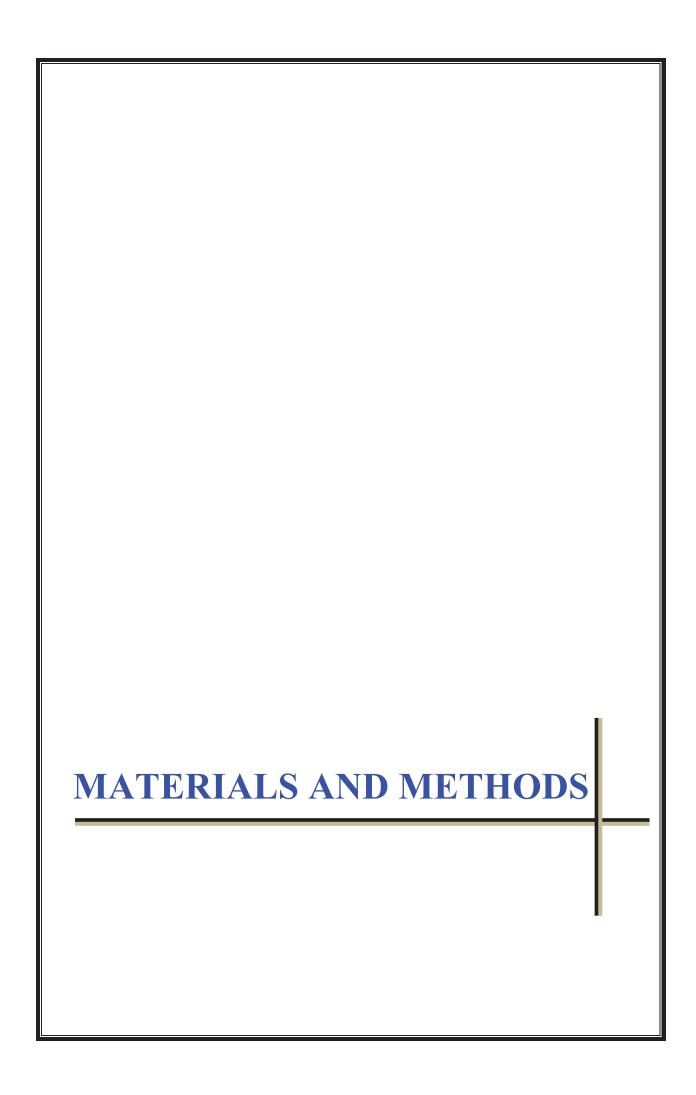
Kim KM et al⁴¹conducted a study analyzing 437 newly diagnosed HCC patients found that the MELD-Na score effectively predicts mortality in first, second and third year, particularly mortality in first year. The score increased notably after treatment in patients that received best supportive care, TACE, and other treatments. However, for patients with advanced tumor stage and "MELD-Na score" ≥ twelve, Hepatocellular carcinoma-specific treatment would not provide more favourable prognosis as compared to the best supportive care. "MELD-Na" can identify functional reserve of liver and prognosis of Hepatocellular carcinoma patients having ascites, and when combined with staging of tumor, it may help in establishing strategy for their therapy.

Mallik M et al⁴²conducted a longitudinal study analyzing 140 patients with liver cirrhosis at the "All India Institute of Medical Sciences Bhopal between July 2019 and July 2020". Follow up of the participants was done over 3 months in order to study the outcomes in the short-term. Haematological tests were conducted on presentation and after three months in survivors. most of the patients i.e 47% fell into "Child-Pugh class C". The mean "MELD score was 13.54", "LMR score was 1.96", and "CLIF-SOFA score was 5". The non-surviving group had significantly higher albumin levels, CLIF-SOFA, MELD, CPS, absolute monocyte count, INR, sr. creatinine, total leukocyte count and total bilirubin compared to the surviving group. "LMR" and "CLIF-SOFA" were notable independent risk factors for mortality, after adjusting for confounding factors. "CLIF-SOFA" was the best prognosticator for mortality at above 5 cut-off point, with chances of mortality prediction at 80.8%. "The

important mortality and morbidity indicators were high total bilirubin, creatinine, INR, TLC, and low platelet count and albumin levels".

Chen B et al⁴³conducted a study and found that non-surviving patients showed lower lymphocyte markers ("LMR"), which were independent risk factors for 3-month mortality. "Zhang et al." found that "LMR" was statistically low in non-surviving group & closely correlated to "MELD score". A 2019 study showed that low "LMR" is associated with increased 1-month mortality in cirrhosis patients. Lower "LMR" may be secondary to inflammatory response in liver cirrhosis, which stimulates release of monocytes from bone marrow into peripheral blood and blood monocytes differentiation into tissue macrophages. "The AUC for MELD, CPS, LMR, and CLIF-SOFA is 0.765, 0.792, 0.75, and 0.808, respectively". "CLIF-SOFA" is a better mortality predictor but is not significant statistically when compared with other scores.

In a study by **Kim JH et al.,**⁴⁴ "CLIF-SOFA" was an important factor for mortality in 30 days. The cut-off sensitivity and specificity of "MELD" and "LMR" were 20.49, 55.38%, and 93.33%, respectively.



"MATERIALS AND METHODS"

"PLACE OF THE STUDY":

The current study was carried out on inpatients in "Sri devraj URS academy of higher education and research".

"TYPE OF STUDY":

"The current study was a prospective observational study".

"DURATION OF STUDY":

The study was carried out for a period of 24 months from June 2022 to September 2024.

SAMPLE SIZE:

The study was conducted on 136 patients.

"INCLUSION CRITERIA":

Patients meeting below criteria have been included in this study.

- Patients having clinical features suggestive of liver cell failure and portal hypertension.
- Patients with ultrasonographical findings suggesting "chronic liver parenchymal disease"

- Patients with previous hospitalizations due to similar complications and presence of relevant past medical records.
- Patients willing to give consent.
- Patients willing to participate.

"EXCLUSION CRITERIA":

Patients meeting the below criteria have been excluded from this study.

- Patients having age < 18 years.
- Patients having hepato-cellular carcinoma.
- Patients having other concurrent disease causing alteration of "LMR", like hematological malignancy, autoimmune disease or any chronic infection.
- patients unwilling to give consent.
- Patients not willing to participate.

"INFORMED CONSENT"

the details of disease process, treatment options, final outcome, complications, probable effects, and chances of recurrence in both the procedure were explained to patients fulfilling the criteria for selection and an informed consent in written format was obtained before enrolment. Information about their right to withdrawal from the study at any stage was communicated.

"DATA COLLECTION"

- A detailed clinical history was obtained and general and systemic examinations were carried out on patients followed by a thorough review of their hospital records.
- All patients meeting inclusion criteria have been included in this study.
- The data was recorded in "master charts".
- the data was analyzed by subjecting it to statistical analysis.

"STATISTICAL ANALYSIS"

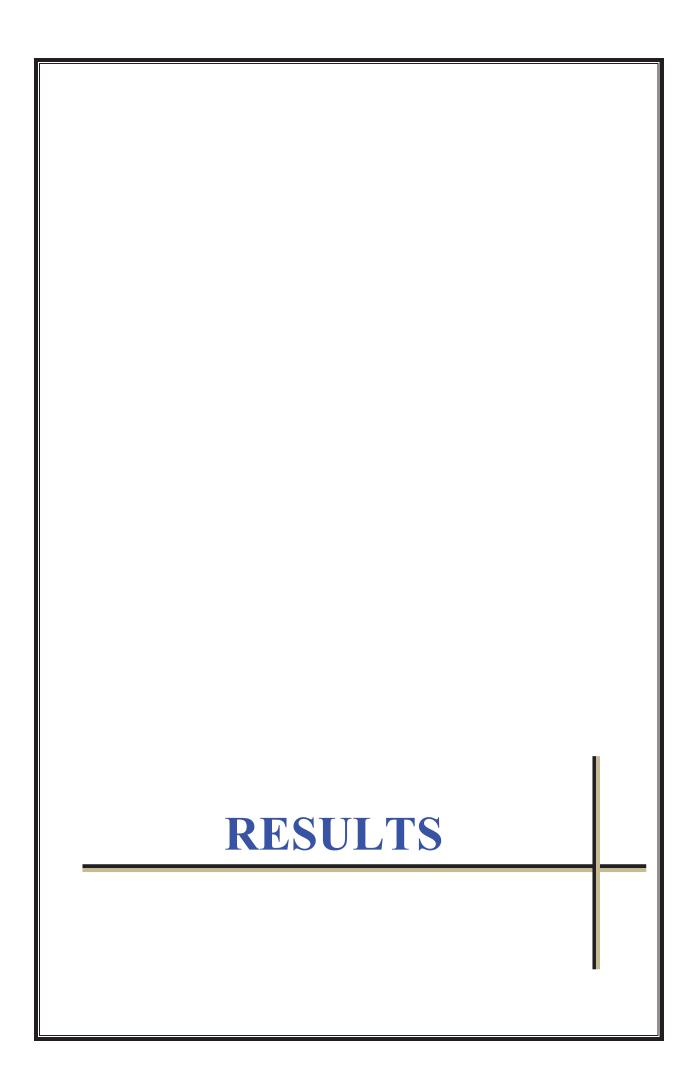
The collected data was entered in "Microsoft Excel Worksheet-2010" and was taken into "IBM SPSS Statistic for windows, version 24 (IBM Corp., Armonk, N.Y., USA) software" for calculating probability value, "standard deviation", mean, percentage & frequency.

Qualitative data has been represented in form of percentage & frequency.

- Association among qualitative variables has been assessed by using "Chi Square test" with continuity correction for the 2 x 2 tables and
- o "Fisher's exact test" for all 2 x 2 tables, where P value of "chi square test" was not valid due to small counts.

Quantitative data has been represented by mean and standard deviation.

- quantitative data Analysis within the groups has been done by using "paired t test" if data passes 'Normality test'.
- o 'One Way Analysis (ANOVA)' was used to compare more than two groups.
 - * A 'P' value < 0.05 has been considered to be significant statistically.



RESULTS

present prospective observational study has been conducted on 136 patients suffering from liver cirrhosis in 'Sri Devaraj Urs Academy of Higher Education and Research' for a period of 24 months from June 2022 to September 2024.

The following were the study results:

Table 4: Age wise distribution of subjects.

Age Group	Number of subjects	Percentage
(years)	(N)	(%)
18 to 30	11	8.09
31 to 40	18	13.23
41 to 50	40	29.41
51 to 60	32	23.53
> 60	35	25.74
Total	136	100

In the present study, participants were segregated into five age groups. The table above gives data on agewise distribution of participants.

Majority subjects fell into ages between of 41 & 50 years i.e. 40 (29.41%) subjects followed by 35 (25.74%) subjects with age >60 years, 32 (23.52%) subjects with age

between 51 and 60 years, 18 (13.23%) subjects in ages between 31 and 40 years and finally 11 (8.09%) subjects of age 18 to 30 years.

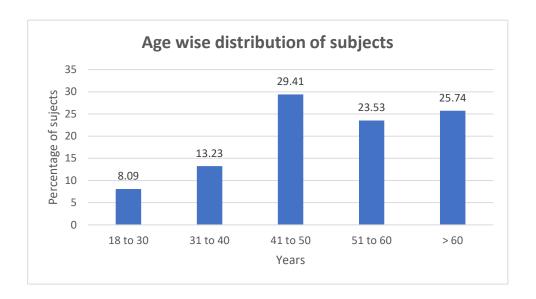


Figure-5: Age wise distribution of subjects

Table 5: Distribution of participants accoring to their gender.

Gender	Number of subjects	Percentage
	(N)	(%)
Male	85	62.5
Female	51	37.5
Total	136	100

Most of the participants were males i.e., 85 (62.5 %) followed by 51 (37.5 %) females.

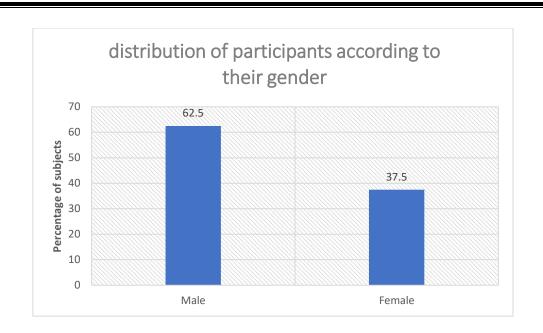


Figure-6: Distribution of participants accoring to their gender

Table 6: Distribution of participants according to the causes of cirrhosis.

Causes of	Number of subjects	Percentage
cirrhosis	(N)	(%)
Alcohol abuse	126	92.65
Hepatitis virus	3	2.21
Steatosis	7	5.14
Total	136	100

The above mentioned table provides data on the distribution of participants based on the causes or cirrhosis.

Majority of subjects had alcohol abuse i.e., 126 (92.65 %) followed by 7 (5.14%) subjects who had steatosis. and finally 3 (2.21%) subjects who had hepatitis virus

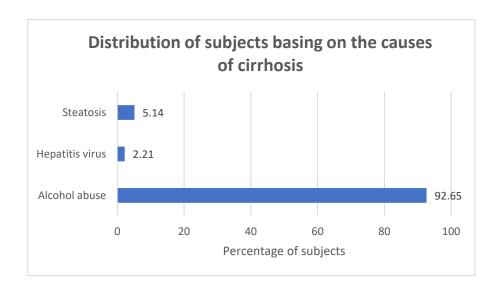


Figure-7: Distribution of participants accoring to cause of cirrhosis

Table 7: Distribution of participants according to time from being diagnosed with cirrhosis.

Time from being	Number of subjects	Percentage
diagnosed as cirrhosis	(N)	(%)
First diagnosis	38	27.94
0 to 5 years	89	65.44
6–10 years	15	11.02
>10 years	6	4.41
Total	136	100

Above table provides data about distribution of patients according to the time from being diagnosed with cirrhosis.

