"EVALUATION OF HEMATOLOGICAL AND BIOCHEMICAL PARAMETERS IN CHRONIC LIVER DISEASE PATIENTS"

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REG NO: BN0121012

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This is to certify that the dissertation **entitled "EVALUATION OF HEMATOLOGICAL AND BIOCHEMICAL PARAMETERS IN CHRONIC LIVER DISEASE PATIENTS"** is a bonafide research work done by the candidate**, Registration number: BN0121012.**

DR. Vijayalaxmi Dhorigol
Professor and HOD
Department of Pathology,
J. N. Medical College,
Belagavi, Karnataka

Professor & Head
Department of Pathology
J.N. Medical College,
BELAGAVI.

Date: 28/6/24
Place: Belagavi.

DR.(Mrs) N. S. Mahantashetti MD (Paed).
Principal
J. N. Medical College,
Belagavi, Karnataka.

PRINCIPAL
J.N. Medical College
BELAGAVI- 590 010

KAHER
BELAGAVI
J.N. MEDICAL COLLEGE

Date: 28/06/2024
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Accredited 'A+' Grade by NAAC (3rd Cycle) Placed in Category 'A' by MoE (GoI)
Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA
0831 - 2471350 0831 - 2470759 www.jnmc.edu rincipal@jnmc.edu

Ref No: MDC/PG/ Date: 25-06-2024

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Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BN0121012
Postgraduate Student,
2021-22 Batch,
Department of Pathology
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)
JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/134 **Date: 27/09/2022**

To.

REG NO: BN0121012
PG Student in Pathology,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

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(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS:

A: G	-	Albumin: Globin ratio
ALB	-	Albumin
ALP	-	Alkaline phosphatase
BIL	-	Bilirubin
BUN	-	Blood urea nitrogen
CLD	-	Chronic liver disease
CREAT	-	Creatinine
F	-	Female
GGT	-	Gamma glutamyl transferase
Hb	-	Hemoglobin
K	-	Potassium
M	-	Male
MCH	-	Mean corpuscular haemoglobin
MCHC	-	Mean corpuscular haemoglobin
MCV	-	Mean corpuscular volume
Na	-	Sodium
NAFLD	-	Non-alcoholic fatty liver disease
NT	-	5 nucleotidase
PLT	-	Platelet count
PT	-	Prothrombin time

RBC	-	Red cell count
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic pyruvic transaminase
T PROT	-	Total protein
WBC	-	White blood cell

ABSTRACT

Background and Objectives:

Chronic liver disease is a progressive deterioration of the normal liver functions. The proper evaluation of hematological and biochemical parameters becomes important for evaluating disease severity and to provide further treatment. Our aim is to evaluate hematological and biochemical parameters in Chronic liver disease patients and to correlate the changes with severity of disease.

Methods:

A prospective cross-sectional study was conducted evaluating all the Hematological and biochemical parameters in 90 Chronic Liver disease patients admitted for evaluation in KLE'S DR. PRABHAKAR KORE HOSPITAL within the study period of one year in the department of Pathology and Biochemistry. Blood samples were collected after taking all aseptic precautions from the chronic liver disease patients in EDTA, citrate and plain vacutainer and then analysed for Hematological and biochemical values.

Results:

In our study the most common age group affected was 41-50 years with male predominance (85%). The common etiology among all chronic liver disease was found to be alcoholic liver disease (44%), chronic hepatitis (32%), non-alcoholic fatty liver disease (17%) and 7% liver failure. In hematological investigations anemia (91%) with predominantly being moderate type was common. Normocytic normochromic RBC morphology was seen in 42% cases. Total WBC count was

normal in 72% cases, thrombocytopenia (70%) predominantly being moderate type. Prothrombin time was elevated in 80% cases.

Among biochemical markers SGPT was normal in 83% of cases. SGOT is mildly raised (51%). ALP (56%) and GGT (64%) were normal. There was mild increase in total bilirubin (79%). There was hypoproteinaemia in 60% and hypoalbuminemia in 86% with decreased A/G ratio. Creatinine and BUN were raised in 60%. Most of the cases had normal sodium and potassium levels. Few cases showed hyponatremia (32%) and hypokalaemia (30%).

Conclusion:

The above findings are useful for in patient management as early detection of hematological and biochemical parameters would aid to know disease progression and severity and hence treatment can be installed at the earliest which shall improve the prognosis in chronic liver disease.

Keywords:

Chronic liver disease; anemia; prothrombin; SGPT; SGOT; ALP; GGT; bilirubin; creatinine; BUN.

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INTRODUCTION

One of the major causes of morbidity and mortality is Chronic liver disease or CLD. It is 11th leading cause of death globally and 10th in India. Nearly 2 million deaths occur worldwide due to chronic liver disease and its related complications. India contributes 18% of all the deaths due to chronic liver disease in the world. Two thirds of all the mortality are among the males mainly in the age group of 45 to 61 years.[1,2] Out of all the causes Viral hepatitis is the leading cause followed by alcoholic liver disease and non-alcoholic fatty liver disease associated with obesity, hyperlipidaemia, and diabetes mellitus, Chronic Viral Hepatitis- B, C, and D, Alpha-1 antitrypsin deficiency, Hereditary hemochromatosis, Wilson disease, Autoimmune hepatitis (AIH). Other causes include Drug induced, Budd-Chiari syndrome, right sided heart failure, veno-occlusive disease and Idiopathic/cryptogenic.[4] Alcohol abuse is rapidly increasing causing alcoholic liver disease which is showing trends to replace Hepatitis as the predominant cause of CLD. Chronic liver disease causes in children include Metabolic liver disease, Hepatitis, Autoimmune hepatitis, Neonatal hepatitis, Tuberculosis, TORCH hepatitis, Extrahepatic biliary obstruction and miscellaneous.[3] Chronic liver disease is progressive deterioration of liver functions for more than 6 months. It is a continuous process of inflammation, destruction, regeneration of liver parenchyma. Cirrhosis is the final stage of CLD. Compensated liver cirrhosis patients have cirrhosis without any clinical symptoms or complications. Decompensated liver cirrhosis patients have cirrhosis related complications like ascites, variceal bleeding, hepatic encephalopathy or non-obstructive jaundice.[4]

The proper evaluation of hematological and biochemical parameters becomes important for evaluating disease severity and to provide further treatment. The early detection of hematological and biochemical derangements would avoid disease complications and enhance patient quality of life with better survival rates in chronic liver disease patients.

OBJECTIVES OF THE STUDY

1) To evaluate Haematological and Biochemical parameters in Chronic liver disease patients.
2) To correlate the changes with severity of disease.

REVIEW OF LITERATURE

Liver is a critical organ of the body. It comprises 2% of the total body weight and is responsible for proteins synthesis and secretion, maintenance of glucose levels, storage of bile and vitamins, dietary compounds metabolism, bilirubin metabolism, detoxification, exocrine and endocrine functions. Liver has 2 lobes situated in right upper quadrant of abdomen. Liver comprises of hepatocytes and biliary epithelial cells (or cholangiocytes). Both derived from endoderm. Mesoderm derivatives are Kupffer cells, Stromal cells and blood vessels.[5,6] Liver has dual blood supply from portal vein (75%) and hepatic artery (25%). From the beginning of the fetal life and further into postnatal life it plays major role in maintenance of hematological parameters thus maintaining blood homeostasis.[7]

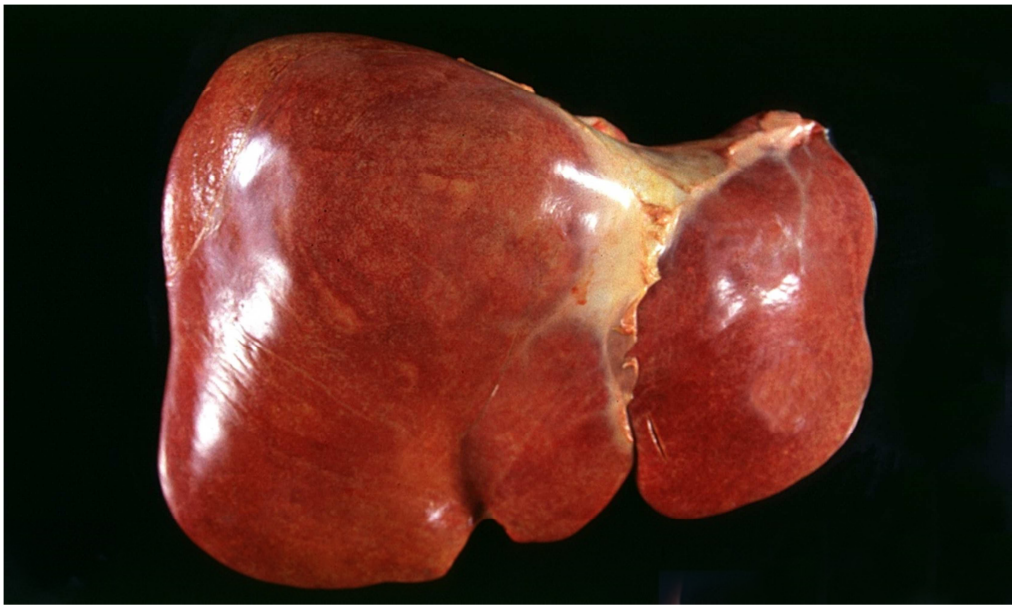


Fig. (1) Liver gross anatomy[8]

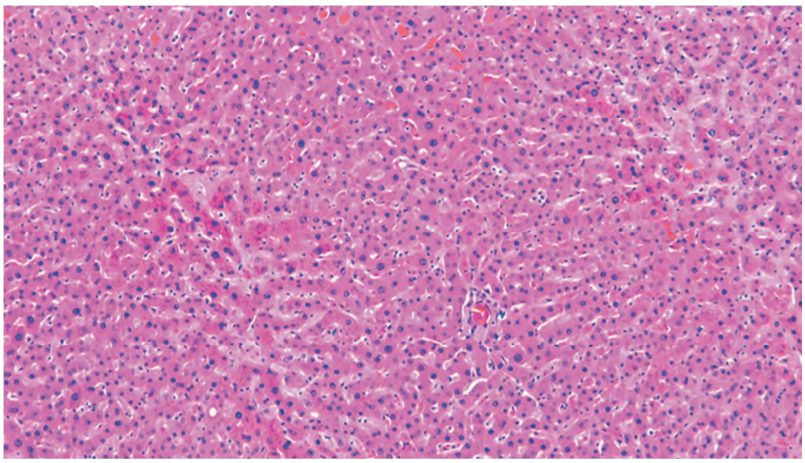


Fig. (2) Liver normal parenchyma, H&E (10x)[9]

On the basis of perfusion, the liver architecture is divided into 3 zones. Zone 1- Periportal- oxygen rich and first to regenerate and is mostly responsible for oxidative metabolisms, gluconeogenesis, bile formation, cholesterol formation and amino acid catabolism. Zone2-pericentral is between 1 and 3. Zone 3- farthest from the portal triad and receives lowest perfusion. Mostly responsible for detoxification, biotransformation of drugs, ketogenesis, glycolysis, lipogenesis, glycogen synthesis, and glutamine formation. The hepatocytes are arranged in single cell sheets known as hepatic plates, separated by sinusoidal spaces connected to basement membrane of hepatocyte. Metabolites and toxins get absorbed through these spaces[10]. Bile is secreted from the apical surface of hepatocytes into the bile canaliculi and then flows through the intra hepatic bile duct to extrahepatic bile ducts and into the gall is bladder where it is stored. The space of Disse is between the sinusoidal lumen and the basolateral membrane of hepatocytes. It contains Kupffer cells which are a form of macrophages and Ito cells or stellate cells. The Kupffer cells sit in the space to filter out pathologic material from the circulation. The Ito cells serve as storage for fat and vitamin A.[10,11]

