"COMPARISION OF HAEMATOLOGICAL PARAMETERS IN ALCOHOLICS AND NON-ALCOHOLICS"

BY

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Sub: Institutional Ethical Clearance for the study.

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LIST OF ABBREVIATIONS USED

RBC : RED BLOOD CELL

DPG : DI PHOSPHO GLYCERATE

HBA : HEMOGLOBIN A

WBC : WHITE BLOOD CELL

HB : HEMOGLOBIN.

RDW : RED BLOOD CELL DISTRIBUTION WIDTH

PCV : PACKED CELL VOLUME

MCV : MEAN CORPUSCULAR VOLUME

MCH : MEAN CORPUSCULAR HEMOGLOBIN

MCHC : MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION

LFT : LIVER FUNCTION TEST.

RFT : RENAL FUNCTION TEST

PT : PROTHROMBIN TIME

INR : INTERNATIONAL NORMALIZED RATIO

NIAAA : NATIONAL INSTITUTE ON ALCOHOL ABUSE AND ALCOHOLISM

GDP : GROSS DOMESTIC PRODUCT

AUD : ALCOHOL USE DISORDER

WHO : WORLD HEALTH ORGANIZATION

ADH : ALCOHOL DEHYDROGENASE

ALDH : ALDEHYDE DEHYDROGENASE

TNF : TUMOR NECROSIS FACTOR

ATP : ADENOSINE TRIPHOSPHATE

ALD : ALCOHOLIC LIVER DISEASE

HCC : HEPATOCELLULAR CARCINOMA

HCV : HEPATITIS C VIRUS

HSC : HEMATOPOIETIC STEM CELLS

SGOT : SERUM GLUTAMIC-OXALOACETIC TRANSAMINASE

SGPT : SERUM GLUTAMIC-PYRUVIC TRANSAMINASE

ALP : ALKALINE PHOSPHATASE

ABSTRACT

INTRODUCTION:

Alcohol abuse has increasingly become a significant public health concern among both youth and adults. There is currently limited research in India comparing hematological parameters between alcoholics and non-alcoholics. Detecting early changes in hematological parameters among chronic alcoholics and providing timely counseling and treatment for alcohol dependence can mitigate future complications associated with alcoholism, ultimately reducing morbidity and mortality rates. The objective of our study is to study the hematological manifestations among alcoholics based on the quantity and duration of alcohol intake and compare them with the non-alcoholics to the patients admitted to Dr. Prabhakar Kore Hospital and MRC, Belagavi.

METHODOLOGY:

The present one-year hospital based cross sectional Study from January 2023 to December 2023 included a total of 60 alcoholics (more than 7 standard drinks per week for women and more than 14 standard drinks per week for men) and 60 non alcoholics meeting inclusion and exclusion criteria. Following informed consent patients were subjected to laboratory work up was done.

RESULTS:

Among total 120 patients who were enrolled for the present study, 60 are alcoholic and 60 are nonalcoholic. All 120 patients were males with no female patients. Majority of our patients consume alcohol for the duration more than 10 years (56.66%) and all the 60 alcoholic patients are severe alcoholics who consume ≥14 standard drinks/week. Our study reflected statistically significant low RBC count, hemoglobin %, platelet count, PCV, mean

MCV, all parameters of liver function tests and prothrombin time in the alcoholic group while there is no positive correlation with parameters like WBC count, MCH, MCHC, blood urea and creatinine.

CONCLUSION:

Our present study results shows there is a positive correlation with the alcoholics group as far as haemoglobin %, RBC count, PCV, Platelet count, MCV, all the parameters of liver function tests and prothrombin time were concerned. There is no significant correlation with the parameters like WBC count, MCH, MCHC, blood urea and creatinine between the groups. These simple cost-effective parameters aid us in detecting haematological changes early in chronic and severe alcoholics and helps to decrease the morbidity and mortality associated with alcoholism by facilitating timely intervention and counseling for alcohol dependence.

KEY WORDS:

Haematological parameters, alcoholics, nonalcoholics.

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INTRODUCTION

In recent years, alcohol abuse is emerged as one of the important public health concerns among the youth and adults of society.[1,2] Alcohol consumption in India is amounted to about 5 billion liters in 2020 and was estimated to reach about 6.21 billion liters by 2024. [3-5] Alcohol consumption is widespread in all the states of India and an estimated 160 million consume alcohol. [3]

With increase in trend of socialization and increase in rate of social acceptability of alcohol consumption among both males and females especially in the younger generation increased the number of alcoholic consumers. As a result, the burden of alcohol related problems are also increasing trend parallel to alcohol consumption.