Majority of subjects had been diagnosed in 0 to 5 years i.e., 89 (65.44 %) followed by 38 (27.94%) subjects who had first diagnosis, 15 (11.02%) subjects who had been diagnosed in 6 to 10 years and 6 (4.41%) subjects who had been diagnosed >10 years...

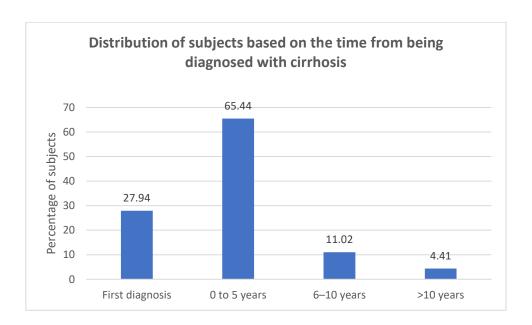


Figure-8:Distribution of subjects based on the time from being diagnosed with cirrhosis

Table 8: Distribution of patients according to their symptoms.

Symptoms	Number of subjects	Percentage
	(N)	(%)
Fatigue	124	91.17
Anorexia	115	84.55
Insomnia	81	59.55
Stomachache	63	46.32

Nausea	15	11.02
Loose stools	27	19.85
Constipation	22	16.17
Ascites	65	47.79
Perceptual	34	25
disturbances		
Nevus araneus	58	42.64
Palmar	55	40.44
erythema		
Leg edema	78	57.35
Gynecomastia	3	2.20
Mucosal	12	8.82
bleeding		
Jaundice	95	69.85

Above table provides data about distribution of participants based on their symptoms.

Most of the participants had fatigue i.e. 123 (91.17 %) followed by 115 (84.55%) subjects having anorexia, 95 (69.85%) subjects having jaundice, 81 (59.55%) subjects having insomnia, 78 (57.35%) subjects having leg edema, 65 (47.79%) subjects having ascites, 63

(46.42%) subjects having stomach ache, 58 (42.64%) subjects having nervous araneus, 55 (40.44%) subjects having palmar erythema, 34 (25%) subjects having perceptual disturbances, 27 (19.85%) subjects having loose stools, 22 (16.17%) subjects having constipation, 15 (11.02%) subjects having nausea, 12 (8.82%) subjects having mucosal bleeding, 3 (2.20%) subjects having gynecomastia

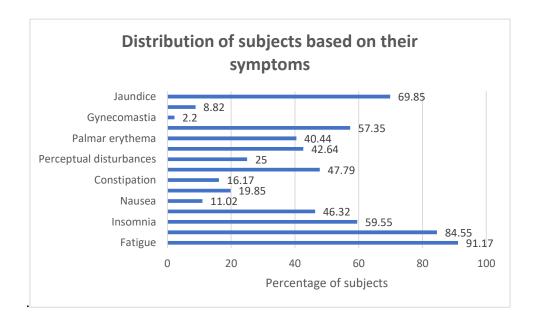


Figure-9:Distribution of participants according to their symptoms

Table 9: Distribution of participants according to their body temperature.

Body	Number of subjects	Percentage
temperature	(N)	(%)
(F)		
98.5	123	90.44
98.5 to 105	9	6.61
> 105	4	2.94
Total	136	100

Above table provides data about distribution of patients based on the body temperature.

Most patients had temperature of 88.5~F i.e., 123~(90.44~%) followed by 9~(6.61%) subjects who had temperature of 98.5~to~105~F and finally 7~(5.14%) subjects who had temperature >105~F.

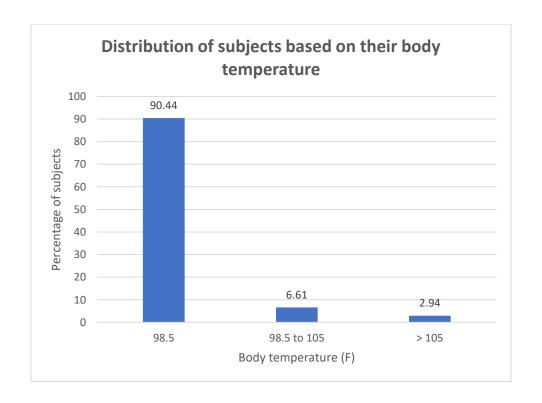


Figure-10:Distribution of participants according to their body temperature

Table 10: Distribution of participants according to their 'blood pressure'.

Blood pressure	Number of subjects	Percentage
(mmHg)	(N)	(%)
<100	13	9.55
100 to 140	108	79.41
>140	15	11.02
Total	136	100

Above table provides data about distribution of patients according to their blood pressure.

Most participants had blood pressure between 100 to 140 mmHg i.e., 108 (79.41 %) followed by 15 (11.02%) subjects who had blood pressure >140 mmHg and finally 13 (9.55%) subjects who had blood pressure <100 mmHg.

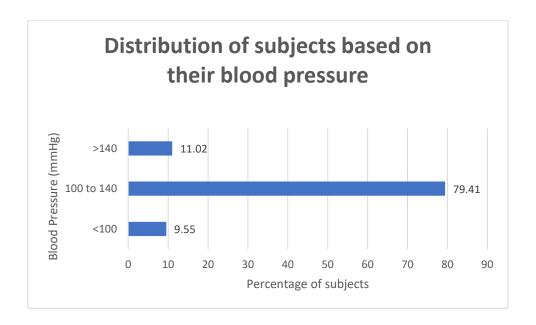


Figure-11: Distribution of participants according to their blood pressure

Table 11: Distribution of participants according to their pulse rate.

Pulse rate	Number of subjects	Percentage
(bpm)	(N)	(%)
<100	94	69.11
100 to 120	37	27.20
>120	5	3.67
Total	136	100

Above table provides data about distribution of participants according to their pulse rate.

Most of the patients had pulse rate <100 bpm i.e., 94 (69.11%) followed by 37 (27.20%) subjects who had pulse rate between 100 to 120 bpm and 5 (3.67%) subjects who had pulse rate >120 bpm.

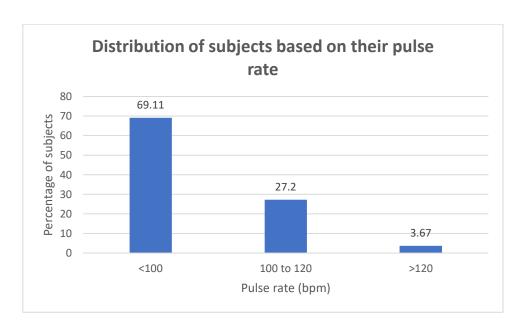


Figure-12:Distribution of participants according to their pulse rate

Table 12: Mean of haematological parameters of patients.

Haematological	Mean	SD
parameters		
Hemoglobin (g/dL)	9.85	2.13
Red blood cell count (cells/mm³)	3.11	0.53
Hematocrit (%)	32.32	6.11
MCV (fL)	92.95	11.91
MCH (g)	29.11	5.63
WBC (cells/mm³)	8500	1214

Above table provides data about distribution of patients according to their mean haematological parameters.

The hemoglobin levels were 9.85 ± 2.13 g/dL, red blood cell count was 3.11 ± 0.53 cells/mm³, hematocrit levels were $32.32\pm6.11\%$, MCV levels were 92.95 ± 11.91 fL, MCH levels were $29.11\pm5,63$ and WBC count was 8500 ± 1214 cells/mm³.

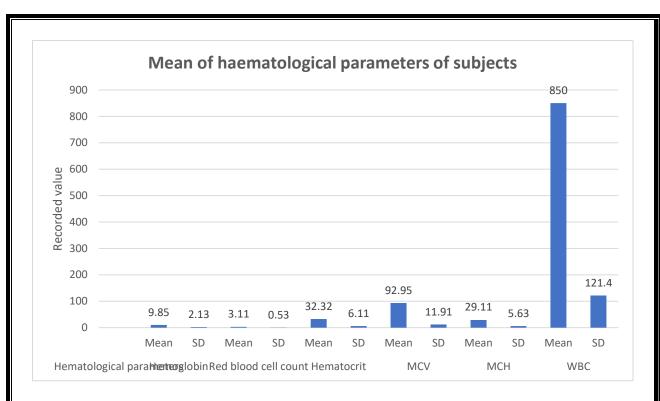


Figure-13:Mean of haematological parameters of subjects

Table 13: Mean values of peripheral blood analysis of subjects.

Parameters	Mean	SD
Platelets	1.3	0.8
(lakhs/mm³)		
Prothrombin rate	58.30	20.11
(%)		
INR	1.85	0.13
APTT	39.12	9.11
(seconds)		

Above table provides data about distribution of patients according to their mean peripheral analysis of blood.

The platelet count was 1.3 ± 0.8 lakhs/mm³, prothrombin rate was $58.30\pm20.11\%$, INR levels were 1.85 ± 0.13 and APTT was 39.12 ± 9.11 seconds.

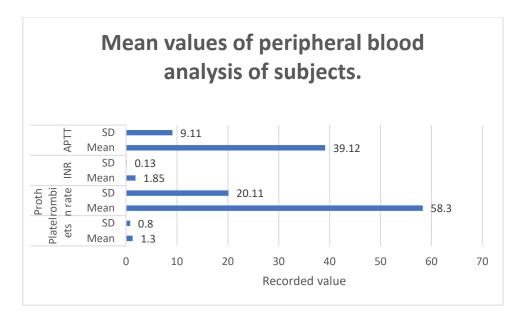


Figure-14:Mean values of peripheral blood analysis of subjects.

Table 14: Mean values of liver function tests of subjects.

Parameters	Mean	SD	Normal values
Albumin	28.13	5.12	34 to 54
(g/L)			
Protein	66.85	9.01	60 to 83
(g/L)			
Total bilirubin	110.12	88.45	1.71 to 20.5
(µmol/L)			
Direct bilirubin	81.93	58.49	<5.1
(µmol/L)			
SGOT	128.58	24.15	8 to 45
(U/L)			
SGPT	75.32	18.96	7 to 56
(U/L)			

Above table provides data about distribution of patients according to the liver function tests.

The albumin was 28.13 ± 5.12 g/L, protein was 66.85 ± 9.01 g/L, total bilirubin was 110.12 ± 88.45 µmol/L, direct bilirubin was 81.93 ± 58.49 µmol/L, SGOT was 128.58 ± 24.15 U/L and SGPT was 75.32 ± 18.96 U/L.

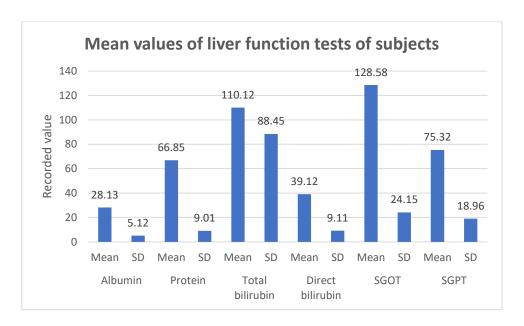


Figure-15: Mean values of liver function tests of subjects

Table 15: Mean biochemical values of subjects.

Parameters	Mean	SD
Glucose (mmol/L)	7.85	4.19
Blood Urea	5.61	3.33
Serum Creatinine	93.12	6.13
(μmol/L)		

Above table provides data about distribution of patients according to their mean biochemical values.

The glucose levels were 7.85 ± 4.19 mmol/L, urea levels were 5.61 ± 3.33 mmol/L, and creatinine levels were 93.12 ± 6.13 µmol/L.

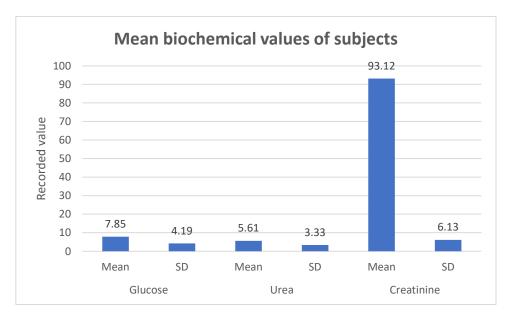


Figure-16: Mean biochemical values of subjects

Table 16: Mean serum electrolyte levels of subjects.

Parameters	Mean	SD	
(mmol/L)			
Sodium	132.78	5.12	
Potassium	3.77	0.59	
Chlorine	96.11	4.32	
Calcium	2.11	0.11	

Above table provides data about distribution of patients according to their mean serum electrolyte levels.

The sodium levels were 132.78 ± 5.12 mmol/L, potassium levels were 3.77 ± 0.59 mmol/L, chlorine levels were 96.11 ± 4.32 mmol/L and calcium levels were 2.11 ± 0.11 mmol/L.

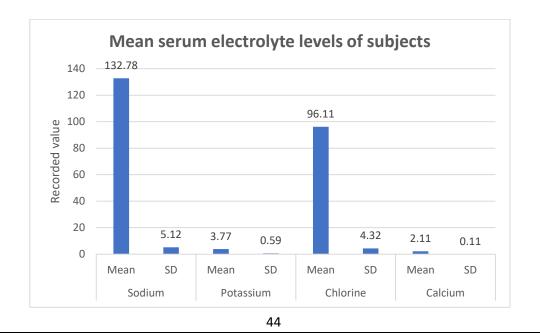


Figure-17:Mean serum electrolyte levels of subjects

Table 17: Distribution of patients according to their Child-Pugh (CP) score.

Child-Pugh	Number of subjects	Percentage
group	(N)	(%)
Group A	12	8.82
Group B	59	43.38
Group C	65	47.79
Total	136	100

Above table provides data about distribution of patients according to their Child-Pugh (CP) score.

Majority of subjects belonged to Group C i.e. 65 (47.79%) subjects, followed by 59 (43.38%) subjects belonging to Group B and 12 (8.82%) subjects belonging to group A.