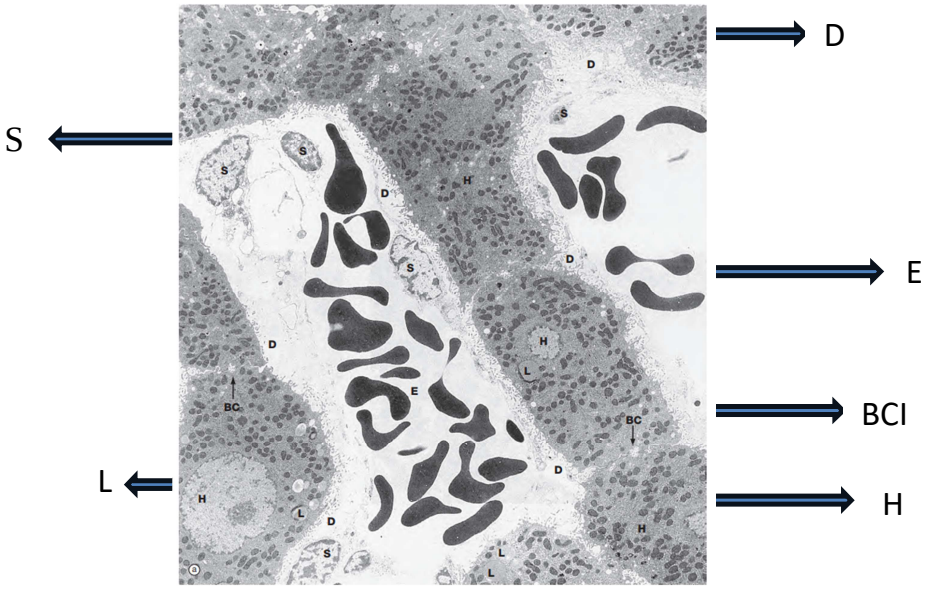
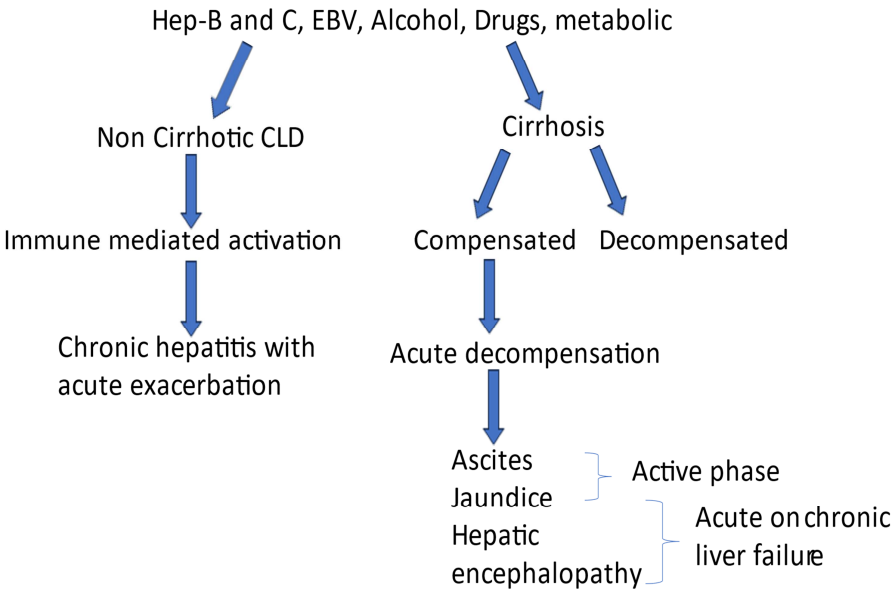


Fig. (3) Liver Electron Microscopy, 4400x[9]

BC- bile canaliculus, D-space of Disse, E- erythrocytes, H-hepatocyte, L-lipid droplet, reticulum, S-sinusoid lining.

PATHOPHYSIOLOGY OF CHRONIC LIVER DISEASE

In response to chronic inflammation stellate cells get activated into proliferative myofibroblasts and increase upregulation of inflammatory receptor expression leading to accumulation of extracellular matrix and fibrosis. Fibrosis can be Perivenular, Periportal, Bridging or Cirrhosis.[11,12,13]



Stages of Chronic liver disease[14,15]

1. Hepatitis or steatosis or hepatosteatosis
2. Fibrosis
3. Cirrhosis
4. Liver failure

Histological staging[16,17]

0- No fibrosis

1- Zone 3 Perivenular fibrosis

2- Perivenular and Periportal fibrosis

3- Bridging fibrosis

4- Cirrhosis

Table no 1: The Child Pugh score of cirrhosis[17,18]

Parameters	Points assign		
	1	2	3
Ascites	Absent	Mild	Moderate
Bilirubin	<2	2-3	>3
Albumin	>3.5	2.8-3.5	<2.8
Prothrombin time/INR	<4/<1.7	4-6/1.7-2.3	>6/>2.3
Encephalopathy	None	Grade 1 or 2	Grade 3 or 4

Class A: 5-6 Class B: 7-9 Class C: 10-15

Haematological Parameters

1) Haemoglobin concentration (Hb)

Hemoglobin is generally decreased. In compensated chronic liver disease Hb is mild to moderately decreased whereas decompensated type there is moderate to severe decrease in haemoglobin.[18,19]

2) Red blood cell (RBC)

RBC counts are mostly normal to low. Macrocytosis is found in chronic liver disease associated with alcohol, vitamin B 12 and folic acid deficiency. Microcytes are found in iron deficiency state of body associated with malnutrition, intestinal malabsorption and acute blood loss in chronic liver disease patients.[18,19]

3) White blood cells (WBC)

WBC counts are normal to increase in chronic liver disease patients. Leucocytosis is found in hepatitis in association with secondary infection like urinary tract infection and spontaneous bacterial peritonitis.[19] Counts can be low i.e. leukopenia in cases associated with of marrow aplasia, malnutrition, hypersplenism and chronic infections.[20,21]

4) Platelet count (PLT)

Counts are normal to low. Increase number of immature platelets and larger mean platelet volume both of which reflects liver dysfunction. Thrombopoietin (TPO) plays an important role in thrombopoiesis in platelet maturation and its subsequent release in circulation. Chronic liver disease decreases TPO production and result in thrombocytopenia. Thrombocytopenia in majority cases of chronic liver disease patients associated with acute blood loss, hypersplenism, hepatitis, cirrhosis and hepatocellular carcinoma and Splenic platelet sequestration.[19]

Thrombocytopenia may be Mild-100000-149000 cells/mm3), Moderate-50000-99000 cells/mm3) and Severe (<50000cells/mm3).[21,22]

5) Prothrombin time (PT)- (normal value 11-13 seconds)

Prothrombin time measures prothrombin to thrombin conversion. Vitamin K is required for synthesis of coagulation factors II, V, VII and X. Synthesis of these factors is deficient in hepatocellular disease. PT measures II, VII, X and is prolonged in chronic liver disease mainly in cirrhotic and failure stage.[22] Elevated levels of thrombin antithrombin complexes is found in chronic active hepatitis, decompensated liver disease, end stage liver disease and fulminant liver failure. To differentiate

between a prolonged PT due to hepatocellular disease from that due to cholestasis with fat malabsorption, PT is repeated after vitamin K administration. Reduction occurs in cholestatic liver disease and not in hepatocellular disease. Prolonged PT generally occurs in advanced liver disease.[23,24]

Biochemical parameters

1) Serum bilirubin-

Normal total bilirubin level is 0.3 to 1.2 mg/dl. Direct/conjugated bilirubin measures 0-0.3 mg/dl. Indirect/unconjugated bilirubin measures 0.2-0.8 mg/dl. It is a marker for liver dysfunction. Non-specific and non-sensitive.[26] Normally Conjugated bilirubin is 10% and unconjugated is 90% of the total Bilirubin. Conjugated bilirubin is composed of glucuronide, bilirubin diglucuronide and delta bilirubin (albumin bound to bilirubin) which is water soluble. In cholestasis and hepatocellular injury delta bilirubin is increased. In post hepatic type of jaundice conjugated bilirubin predominates (>50% of the total). In hepatocellular jaundice conjugated bilirubin is 20%-40%. Unconjugated bilirubin predominates in hemolysis. Persistent bilirubin elevation can be seen in cirrhosis and reflects severe liver damage.[27,28]

2) Serum aminotransaminases- SGOT and SGPT

Normal values: SGPT(ALT) -7 to 55 U/L, SGOT(AST)- 8-48 U/L.

SGPT and SGOT are markers of hepatocellular injury. AST is cytosolic and mitochondrial enzyme. It is found in liver, cardiac muscle, skeletal muscle, kidney, brain, pancreas, lungs, leucocytes, red cells.[26]

ALT is a cytosolic enzyme. Marked elevations in AST and ALT (> 15 times) is seen in viral hepatitis, toxin induced hepatic injury, acute biliary tract obstruction.[26,27]

Moderate elevations (5-15 times) occur in chronic hepatitis, autoimmune hepatitis, alcoholic hepatitis, acute biliary obstruction and drug induced hepatitis.

Mild elevations (1-3 times) seen in cirrhosis.[28]

These enzymes can be used to differentiate hepatocellular and cholestatic jaundice. Normal AST/ALT ratio is 0.7 to 1.4. Increased ratio (>2) is highly suggestive of alcoholic hepatitis, while ratio < 1.0 is seen in acute viral hepatitis. Increased transaminases beyond 6months indicate chronicity in case of hepatitis.[29]

3) **Serum alkaline phosphatase (ALP) -**Normal values- 47-127 U/L

ALP is distributed in many tissues like liver, bones, intestine, kidney and placenta. ALP is higher in osteoblastic activity. In liver it is located along the canalicular surface of hepatocyte. It is a Non-specific marker. Simultaneous measurement of serum GGT and serum 5NT is used to ascertain hepatic origin of ALP increase. Diseases that affect mainly hepatocyte secretion have elevated levels of ALP. It is raised in chronic liver disease. Cholestasis and hepatic injury can be differentiated using ALP levels.[33]

4) **Serum gamma glutamyl transferase (GGT)-** Normal values-8 to 61 U/L

GGT is found in liver, pancreas, kidney and prostate. Specific parameter to detect liver injury is GGT. Increased enzyme activity is seen in alcoholics. It is useful for follow up of patients with alcoholic hepatitis. Elevation of both GGT and ALP

indicates chronic liver disease. The serum gamma glutamyl transferase level is elevated more consistently along with the alanine transaminase level in anicteric alcoholic chronic liver diseases.[3]

5) **Total Proteins -**Normal value-6-8.3 gm/dl

Test for proteins in liver disease include total Serum proteins, serum albumin and albumin/globulin ratio (normal >1.5) to measure synthetic activity of liver.

Total serum protein level is affected by both albumin and gamma globulins. In cirrhosis decrease in level of albumin is compensated by increase in levels of gamma globulins.[33]

6) **Albumin -**Normal value- 3.5-5.5 gm/dl

Albumin is synthesised only in liver and constitutes 60% of total proteins. Serum albumin level is low in chronic liver disease and correlates with synthetic activity of liver and therefore helpful in following progression of cirrhosis and chronic hepatitis. Serum albumin is sensitive marker but non-specific for liver disease[28].

In cirrhosis and chronic active hepatitis serum globulins are increased due to inflammation which causes reversal of A/G ratio.[32]

7) **Serum Electrolytes**

Electrolyte imbalance is associated with severity, complications and outcome of chronic liver disease.

Both hypokalaemia and hyperkalaemia can occur in chronic liver disease but mostly normal values are observed. Hyponatremia is due to hyperaldosteronism.