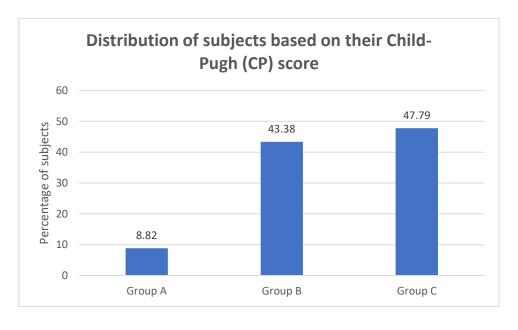


Figure-18: Distribution of subjects based on their Child-Pugh (CP) score

Table 18: Child-Pugh (CP) score of subjects Vs their "MELD-Na score" and "lymphocyte to monocyte ratio".

'Child-Pugh' group	'MELD-Na score' (Mean ± SD)	'Lymphocyte to monocyte ratio' (Mean ± SD)
Group A	9.1 ± 2.5	2.75 ±1.5
Group B	16.8 ±4.8	2.03 ±1.12
Group C	28.1 ± 7.9	1.15 ±1.01

The above table provides data for comparison of Child-Pugh (CP) score of subjects

Vs their MELD Na score and lymphocyte to monocyte ratio.

Based on MELD-Na score, majority of score was in Group C i.e. 28.1 ± 7.9 , followed by 16.8 ± 4.8 score in Group B and 9.1 ± 2.5 score in Group A.

Based on lymphocyte to monocyte ratio, Group A had highest ratio i.e. 2.75 ± 1.5 , followed by Group B i.e. 2.03 ± 1.12 and finally Group C i.e. 1.15 ± 1.01 .

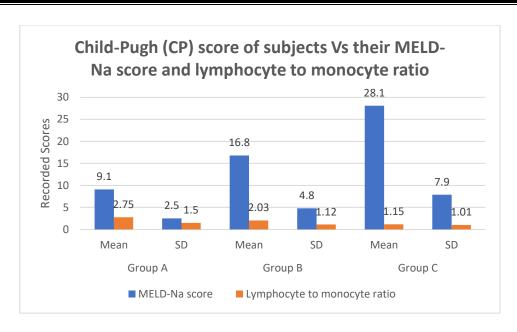


Figure-19: Child-Pugh (CP) score of subjects Vs their MELD-Na score and lymphocyte to monocyte ratio

Table 19: Co-relation analysis between LMR, MELD-Na and CP scores.

Score	СР	MELD-Na	LMR
СР	1	<0.001	- 0.05
MELD-Na	<0.001	1	- 0.03

The above table gives data on co-relation analysis between LMR, MELD-Na and CP scores.

Child-Pugh score had a negative and statistically significant co-relation with lymphocyte to monocyte ratio (r:- **0.05**), indicating that an increase in the Child-Pugh score, lymphocyte to monocyte ratio decreases significantly.

Child-Pugh score had a positive and statistically highly significant co-relation with 'MELD-Na score' (r:- <0.001)indicating that an increase in the "Child-Pugh score", "MELD-Na score" increases significantly.

"MELD-Na score" had a negative & statistically significant co-relation with lymphocyte to monocyte ratio (r:- 0.03) indicating that an increase in the MELD-Na score, lymphocyte to monocyte ratio decreased significantly.

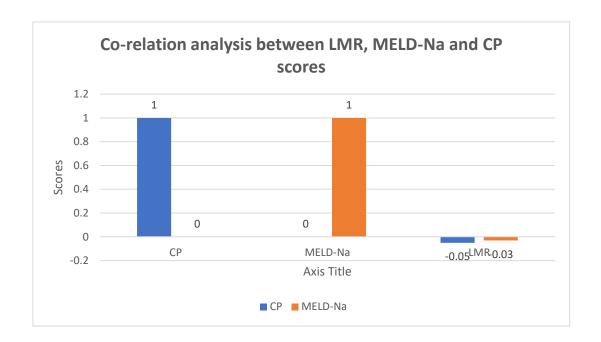
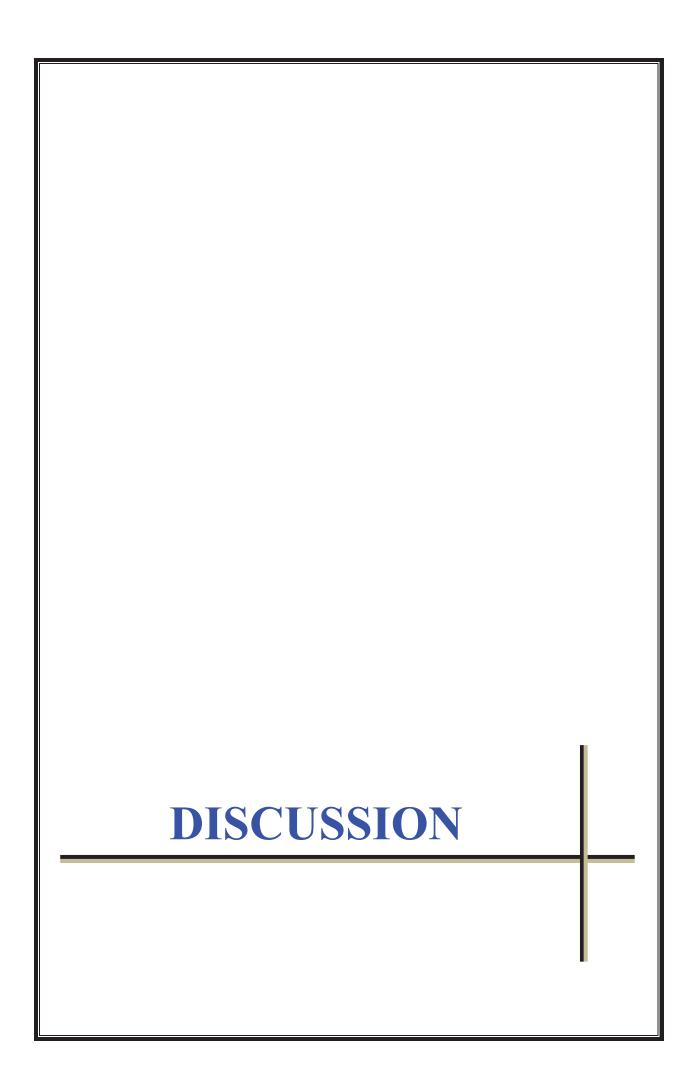


Figure-20:Co-relation analysis between LMR, MELD-Na and CP scores



DISCUSSION

In the majority of countries, liver cirrhosis is the most common hepatobiliary illness. Hepatitis C and B viruses, alcohol, & biliary tract disorders like autoimmune hepatitis, 'sclerosing cholangitis', gallstones etc. are significant causes of cirrhosis. 45,46 Unfortunately, because cirrhosis frequently presents silently, precise data regarding the percentage of people with this disease are lacking. Since cirrhosis is typically discovered only after complications have arisen, the death rate among these patients has rarely been completely explained. Encephalopathy, gastrointestinal haemorrhage, hepatorenal syndrome, malignancy, infections, etc. are the main reasons of death in cirrhosis patients. 47

Although liver transplantation is a revolutionary treatment for cirrhosis, it is primarily performed in industrialized nations. The amount of patients awaiting liver transplantation is rising, therefore it's critical to identify those who require transplantation as soon as possible with a clear-cut prognosis. The Child-Pugh classification has been extensively utilised for the past 40 years to estimate mortality in cirrhosis patients; alternative scores have also been mentioned. To replace the "Child-Pugh score" in the classification of patients on the waiting list for liver transplantation, "MELD (Model for End-Stage Liver Disease) index" has been studied as a predictor for mortality of cirrhosis patients waiting for transplantation of liver in America and Europe. ^{48,49} Recently, patients with low serum sodium levels who are cirrhotic have been treated with the MELD-Na, a further development of the MELD index. ^{50,51}

Alternatively, because systemic inflammation and immune system dysregulation are integrated into the physiopathological route of advanced cirrhosis, the "lymphocyte-to-monocyte ratio" is now a prognosticating marker in this disease. This begins with a local insult to the liver, which, after being overcompensated, results in a decrease in the manufacture of immune proteins and recognition receptors, hence decreasing the innate immune system's ability to kill bacteria. Secondarily, an increased enteric bacterial load

damages the gut and the lymphoid tissue that surrounds it, which serves as a barrier against intestinal pathogens.

The ultimate outcome of such a prolonged inflammation is the tiredness and re-programming of different immune associated cell lines, including "LMR" modification. As cirrhosis progresses, other clinical and biochemical indicators are also altered, including body temperature, heart and respiration rates, and portal blood pressure.⁵² White blood cell count has been demonstrated in recent research to be a separate component of systemic inflammation and to be a reliable indicator of the onset, severity, and related mortality of acute on chronic liver failure.⁵³⁻⁵⁶Furthermore, the "LMR" has demonstrated a function in prognosticating survival of various patients, including those with Crohn's disease, cancer, and cardiovascular disease.⁵⁷⁻⁵⁹

The LMR has not been sufficiently researched, but it might be a marker for prognosis in patients having liver cirrhosis because it is a cheap, easily obtained, repeatable, and widely available biomarker. Therefore, purpose of this study has been to identify clinical along with laboratory features of cirrhosis patients in order to investigate the relationship between LMR and the prognostic instruments that are currently being used, such as "Child-Pugh" and "MELD/MELDNa scores".

AGE:

Majority of subjects were aged between 41 and 50 years which was 29.41% patients followed by 25.74% patients with age of >60 years, 23.52% patients with age between 51 & 60 years, 13.23% patients in age between 31 & 40 years and finally 8.09% subjects of age 18 to 30 years.

Our study was similar to Nguyen DT et al³⁶, Sajja KC et al⁶⁰

Study by	Majority (Years)
Nguyen DT et al ³⁶	60 (51.6%)
Sajja KC et al ⁶⁰	42-52 (32.12%)
Present study	41 to 50 (29.41%)

"GENDER":

Most of patients were males which was 62.5 % followed by 37.5 % female patients.

Our study was in correlation with Nguyen DT et al³⁶, Fornari F et al⁶¹, John BV et al⁶²

Majority (Percentage)
Male (66.7%)
Male (61.9%)
Male (59.5%)
Male (62.5%)

CAUSE OF CIRRHOSIS:

Majority of subjects had alcohol abuse which was 92.65 % patients followed by 5.14% patients who had steatosis and lastly 2.21% subjects who had viral hepatitis.

Our study was similar to Nguyen DT et al³⁶, Starr SPet al⁶³, Roerecke M et al⁶⁴, Micu SI et al⁶⁵

Study by	Majority (Percentage)
Nguyen DT et al ³⁶	Alcohol Abuse (35.3%)
Starr SPet al ⁶³	Alcohol Abuse (61.2%)
Roerecke M et al ⁶⁴	Alcohol Abuse (88.4%)
Micu SI et al ⁶⁵	Alcohol Abuse (40.9%)
Present study	Alcohol Abuse (55.15 %)

TIME FROM DIAGNOSIS WITH CIRRHOSIS:

Majority of subjects had been diagnosed in 0 to 5 years which was 65.44 % patients followed by 27.94% patients who had first diagnosis, 11.02% subjects who had been diagnosed in 6 to 10 years and 4.41% subjects who had been diagnosed >10 years.

Our study was in similar to Nguyen DT et al³⁶, Smith A et al⁶⁶, Wiegand J et al⁶⁷

Study by	Majority (Years)
Nguyen DT et al ³⁶	5 (62.1%)
Smith A et al ⁶⁶	<5 years (55.7%)
Wiegand J et al ⁶⁷	2-5 (59.4%)
Present study	0 to 5 (65.44 %)

SYMPTOMS:

Majority of subjects had fatigue i.e., 91.17 % subjects followed by 84.55% subjects having anorexia, 69.85% subjects having jaundice, 59.55% subjects having insomnia, 57.35% subjects having leg edema, 47.79% subjects having ascites, 46.42% subjects having stomachache, 42.64% subjects having nervous araneus, 40.44% subjects having palmar

erythema, 25% subjects having perceptual disturbances, 19.85% subjects having loose stools, 16.17% subjects having constipation, 11.02% subjects having nausea, 8.82% subjects having mucosal bleeding, 2.20% subjects having gynecomastia.

Our study was similar to Nguyen DT et al³⁶, Bhandari K et al⁶⁸, Gerber LH et al⁶⁹

Study by	Majority (Percentage)
Nguyen DT et al ³⁶	Fatigue (90.2%)
Bhandari K et al ⁶⁸	Fatigue (75%)
Gerber LH et al ⁶⁹	Fatigue (84.7%)
Present study	Fatigue (91.17%)

BODY TEMPERATURE:

Majority of subjects had temperature of 88.5 F i.e., 90.44 % subjects followed by 6.61% subjects who had temperature of 98.5 to 105 F and finally 5.14% subjects who had temperature >105 F.

Our study was similar to Müller MJ et al⁷⁰, Haddadian Z et al⁷¹

81.4%)
78.4%)
00.44%)
•

BLOOD PRESSURE:

Majority of subjects had blood pressure between 100 to 140 mmHg i.e., 79.41 % subjects followed by 11.02% subjects who had blood pressure >140 mmHg and finally 9.55% subjects who had blood pressure between <100 mmHg.

Our study was similar to Tergast TL et al⁷², Blendis L et al⁷³

Study by	Majority (mmHg)
Tergast TL et al ⁷²	100-140 (68.6%)
Blendis L et al ⁷³	100-120 (71.8%)
Present study	100 to 140 (79.41 %)

PULSE RATE:

Majority of subjects had pulse rate <100 bpm i.e., 69.11% subjects followed by 27.20% subjects who had pulse rate between 100 to 120 bpm and 3.67% subjects who had pulse rate >120 bpm.