Hypokalaemia is due to low dietary intake, GI losses and diuresis. Hyperkalaemia can be seen in severe cases of CLD along with hypoalbuminemia and increased levels of creatinine associated with kidney dysfunction.[37] More than 55% of mortality in chronic liver disease is associated with hyponatremia and 15% hypokalaemia.[38]

8) **Serum Creatinine -** Normal values 0.6-1.3 mg/dl

Creatinine production in liver undergoes using three amino acids glycine, methionine and arginine. Creatinine is the end product of creatine metabolism in the kidney. Abnormal serum creatinine in CLD is non-specific but can be further evaluated to rule out acute kidney injury, chronic renal injury or hepatorenal syndrome. Serum creatinine is useful for prognostication and liver transplant prioritization in liver disease patients.[38]

9) **BUN -**Normal values-8-20 mg/dl

Normal values-8-26 mg/dl. Urea is the end product of protein metabolism and a nitrogenous waste product. Low levels of urea indicate poor liver function. Elevated BUN levels reflect impaired function of kidney. Urea synthesis is impaired in hepatic fibrosis therefore CLD patients with low urea levels have higher risk of disease progression.[39,41]

The other investigations which can be done in chronic liver disease are

Bone marrow studies:

Bone marrow findings correlate in majority of cases. The bone marrow shows normal or increased cellularity, with normal or increased erythrocytogenesis, megakaryocytogenesis independent of blood loss. Peripheral cytopenia mostly

anaemia and thrombocytopenia with normal marrow findings indicate hypersplenism in hepatic cirrhosis.[40]

In cases of chronic haemorrhage blood and bone marrow pictures are suggestive of iron deficiency anemias. Most of the patients present with macrocytic/ Dimorphic blood picture with Megaloblastic bone marrow. Few cases of Aplastic anemia also have been reported.[41]

Imaging -

Ultrasonography, CT, MRI, Transient elastography (fibro scan)

Ultrasonography- It is non-invasive, affordable screening method for fatty liver, cirrhosis, hepatocellular carcinoma. Increased echogenicity and nodularity indicate fatty liver and Cirrhosis. Duplex doppler ultrasonography can be done to assess patency of hepatic, portal and mesenteric veins.[39]

CT and MRI- Over all 65- 95% sensitivity for liver disease. In case of cirrhosis the imaging shows variation in size, surface nodularity, generalized heterogenicity and attenuation of hepatic vasculature. CT is more sensitive than MRI.[38,39] MRI is useful for detection of the disease and not for staging. Magnetic resonance elastography (MRE) is emerging tool to detect cirrhosis which uses the concept of liver parenchyma stiffness which advances with fibrosis and the findings are not hampered by ascites, steatosis or obesity.[48]

Liver biopsy- gold standard for diagnosis of cirrhosis. Also to assess degree of inflammation (grade) and fibrosis (stage). Presence of fibrosis and nodules indicates cirrhosis which can be micronodular, macronodular and mixed[46,47].

Fibro scan- non-invasive, measures liver stiffness corresponding to fibrosis. Colloid liver spleen scan using tech-99m sulphur colloid show increase uptake in spleen and bone marrow.[49]

Esophagogastroduodenoscopy show varices in oesophagus or stomach suggest portal hypertension.[50]

MATERIALS AND METHODS

A prospective cross-sectional study was conducted evaluating all the Hematological and biochemical parameters on 90 Chronic Liver disease patients admitted for evaluation in KLE'S DR. PRABHAKAR KORE HOSPITAL within the study period of one year in the department of Pathology and Biochemistry.

Inclusion Criteria: All the adults admitted under evaluation for Chronic liver disease in KLEs Dr Prabhakar Kore Hospital, Belgaum with signs and symptoms persisting for more than 6 months, alcoholic liver disease, non-alcoholic fatty liver disease, chronic hepatitis and other metabolic cause of cirrhosis included.

Exclusion Criteria: Patients with Chronic Liver disease, aged less than 18years, acute liver disease, pregnant females, known case of coagulation disorder, malignancy or Septicaemia.

Blood samples were collected after taking all aseptic precautions from the chronic liver disease patients under in EDTA, citrate and plain vacutainer and then analysed for Hematological and biochemical values. The investigations performed were-

HEMTOLOGICAL MARKERS

- Hemoglobin concentration
- Red cell count
- Mean corpuscular volume (MCV)
- Mean concentration of haemoglobin (MCH)
- Mean corpuscular haemoglobin concentration (MCHC)

- white blood cell count (WBC)
- Platelet count (PLT)

BIOCHEMICAL MARKERS

- SGOT
- SGPT
- ALP
- Total bilirubin
- Total Proteins
- Albumin
- A:G ratio
- GGT

COAGULATION TESTS

- Prothrombin time (PT)

ELECTROLYTES

- Sodium
- Potassium

OTHERS - Serum creatinine and BUN.

1) **Automated hematology analyzer** - The Sysmex XN-1500 automated hematology analyzer uses the fluorescence flowcytometry hydro dynamic focusing method (DC detection) for analyzing the RBCs, WBCs and platelet counts. The hydro dynamic focusing method improves the blood count

accuracy and reproducibility. As the blood cells pass through the aperture in a line, it also prevents the generation of abnormal blood cell pulses.

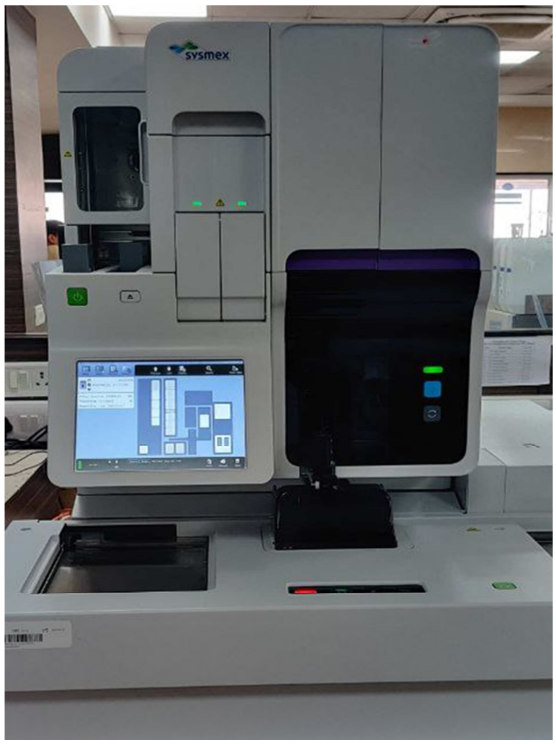


Fig (4) Sysmex XN-1500 automated hematology analyzer

2) **For Biochemical and electrolytes parameters-** semi-automatic or fully automated analysers, which are based on the principle of photometry. Photometry is the measurement of light absorbed in the ultraviolet (UV) to visible (VIS) to infrared (IR) range. It works on principle of **Beer–Lambert's law** which calculates coefficients obtained from the transmittance measurement. Accurate measurements are achieved by a test specific calibration which correlates absorbance and analyte concentration

Fig (5) Biochemical Analyzer

Fig (6) Electrolyte Analyzer

Data processing and analysis/statistical analysis: Testing of hematological markers, liver function and liver injury markers were done followed by statistical analysis to obtain the distribution of these parameters in different groups. All the data were compiled in MS Excel and analyzed using commercially available statistical software (SPSS version 2.0). Data was expressed as mean±standard deviation. Multivariable generalized linear models were used to examine the association between the parameters and severity of the chronic liver disease. The p-value was calculated for each parameter and $p < 0.005$ is considered statistically significant.

RESULTS AND OBSEVATIONS

1) Distribution according to age (table no.2)

AGE (Years)	Number of cases	Percentage %
31-40	20	22
41-50	30	33
51-60	20	22
61-70	13	14
71-80	04	04
81-90	02	04
91-100	01	01

2) **Distribution according to gender (table no 3)**

Gender	Percentage (%)
Males	85
Females	15

Among 90 patients with chronic liver disease under evaluation, the average age was 50 years which ranges from 25 to 97 years old. Highest cases were found in the age group of 41-50 years old. Majority of the patients are males (85%).

3) **Distribution according clinical classification (table no 4)**

Number of patients %	Type of CLD
35%	Compensated ALD
32%	Chronic hepatitis
17%	NAFLD
9%	Decompensated ALD
7%	Liver failure

Based on clinical classification of chronic liver disease. Alcoholic liver disease cases were highest with 35% compensated ALD and 9 % decompensated ALD followed by 32% chronic hepatitis, 17% NAFLD, and 7% end stage liver disease or failure.

Hematological parameters

1) Hemoglobin levels-(table no 5)

Hemoglobin mg/dl	Number of patients %
>11	16
9-10	34
8-9	32
7-8	10
<7	08

34% of cases show haemoglobin levels between 9-10 mg/dl. 32% of cases show 8-9 mg/dl, 16% cases show >11 mg/dl, 10% cases show values 7-8 and least number of cases show haemoglobin value <7mg/dl.

Hb10-normal Hb for that age mg/dl (mild)	**Hb 7-10 mg/dl (moderate)**	**Hb <7 mg/dl (severe)**
14% (n=13)	70 % (n=63)	07% (n=6)

(table no. 6)

70% of cases show moderate anemia, 14% mild and 8% severe anemia. This comparison is non significant with a P value of 0.07 (P value>0.05).

CLD	**Number of patients %**	**Hemoglobin (mg/dl)**
NAFLD	60% (n=9)	>10
Chronic hepatitis	76% (n=22)	7-10
Compensated ALD	80% (n=25)	7-10
Decompensated ALD	60% (n=5)	7-10
Liver failure	63% (n=4)	<7

(table no. 7)

60% of NAFLD has Hb >10mg/dl, (76%) chronic hepatitis, (80%) compensated ALD, (60%) decompensated ALD showed Hb 7-10mg/dl and (63%) liver failure showed Hb <7 mg/dl. Hence majority of the chronic liver disease had moderate anemia. Most of the liver failure cases had severe anemia. Hence severe anemia in chronic liver disease has bad prognosis.

2) Red cell morphology-

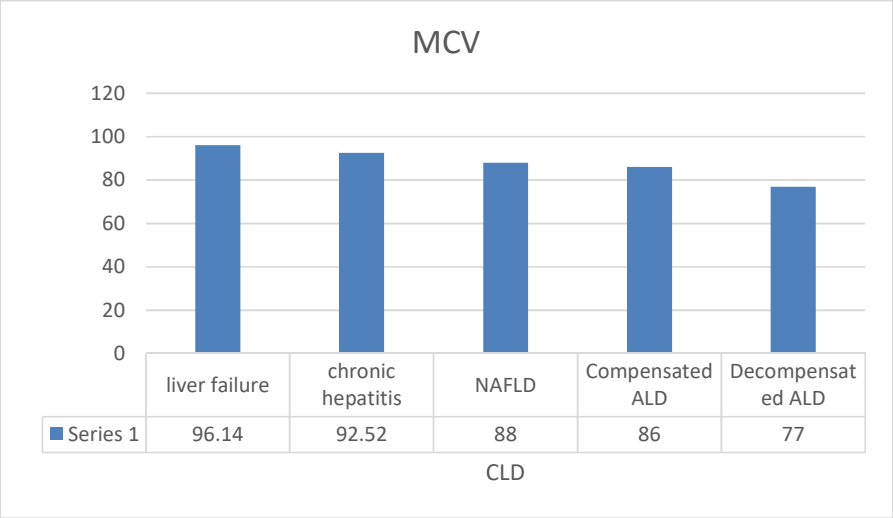
MCV:

On peripheral smear the morphology of RBC-

Type of Anemia	Number of Patients %
Normocytic Normochromic	42%
Macrocytic	39%
Microcytic Hypochromic	19%

(table no. 8)

Evaluation of Anemia in chronic liver disease patients revealed 42% of cases with normocytic normochromic anemia, Macrocytic anemia in 39% of cases and 19% of cases with Microcytic Hypochromic anemia.



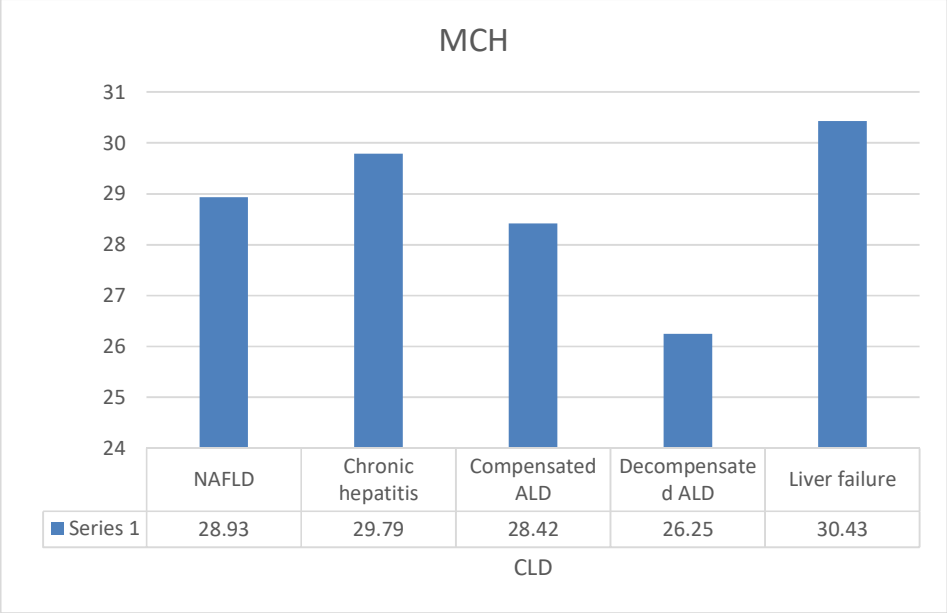
Graph (1) distribution of MCV in different CLD

MCV	**NAFLD**	**88.4**
	Chronic hepatitis	92.5
	Compensated	86.94
	Decompensated	77
	Liver failure	96.14

(table no. 9)

On comparison between the five groups the mean value of MCV in liver failure or end stage disease was the highest followed by chronic hepatitis, NAFLD, compensated ALD and least in decompensated. The comparison is statistically significant with P-value of 0.02.

MCH-



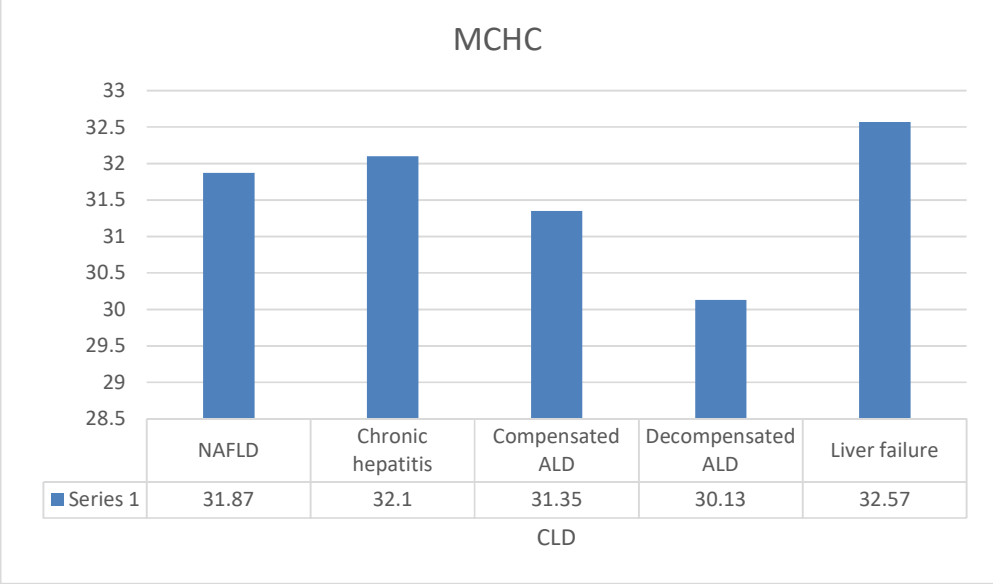
Graph (2) distribution of MCH in different CLD

MCH	**NAFLD**	**28.93**
	Chronic hepatitis	29.79
	Compensated	28.42
	Decompensated	26.25
	Liver failure	30.43

(table no.10)

The mean value of MCH in liver failure or end stage liver disease was the highest followed by chronic hepatitis, NAFLD, compensated ALD and least in decompensated. The comparison of MCH is statistically significant with P-value of 0.017.

MCHC-



Graph (3) distribution of MCHC in different CLD

MCHC	NAFLD	31.87
	Chronic hepatitis	32.1
	Compensated	31.35
	Decompensated	30.13
	Liver failure	32.57

(table no.11)

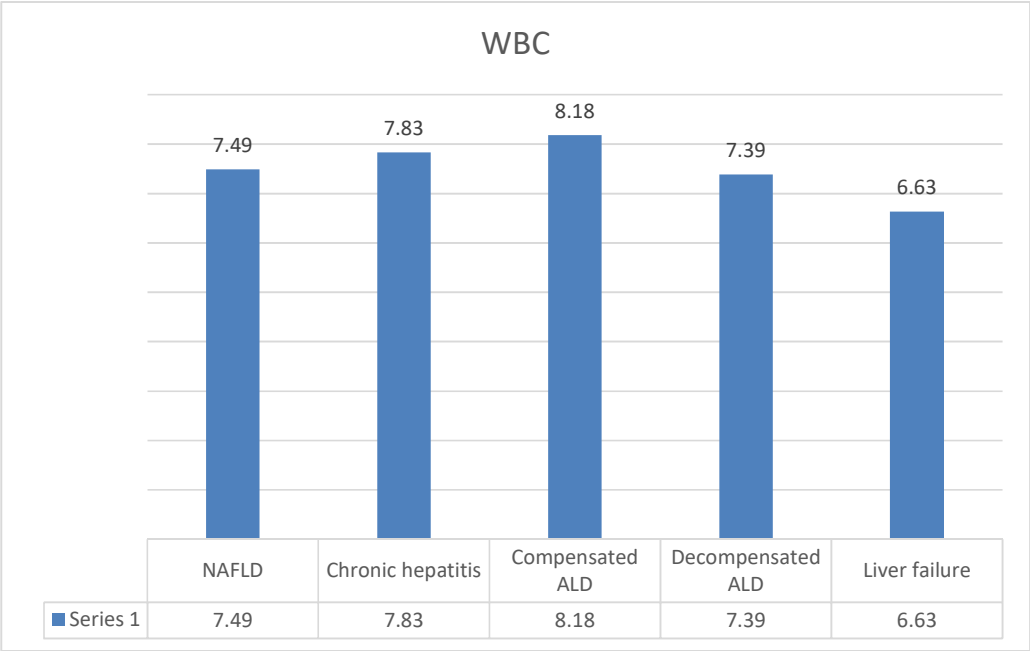
On comparison of MCHC the mean value in liver failure cases was the highest followed by chronic hepatitis, NAFLD, compensated ALD, and least in decompensated ALD. The comparison is statistically not significant with P-value of 0.08.

3) WBC-

Total WBC counts cells/mmm3	**Number of patients %**
<4000	10%
>4000-11000	72%
>11000	18%

(table no.12)

Among all the cases of chronic liver disease 72% of cases had normal counts, 18% cases had Leucocytosis and 10% had leukopenia.



Graph (4) distribution of WBC in different CLD

TC	**NAFLD**	**7.49**
	Chronic hepatitis	7.83
	Compensated	8.18
	Decompensated	7.39
	Liver failure	6.63

(table no.13)

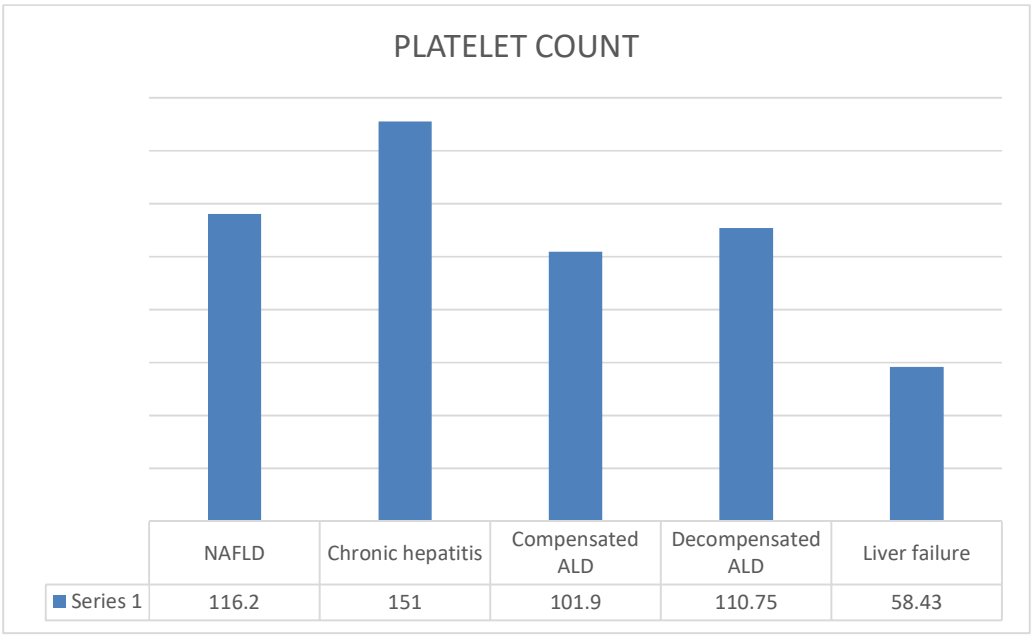
Majority of chronic liver disease had normal counts (72%). Few of the compensated ALD cases showed leucocytosis signifying good clinical response. The comparison is not significant with P value of 0.95 (P-value>0.05).

4) Platelet count -

Number of patients %	Platelet count cells/mm3
30% (28)	150000-450000 (normal)
27% (24)	100000-150000 (mild)
30% (27)	50000-99000 (moderate)
13% (11)	<50000 (severe)

(table no.14)

On comparison of Platelet counts 70% cases had thrombocytopenia. Out of these 27% had mild thrombocytopenia, 30% had moderate thrombocytopenia and 13% had severe thrombocytopenia. Thus, majority were having moderate thrombocytopenia.



Graph (5) distribution of platelet count in different CLD

PLT	**NAFLD**	**116**
	Chronic hepatitis	151
	Compensated	101
	Decompensated	110
	Liver failure	58

(table no.15)

The mean value of chronic hepatitis was the highest followed by NAFLD, decompensated ALD, compensated ALD and least in liver failure. The comparison was not statistically significant with P-value 0.08 (P-value >0.05).

5) Prothrombin Time

Prothrombin Time (normal value 11-13 seconds)	**Number of patients %**
>13.6 secs	88% (n=80)
11-13.5 secs	12% (n=10)

(table no. 16)

Prothrombin time is >13.6 seconds in 88% cases and in 12% within normal range.

CLD	Number of patients %	Prothrombin time (seconds)
Liver failure	100% (n=7)	>13.6 sec
Chronic hepatitis	89% (n=25)	>13.6 sec
NAFLD	80% (n=12)	>13.6 sec
Compensated ALD	80% (n=25)	>13.6 sec
Decompensated ALD	66% (n=5)	>13.6 sec

(table no. 17)

On comparison of PT was raised in all cases of liver failure followed by chronic hepatitis, compensated ALD, decompensated ALD and least in NAFLD. PT values on comparison is significant with P-value 0.014 (P-value <0.05)

6) SGPT-

SGPT (U/L) (normal values SGPT(ALT) -7 to 55 U/L)	**Number of patients %**
7-55	83% (n=75)
56-100	12% (n=11)
>101	5% (n=4)

(table no. 18)

83% of cases had normal values between 7-55 U/l, 12% have values between 56-100 U/L and 5% have values >101 U/L.

Type of CLD	Number of patients % (N)	SGPT U/L
NAFLD (N=15)	67% (N=10)	7-55 U/L
Chronic hepatitis (N=29)	77% (N=20)	56-100 U/L
Compensated ALD (N=34)	31% (N=29)	7-55U/L
Decompensated ALD (N=8)	88% (N=7)	7-55U/L
Liver failure (N=7)	100% (N=7)	7-55 U/L

(table no. 19)

The SGPT values were 7-55 U/l in 100% cases of liver failure followed by decompensated ALD (88%), NAFLD (67%) and compensated ALD (31%). In chronic hepatitis (77%) patients had SGPT between 56-100U/L. The comparison is non significant with a P Value of 0.09 (P value >0.05).

7) SGOT-

SGOT (U/L) (normal values SGOT(AST)- 8-48 U/L)	Number of patients %
8-45	51% (n=46)
46-100	30% (n=27)
101-200	13% (n=12)
> 201	6% (n=05)

(table no. 20)

51% of cases show normal values between 8-45 U/L, 30% cases had values between 46-100 U/L, 13% of cases had values between 101-200 U/L and 6% cases had values >201 U/L.

CLD	Number of patients %	SGOT U/L
NALFD	60% (n=9)	8-54
Chronic hepatitis	45% (n=13)	46-100
Compensated ALD	42% (n=13)	8-45
Decompensated ALD	75% (n=06)	8-45
Liver failure	86% (n=06)	46-100

(table no. 21)

The values were high in chronic hepatitis (45%) and liver failure (86%) between 46-100 U/L. In the remaining cases SGOT was within the normal range. The comparison is non significant with a P Value of 0.7 (P value >0.05).

8) Alkaline phosphatase-

Alkaline Phosphatase U/L (normal values- 47-127 U/L	**Number of patients %**
47-147	89% (n=80)
>200	11% (n=10)

(table no. 22)

89% of CLD cases had values 40-200 U/L. 11% had values more than 200 U/L.

CLD	Number of patients %	Alkaline phosphatase U/l
Decompensated ALD	75%	>200
NAFLD	60%	40-200
Chronic hepatitis	41%	40-200
Compensated ALD	52%	40-200
Liver failure	83%	40-200

(table no. 23)

Majority of Decompensated ALD cases (75%) had values >200 U/L. On comparison P value of 0.17 is not significant (P value>0.05).