Our study was similar to Møller S et al⁷⁴

Study by	Majority (bpm)
Møller S et al ⁷⁴	80-100 (65.9%)
Present study	<100 (69.11)

HEMATOLOGICAL PARAMETERS:

The hemoglobin levels were 9.85 ± 2.13 g/dL, red blood cell count was 3.11 ± 0.53 cells/mm³, hematocrit levels were $32.32\pm6.11\%$, MCV levels were 92.95 ± 11.91 fL, MCH levels were $29.11\pm5,63$ and WBC count was 8500 ± 1214 cells/mm³.

Our study was similar to Deshpande N et al⁷⁵, Behera BP et al⁷⁶

PERIPHERAL BLOOD ANALYSIS:

The platelet count was 1.3 ± 0.8 lakhs/mm³, prothrombin rate was $58.30\pm20.11\%$, INR levels were 1.85 ± 0.13 and APTT was 39.12 ± 9.11 seconds.

Our study was similar to Smith A et al⁶⁶, Blendis L et al⁷³, Smith A et al⁶⁶

LIVER FUNCTION TESTS:

The albumin was 28.13 ± 5.12 g/L, protein was 66.85 ± 9.01 g/L, total bilirubin was $110.12\pm88.45\mu$ mol/L, direct bilirubin was $81.93\pm58.49\mu$ mol/L, SGOT was 128.58 ± 24.15 U/L and SGPT was 75.32 ± 18.96 U/L

Our study was similar to Blendis L et al⁷³, Sharma P et al⁷⁷, Agrawal S et al⁷⁸

MEAN BIOCHEMICAL PARAMETERS:

The glucose levels were 7.85 ± 4.19 mmol/L, urea levels were 5.61 ± 3.33 mmol/L, and creatinine levels were 93.12 ± 6.13 µmol/L.

Our study was similar to Müller MJ et al⁷⁰, Haddadian Z et al⁷¹, Chaudhry A et al⁷⁹

SERUM ELECTROLYTE LEVELS:

The sodium levels were 132.78 ± 5.12 mmol/L, potassium levels were 3.77 ± 0.59 mmol/L, chlorine levels were 96.11 ± 4.32 mmol/L and calcium levels were 2.11 ± 0.11 mmol/L.

Our study was similar to Musso CG et al⁸⁰, Lalama MA et al⁸¹

CHILD-PUGH SCORE:

Majority of subjects belonged to Group C i.e. 47.79% subjects, followed by 43.38% subjects belonging to Group B and 8.82% subjects belonging to group A.

Our study was similar to Rahman M et al³⁷, Tsoris A et al⁸²

Study by	Majority (Percentage)
Rahman M et al ³⁷	Group C (56.4%)
Tsoris A et al ⁸²	Group C (45.2%)
Present study	Group C (47.79%)

"CHILD-PUGH SCORE" VS "MELD_Na SCORE":

Based on "MELD-Na score", majority of scores were in Group C i.e. 28.1 ± 7.9 , followed by 16.8 ± 4.8 score in Group B and 9.1 ± 2.5 score in Group A.

Based on lymphocyte to monocyte ratio, Group A had highest ratio i.e. 2.75 ± 1.5 , followed by Group B i.e. 2.03 ± 1.12 and finally Group C i.e. 1.15 ± 1.01 .

Our study was similar to Rahman M et al³⁷, Prasad R et al³⁸, Kim KM et al⁴⁷

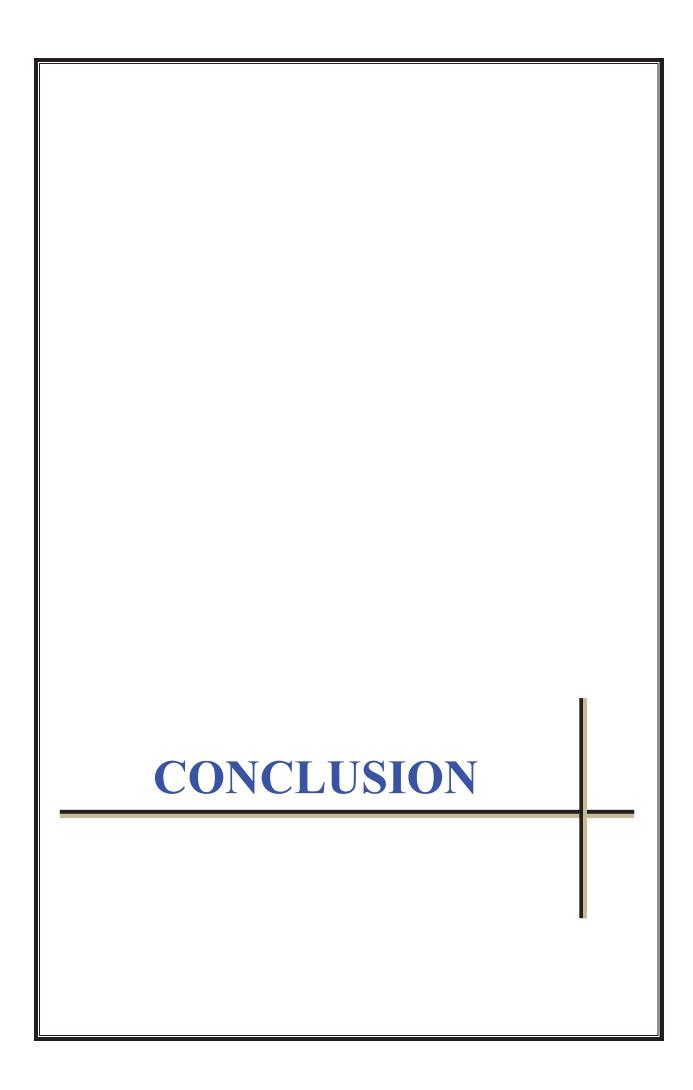
CORRELATION ANALYSIS BETWEEN LMR, MELD-Na AND CP SCORES:

Child-Pugh score had a negative and statistically significant co-relation with lymphocyte to monocyte ratio (r:- **0.05**). That indicates that with an increase in "Child-Pugh score", lymphocyte to monocyte ratio decreases significantly.

Child-Pugh score had a positive and statistically highly significant co-relation with 'MELD-Na score' (r:- <0.001). That indicates that with an increase in the "Child-Pugh score", "MELD-Na score" increases significantly.

'MELD-Na score' had a negative and statistically significant co-relation with lymphocyte to monocyte ratio (r:- 0.03). That indicates that with an increase in the MELD-Na score, lymphocyte to monocyte ratio decreases significantly.

Our study was similar to Rahman M et al³⁷, Prasad R et al³⁸, Kim KM et al⁴⁷

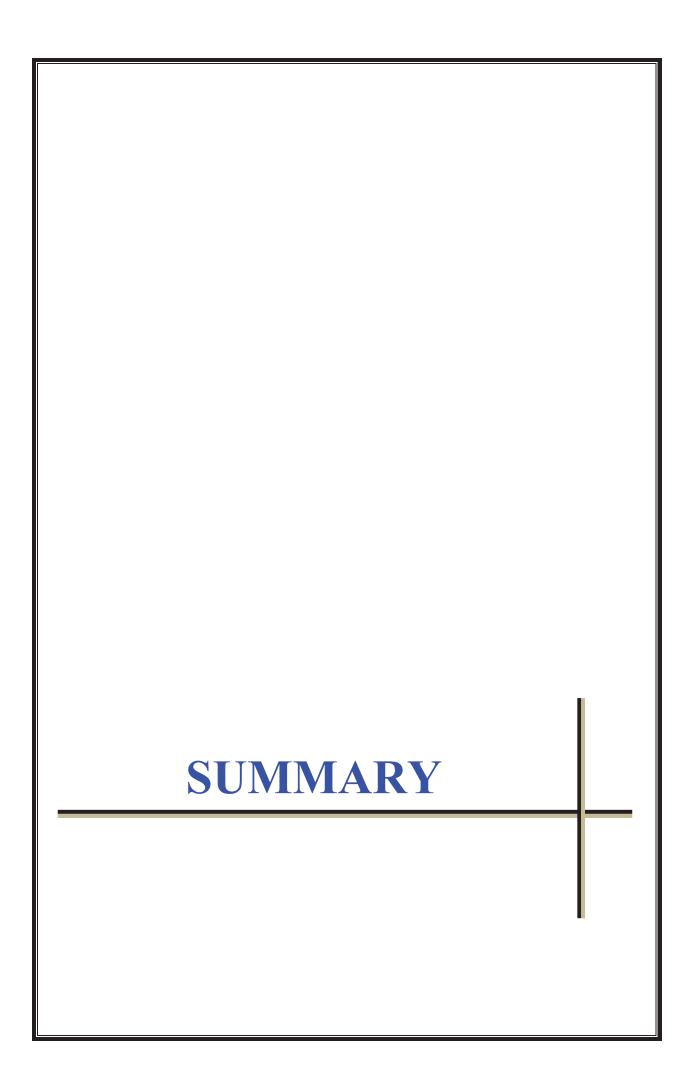


CONCLUSION

The distribution of subjects according to the Child-Pugh classification revealed that the majority of patients were classified as Group C, which indicates advanced liver disease. Similarly, based on MELD-Na scores, a considerable proportion of individuals fell into Group C, indicating a more severe liver dysfunction.

When comparing the lymphocyte/monocyte ratios between groups, Group A had the highest ratio, followed by Group B and lastly Group C. This shows an inverse link between LMR and the severity of liver cirrhosis, with greater ratios indicating less severe disease.

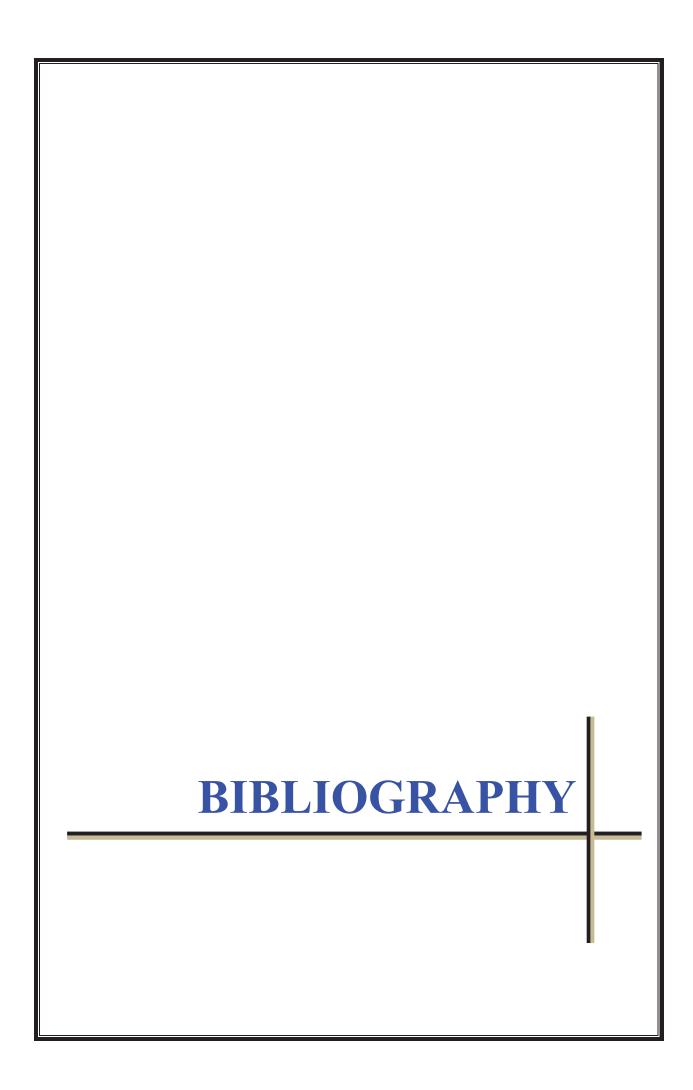
In conclusion, the study findings suggest that LMR could be a good biomarker for measuring liver cirrhosis severity, perhaps complementing or even surpassing established grading systems such as MELD-Na and Child-Pugh. Additional study is needed to validate these findings and investigate the clinical consequences of introducing LMR into standard diagnostic processes for liver cirrhosis patients.



SUMMARY

- Most of subjects were between ages of 41 and 50 years which was 29.41% patients followed by 25.74% patients with age of >60 years, 23.52% patients with age between 51 & 60 years, 13.23% patients with age between 31 & 40 years & lastly 8.09% subjects of age 18 to 30 years.
- Majority of patients were males which was 62.5 % patients followed by 37.5 % female subjects.
- Majority subjects had alcohol abuse i.e., 55.15 % subjects followed by 39.71% subjects who had hepatitis virus and finally 5.14% subjects who had steatosis.
- Majority of subjects had been diagnosed in 0 to 5 years which was 65.44 % patients followed by 27.94% patients who had first diagnosis, 11.02% subjects who had been diagnosed in 6 to 10 years and 4.41% subjects who had been diagnosed >10 years..
- Majority of subjects had fatigue i.e., 91.17 % subjects followed by 84.55% subjects having anorexia, 69.85% subjects having jaundice, 59.55% subjects having insomnia, 57.35% subjects having leg edema, 47.79% subjects having ascites, 46.42% subjects having stomachache, 42.64% subjects having nervous araneus, 40.44% subjects having palmar erythema, 25% subjects having perceptual disturbances, 19.85% subjects having loose stools, 16.17% subjects having constipation, 11.02% subjects having nausea, 8.82% subjects having mucosal bleeding, 2.20% subjects having gynecomastia.
- Majority of subjects had temperature of 88.5 F i.e., 90.44 % subjects followed by 6.61% subjects who had temperature of 98.5 to 105 F and finally 5.14% subjects who had temperature >105 F.