9) Gamma glutamyl transferase

GGT U/L (normal values- 8 to 61 U/L)	**Number of Patients %**
8-61	64% (n=58)
62- 100	17% (n=15)
101-200	8% (n=7)
>201	11% (n=10)

(table no.24)

Majority of Decompensated ALD cases (75%) had values >200 U/L. On comparison P value of 0.17 is not significant (P value>0.05).

CLD	Number of patients %	Gamma Glutamyl transferase U/L
Liver failure	86% (n=6)	8-61
Decompensated ALD	78% (n=7)	8-61
Chronic hepatitis	67% (n=19)	8-61
Compensated ALD	62% (n=19)	8-61
NAFLD	53% (n=8)	8-61

(table no. 25)

In all the cases of chronic liver disease the GGT values ranged between 8-61UL. This comparison is non significant with a P value of 0.4 (P value >0.05).

10) Total Bilirubin-

Total Bilirubin mg/dl (Normal level-0-1.2mg/dl	**Number of patients %**
<1.2	21% (n=19)
>1.2	79% (n=71)

(table no. 26)

Most of the cases (79%) had total bilirubin >1.5mg/dl and 21% cases had total bilirubin <1.2 mg/dl.

CLD	Number of patients %	Total Bilirubin mg/dl
Chronic hepatitis	100% (n=31)	>1.5
Compensated ALD	87%(n=28)	>1.5
Liver failure	71% (n=5)	>1.5
Decompensated ALD	66%(n=6)	>1.5
NAFLD	66% (n=10)	>1.5

(table no. 27)

The total bilirubin value was >1.5 mg/dl in all cases of chronic hepatitis, compensated ALD (87%), liver failure (71%), decompensated ALD (66%), and NAFLD (66%). The comparison is not significant with a P value of 0.272 (P value >0.05).

11) Total proteins-

Total Proteins (Normal value-6-8.3 gm/dl)	**Number of patients %**
<6.0	60 (n=46)
> 6.0	40 (n=44)

(table no. 28)

The total protein value was less than <6.5 gm/dl in 60% of cases and >6.5 gm/dl in 40% cases.

CLD	Number of patients % (n)	Total proteins gm/dl
Liver failure	100% (n=7)	<6.0
Compensated ALD	68% (n=21)	<6.0
NAFLD	67% (n=10)	<6.0
Decompensated ALD	63% (n=5)	<6.0
Chronic hepatitis	62% (n=18)	<6.0

(table no. 29)

The total protein value is <6.0 mg/dl in all the cases of CLD.

All the cases of liver failure followed by compensated ALD (68%), NAFLD (67%), Decompensated ALD (63%) and chronic hepatitis (62%) had values less than 6.5 mg/dl. On comparison P-value is 0.31 which is not significant (P value >0.05).

12) Albumin-

Albumin gm/dl (Normal value- 3.5-5.5 gm/dl)	**Number of patients %**
<3.5	86% (n=77)
>3.5	14% (n=13)

(table no.30)

86% of CLD cases had <3.5 gm/dl (hypoalbuminemia) and 14% cases had within normal range.

CLD	Number of patients %	Albumin (g/dl)
Liver failure	100% (n=7)	<3.5
NAFLD	100% (n=15)	>3.5
Decompensated ALD	80% (n=70)	<3.5
Compensated ALD	77% (n=100	>3.5
Chronic hepatitis	75% (n=24)	<3.5

(table no.31)

All the cases of liver failure, decompensated ALD (80%) and chronic hepatitis (75%) had albumin value of <3.5 gm/dl. All NAFLD cases and compensated ALD (77%) had >3.5 gm/dl.

13) A: G RATIO

A/G ratio (normal A/G ratio is 1.5 to 2.5:1)	**Number of patients %**
<1.5:1	86% (n=77)
>1.5:1	14% (n=13)

(table no.32)

86% of cases had A/G ratio <1.5:1 and remaining 14% had >1.5:1.

CLD	Number of patients %	A:G ratio
Liver failure	88% (n=5)	<1.5:1
Decompensated ALD	70% (n=6)	<1.5:1
Chronic hepatitis	68% (n=21)	<1.5:1
Compensated ALD	65% (n=18)	<1.5:1
NAFLD	60% (n=9)	<1.5:1

(table no.33)

Majority cases had A:G ratio <1.5:1, liver failure (88%) followed by decompensated ALD (70%), chronic hepatitis (68%), compensated ALD (65%) and NAFLD (60%). On comparison P value is 0.35 which is not significant (P value >0.05).

Serum Creatinine-

Serum Creatinine mg/dl (normal values 0.6-1.3 mg/dl)	**Number of patients%**
0.6-1.2	40% (n=36)
>1.3	60% (n=54)

(table no.34)

60% had values >1.3 mg/dl and 40% cases had values of 0.6-1.3 mg/dl.

CLD	Number of patients %	Sr creatinine (mg/dl)
Liver failure	80% (n=5)	>1.3
Decompensated ALD	65% (n=20)	>1.3
Compensated ALD	50% (n=15)	>1.3
Chronic hepatitis	45% (n=13)	>1.3
NAFLD	45% (n=4)	>1.3

(table no. 35)

Most of the cases had values >1.3 mg/dl. Liver failure (80%) followed by decompensated ALD (65%), compensated ALD (50%), chronic hepatitis (45%) and NAFLD (45%). The comparison is statistically significant with P-value 0.01 (P-value<0.05).

14) Blood Urea Nitrogen (BUN)-normal value-8-20mg/dl

BUN (normal values-8-20 mg/dl)	**Number of patients %**
<8	26 % (n=23)
8-20	14% (n=13)
>20	60% (n=64)

(table no.36)

60% of cases had BUN values >20 mg/dl, 14% of cases had values 8-20 mg/dl and 26% of cases show value <8mg/dl of BUN.

CLD	Number of patients %	BUN mg/dl
Liver failure	85% (n=4)	<8
Decompensated ALD	67% (n=6)	>20
Compensated ALD	62%(n=18)	8-20
Chronic hepatitis	48% (n=15)	<8
NAFLD	40% (n=6)	>20

(table no.37)

Among all the cases decompensated ALD (67%) and NAFLD (40%) had value >20 mg/dl

Liver failure (85%) and chronic hepatitis (48%) had values <8mg/dl.

The value for compensated ALD (62%) ranged between 8-20 mg/dl. The comparison is significant with a P value of 0.003 (P value <0.05).

15) Serum Electrolytes-

Sodium- normal values 135-145 meq/l

Sodium (meq/L)	Number of patients%
<130	32% (n=28)
131-135	44% (n=40)
>136	24% (n=22)

(table no.38)

Electrolyte studies reveal 32% of cases with low sodium levels of <130 meq/l, 44% of cases ranged from 131-135 meq/L and 24% of cases had value >136 meq/L.

CLD	Number of patients %	Sodium meq/L
Decompensated ALD	63% (n=5)	131-135
Liver failure	57% (n=4)	<130
NAFLD	53% (n=8)	131-135
Chronic hepatitis	37% (n=11)	<130
Compensated ALD	60% (n=16)	<130

(table no.39)

On comparison decompensated ALD (63%) and NAFLD (53%) cases have value 131-135 meq/L, compensated ALD (60%), liver failure (57%) and chronic hepatitis (37%) have value <130meq/L.

On comparison the values are not statistically significant with P-value 0.7 (P-value>0.05).

Potassium-normal values 3.5-5.5 meq/l

Potassium (meq/l)	Number of patients %
>3.5 (normal)	70%(n=63)
3-3.5 (mild)	19% (n=17)
2.5-2.9 (moderate)	11% (n=10)
<2.5 (severe)	00%

(table no. 40)

Serum potassium levels are normal in most (70%) of the cases.

Mild type of hypokalemia is seen in 19% and moderate hypokalemia in 11% of the cases.

CLD	Number of patients % with Potassium (< 3.5meq/l)
Decompensated ALD	38% (n=3)
Compensated ALD	32% (n=10)
Liver failure	29% (n=3)
Chronic hepatitis	28% (n=8)
NAFLD	6.6% (n=3)

(table no.41)

Most of the hypokalemia cases were seen in decompensated ALD (38%) followed by compensated ALD (32%), liver failure (29%), chronic hepatitis (28%) and NAFLD (6.6%). Potassium levels show non significant variation with P-value 0.2 (P-value>0.05).

DISCUSSION

The above prospective cross-sectional study conducted on patients of chronic liver disease for the evaluation of hematological and biochemical parameters showed all the variations and trends related to the severity of the disease.

In present study most of the cases were between 41-50 years of age group, among all these chronic hepatitis 32%, compensated alcoholic liver disease 35%, NAFLD 17%, decompensated alcoholic liver disease 9% and liver failure or end stage liver disease 7%. (table no 4)

Our study showed 88% male predominance which is comparable to other studies. 12% were women which can be explained by the fact that CLD prevalence is larger due to higher alcohol consumption in males. (table no.3)

1) According to age group and gender:

Studies done by	Common age group affected (years)	Number of cases (n)	Males (n)	Females (n)
Tara V (2019)[60]	41-50	40%(n=100)	93% (93)	07% (7)
Kesavadas SM et al (2017)[62]	41-50	40%(n=75)	85% (64)	15% (11)
E Halleys Kumar et al (2021)[62]	40-49	68%(n=100)	70% (70)	30% (30)
Present study (2023)	41-50	33% (n=90)	85% (76)	15% (14)

(table no.42)

In our region Alcoholic liver disease is predominant CLD commonly seen in the fifth decade as it is observed that alcohol consumption begins in second to third decade. This occurrence of age is seen comparable with the above -mentioned studies.

A) HEMATOLOGICAL PARAMETERS:

1) Hemoglobin:

Studies done by	Anemia (<12mg/dl)
E Halleys Kumar et al (2021)[62] (n=100)	86% (n=86)
Kaur J et al (2021)[68] (n=90)	86% (n=78)
Present study (2023) (n=90)	91% (n=82)

(table no.43)

Most of the chronic liver disease shows anemia (91%) which is comparable to other studies mentioned above.

Studies done by	Hb>10mg/dl (mild)	Hb 7-10 mg/dl (moderate)	Hb <7 mg/dl (severe)
Kaur J et al (2021)[68]	14% (n=10)	32% (n=29)	44% (n=39)
E Halleys Kumar et al (2021)[62]	14% (n=14)	42% (n=42)	16% (n=16)
Present study (2023)	14% (n=13)	70 % (n=63)	07% (n=6)

(table no.44)

Our study reported 70% had moderate anemia and 7% had severe anemia mostly occurring in all CLD except NAFLD which is comparable to the study done by **E Halleys Kumar et al.[62]** In a study done by **Kaur J et al[68]** 43.3% of cases have

severe anemia which is higher than the present study. Hence in our study moderate and severe anemia are seen in advanced and severe liver disorders. All cases of severe anemia (7%) occurred in liver failure. Anemia in CLD is mainly due to malnutrition, chronic alcoholism, hypersplenism and blood loss. (table no.6)

2) RBC morphology:

Study	Normocytic normochromic	Macrocytic	Microcytic hypochromic	Dimorphic
Kesavadas et al (2017)[61]	40.9%	28.8%	22.7%	04%
Tara V (2019)[60]	25.8%	22.5%	16.1%	12.9%
Joshi et al (2023)[67]	28.7%	26%	13.3%	-
Kaur J et al (2021)[68]	64.4%	14.5%	21.1%	-
Present study (2023)	42%	39%	19%	-

(table no.45)

In our study on peripheral smear 42% of cases had normocytic normochromic blood picture, 39% had macrocytic and 19% had microcytic hypochromic anemia which is comparable with the above studies (table no.8).

3) WBC counts:

Study done by	**Normal (4000-11000cells/mm3)**	**Leucopenia (<4000 cells/mm3)**	**Leucocytosis (>11000cells/mm3**
Kesavadas SM et al (2017)[71] (n=75)	53%	28%	19%
Kaur J et al (2021)[68] (n=90)	38.8%	15.6%	45.6%
E Halleys Kumar et al (2021)[62] (n=100)	60%	4%	36%
Present study (2023) (n=90)	72%	10%	18%

(table no.46)

In the present study WBC counts were normal in 72% cases which is comparable to the study done by **E Halleys Kumar et al[62]** and **Kesavadas SM et al.[61]** In our study leukocytosis was reported in 18% of the cases which is comparable to study done by **Kesavadas SM et al[61] (18.7%)**. Leucopenia was seen in 10% of cases which is comparable to the study done by **Kaur J et al[68] (15.6%)** (table no.12). Few of the compensated ALD cases showed leucocytosis signifying good clinical response. (table no.13)

Leukocytosis in the CLD patients is associated with the secondary infections. Causes like hypersplenism and chronic bleeding can result in leucopenia. Hence it is observed that leukocytosis and leucopenia indicate disease progression towards severity. Overall, the WBC counts are comparable with the above -mentioned studies.