- Majority of subjects had blood pressure between 100 to 140 mmHg i.e., 79.41 % subjects followed by 11.02% subjects who had blood pressure >140 mmHg and finally 9.55% subjects who had blood pressure between <100 mmHg.
- Majority of subjects had pulse rate <100 bpm i.e., 69.11% subjects followed by 27.20% subjects who had pulse rate between 100 to 120 bpm and 3.67% subjects who had pulse rate >120 bpm.
- The hemoglobin levels were 9.85 ± 2.13 g/dL, red blood cell count was 3.11 ± 0.53 cells/mm³, hematocrit levels were $32.32\pm6.11\%$, MCV levels were 92.95 ± 11.91 fL, MCH levels were 29.11 ± 5.63 and WBC count was 8500 ± 1214 cells/mm³.
- The platelet count was 1.3±0.8 lakhs/mm³, prothrombin rate was 58.30±20.11%, INR levels were 1.85±0.13 and APTT was 39.12±9.11 seconds.
- The albumin was 28.13 ± 5.12 g/L, protein was 66.85 ± 9.01 g/L, total bilirubin was 110.12 ± 88.45 µmol/L, direct bilirubin was 81.93 ± 58.49 µmol/L, SGOT was 128.58 ± 24.15 U/L and SGPT was 75.32 ± 18.96 U/L
- The glucose levels were 7.85±4.19 mmol/L, urea levels were 5.61±3.33 mmol/L, and creatinine levels were 93.12±6.13 μmol/L.
- The sodium levels were 132.78±5.12 mmol/L, potassium levels were 3.77±0.59 mmol/L, chlorine levels were 96.11±4.32 mmol/L and calcium levels were 2.11±0.11 mmol/L.
- Majority of subjects belonged to Group C i.e. 47.79% subjects, followed by 43.38% subjects belonging to Group B and 8.82% subjects belonging to group A.
- Based on MELD-Na score, majority of score was in Group C i.e. 28.1 ± 7.9, followed by 16.8 ±4.8 score in Group B and 9.1 ± 2.5 score in Group A.
- Based on lymphocyte to monocyte ratio, Group A had highest ratio i.e. 2.75 ± 1.5 , followed by Group B i.e. 2.03 ± 1.12 and finally Group C i.e. 1.15 ± 1.01 .



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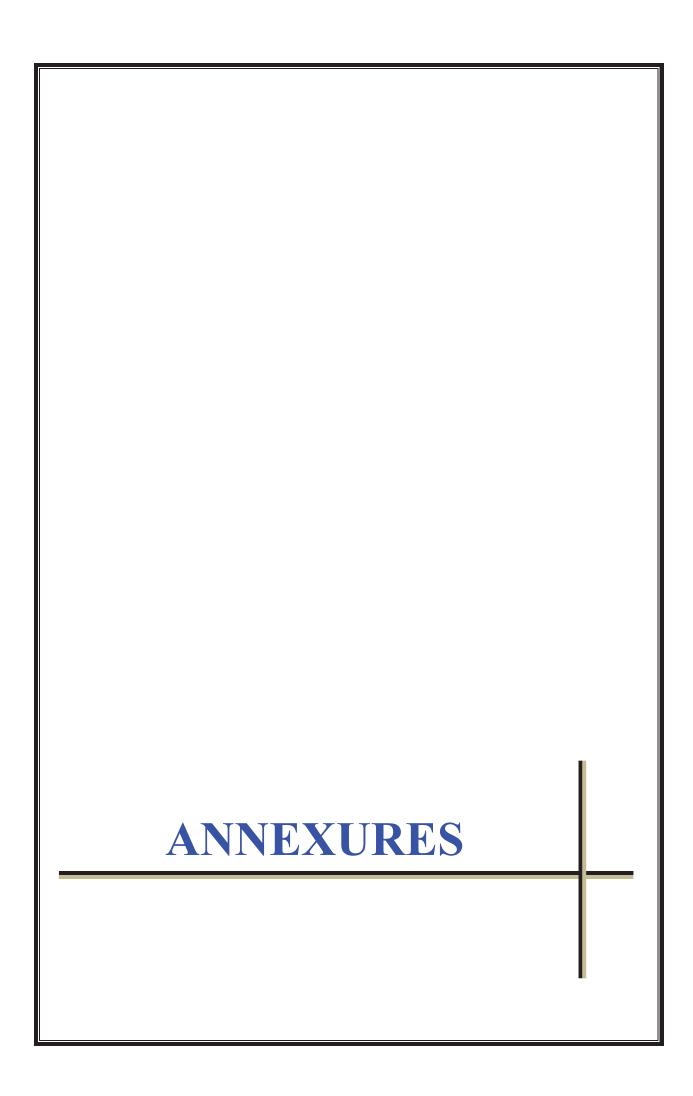
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ANNEXURES

PATIENT INFORMATION SHEET

STUDY TITLE: "EVALUATION OF THYROID FUNCTION TESTS INPATIENTS WITH CHRONIC KIDNEY DISEASE"

GUIDE:DR.B. N. RAGHAVENDRA PRASAD

STUDY CONDUCTED BY: DR. S. A. GAGAN

STUDY LOCATION: R L Jalappa Hospital and Research Centre attached to Sri DevarajUrs Medical College, Tamaka, Kolar

The relation between thyroid dysfunction and the severity of CKD is unclear. The prevalence of hypothyroidism in end-stage renal disease (ESRD) has been estimated between 0 and 9%. There is also an increased prevalence of goitre in patients with ESRD.

In view of the variability of thyroid function tests in CKD patients in previous studies, a study on thyroid function in CKD patients is being undertaken

All Patients diagnosed with chronic kidney disease will be included in this study. Patients in this study will undergo routine investigations RFT, TFT. The principal investigator will bear the expenses of special investigations required for the study .

Your participation in the study will help us to use the outcomes of this study for future subjects and will bring to limelight the importance and potentiate the clinical application ofthyroid function test in the chronic kidney disease patients.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee.

There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.
The purpose of the study is explained in detail to us and all information collected is for study purpose only. The data collected is submitted to the department of General Medicine, SDUMC, Kolar and confidentiality ensured .The merits and demerits have been explained briefly to us

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಮಾರ್ಗದರ್ಶಿ: ಡಾ. ಬಿಎನ್. ರಾಘವೇಂದ್ರ ಪ್ರಸಾದ್

ಸಂಶೋಧಕ:ಡಾ. ಎಸ್.ಎ.ಗಗನ್

ಅಧ್ಯಯನ ಸ್ಥಳ: ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿಗೆ ಲಗತ್ತಿಸಲಾಗಿದಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು, ಟಮಕ, ಕೋಲಾರ ಥೈರಾಯ್ಡ್ ಅಪಸಾಮಾನ್ಯ ಕ್ರಿಯೆ ಮತ್ತು ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯ ತೀವ್ರತೆಯ ನಡುವಿನ ಸಂಬಂಧವು ಅಸ್ಪಷ್ಟವಾಗಿದೆ. ಅಂತಿಮ ಹಂತದ ಮೂತ್ರಪಿಂಡದ ಕಾಯಿಲೆಯಲ್ಲಿ (ಇ ಎಸ್ ಆರ್ ಡಿ) ಹೈಪೋಥೈರಾಯ್ಡಿಸಮ್ನ ಹರಡುವಿಕೆಯು 0 ಮತ್ತು 9% ರ ನಡುವೆ ಅಂದಾಜಿಸಲಾಗಿದೆ.

ಅಂತಿಮ ಹಂತದ ಮೂತ್ರಪಿಂಡದ ಕಾಯಿಲೆಯರೋಗಿಗಳಲ್ಲಿ ಗಾಯಿಟರ್ನ ಹೆಚ್ಚಿದ ಹರಡುವಿಕೆಯೂ ಇದೆ.

ಹಿಂದಿನ ಅಧ್ಯಯನಗಳಲ್ಲಿ ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯರೋಗಿಗಳಲ್ಲಿ ಥೈರಾಯ್ಡ್ ಕ್ರಿಯೆಯ ಪರೀಕ್ಷೆಗಳ ವ್ಯತ್ಯಾಸದ ದೃಷ್ಟಿಯಿಂದ, ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯರೋಗಿಗಳಲ್ಲಿ ಥೈರಾಯ್ಡ್ ಕ್ರಿಯೆಯ ಕುರಿತು ಅಧ್ಯಯನವನ್ನು ಕೈಗೊಳ್ಳಲಾಗುತ್ತಿದೆ ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯಿಂದ ಬಳಲುತ್ತಿರುವ ಎಲ್ಲಾ ರೋಗಿಗಳನ್ನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ರೋಗಿಗಳು ದಿನಚರಿ ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗುತ್ತಾರೆ ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯ ಪರೀಕ್ಷೆ

, ಥೈರಾಯ್ಡ್ ಕಾರ್ಯ ಪರೀಕ್ಷೆ. ಅಧ್ಯಯನಕ್ಕೆ ಅಗತ್ಯವಾದ ವಿಶೇಷ ತನಿಖೆಗಳ ವೆಚ್ಚವನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯು ಭರಿಸುತ್ತಾನೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ಭವಿಷ್ಯದ ವಿಷಯಗಳಿಗೆ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಬಳಸಲು ನಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ ಮತ್ತು ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯ ರೋಗಿಗಳಲ್ಲಿ ಥೈರಾಯ್ಡ್ ಕ್ರಿಯೆಯ ಪರೀಕ್ಷೆಯ ಪ್ರಾಯೋಗಿಕ ಬಳಕೆಗೆ ಪ್ರಾಮುಖ್ಯತೆಯನ್ನು ನೀಡುತ್ತದೆ.

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಅಥವಾನಿಮ್ಮಿಬ್ಬರಿಂದಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ.

ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನಿಮಗೆ ಸಿಗುವ ಚಿಕಿತ್ಸೆನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ನಮಗೆ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯು ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರಬಳಸಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಡೇಟಾವನ್ನು ಜನರಲ್ ಮೆಡಿಸಿನ್, ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು, ಕೋಲಾರ ಇಲಾಖೆಗೆ ಸಲ್ಲಿಸಲಾಗಿದೆ ಮತ್ತು ಗೌಪ್ಯತೆಯನ್ನು ರಕ್ಷಿಸಲಾಗುವುದು. ಅರ್ಹತೆ ಮತ್ತು ದೋಷಗಳನ್ನು ನಮಗೆ ಸಂಕ್ಷಿಪ್ತವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

<u>Title:</u>EVALUATION OF THYROID FUNCTION TESTS INPATIENTS WITH CHRONIC KIDNEY DISEASE

Principal investigator: Dr. S. A. Gagan
I, Mr./Mrs./Miss have been explained in my own understandable language,that I will be included in the above mentioned study ,being conducted in RL JALAPPA HOSPITAL.
I have been explained that my clinical findings, investigations, treatment and prognosis will be assessed and documented for study purpose.
I have been explained my participation in this study is entirely voluntaryand I can withdraw from the study any time and this will not affect my relation with my doctor or treatment for my ailment.
I have been explained about the risk/benefit of the study.
I understand that the medical information produced by this study will become part of institutional records and will be kept confidential by my said institute.
I agree not to restrict the use of any data or result that arise from this study provided such a use is only for scientific purpose(s).

I have principal investigator mobile number for enquiries.

I have been informed that standard of car	re will be maintained throughout the
treatment period.	

I in my sound mind give full consent to be added in the part of this study.

Investigator: Dr. S. A. Gagan

Phone number: 9945548806

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

ಶ್ರೀದೇವರಾಜ್ಅರಸ್ಉನ್ನತಶಿಕ್ಷಣಮತ್ತುಸಂಶೋಧನೆಯಅಕಾಡೆಮಿ, ತಮಕಾ, ಕೋಲಾರ - ೫೬೩೧೦೧<u>.</u>

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

<u>ಶೀರ್ಷಿಕೆ</u>: ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆ ಇರುವ ರೋಗಿಗಳಲ್ಲಿ ಥೈರಾಯ್ಡ್ ಕ್ರಿಯೆಯ ಪರೀಕ್ಷೆಗಳ ಮೌಲ್ಯಮಾಪನ

ಸಂಶೋಧಕ:ಡಾ.ಎಸ್.ಎ.ಗಗನ್

ನಾನು, ಶ್ರೀ/ಶ್ರೀಮತಿ/ಕುಮಾರಿ......ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿನಡೆಸಲಾಗುತ್ತಿರುವ, ಮೇಲೆತಿಳಿಸಿದಅಧ್ಯಯನದಲ್ಲಿನನ್ನನ್ನುಸೇರಿಸಿಕೊಳ್ಳಲಾಗುವುದು ಎಂದುನನ್ನದೇಅರ್ಥವಾಗುವಭಾಷೆಯಲ್ಲಿವಿವರಿಸಲಾಗಿದೆ.

ನನ್ನಭೌತಿಕಸಂಶೋಧನೆಗಳು, ತನಿಖೆಗಳು, ಚಿಕಿತ್ಸೆಮತ್ತುಮುನ್ನರಿವುಗಳನ್ನುಮೌಲ್ಯಮಾಪನಮಾಡಲಾಗುತ್ತದೆಮತ್ತುಅಧ್ಯಯನದಉದ್ದೇಶಕ್ಕಾಗಿ ದಾಖಲಿಸಲಾಗುತ್ತದೆಎಂದುನನಗೆವಿವರಿಸಲಾಗಿದೆ.