4) Platelet counts:

Study done by	**Platelets (thrombocytopenia) counts <1.5lac/mm3**
E Halleys Kumar et al (2021)[62]	Thrombocytopenia (56%)
Qamar AA et al (2009)[69]	Thrombocytopenia (77%)
Present study (2023)	Thrombocytopenia (70%)

(table no.47)

Study done by	**Mild (100000-149000 cells/mm3)**	**Moderate (50000-99000 cells/mm3)**	**Severe (<50000cells/mm3)**
E Halleys Kumar et al (2021)[62]	17%	17%	22%
Afdhal N (2008)[72]	62%	13%	1%
Present study (2023)	27%	30%	13%

(table no.48)

On comparison of Platelet counts 70% cases of thrombocytopenia were reported (**table No:12).** 27% had mild thrombocytopenia, 30% moderate thrombocytopenia and 13% had severe thrombocytopenia which is comparable with the above studies. (table no.12)

In most of the studies thrombocytopenia is the common finding in CLD. Alcohol induced bone marrow suppression, hypersplenism, reduced TPO production, DIC induced by infections or drugs are the common causes of low platelet counts in CLD. The platelet counts can be severely reduced towards later stages of the disease.

5) Prothrombin time

Study done by	**Number of patients %**	**Prothrombin time-seconds (mean values)**
Joshi D et al (2023)[67] (n=155)	72% (n=112)	17.44 sec
Kaur et al (2021)[68] (n=90)	89% (n=81)	20.4 sec
Present study (2023) (n=90)	88% (n=80)	19.12sec

(table no.49)

In our study PT is raised in 88% of cases which is comparable to the study done by **Kaur J et al.[68]** It significantly increases with the severity of the disease. The increased PT reflects deranged coagulation system which results in increased bleeding tendency in CLD cases. Raised PT indicates decrease in synthetic activity of the liver.

B) Table for biochemical parameters:

1) Serum transaminases: AST or SGOT, ALT or SGPT

SGPT:

Studies done by	SGPT (mean values)
Ahmed et al (2018)[75] (n=771)	56.5U/L
S Akter et al (2021)[76] (n=100)	42.1U/L
R Anju et al (2017)[77] (n=40)	43 U/L
Present study (n=90)	35.61U/L

(table no.50)

SGPT (U/L)	Number of patients %
7-55	83% (n=75)
>56-100	17% (n=15)

(table no.51)

Studies done by	Number of patients %	SGPT U/L
Ray G et al (2014)[78] (n=395)	69% (n=273)	7-55
S Akter et al (2021)[76] (n=100)	50% (n=50)	7-55
Present study (2023) (n=90)	83% (n=75)	7-55

(table no.52)

In our study most of the cases (83%) have SGPT levels ranged between 7-55 U/L.

17% cases have values more than >56 U/L which is comparable with the above-mentioned studies.[66,68]

SGPT is found specifically in liver and is more specific for hepatocellular injury. The value of SGPT is lower than the above studies. The cause of the low mean values can be attributed to high number of Alcoholic Liver Disease patients in which values are near to normal range (table no.17). The deficiency of pyridoxine causes decrease in SGPT more than SGOT in alcoholic liver disease.

SGOT

Study done by	SGOT (mean values)
Ahmed et al (2018)[75] (n=771)	78.5U/L
S Akter et al (2021)[76] (n=100)	52.8U/L
R Anju et al (2017)[77] (n=40)	70 U/L
Present study (2023) (n=90)	71.4U/L

(table no.53)

SGOT (U/L)	Number of patients %
8-45	51% (n=46)
>46	49% (n=44)

(table no.54)

Studies done by	Number of patients %	SGOT
S Akter et al (2021)[76] (n=100)	50% (n=50)	8-45 U/L
Present study (2023) (n=90)	51% (n=46)	8-45 U/L

(table no.55)

In our study 51% of the cases had SGOT value between 8-45 U/L and 49% had increased levels which is comparable with other studies.

In our study the mean value of SGOT is 71.4U/L. which is mildly increased and is comparable to the study done by **Ahmed et al[75]** and **Anju et al.[77]** SGOT is present in organs other than liver such as cardiac muscle, skeletal muscle, brain and kidney and hence it is less sensitive. The higher value of SGOT than SGPT can be attributed to more muscle damage in CLD patients.

Hence, SGOT showed some progressive increase in advanced liver disease whereas SGPT which is more liver specific did not show this correlation.

Serum Alkaline Phosphatase:

Study done by	Alkaline Phosphatase (Normal level-47-147 U/L) (mean values)
S Akter et al (2021)[76] (n=100)	181 U/L
Ahmed et al (2018)[75] (n=771)	171 U/L
Present study (2023) (n=90)	136 U/L

(table no.56)

Alkaline Phosphatase U/L	Number of patients %
47-147	89% (n=50)
>147	11% (n=40)

(table no.57)

Studies done by	Number of patients %	ALP U/L
S Akter et al (2021)[76] (n=100)	100%(n=100)	47-147 U/L
Ahmed et al (2018)[75] (n=771)	79% (n=609)	47-147 U/L
Present study (2023) (n=90)	89% (n=80)	47-147 U/L

(table no.58)

In our study most cases (89%) had value between 47-147 U/l which is normal and comparable with the above studies.

Serum alkaline phosphatase is a nonspecific marker for assessment of liver injury due to its extrahepatic production but if raised it suggest extrahepatic biliary obstruction, cholestasis and primary liver cirrhosis. In our study 89% cases have values between 47-147 U/L. The mild increase in chronic liver disease is because of intrahepatic cholestasis and other nonspecific causes. Since ALP lacks specificity for liver diseases GGT screening can be utilized with other abnormal liver chemistries. The ALP values in our study were comparable to the study done by **Ahmed et al.**[75]

6) Gamma glutamyl transferase: GGT

Study done by	**GGT (Normal values- 8 to 61 U/L) (mean value)**
Krishnamurthy HA (2013)[79] (n=50)	49 U/L
Xing M et al (2022)[80] (n=515)	62 U/L
Present study (2023) (n=90)	88.34U/L

(table no.59)

GGT U/L	**Number of Patients %**
8-61	64% (n=58)
>62	36% (n=32)

(table no.60)

Studies done by	Number of patients %	GGT U/L
Krishnamurthy HA (2013)[79] (n=50)	100% (n=50)	8-61 U/l
Present study (2023) (n=90)	64% (n=58)	8-61 U/L

(table no.61)

In our study most of the cases (64%) had value ranged between 8-61 U/L which is comparable with the study done by Krishnamurthy HA.[79]

Gamma glutamyl transpeptidase can be used as a hepatic injury marker in view of its persistent elevation. Our study showed increased value since the initial stages of liver diseases. The value of GGT is higher in alcoholic liver disease due to oxidative stress caused by ethanol and its metabolites. The mean value was reported to be higher in chronic hepatitis, followed by alcoholic liver disease (table no.23). The lowest value was reported in NAFLD (53%) which is comparable to the study done by **Xing M et al**.[80] Thus, GGT values are significant in early stages of alcoholic liver disease rather than well established and progressive CLD.

7) Serum bilirubin:

Study done by	Serum bilirubin (mean values) (Normal level-0-1.2mg/dl)
S Akter et al (2021)[76] (n=100)	2.46 mg/dl
Ahmed et al (2018)[75] (n=771)	2.14 mg/dl
R Wadekar (2021)[81] (n=93)	4.9 mg/dl
Present study (n=90)	3.7 mg/dl

(table no.62)

Total Bilirubin mg/dl	Number of patients %	Total bilirubin mg/dl
S Akter et al (2021)[76] (n=100)	75% (n=75)	>1.5
Present study (2023) (n=90)	79% (n=65)	>1.5

(table no.63)

In our study most of the cases (79%) had value >1.5 mg/dl which is comparable to the study done by S Akter et al.[76]

Serum bilirubin level is indicator of liver uptake, conjugation and secretory function. The mean value of total bilirubin in our study is 3.7mg/dl.

As per study done by **S Akter et al[76]** the serum bilirubin rises significantly in hepatitis and increasing severity of the hepatocellular injury. In our study the serum bilirubin was found to be raised in most of the cases of CLD and 100% cases of chronic hepatitis (table no. 25). The values of serum bilirubin remained elevated in CLDs but were not as high as in acute hepatitis.

8) Total proteins

Study done by	Total proteins (gm/dl) (mean values)
S Akter et al (2021)[56](n=25)	6.55
Wadekar R (2021)[71] (n=93)	6.13
Present study (2023) (n=90)	6.37

(table no.64)

Serum total proteins and albumin are indirect measure of synthetic function of liver. The chronic inflammation of the hepatic parenchyma and lack of nutrients leads to impaired synthetic function. Serum albumin levels reflect nutritional status as well as severity of inflammation.

Studies done by	Number of patients %	Total proteins gm/dl
R Wadekar (2021)[81](n=93)	100% (n=93)	<6.0
S Akter et al (2021)[76](n=25)	50% (n=50)	<6.0
Present study (2023) (n=90)	60% (n=46)	<6.0

(table no.65)

60% of the cases reported Total proteins <6.0 g/dl in our study which is comparable to the study done by **S Akter et al**.[76] These parameters are positive indicators of chronic liver disease.

9) Albumin:

Study done by	Albumin (< 3.5mg/dl)
Ahmed et al (2018)[75](n=771)	3.88
R Wadekar et al (2021)[81] (n=93)	2.94
S Akter et al (2021)[76] (n=100)	3.37
Present study (2023) (n=90)	3.33

(table no.66)

Studies done by	Number of patients %	Albumin mg/dl
S Akter et al (2021)[76](n=100)	75% (n=75)	<3.5
Present study (2023) (n=90)	86% (n=77)	<3.5

(table no.67)

Serum albumin levels are decreased in all liver failure cases, 80% decompensated ALD and 75% chronic hepatitis. All NAFLD and 77% compensated ALD show albumin value of >3.5 mg/dl which is comparable to the above studies. The albumin level reflects nutritional status as well as severity of the inflammation in the CLD. (table no 29)

10) A/G ratio:

Study done by	A:G ratio (mean value)
S Akter et al (2021)[76] (n=25)	1.10±1
Present study (2023) (n=90)	0.91±0.4

(table no.68)

Studies done by	Number of patients %	A: G ratio (mean values)
S Akter et al (2021)[76] (n=100)	75%(n=75)	<1.5:1
Present study (2023) (n=90)	86% (n=77)	<1.5:1

(table no.69)

86% cases show A/G ratio <1.5:1 which is comparable to the study done by S Akter et al. The A:G ratio is low in 88% liver failure, 70% decompensated ALD, 68% chronic hepatitis, 65% compensated ALD and 60% NAFLD. Impaired albumin synthesis cause decrease in ratio. Reversal of ratio is found in severe inflammation or cirrhosis. (table no.31)

11) Serum creatinine:

Study done by	Serum creatinine (mg/dl)
Ahmed et al (2018)[75] (n=771)	1.10
Kaur J et al (2021)[68] (n=90)	1.69
Present study (2023) (n=90)	1.26

(table no.70)

Serum Creatinine mg/dl	Number of patients%	Serum creatinine md/dl
Kaur J et al (2021)[68] (n=90)	45% (n=45)	>1.3
Present study (2023) (n=90)	60% (n=54)	>1.3

(table no.71)

In present study creatinine values are elevated to >1.3 mg/dl in 60% of cases and is seen significantly associated with the increasing severity of the disease which is comparable to the study done by **Kaur J et al[68] (48%).** The creatinine levels are non-specific but can be further evaluated to rule out kidney injury or hepatorenal syndrome. **Slack et al[98]** study revealed association of CLD with CKD by evaluating renal function parameters and importance of creatinine in the evaluation affected by

CLD. The evaluation of kidney injury biomarkers like creatinine improves the prognosis in post liver transplant patients.[96]

12) BUN

Study done by	BUN (mg/dl) (mean values) (Normal value 8-20 mg/dl)
Kaur J et al (2021)[68] (n=90)	29.16
Ahmed et al (2018)[75] (n=771)	16.49
Present study (2023) (n=90)	19.12

(table no.72)

Studies done by	Number of patients %	BUN mg/dl
Ahmed et al (2018)[75] (n=771)	34% (n=269)	>20
Present study (2023) (n=90)	60% (n=64)	>20

(table no.73)

In present study 60% of cases with severe chronic liver disease have elevated values of BUN, 62% in decompensated ALD and 40% in NAFLD. This is comparable to the study done by **Ahmed et el**.[75] (table no.35)

13) Serum electrolytes:

Serum Sodium:

Study done by	Sodium levels <130 meq/l	131-135 meq/l	>136 meq/l
Singh Y et al[100] (n=100)	47%	8%	45%
Singh N et al[99] (n=120)	30%	33%	37%
Present study	32%	44%	24%

(table no.74)

Serum electrolytes are independent markers of chronic liver disease.