ಈಅಧ್ಯಯನದಲ್ಲಿನನ್ನಭಾಗವಹಿಸುವಿಕೆಯುಸಂಪೂರ್ಣವಾಗಿಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆಮತ್ತುನಾನುಯಾ ಪುದೇಸಮಯದಲ್ಲಿಅಧ್ಯಯನದಿಂದಹಿಂದೆಸರಿಯಬಹುದುಮತ್ತುಇದುನನ್ನವೈದ್ಯರೊಂದಿಗಿನನನ್ನಸಂ ಬಂಧಅಥವಾನನ್ನಕಾಯಿಲೆಯಚಿಕಿತ್ಸೆಯಮೇಲೆಪರಿಣಾಮಬೀರುವುದಿಲ್ಲಎಂದುನನಗೆವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದಅಪಾಯ/ಪ್ರಯೋಜನದಬಗ್ಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಈಅಧ್ಯಯನದಿಂದಉತ್ಪತ್ತಿಯಾಗುವವೈದ್ಯಕೀಯಮಾಹಿತಿಯುಸಾಂಸ್ಥಿಕದಾಖಲೆಗಳಭಾಗವಾಗುತ್ತದೆ ಮತ್ತುಮೇಲೆ ತಿಳಿಸಿದಸಂಸ್ಥೆಯುಗೌಪ್ಯತೆಯನ್ನು ಕಾಪಾಡುತ್ತದೆಎಂದುನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಈಅಧ್ಯಯನದಿಂದಉಂಟಾಗುವಯಾವುದೇಡೇಟಾಅಥವಾಫಲಿತಾಂಶದಬಳಕೆಯನ್ನುವೈಜ್ಞಾನಿಕ ಪ್ರಸ್ತಾಪಗಳಿಗಾಗಿನಿರ್ಬಂಧಿಸದಿರಲುನಾನುಸಮ್ಮತಿಸುತ್ತೇನೆ.

ವಿಚಾರಣೆಗಾಗಿನಾನುಪ್ರಧಾನತನಿಖಾಧಿಕಾರಿಯಮೊಬೈಲ್ಸಂಖ್ಯೆಯನ್ನುಹೊಂದಿದ್ದೇನೆ.

ಚಿಕಿತ್ಸೆಯಅವಧಿಯುದ್ದಕ್ಕೂಆರೈಕೆಯಗುಣಮಟ್ಟವನ್ನುನಿರ್ವಹಿಸಲಾಗುವುದುಎಂದುನನಗೆತಿಳಿಸಲಾಗಿ ದೆ.

ಈಅಧ್ಯಯನದಭಾಗದಲ್ಲಿ ಸೇರಿಸಲುನನ್ನ ಉತ್ತಮಮನಸ್ಸಿನಲ್ಲಿ ನಾನುಸಂಪೂರ್ಣಒಪ್ಪಿಗೆಯನ್ನು ನೀಡು ತ್ತೇನೆ.

ತನಿಖಾಧಿಕಾರಿ: ಡಾ.ಎಸ್.ಎ.ಗಗನ್

ದೂರವಾಣಿಸಂಖ್ಯೆ : ೯೯೪೫೫೪೮೮೦೬

ಹೆಸರು	ಸಹಿ	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿ:			
ಸಾಕ್ಷಿ:			
ಪ್ರಾಥಮಿಕತನಿಖಾಧಿಕಾರಿ/ ವೈದ್ಯ:			

MASTER CHART

			-	Time from being			1	ı				I	мсн	WBC	Platelets	Prothrombin	INR	APTT	A Barrente	Destrie	Total	Discot	SGOT S	CDT	1	1	C 4:	Potassium	Chlorina	Calcium Child-	Lymphocyto
S. No			Causes of cirrhosis	diagnosed with	S	Body	Blood	Pulse rate	Hemogl obin	Red blood cell count	Hematocrit	MOV	MCII	WBC	Tiatelets	rate	INK	ALLI	Albumm	Trotein	bilirubin (umol/L	bilirubin	3001	Glucos		Creatinine	Soulum	1 otassium	Cinorine	Pugh MELD	
5. No	Age	Gender	cirrnosis	cirrnosis	Symptoms	temperature							(g)	(cells/mm³)	(lakhs/mm³)			(seconds)	(g/L)	(g/L)	(µmol/L	(µmol/L	(U/L) (U/L)						group Na scor	re ratio
	(years)				Fatigue, Anorexia, Jaundice, Insomnia, Leg	(F)	(mmHg)	(bpm)	(g/dL)	(cells/mm ³)	(%)	(fL)				(%))		(mmol/	L) (mmol/L	(µmol/L)	(mmol/L)	(mmol/L)	(mmol/L)	(mmol/L)	
,	19	Male A	dcohol abuse	6–10	edema, Constipation, Perceptual disturbances	98.5	>140	>120	9.54	3.21	29.6	98.7	32.08	7921	1.6	61.88	1.91	44.2	22.79	67.60	190.46	120 26	128.58	37.63 4.92	5.89	101.56	136.44	4.21	99.15	2.09 Group B 22.1	3.33
					Fatigue, Anorexia, Jaundice, Mucosal																										
2	24	Male H	lepatitis virus	First diagnosis	Bleeding Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	<100	10.2	3.12	25.9	100.6	29.72	9641	0.5	56.42	1.86	42.01	27.68	61.98	145.34	40.29	135.73	55.98 7.89	6.92	104.33	129.66	3.62	93.21	2.19 Group C 15.7	1.45
2	21	Mala H	Iepatitis virus	0 to 5 years	Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema	98.5	100 to 140	<100	11.98	3.38	27.87	98.5	28.69	9482	2.2	74.57	1.92	38.57	21 97	65.40	90.21	110.42	115.42	32.04 11.02	8.23	97.2	134.23	3.36	94.05	2.02 Group B 20.4	3.15
-	21	iviale 11	epanus virus	0 to 5 years	Fatigue, Anorexia, Jaundice, Insomnia,	70.3	100 to 140	<100	11.70	3.36	27.07	70.3	20.09	2402	2.2	74.57	1.92	36.37	31.07	03.49	09.21	119.42	115345	11.02	0.23	91.2	134.23	3.30	74.03	2.02 Gloup B 20.4	3.13
4	29		lepatitis virus	0 to 5 years	Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema	98.5	>140	<100	9.11	3.47	33.92	105.76	30.92	8693	2	69.45	1.97	41.28	28.37	67.32	166.71	78.38		80.24 7.01		99.25	133.11	3.55	97.58	2.01 Group A 15.3	
5	30	Male A	dcohol abuse	0 to 5 years	Stomachache, Ascites, Leg edema Fatigue, Anorexia, Jaundice, Mucosal	98.5	100 to 140	<100	10.73	3	28.21	87.62	28.28	9742	0.9	73.12	1.87	40.9	33.09	71.1	174.89	58.25	120.21	66.57 9.73	7.45	100.44	131.85	4.21	93.76	2.15 Group C 19.7	1.92
	16	Male A	dcohol abuse	First diagnosis	Bleeding, Nausea, Constipation, Perceptual disturbances	98.5	100 - 140	100 to 120	8.29	3.19	35.43	102.14	33.92	9376	1.6	52.87	1.83	33.85	21.21	50.04	124.62	72.01	122.07	94.18 5.36	5.89	95.78	136.44	3.62	00.02	2.04 Group C 14.9	2.56
0	10	Maie A	diconoi abuse	First diagnosis	Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	100 to 120	8.29	3.19	35.43	102.14	33.92	93/6	1.0	32.87	1.83	33.83	31.21	38.94	134.03	/3.01	132.87	4.18 5.30	5.89	95./8	130.44	3.02	98.02	2.04 Group C 14.9	2.30
7	22	Male A	dcohol abuse	0 to 5 years	Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema	98.5	<100	<100	9.04	3.62	31.04	109.83	27.45	7702	0.5	65.02	1.92	45.06	28.95	70.16	45.24	73.52	152.73 8	85.41 9.24	6.92	94.6	129.66	3.98	91.45	2.08 Group B 21.6	2.98
				•	Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus																										
					Palmar erythema, Leg edema,																										
8	28	Male A	dcohol abuse	0 to 5 years	Constipation, Perceptual disturbances Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	100 to 120	11.22	3.78	29.86	100.25	33.82	7990	2.2	47.76	1.81	44.2	32.78	68.77	185.22	72.64	101.29	57.89 12.18	3.67	90.88	130.91	4.03	94.89	2.12 Group C 18.2	3.27
9	18	Female A	deahal abuse	6–10	Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema	98.5	100 to 140	<100	12.77	3.04	27.75	96.38	28.09	10013	1.2	71.6	1.89	42.01	27.68	72.43	238 68	129.87	130.63	11.75 4.92	4 56	98.01	131.27	3.24	98.63	2.1 Group C 17.1	1.45
					Fatigue, Anorexia, Jaundice, Mucosal	98.5				3.68		112.07			1.1		1.02	47.64	31.87	63.29		45 77		00.62 7.89	7.09	92.76	133 59		92.33	2.22 Group A 22.5	
10	21		dcohol abuse	First diagnosis	Bleeding, Nausea Fatigue, Anorexia, Jaundice, Insomnia, Leg	70.5	>120	100 to 120	7.66	5.00	32.78		28.74	9622	**	61.88			01.01	0.5.27	199.08	45.77		- 110	7.07	72.70	133.37	4.26	72.33	2.22 Gloup II 22.5	2.64
11	25	Male A	dcohol abuse	0 to 5 years	edema Fatigue, Anorexia, Gynecomastia, Leg	98.5	100 to 140	<100	11.19	3.21	31.16	83.25	30.24	10060	1.9	56.42	1.95	37.2	25.75	59.14	48.87	109.18	153.84	71.83 10.74	2.79	91.65	129.01	3.19	95.72	2.09 Group B 13.6	1.29
12	34	Male A	dcohol abuse	6–10	edema, Constipation, Perceptual disturbances	98.5	<100	>120	9.51	2.92	37.03	96.51	26.75	10518	1.4	74 57	1.78	46.22	30.04	68.99	189.77	130.61	112.37	53.45 6.05	6.78	96.82	137.12	3.81	95.28	2.19 Group C 18.7	2.7
13	36	Male A	dcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice, Insomnia	98.5	100 to 140	<100	8.91	3.66	29.91	104.19	27.86	10074	0.7	58.9	1.84	41.76	29.11	64.77	180.46	88.87	134.62 8	88.12 6.43	4.32	93.51	130.05	3.6	99.15	2.03 Group C 17.9	
14 15	37 35	Male A Male A	dcohol abuse	First diagnosis 0 to 5 years	Fatigue, Anorexia Fatigue, Anorexia, Jaundice, Insomnia	98.5 98.5	100 to 140 >140	100 to 120 <100	12.06 9.76	3.32 3.61	31.25 26.6	95.11 108.29	31.32 29.58	9112 7921	2.5 2.1	63.79 49.42	1.88	35.01 44.29				139.36 40.29		70.21 11.72 72.36 10.2			135.76 133.95	3.28 4.01	93.21 94.05	2.07 Group B 21.2 2.16 Group A 16.4	
16	39	Male A	dcohol abuse	First diagnosis	Fatigue, Anorexia, Jaundice	98.5	100 to 140	<100	10.64	3.49	29.67	113.5	26.28	9641	0.4	59.23	1.79	42.67	32.09	75.01	82.97	101.63	126.33 8	80.11 11.66 79.23 10.68	6.96	97.2	132.34	3.72	96.97	2.13 Group C 22.1	2.89
17	33	Male A		0 to 5 years	Fatigue, Jaundice Fatigue, Anorexia, Jaundice, Leg edema,	98.5	>140	<100	9.99	3.88	31.05		34.39	9482	1.8	48.15	1.91	35.45	25110						01.10	,,,,,,	130.45			2.05 Group C 15.7	2.02
18 19	40		dcohol abuse	First diagnosis	Constipation, Perceptual disturbances Fatigue, Anorexia, Jaundice, Insomnia	98.5 98.5	100 to 140 100 to 140	100 to 120	10.42	2.58 3.31	36.71	93.09 110.71	31.49	8693 9742	2.6	70.23 66.18	1.86 1.82	38.81 48.33	26.34	67.58	122.53	90.11		37.19 12.2 54.76 9.81	9.02	89.47	134.68 132.32	4.36 3.97	98.25	2.23 Group B 20.1 2 Group C 17.3	2.23 3.33
20	34	Male A	dcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice, Leg edema	98.5	100 to 140	100 to 120	8.72	3.02	31.64	89.57	27.86	9278	0.5	76.77	1.93	38.76	28.92	72.75	121.35	100.46	119.78	32.99 11.17	6.21	98.9	134.79	4.36	92.97	2.06 Group B 23	1.45
21	38	Female A	decohol abuse	0 to 5 years	Fatigue, Jaundice Fatigue, Anorexia, Jaundice, Insomnia, Leg	98.5	100 to 140	>120	11.64	3.86	30.1	113.01	31.47	7899	1.2	64.92	1.98	44.54	32.47	67.11	53.09	111.52	155.02	93.74 8.42	9.89	103.27	132.15	4.12	96.31	2.18 Group C 18.4	1.9
22	36	Male A	dcohol abuse	First diagnosis	edema Fatigue, Anorexia, Jaundice, Insomnia,	98.5	>140	<100	11.36	3.24	30.76	94.4	25.32	10073	1.9	50.65	1.8	31.92	31.23	59.36	60.36	63.42	105.72	6.28 5.53	4.33	89.21	136.21	3.19	94.88	2.14 Group A 16	3.07
23	32		dcohol abuse	0 to 5 years	Constipation, Perceptual disturbances	98.5	100 to 140	<100	9.08	3.78	32.92	91.62	28.68	8093	1.3	54.83	1.76	48.67	32.01	73.68	159.84	123.27		32.55 8.99		92.94	130.52	4.24	99.82	2.17 Group B 19.5	2.44
24 25		Female A Male A		6-10 0 to 5 years	Fatigue, Anorexia, Jaundice Fatigue, Anorexia, Jaundice, Insomnia	98.5 98.5	<100 100 to 140		11.5 11.05		30.45 33.67			9928 8912	2.4 1.6	59.41 61.47	1.79							7.28 57.44 6.75						2.24 Group C 21.8 2.2 Group C 14.5	
25	21			0. 5	jaundice, Gynecomastia, Constipation, Perceptual disturbances	98.5	100 - 140	100 to 120	11.03	3.29	29.9	101.02	31.22	9813	2.1	56.89	1.91	38.54	20.07	60.17	00.70	75.22	141.07	9.13 10.43	6.67	100.05	134.76	4.29	97.67	2.25 Group B 20.9	1.26
27	40	Female A	dcohol abuse		Fatigue, Anorexia, Jaundice, Insomnia	98.5	100 to 140	<100	10.39	3.52	32.74	91.82	28.21	8527	0.6	67.21	1.86	40.67	26.59	67.69	203.23	53.84	110.14	75.68 7.63	8.09	104.11	131.2	3.6	91.87	2.28 Group A 16.9	3.18
28	38	Male A	dcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice Fatigue, Anorexia, Jaundice, Mucosal	98.5	100 to 140	<100	8.52	3.17	29.54	109.65	33.44	8240	1.1	76.09	1.92	42.81	32.32	61.98	218.76	92.21	137.07	94.72 8.37	7.11	92.01	137.46	4.06	97.14	2.26 Group C 22.8	2.75
29	32	Male A	dcohol abuse	>10	Bleeding, Nausea Fatigue, Anorexia, Jaundice, Insomnia,	98.5	>140	<100	9.92	3.74	31.72	87.75	28.41	10244	2.5	52.01	1.84	33.15	29.91	65.49	144.85	73.14	147.61	8.39 8.92	5.21	99.99	134.09	3.99	92.61	2.27 Group B 13.2	3
30	34	Female A	dcohol abuse	First diagnosis	Constipation, Perceptual disturbances	98.5	100 to 140	100 to 120		3.07	28.08	98.29	27.39	8787	1.9	56.68	1.97	42.57	31.28	67.23	101.28	79.36	104.71	8.66 6.77	8.67	91.09	132.01	4.29	94.34	2.21 Group C 18	1.66
31	36	Male A	dechol abuse	6–10	Fatigue, Anorexia, Jaundice Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	<100	11.18	3.32	30.82	105.41	29.65	8359	2.3	49.12	1.88	41.9	32.54	64.31	172.69	56.39	146.11 5	12.15 8.45	7.22	94.44	135.83	3.86	99.23	2.29 Group B 23.2	1.13
					Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema.																										
32	49	Male A	dcohol abuse	0 to 5 years	Constipation, Perceptual disturbances	98.5	100 to 140	<100	10.41	3.06	30.01	92.19	33.32	8961	1	67.65	1.81	36.88	32.18	65.75	198.05	73.29	113.44	6.91 10.95	5.43	102.24	131.95	3.85	95.02	2.31 Group C 15.1	2.78
					Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus,																										
33	50	Female A	dcohol abuse	First diagnosis	Palmar erythema, Leg edema, Constipation, Perceptual disturbances	98.5	>140	<100	8.81	3.27	32.59	106.67	28.98	9246	2.4	66.84	1.83	48.25	27.46	63.43	170.99	98.02	148.66	32.77 8.88	6.98	98.65	133.97	4.23	98.21	2.3 Group A 19.3	1.41
33		- condc A		- n.s. saaguosis	Fatigue, Anorexia, Jaundice, Insomnia,	20.0	- 140	~200	0.01	2.41	240.27	100.07	20.70	/240	2.7	55.04	1.00	-10.23	27.90	Var.98.3	. 10.23	20.02	1-10.00	0.00	0.90	20.00	203.71	Tokal	20.61	2.0 Group A 19.3	2,793
					Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema,																										
34	41	Male A	dcohol abuse	0 to 5 years	Constipation, Perceptual disturbances Fatigue, Anorexia, Jaundice, Insomnia,	> 105	100 to 140	100 to 120	11.03	3.85	28.86	115.36	30.09	8595	1.4	72.41	1.9	30.27	30.45	68.11	103.11	137.92	121.54	57.35 5.28	4.75	95.8	134.44	3.98	93.89	2.33 Group C 21.5	2.91
					Stomachache, Ascites, Nevus araneus,																										
35	42	Male A	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema, Constipation, Perceptual disturbances	98.5	100 to 140	<100	11.61	3.46	32.87	102.86	30.87	10551	0.8	61.76	1.75	47.06	31.36	66.16	172.55	67.49	139.17	54.78 9.67	6.55	90.39	131.71	3.88	95.69	2.37 Group C 17.7	3.2
				-	Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus.																										
	40			0.5	Palmar erythema, Leg edema,	00 =			10.00	201	22.55	00.20	20.00	0444	2-		100	40.00	22.5	70.00	10	00.51	121.55	-0.20		100.00	125.01	2	06.00	224	2.02
36	43	Female A	dcohol abuse	0 to 5 years	Constipation, Perceptual disturbances Fatigue, Anorexia, Jaundice, Insomnia,	98.5	>140	<100	12.03	3.24	33.76	89.38	29.03	8723	2.7	69.05	1.94	43.23	32.17	72.25	171.34	89.21	131.23	59.29 9.37	8.34	102.25	135.04	3.45	96.09	2.34 Group C 22	3.02
					Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema.																										
37	44	Male A	Alcohol abuse	0 to 5 years	Constipation, Perceptual disturbances	98.5	100 to 140	<100	9.27	3.03	35.57	104.51	30.32	8324	2.2	64.96	1.89	44.16	33.28	64.97	147.32	97.68	114.28	70.45 8.77	5.91	93.41	131.39	3.98	97.63	2.32 Group B 14.2	2.29
					Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus,																										
38	45	Male A	dcohol abuse	First diagnosis	Palmar erythema, Leg edema, Constipation, Perceptual disturbances	98.5	100 to 140	100 to 120	10.49	3.2	32.8	107.51	31.23	8925	1.6	57.18	1 99	40.31	30.65	71.36	115.66	134 74	142.65	81 58 8 15	4.22	97 57	134.62	4 19	93.82	2.36 Group C 19	2.51
	_	- 1																												- 1	

					Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema,																												
39	46	Female	Alcohol abuse	0 to 5 years	Constipation, Perceptual disturbances	98.5	>120	<100	11.12	3.77	31.22	87.65	33.35	9441	1.1	68.02	1.77	40.11	31.95	65.03 129.93	7 132.71	117.32	79.65	9.98	i.21	100.24	130.81	4.03	95.17	2.39	Group B	16.6	2.87
					Fatigue, Anorexia, Jaundice, Insomnia,			1100				07100											13100						,,,,,,				
					Stomachache, Ascites, Nevus araneus,																												
40	47	Male		0. 5	Palmar erythema, Leg edema, Constipation, Perceptual disturbances	00.5	100 - 140	<100	8.91	3.1	30.78	95.74	21.76	9904	1.8	75.22	1.93	45.95	20.57	62.58 131.43	02.67	150.5	06.70		.78	02.22	135.62	4.26	97.19	2.25		20.5	1.25
40	4/	Maie	Alcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	<100	8.91	5.1	30.78	95.74	31./0	9904	1.8	15.22	1.95	45.95	30.37	02.58 131.4:	5 83.07	150.5	80.78	0.59	./8	93.22	133.02	4.20	97.19	2.55	Group C	20.5	1.25
					Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema,																												
41	48	Male	Alcohol abuse	0 to 5 years	Constipation, Perceptual disturbances	98.5	100 to 140	<100	9.2	3.31	33.6	106.64	28.18	8960	2.5	67.65	1.85	38.47	32.42	70.88 164.8	138.72	123.71	63.01	7.09	.45	97.77	133.56	4	92.72	2.38	Group B	23.6	1.91
					Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema,																												
42	49	Female	Alcohol abuse	First diagnosis	Constipation, Perceptual disturbances	98.5	>140	100 to 120	12.66	3.38	28.33	91.67	30.65	9543	0.8	66.84	1.8	40.99	28.47	64.42 177.56	68.84	109.85	90.82	0.65	i.01	100.85	129.77	3.18	98.76	2.41	Group C	15.5	3.01
					Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus,																												
43	50	Male	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema, Constipation, Perceptual disturbances	98.5	100 to 140	<100	10.61	3.75	31.11	115 60	28.76	10482	1.3	72.41	1.96	36.07	31.86	72.92 116.43	96.61	144.96	68.49	0.36	.89	98.6	136.31	3.74	92.39	2.42	C D	18.9	2.21
43	30	Maie	Alcohol abuse	0 to 3 years	Fatigue, Anorexia, Jaundice, Insomnia,	90.3	100 to 140	<100	10.01	3.13	31.11	113.06	20.70	10462	1.3	72.41	1.90	30.07	31.00	72.92 110.4:	90.01	144.90	00.49	0.30	.09	96.0	130.31	3.74	92.39	2.42	Споир в	10.9	2.31
					Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema,																												
44	41	Male	Alcohol abuse	0 to 5 years	Constipation, Perceptual disturbances	98.5	<100	<100	11.01	3.65	28.46	107.1	28.82	9271	2	69.45	1.82	36.35	26.55	63.6 149.6	102.87	106.64	92.16	9.33	.01	92.16	131.15	3.34	98.88	2.43	Group C	22.4	1.39
					Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema, Perceptual																												
45	42	Female	Alcohol abuse	6-10	disturbances	98.5	100 to 140	<100	9.35	3.07	34.31	110.47	30.35	8692	1.5	73.12	1.89	38.85	31.36	65.44 144.79	123.26	152.15	77.02	9.65	.22	101.21	134.27	3.62	94.21	2.4	Group B	13.9	3.05
					Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus,																												
46	42	Male	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema, Perceptual disturbances	98.5	100 to 140	<100	10.67	3.56	35.14	93.3	30.04	8979	2.7	52.87	1.77	43.68	28.34	62.82 161.58	3 137.44	119 02	76.23	1 92	.33	99.08	130.38	4.06	97.41	2.44	Group P	10.9	1.78
40	43	Maie	Alcohol abuse	0 to 3 years	Fatigue, Anorexia, Jaundice, Insomnia,	90.3	100 to 140	<100	10.07	3.30	33.14	93.3	30.04	07/7	2.1	32.61	1.//	43.00	20.34	02.82 101.30	5 137.44	110.93	70.23	1.02 .	.33	99.00	130.36	4.00	97.41	2.44	Споир в	19.0	1./0
					Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema, Perceptual																												
47	44	Male	Alcohol abuse	0 to 5 years	disturbances	98.5	100 to 140	100 to 120	10.45	3.42	29.91	109.54	28.89	8144	1.2	65.02	1.91	34.57	29.92	66.54 111.49	134.64	129.42	64.89	8.73	.09	100.92	136.67	3.88	91.78	2.47	Group C	21.9	2.22
					Fatigue, Anorexia, Jaundice, Insomnia, Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema, Perceptual																												
48	45	Female	Alcohol abuse	First diagnosis	disturbances	98.5	100 to 140	<100	11.78	3.16	32.45	91.73	32.22	9519	2.4	47.76	1.78	38.66	31.43	62.26 89.62	84.97	136.5	72.73	2.13	.67	91.78	132.92	4.05	99.41	2.45	Group C	16.7	2.28
					Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus,																												
49	46	Male	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema, Perceptual disturbances	98.5	100 to 140	100 to 120	11.29	3.94	31.73	93.75	27.15	10302	0.9	71.6	1.99	39.56	29.61	72 93 197 0	80.71	111 58	80.56	9.25	.45	94.05	129.32	3.94	95.56	2.46	Group B	23.4	1.19
	40	.viiic	7 Heorioi dodise	o to 5 years	Fatigue, Anorexia, Jaundice, Insomnia,	70.5	100 to 140	100 to 120	11.27	3.74	31.73	75.15	27.10	10302	0.7	71.0	1.//	37.30	27.01	12.93	00.71	111.50	00.50	7.20	.40	74.05	127.02	3.74	75.50	2.40	Citoup D	20.7	1.17
					Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema, Perceptual																												
50	47	Female	Alcohol abuse	0 to 5 years	disturbances Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	<100	8.31	3.63	30.16	114.01	29.61	10055	1.7	61.88	1.86	40.77	30.72	73.2 190.22	89.81	151.88	66.82	1.09	.56	97.31	136.11	4.14	97.25	2.5	Group C	18	2.67
					Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema, Perceptual																												
51	48	Male	Alcohol abuse	6-10	disturbances	> 105	100 to 140	<100	11.57	3.19	32.81	97.22	32.44	8575	2.3	56.42	1.94	42.94	29.73	69.32 84.42	76.83	124.97	69.92	6.96	i.78	94.32	130.28	4.28	96.64	2.49	Group C	23.2	2.81
					Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema, Perceptual																												
52	49	Male	Alcohol abuse	First diagnosis	disturbances	98.5	100 to 140	100 to 120	10.68	3.81	34.11	90.23	30.43	8877	1.6	74.57	1.81	46.67	33.11	66.8 173.24	137.05	107.21	76.57	9.84	.02	91.34	133.42	4.25	94.76	2.48	Group C	15.1	2.21
				, and the second	Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus,																												
			Alcohol abuse		Palmar erythema, Leg edema, Perceptual disturbances	98.5	<100	<100	8.86	3.29	35.25		27.18	8357	2.1	58.9	1.88	39.83	27.61		3 138.71				5.9	94.38	135.99	3.68	98.53			19.3	2.11
5.5	50	Female	Alcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice, Insomnia,	98.5	<100	<100	8.86	3.29	35.25	113.24	27.18	8357	2.1	58.9	1.88	39.83	27.61	60.85 206.63	3 138.71	140.81	73.92	8.05	5.9	94.38	135.99	3.08	98.53	2.52	Group B	19.3	2.11
					Stomachache, Ascites, Nevus araneus,																												
					Palmar erythema, Leg edema, Perceptual		1																										
54	41	Male	Alcohol abuse	0 to 5 years	disturbances	98.5	100 to 140	100 to 120	8.17	3.52	31.14	102.81	28.73	10256	0.7	63.79	1.83	41.81	30.81	71.97 176.8	84.94	119.78	79.64	1.36	.98	95.14	131.6	4.3	95.97	2.51	Group C	21.5	1.97
					Fatigue, Anorexia, Jaundice, Insomnia,																												
					Stomachache, Ascites, Nevus araneus, Palmar erythema, Leg edema, Perceptual																												
55	42	Female	Alcohol abuse	>10	disturbances	98.5	100 to 140	<100	11.31	3.04	31.53	115.02	29.84	9357	1.2	49.42	1.97	43.03	28.45	67.38 183 %	134.27	155.02	87.88	6.46	.56	102.04	136.76	3.42	99.39	2.55	Group B	17.7	2.41
					Fatigue, Anorexia, Jaundice, Insomnia,																							-		-			
					Stomachache, Ascites, Nevus araneus,		1																										
	43	Male		0. 5	Palmar erythema, Leg edema, Perceptual	98.5	100 to 140	<100	11.01	3.39	28.35	93.18	26.48	8701		59.23	1.74	36.82	29.58	67.51 121.15	5 85.63	105.72	66.11	9.61	.21	05.00	132.89	3.99	96.55	2.53	G D	22	
56	43	Male	Alcohol abuse	0 to 5 years	disturbances Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	<100	11.01	3.39	28.35	93.18	26.48	8/01	2	59.23	1.74	36.82	29.58	07.51 121.15	85.63	105.72	00.11	9.01		95.89	152.89	3.99	96.55	2.53	Group B	-22	2.1
					Stomachache, Ascites, Nevus araneus,		1																										
57	44	Male	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema	98.5	100 to 140	<100	8.53	3.8	29.82	111.6	33.12	9085	1.5	48.15	1.92	43.64	31.89	60.77 149.16	135.51	125.26	81.17	9.12	.54	98.7	130.13	4.23	92.16	2.57	Group C	14.2	1.79
					Fatigue, Anorexia, Jaundice, Insomnia,																												
50	45			Fr F	Stomachache, Ascites, Nevus araneus,	00.5	100 - 110	100 . 100	11.00	2.05	22.51	102.00	20.41	0.420	2.4	70.22	1.70	20.26	20.42	62.72	02.74	120.05	02.21			02.64	124.02	4.	07.01	254		10	2.24
58	45	Male	Alcohol abuse	First diagnosis	Palmar erythema, Leg edema Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	100 to 120	11.29	3.05	32.51	102.09	28.41	8420	2.4	70.23	1.79	38.36	28.43	0.5.72 154.35	83.74	158.96	93.21	1.52	.56	93.64	134.92	4.1	97.91	2.54	Group C	19	2.54
					Stomachache, Ascites, Nevus araneus,		1																										
59	46	Female	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema	98.5	100 to 140	<100	9.15	3.14	28.83	95.72	30.76	8454	1.8	66.18	1.85	41.74	32.21	69.92 117.93	83.76	108.77	79.82	7.75	.67	95.01	132.52	3.58	93.14	2.56	Group B	16.6	1.87
					Fatigue, Anorexia, Jaundice, Insomnia,																		П	T	T								
	477	F 1	C+ ·	0 5	Stomachache, Ascites, Nevus araneus,	00.5	100 - 100	100 4 120	11.10	3.49	33.52	113.14	26.61	9226	2.3	76.77	1.98	32.02	22.65	60.16	7 87.53	141.07	71.76	8.81	.45	00.22	136.98	3.49	97.86	2.50	c c	20.5	1.79
60	4/	Female	Steatosis	0 to 5 years	Palmar erythema, Leg edema Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	100 to 120	11.18	3.49	35.52	113.14	26.61	9226	2.3	76.77	1.98	32.02	32.66	69.16 168.57	87.53	141.07	/1./6	8.81	.45	98.22	136.98	3.49	97.86	2.59	Group C	20.5	1.79
					Stomachache, Ascites, Nevus araneus,																												
61	48	Male	Alcohol abuse	6-10	Palmar erythema, Leg edema	98.5	100 to 140	<100	9.78	3.27	32.85	89.67	32.31	9301	1.1	64.92	1.87	35.69	26.92	69.41 194.43	78.29	110.14	88.33	0.02	.21	91.82	129.89	3.4	94.43	2.58	Group B	23.6	3.1
					Fatigue, Anorexia, Jaundice, Insomnia,																		П	T	T								
	49			0. 5	Stomachache, Ascites, Nevus araneus,	00.5	100 - 110	-100	11.22	2.70	21.70	110.25	27.07	0620		50.65	1.73	20.44	22.22	64.05		127.07	70.70	0.02	70	00.12	122.20	2.5	00.25	2.5	G D	15.5	2.62
62	49	Male	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema Fatigue, Anorexia, Jaundice, Insomnia,	98.5	100 to 140	<100	11.32	3.79	31.79	110.25	27.07	9628	1	50.65	1.73	39.44	33.32	0+.05 156.16	5 111.63	137.07	10.39	0.92	./8	98.13	133.38	3.6	98.36	2.6	Group B	13.3	2.62
					Stomachache, Ascites, Nevus araneus,																												
63	50	Female	Alcohol abuse	0 to 5 years	Palmar erythema, Leg edema	98.5	100 to 140	100 to 120	8.51	3.09	32.14	91.8	32.35	10621	2.6	54.83	1.9	41.66	29.05	69.26 174.22	138.42	147.61	74.52	8.31	.43	100.39	135.31	3.42	91.57	2.62	Group C	18.9	1.06

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115	67	Female	Alcohol abuse	First diagnosis	Stomachache, Ascites	98.5	>140	<100	11.81	3.42	30.16	94.1	28.86	9901	2.5	70.23	1.98	40.29	31.11	71.01	161.89	138.57 119	78 66.2	9.02	7.01	92.51	131.14	3.88	91.73	3.13	Group C	19.3	2.58
					Fatigue, Anorexia, Jaundice, Insomnia,																												
116	70	Female	Steatosis	0 to 5 years	Stomachache, Ascites, Leg edema	98.5	100 to 140	<100	9.7	3.85	31.64	108.9	27.42	8566	2.1	66.18	1.83	40.73	29.47	69.49	184.33	90.12 15:	02 90.1	8.49	5.67	96.36	137.16	4.16	97.37	3.15	Group C	21.5	2.07
					Fatigue, Anorexia, Jaundice, Insomnia,																										i I		
117	75	Male	Alcohol abuse	First diagnosis	Stomachache, Ascites, Leg edema	98.5	100 to 140	<100	10.16	3.14	32.57	91.25	27.83	10017	0.4	76.77	1.89	40.11	32.22	63.19	167.22	110.94 10:	72 75.5	11.91	6.89	98.84	134.39	4.27	92.91	3.14	Group A	17.7	2.15
					Fatigue, Anorexia, Jaundice, Insomnia,																										1 1		
					Stomachache, Ascites, Nevus araneus, Leg																										1		
118	80	Female	Alcohol abuse	0 to 5 years	edema	98.5	100 to 140	<100	10.84	3.72	32.24	113.09	28.61	9099	1.8	64.92	1.75	45.82	31.59	63.52	182.69	130.17 12:	26 88.3	8.6	6.67	92.31	132.79	4.27	96.38	3.16	Group A	22	2.93
					Fatigue, Anorexia, Jaundice, Insomnia,																										1 1		
119	63	Male	Alcohol abuse	First diagnosis	Stomachache, Ascites, Leg edema	98.5	<100	<100	11.32	3.29	32.21	94.82	32.12	9732	2.6	52.87	1.94	36.27	29.39		179.58		96 67.4	6.99	7.45	97.66	136.12	3.43	95.09	3.17	Group B	14.2	2.77
120	66	Female	Alcohol abuse	0 to 5 years	Fatigue, Nausea	98.5	100 to 140	100 to 120	10.78	3.63	30.34	110.27	32.86	8993	2.2	65.02	1.81	38.47	28.22	70.13	176.91	82.49 103	77 78.6	9.13	8.21	101.01	130.86	4.33	98.15	3.18	Group C	19	2.71
121	71	Female	Alcohol abuse	First diagnosis	Fatigue, Anorexia, Nausea, Leg edema	98.5	100 to 140	<100	10.91	3.4	32.86	89.9	28.18	10327	0.5	47.76	1.87	40.61	29.87	62.68	207.05	120.07 14	07 80.7	9.2	5.78	92.84	133.7	4.29	93.11	3.2	Group B	16.6	2.36
122	76	Male	Steatosis	0 to 5 years	Fatigue, Anorexia	98.5	100 to 140	<100	10.16	3.04	30.23	106.12	32.72	10524	1.2	71.6	1.73	42.73	31.42	71.47	168.3	82.97 110	14 81.8	11.38	7.43	101.39	135.34	3.57	96.47	3.19	Group A	20.5	1.69
123	79	Female	Alcohol abuse	First diagnosis	Fatigue, Anorexia, Nausea, Leg edema	98.5 to 105	<100	100 to 120	10.98	3.82	32.53	114.65	28.39	10726	1.9	61.88	1.96	39.86	30.76	67.36	136.97	96.46 13	07 85.3	9.3	8.01	93.1	131.91	3.82	97.29	3.21	Group C	23.6	2.72
124	65	Male	Alcohol abuse	0 to 5 years	Fatigue, Anorexia	98.5	100 to 140	<100	8.6	3.55	30.53	90.94	31.16	8628	1.3	56.42	1.8	41.11	29.51	62.44	200.03	95.33 14	61 76.2	7.08	6.98	100	137.01	4.34	91.99	3.22	Group B	15.5	2.94
125	69	Female	Alcohol abuse	6-10	Jaundice, Insomnia	98.5	100 to 140	<100	11.06	3.21	31.22	105.63	32.08	8925	2.4	74.57	1.93	39.8	31.21	70.03	150.78	99.14 10-	71 89.4	11.18	4.56	95.94	136.65	3.62	94.55	3.23	Group C	18.9	1.48
126	73	Male	Alcohol abuse	First diagnosis	Fatigue, Anorexia, Nausea, Leg edema	98.5	>140	<100	8.4	3.67	32.28	111.12	29.72	9797	1.6	58.9	1.77	37.24	29.83	72.88	216.4	135.92 146	11 84.1	5 11.23	6.34	93.12	131.22	4.01	98.93	3.25	Group B	22.4	2.74
127	77	Female	Alcohol abuse	0 to 5 years	Fatigue, Anorexia, Leg edema	98.5	100 to 140	>120	10.8	3.11	34.16	100.25	28.69	9774	2.1	63.79	1.75	41.1	31.35	67.29	115.74	98.81 13	07 69.9	10.47	7.45	99.73	134.59	4.06	91.65	3.24	Group A	13.9	2.57
128	81	Male	Alcohol abuse	0 to 5 years	Fatigue, Jaundice	98.5 to 105	>140	<100	11.33	3.48	30.66	96.38	30.92	9156	0.6	52.87	1.94	37.78	30.92	63.43	198.01	98.29 14	61 79.8	9.37	5.78	101.16	132.11	3.91	96.74	3.26	Group C	19.8	2.69
																															1 1		
					Fatigue, Anorexia, Jaundice, Nevus																										1		
129	62	Female	Alcohol abuse	First diagnosis	araneus	98.5	100 to 140	100 to 120	11.26	3.74	32.53	112.07	31.54	9367	1.1	65.02	1.81	42.46			160.27				9.01	94.52	135.75	3.78	93.92		Group B	21.9	1.32
130	74	Male	Alcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice, Leg edema	98.5	100 to 140		11.02	3.27	34.01	83.25		9601	2.5	47.76	1.87	38.91			125.75				7.45	91.02	131.86	3.68	97.89		Group C	16.7	2.63
131	83	Female	Steatosis	>10	Fatigue, Anorexia, Jaundice, Leg edema	98.5	100 to 140	<100	10.23	3.89	33.16	96.51	31.94	10324	1.9	71.6	1.73	42.3	29.85	64.88	184.71	112.97 11	58 71.8	7.84	8	97.16	133.92	4.36	92.04	3.29	Group B	23.4	2.19
132	85	Male	Alcohol abuse	0 to 5 years	Fatigue, Anorexia, Jaundice	98.5	100 to 140	<100	11.39	3.4	31.14	104.19	28.29	10584	2.3	61.88	1.96	41.55	28.84	63.32	154.6	106.36 15	88 79.8	10.77	5.32	102.12	136.43	3.77	99.18	3.3	Group A	18	2.37
133	89	Female	Alcohol abuse	First diagnosis	Jaundice, Nausea	98.5 to 105	>140	100 to 120	8.9	3.04	30.46	95.11	31.52	9714	1.9	56.42	1.8	40.69	32.42	64.77	173.79	100.43	97 72.9	8.75	7.76	93.83	130.97	4.29	94.34	3.31	Group B	21.9	1.88
134	91	Male	Alcohol abuse	0 to 5 years	Fatigue, Leg edema	98.5	100 to 140	>120	11.48	3.82	30.63	108.29	28.74	8694	1.3	74.57	1.93	41.25	28.35	62.11	207.8	115.12 10	21 89.1	10.75	8.12	91.93	134.73	4.19	98.41	3.32	Group B	16.7	1.93
135	94	Female	Alcohol abuse	6-10	Fatigue	98.5	100 to 140	<100	9.29	3.55	34.02	113.5	32.79	9967	2.4	58.9	1.77	42.64	29.94	65.21	200.64	133.32 116	99 71.8	11.56	6.45	97.47	130.86	4.07	95.28	3.33	Group B	23.4	1.09
136	96	Male	Alcohol abuse	First diagnosis	Fatigue, Nausea, Leg edema	98.5	>140	100 to 120	9.79	3.21	31.19	105.63	31.25	8387	1.6	63.79	1.76	40.81	32.28	70.97	203.68	105.15 120	21 81.2	8.89	4.79	92.44	133.7	3.94	99.09	3.34	Group C	18	1.4
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