These markers can get greatly influenced by drug intake, kidney injury, chronic alcoholism. Alcohol causes diuretic effect which depletes the electrolyte stores of body. Hyponatremia is the common finding in CLD cases. The low sodium levels can play a major role in development of hepatic encephalopathy.

In our study 32% Hyponatremia was reported which is comparable to study done by **Singh N et. Al.[99]** However, the common finding was sodium between 131-135 meq/l (44%) along with hypokalaemia in few cases (30%).

57% liver failure and 37% chronic hepatitis show sodium levels <130 meq/mol. 63% Decompensated ALD and 53% NAFLD showed values between 131-135 meq/l. The electrolyte values are significantly associated with the disease outcome rather than severity. (table no.37)

Serum potassium

Studies done by	Potassium levels <3.5 meq/l
Kashyap KC et al (2016)[97] (n=40)	15% (n=6)
Singh Y et al (2022)[100] (n=100)	30%(n=30)
Present study (2023) (n=90)	30% (n=27)

(table no.75)

In our study Serum potassium levels were normal in most (70%) of the cases. Hypokalaemia was seen in 30% cases which is comparable to the study done by **Singh Y et al.[100]** On comparison decompensated ALD (38%) had increased cases of hypokalaemia followed by compensated ALD (32%), liver failure (29%), chronic hepatitis (28%) and NAFLD (6.6%) (table no.39).

The body stores of potassium are low in chronic alcoholics. The serum potassium levels can be affected by GI loss, diet, hyperaldosteronism and diuretic-treatment. It also plays an important role in hepatic encephalopathy in cirrhotic patients. Hence, electrolyte regulation is a priority in chronic liver disease patients.

CONCLUSION

Hematological and biochemical parameters were studied in chronic liver disease in a tertiary care center. The most common etiology of CLD was alcoholic liver disease mostly seen in males.

Majority of the patients of CLD had moderate type of anemia. Severe anemia was seen with increased severity of the disease like liver failure.

Most of the cases had normal WBC count. However, Leukocytosis is associated with infections and Leucopenia and thrombocytopenia suggests chronicity of the disease with bone marrow suppression secondary to malnutrition and hypersplenism.

Majority of the cases had elevated prothrombin time which signifies decreased synthetic function of the liver which attributes to progression and severity of the disease.

SGPT, SGOT, alkaline phosphatase and GGT are not of much significance in assessing the progression and severity of the disease. However, GGT is specific for alcoholic liver disease.

Severe forms of CLD are always associated with raised bilirubin, hypoproteinemia, hypoalbuminemia with decreased A/G ratio which correlates with disease progression.

Raised Serum creatinine and BUN are associated with majority of the CLDs which also determines the disease progression and severity.

Serum sodium and potassium levels are normal in majority of the cases. Hyponatremia and hypokalemia are associated with advanced CLDs like liver failure.

Thus, the early detection of the above parameters can help to detect the disease progression and severity. Hence help in treatment modification for better outcome in chronic liver disease.

SUMMARY

A prospective cross- sectional study was conducted in the tertiary care hospital's department of pathology from January 2023 to December 2023.

90 cases of chronic liver disease were studied for the evaluation of hematological and biochemical parameters.

The most common age group affected is 41-50 years with male predominance (85%).

The most common chronic liver disease based on etiology was alcoholic liver disease which was seen in 44% cases.

The common finding in the hematological parameters was anemia seen in 91% cases with predominantly being moderate type of anemia.

Normocytic normochromic RBC morphology was seen in 42% of cases.

WBC count was normal in 72% of the cases. Leucocytosis was seen in 18% cases. Leucopenia was a finding in 10% cases.

Thrombocytopenia was seen in 70% cases with moderate type of thrombocytopenia in 30%.

Prothrombin time was elevated in 88% of cases.

The biochemical parameters show not much variation with disease severity. SGPT is normal in 83% of cases.

SGOT show mildly raised values in 49% cases.

89% cases show normal Alkaline phosphatase levels.

GGT is seen within normal range in 64% of cases with mild increase in alcoholic liver disease.

79% of the cases showed total bilirubin >1.2 mg/dl.

There was hypoproteinaemia in 60% of the cases and hypoalbuminemia in 86% of the cases with decreased A/G ratio.

Creatinine levels and BUN were raised in 60% of the cases.

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ANNEXURE-I

INFORMED CONSENT FORM

"EVALUATION OF HAEMATOLOGICAL AND BIOCHEMICAL PARAMETERS IN CHRONIC LIVER DISEASE PATIENTS"

Objective: To evaluate hematological and biochemical parameters in chronic liver disease patients and to correlate the changes with severity of the disease.

Introduction: Chronic liver disease is one of the frequent causes of the deaths especially in developing countries. There has been noted an increase in prevalence of the disease in the recent times.

The proper evaluation of hematological and biochemical parameters becomes important for evaluating disease severity and to provide further treatment. The early detection of hematological and biochemical derangements would avoid disease complications and enhance patient quality of life with better survival rates in chronic liver disease patients.

Explanation of procedure: Blood samples from the chronic liver disease patients under evaluation will be collected and analysed for hematological and biochemical parameters. The changes would be correlated with the severity of the disease.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**EVALUATION OF HAEMATOLOGICAL AND BIOCHEMICAL PARAMETERS IN CHRONIC LIVER DISEASE PATIENTS**". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE-2

PROFORMA

PATIENT HISTORY

- Name:
- Age:
- IP no:
- Presenting complaints:
- History of present illness:
- Past medical/surgical history:
- Family history:
- Personal history:

General Physical Examination

- Build:
- Nutrition:
- Height:
- Weight:
- Pallor:
- Jaundice:
- Oedema of legs:
- Pulse:
- Blood pressure:
- Loss of hair
- Spider nevi
- Testicular atrophy
- Clubbing
- Gynaecomastia

Systemic Examination

CNS, CVS, RS, PA

Routine investigation- Blood

Hematological investigation

- Haemoglobin concentration (Hb)
- Red cell count (RBC)
- Mean corpuscular volume (MCV)
- Mean concentration of haemoglobin (MCH)
- Mean corpuscular haemoglobin concentration (MCHC)
- White blood cell count
- Platelet count (PLT)
- Prothrombin time

Biochemical parameters

- SGOT
- SGPT
- ALP
- GGT
- total bilirubin
- total proteins
- albumin
- A:G ratio
- Serum creatinine
- BUN

ELECTROLYTES

- Sodium
- Potassium

ANNEXURE-II

KEY TO MASTER CHART

ACRONYM	**DESCRIPTION**
IP NO	In patient number
M	Male
F	Female
Hb	Hemoglobin
RBC	Red cell count
MCV	Mean corpuscular volume
MCH	Mean corpuscular haemoglobin
MCHC	Mean corpuscular haemoglobin
WBC	White blood cell
PLT	Platelet count
PT	Prothrombin time
SGPT	Serum glutamic pyruvic transaminase
SGOT	Serum glutamic oxaloacetic transaminase
ALP	Alkaline phosphatase
GGT	Gamma glutamyl transferase
BIL	Bilirubin
CREAT	Creatinine

T PROT	Total protein
ALB	Albumin
A: G	Albumin: Globin ratio
BUN	Blood urea nitrogen
NA	Sodium
K	Potassium
NAFLD	Non-alcoholic fatty liver disease
COMP ALD	Compensated alcoholic liver disease
DECOMP ALD	Decompensated Alcoholic liver disease
CHR HEPAT	Chronic hepatitis

ANNEXURE-III MASTER CHART

IP NO.	AGE	GENDER	HB	MCV	MCH	MCHC	RBC	TC	PLT	SGOT	SGPT	ALP	GTT	T BIL	SR CREAT	BUN	PT	NA	K	T PROT	ALBUMIN	A/G RATIO	DIAGNOSIS
1194828	37	M	7	79	27	30	2.3	35	169	87	24	210	252	14	1.43	22	23.8	135	3.89	6.2	3	0.7	Comp ALD
1196235	50	M	8	60	22	26	2.28	22	134	186	130	109	18	8.6	0.9	84	39	127	5.5	4.8	2.2	1	Comp ALD
1179079	52	M	7.2	88	29	32	2.04	18	68	37	27	60	19	7.73	1.67	29	41	127	2.98	5.3	1.2	0.9	Comp ALD
1204927	44	M	11	90	29	33	3.3	17.4	265	96	29	116	80	7.94	0.55	5.14	18	133	4.56	7	2	0.7	CHR HEPAT
1179420	57	M	9.1	86	28	33	3.6	15.6	91	30	10	184	60	6.17	1.5	42	13.2	134	2.8	7.2	1	0.9	DECOMP ALD
1166791	70	M	11.4	80	32	31	3.88	14.7	357	84	27	344	200	8.82	1.45	14	15	136	4.8	6	4	0.6	CHR HEPAT
1171983	39	M	6.7	105	35	32	2.39	14.1	122	60	31	214	35	0.8	1.01	7.94	22	137	3.7	7.5	4.2	1.3	CHR HEPAT
1208510	38	M	9.5	92	28	32	4.59	14	636	39	56	78	26	0.8	0.66	18	16	133	4.2	4.3	4	0.7	CHR HEPAT
1176794	52	M	10.9	90	30	32	2.97	13.9	179	266	75	148	189	2.8	1.73	38	15.5	137	3.89	5.5	5	1.8	NAFLD
1173010	70	M	9.7	82	30	32	3.24	13.9	192	32	18	123	16	1.35	2.47	29	15	129	4.49	6.2	3.5	0.6	CHR HEPAT
1201697	32	M	7.8	99	28	32	2.22	13.8	91	50	34	186	39	8.68	2.4	14	32	127	4.5	7.2	3.8	1.1	Comp ALD
1204421	60	M	7.6	104	30	33	3.02	13.4	98	90	73	105	87	0.5	1.22	22	11.8	138	4	6.2	4.2	0.4	NAFLD
1204611	34	F	9.8	109	30	33	2.49	13	75	111	41	131	45	9.75	0.7	14	44	125	2.5	6.2	3.8	1.3	COMP ALD
1204272	52	M	11.2	60	22	27	3.32	12.8	154	36	24	149	87	1.09	1.71	24	13.4	130	6.3	7	3.4	0.8	NAFLD
1170188	41	F	9.1	109	36	33	2.47	11.7	236	106	21	239	754	7.26	0.53	6.54	16.8	136	2.57	8.2	5	1.1	CHR HEPAT
1200973	37	M	8.9	84	30	33	3.03	11.6	78	91	51	117	119	3.44	1.52	20	16.8	138	4.6	6.2	4	0.8	Comp ALD
1179263	49	M	11.6	90	30	32	4.07	11.4	127	40	17	129	59	2	1.41	25	14	140	3.7	6.9	3.5	1.5	NAFLD
1195552	50	M	12.3	88	29	32	3.67	11.4	46	50	35	156	33	4.19	1.47	26	32.6	130	4.06	5.7	4	0.6	LIVER FAILURE
1167271	53	M	13.4	86	32	33	4.86	10.8	269	52	82	51	20	0.53	1.05	12.61	10	136	4.6	6	5	1.8	Comp ALD
1207603	43	M	8.2	92	28	31	2.75	10.3	215	162	23	144	18	6.9	0.79	11.68	14	133	3.07	7.5	5	0.5	CHR HEPAT
1171559	40	M	11.6	100	30	33	4	9.9	215	20	107	14	393	0.34	0.94	5.6	10.5	136	4.5	8.2	3	0.7	Comp ALD
1176377	72	M	11.8	66	24	29	4.11	9.5	153	34	24	82	55	0.39	5.67	65	12.5	131	6	5.3	3.5	0.4	DECOMP ALD
1205779	57	M	10.3	110	33	34	3.39	9.4	43	43	28	144	22	5.16	0.85	9.8	16	133	3.25	5	2.2	1.2	CHR HEPAT
1205898	42	F	8.8	89	28	32	3.25	9.3	288	163	116	302	528	8.6	0.54	4.6	16.7	132	5.16	5	4	1	CHR HEPAT
1180617	63	M	13.2	110	34	34	4.3	9.1	329	14	15	78	25	0.85	0.86	5	14.7	138	4.3	5.3	3.3	0.7	NAFLD
1206972	38	M	8.5	90	27	31	2.9	9	175	50	91	142	233	2.2	3.7	48	24.2	122	4.45	7	3.4	0.7	CHR HEPAT
1164707	38	M	10.6	72	23	29	3.11	8.7	190	348	80	191	40	2.2	0.66	6.07	15	133	2.7	5.2	2	1.2	Comp ALD
1190163	37	F	6.5	86	30	31	1.97	8.4	90	41	17	38	73	5.7	0.8	7.94	22.7	130	4.44	5.7	1	0.6	Comp ALD
1208012	37	M	10.6	88	27	32	3.4	8.4	123	30	12	86	50	2.42	0.64	5.14	17	135	4.2	7.9	5	1.3	DECOMP ALD

1205470	48	M	11.7	89	27	32	3.6	8.4	104	66	36	97	35	4.43	0.8	10	18.2	129	2.6	7.4	4	0.7	Comp ALD
1171525	46	M	12.7	93	30	32	4.2	8.3	273	23	22	137	75	0.9	1.27	13	15.8	137	3.76	6.7	4.5	1.8	CHR HEPAT
1201811	52	M	9.2	66	27	29	2.52	8.2	68	48	41	136	60	4.78	1.03	6.54	31	131	3	5.3	5.5	0.4	Comp ALD
1201740	64	M	8.6	69	25	29	2.42	8	35	43	21	104	40	4.8	1.11	12.14	18.6	138	4	6.2	5	0.7	CHR HEPAT
1169701	45	M	9.7	77	30	32	2.64	7.7	106	38	16	110	16	4.45	0.83	7	21	132	3.88	6.5	3	0.7	NAFLD
1203175	36	M	8.5	107	35	36	4.41	7.5	143	33	26	145	69	3.03	0.77	15	16	136	4.06	6	0.3	0.7	CHR HEPAT
1207695	43	M	9.5	103	31	33	3.04	7.5	51	24	18	91	61	3.6	0.67	8.4	21	120	4.44	5.9	4	0.5	LIVER FAILURE
1166665	78	M	12	92	29	32	3.74	7.4	136	62	43	248	230	4.56	2.01	24	15	128	2.92	5.9	2.2	0.8	COMP ALD
1179810	57	F	8.7	89	30	33	2.88	7	95	22	10	146	16	1.07	1.72	15	12	131	3.5	5	4	0.6	COMP ALD
1170468	58	M	10.7	86	29	33	3.53	6.9	131	69	43	113	18	1.64	1.67	20	18	127	4.16	7	3.8	0.9	CHR HEPAT
1167227	43	F	8.3	98	34	34	2.3	6.8	18	100	21	66	35	7.16	1.54	14.48	21	134	2.5	6.1	4	1.3	DECOMP ALD
1179235	50	M	10.2	82	26	30	4.37	6.8	125	64	32	115	16	4.39	0.63	5.14	15.6	132	3.8	5.7	6	0.5	CHR HEPAT
1203133	52	M	9.4	100	29	32	2.7	6.7	98	57	27	98	55	2.97	1.86	21.4	22	137	4.2	5	6	0.7	Comp ALD
1202605	47	F	7.2	69	27	29	2.63	6.6	91	39	20	137	24	3.86	1.65	11.68	18	138	4	7.4	3.8	1.1	COMP ALD
1170164	56	M	10.2	82	30	33	3.46	6.5	202	22	11	148	200	0.62	0.72	6	13	132	4.06	7.5	4	0.7	NAFLD
1194120	47	M	11.3	88	28	32	3.64	6.5	48	150	30	228	38	3.43	0.72	4.67	18.5	134	3.6	6	2	0.6	NAFLD
1203181	33	F	10.5	100	29	33	3.05	6.4	111	98	35	143	529	4.03	0.5	5.14	14.8	135	3.4	6.3	1.4	1.2	CHR HEPAT
1180301	40	F	8.6	94	30	33	4	6.3	95	23	34	106	33	1.05	1.94	61	18	133	6.7	6.6	1.8	1	DECOMP ALD
1178189	80	F	9.7	86	30	31	3.39	6.2	176	30	15	82	50	1.89	3.46	51	17	133	4.2	6.4	5	1.9	DECOMP ALD
1197949	53	M	7.3	90	27	30	1.07	6.2	50	14	14	143	42	4.05	1.83	26	19	118	3.7	5.8	4	0.5	CHR HEPAT
1201078	60	M	9.7	86	29	32	3.06	6	59	39	12	281	30	1.9	1.27	8.9	33	134	4	5.3	3	0.6	LIVER FAILURE
1190039	38	M	8.3	105	33	34	2.09	6	44	66	41	209	7	7.9	0.7	19	31	142	3	5.8	3.8	0.5	LIVER FAILURE
1172250	66	M	10.1	97	32	34	4.5	5.9	123	26	26	106	41	0.38	1.06	9.34	12	136	3.58	6.5	4	1.3	CHR HEPAT
1204083	65	M	8.2	88	29	32	3.38	5.8	78	39	26	103	21	1.01	1.15	18	17	135	4.4	7.9	5	1.1	Comp ALD
1181807	47	M	7.9	106	31	33	2.3	5.7	51	35	30	210	24	4.91	0.75	5.6	24.2	137	4.06	7.6	6	0.4	CHR HEPAT
1180588	43	M	10.9	102	32	32	3.42	5.6	58	76	66	155	225	7.73	0.83	15	25	137	3.78	5	7.3	1.3	CHR HEPAT
1196185	47	F	11.5	99	27	32	3.96	5.6	121	51	25	169	39	1.39	0.95	7	18.6	130	4.46	6.3	1.5	0.8	LIVER FAILURE
1178738	37	M	6.3	108	32	35	1.88	5.5	41	19	29	132	30	5.85	0.53	5.6	18.3	128	4	8.7	7.8	0.4	Comp ALD
1202334	47	M	11.1	78	32	34	2.81	5.4	65	108	31	131	219	13.39	1.19	8.87	22	133	3.8	5.8	4.4	0.8	CHR HEPAT
1172280	59	M	11.3	88	30	33	4.02	5.3	106	16	11	107	154	1.03	0.69	5.14	13	132	3.8	7	2.8	0.8	Comp ALD
1166653	58	M	11	100	30	33	3.17	5.3	57	79	46	145	80	4.7	0.6	16	22	124	3	4.7	3.4	0.8	Comp ALD
1169610	63	M	10.2	87	29	33	2.3	5.2	109	44	23	67	30	3.48	1.67	18.6	14	125	3.86	7.3	4.8	0.9	NAFLD
1206300	50	M	6.1	63	23	27	2.45	5.2	106	24	19	142	16	2.96	0.73	6.5	15.6	132	3.5	6.4	6.8	0.4	Comp ALD
1188281	40	F	9	70	24	27	3	5	100	202	57	151	298	8	0.93	9.3	25	135	4.8	7	5.5	2.6	DECOMP ALD
1188691	40	M	7.9	70	24	29	3.29	5	114	533	32	144	200	3.64	0.91	9.4	15.6	134	3.6	7.6	5	0.6	Comp ALD
1204482	44	M	9	110	34	36	2.71	5	40	35	39	149	100	2.92	1.58	20	21	135	4	4.9	4	1.1	NAFLD
1208753	37	M	8	100	27	31	2.65	5	79	64	33	66	16	2.96	2.74	31	16.5	130	3.4	6.1	3	1.3	NAFLD
1206171	61	M	19.6	88	29	32	6.7	4.8	191	128	52	55	20	3.18	1.81	13	23	151	4	7.3	63	0.8	CHR HEPAT
1181550	45	M	12.2	100	30	32	4.7	4.7	66	72	92	262	80	3.32	0.64	7.47	16.8	138	3.06	6	5.8	1	CHR HEPAT
1169097	70	M	12	72	25	29	3.83	4.7	115	60	43	124	26	1.11	0.9	11.6	14.4	136	4.12	6.2	6	1.8	NAFLD

1167316	55	M	12.9	78	30	33	3.2	4.6	46	457	108	146	30	4.3	0.79	7.9	14	132	4.6	7.8	4	1.9	NAFLD
1185682	63	M	8.5	60	22	26	3.06	4.5	40	15	20	69	22	0.94	2.42	38	14.9	129	4.3	6.5	6	0.6	DECOMP ALD
1174456	36	M	12.6	97	30	32	3.86	4.3	120	55	18	111	54	2.72	3.49	23	17	128	3.8	6.1	4	1	CHR HEPAT
1202212	47	F	10.9	77	29	33	3	4.3	109	17	10	70	30	0.42	0.65	8.87	11.7	135	3	6.5	2.2	0.7	Comp ALD
1175031	53	M	8.8	102	33	32	2.45	4	186	55	33	101	30	1.06	0.7	11.68	15	117	4.8	5.8	0.5	0.6	Comp ALD
1196792	32	M	8	70	25	29	2.42	3.7	14	47	29	113	70	7.9	0.87	12	24	103	5.98	6.5	4	0.7	CHR HEPAT
1204349	65	M	6.3	90	29	32	3.56	3.7	50	30	26	106	62	0.43	1.09	11	11.6	141	4	5.9		0.8	Comp ALD
1206874	43	M	5.8	94	29	30	2.31	3.7	62	44	22	117	14	2.8	0.73	10.2	18.3	130	4.04	6.7	5	0.9	Comp ALD
1182150	44	F	11.6	72	26	29	4.79	3.6	39	32	25	158	86	2.91	0.89	21	18.4	128	3.3	6.4	3.8	0.5	NAFLD
1203139	50	M	12.6	66	25	30	3.76	3.6	108	23	20	85	50	1.72	1.11	6.54	12.4	139	4.56	6.6	5	1.3	DECOMP ALD
1193700	73	M	10	107	29	33	3.7	3.5	112	29	14	109	28	0.57	0.83	9.8	13.4	136	3.7	7.2	8	0.6	CHR HEPAT
1168223	97	M	7.8	100	32	34	3.04	3.5	58	23	12	117	20	1.13	1.1	20	14	131	4.3	6.3	6.7	0.9	CHR HEPAT
1195667	43	M	7.5	86	26	30	1.62	3.3	64	62	38	139	30	3.4	0.95	18	31	128	3.26	4.4	5	0.9	CHR HEPAT
1173357	41	F	8	94	30	31	2.55	3.1	70	41	21	141	123	2.5	0.88	4.67	21	134	3.5	6.1	3.2	1	LIVER FAILURE
1202511	46	M	9.5	106	29	32	3.13	3	72	25	18	131	62	2.32	1.04	16.8	17.4	132	4	9.2	6	0.8	NAFLD
1170717	28	M	11.9	86	29	32	4.9	2.7	62	60	45	197	90	2.25	0.74	4.6	14.8	135	4.5	6.9	5	0.8	CHR HEPAT
1191069	46	M	7.9	110	30	34	3.5	2.6	43	35	65	152	27	2.51	1.53	25	25	123	5.55	6.7	2	0.7	Comp ALD
1187719	25	M	7.7	88	29	32	3.1	2.2	63	91	41	147	88	8.39	1.28	15	45	136	4.89	6.7	4	0.9	Comp ALD
1170540	45	F	11.6	63	24	27	4.6	2.1	37	22	18	304	41	0.95	0.56	6	13.8	137	4.22	7	7	1.5	Comp ALD
1170961	64	F	8.5	96	30	31	2.57	1.8	128	47	27	139	80	3.12	1.02	29	15.8	134	4.55	7.3	2.5	1	Comp ALD
1201679	44	M	7.5	90	30	33	2.03	1.6	10	31	29	104	41	1.92	1.15	1.36	18	133	5.7	4.8	3	0.4	Comp ALD