The consequences of alcohol use on an individual's health depend on the quantity and duration of consumption. [4,5] Heavy alcohol consumption effects the hematological parameters by effecting the various cell lineages which is either directly by bone marrow suppression[6,7] or indirectly by nutritional deficiencies seen in alcoholics because of the decreased absorption of nutrients by alcohol, decreased nutrient activation and poor dietary habits.[8,9]

Liver is the organ that is primarily affected by alcohol.[10] Alcoholic Liver disease is the cause of an increased morbidity and mortality among these people and accounts for

elevated social and economic costs.[11] The other major abnormalities that were identified with the alcohol consumption are:

(1) The restriction of cell production secondary to tissue inflammation,[7]

(2) Interference with folic acid absorption and metabolism,[12]

(3) Disruption of mitochondrial function, normal PLP metabolism, and heme synthesis, [13] and

(4) The development of membrane abnormalities which shorten cell life span.[14,15]

Need for the study

Alcoholism is a worldwide social and medical problem. As of today, very few studies have been done in India to compare the hematological parameters in alcoholics and non-alcoholics.

In this regard, this study was therefore designed to evaluate the effects of consumption of alcohol depending on both quantity and duration on cellular components of blood in the Indian population. Detecting the changes in hematological parameters among the chronic alcoholics in the earlier stage and providing appropriate counseling and treatment for alcohol dependence timely and properly will decrease the future complications associated with alcoholism and also reduce morbidity and mortality in alcoholics.

AIM AND OBJECTIVE

The objective of the study is:

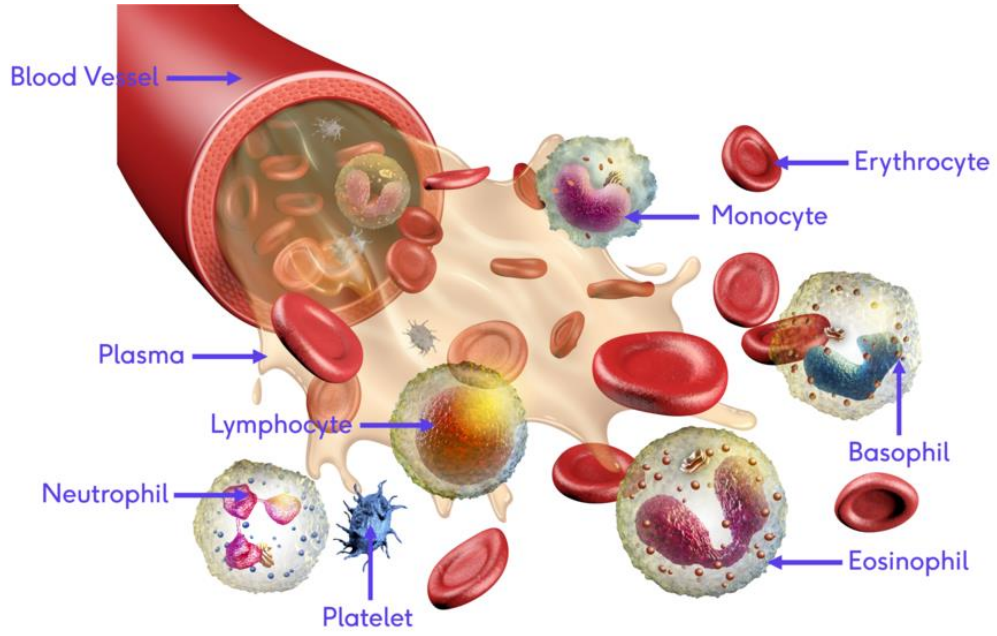
- To study the hematological manifestations among alcoholics based on the quantity and duration of alcohol intake and compare them with the non-alcoholics to the patients admitted to Dr. Prabhakar Kore Hospital and MRC, Belagavi.

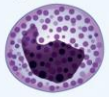
REVIEW OF LITERATURE

THE COMPONENTS AND VOLUME OF WHOLE BLOOD

Blood is a body fluid that is composed of plasma and cellular components. The cellular components being red blood cells, leukocytes and platelets remain suspended in the aqueous medium that is known as Plasma which amounts to 60% of the total blood volume. Plasma is approximately 91 percent water with solid components such as Coagulants, Plasma proteins (albumin and globulin), Electrolytes (sodium, potassium, bicarbonate, chloride, magnesium and calcium) and other components in small amounts such as enzymes, hormones, and vitamins and Immunoglobulins.[16,17]

FIGURE 1: BLOOD COMPONENTS AND THEIR FUNCTION:



Formed element	Major subtypes	Numbers present per microliter (μL) and mean (range)	Appearance in a standard blood smear	Summary of functions	Comments
Erythrocytes (red blood cells)		5.2 million (4.4–6.0 million)	Flattened biconcave disk; no nucleus; pale red color	Transport oxygen and some carbon dioxide between tissues and lungs	Lifespan of approximately 120 days
Leukocytes (white blood cells)		7000 (5000–10,000)	Obvious dark-staining nucleus	All function in body defenses	Exit capillaries and move into tissues; lifespan of usually a few hours or days
	Granulocytes including neutrophils, eosinophils, and basophils	4360 (1800–9950)	Abundant granules in cytoplasm; nucleus normally lobed	Nonspecific (innate) resistance to disease	Classified according to membrane-bound granules in cytoplasm
	Neutrophils 	4150 (1800–7300)	Nuclear lobes increase with age; pale lilac granules	Phagocytic; particularly effective against bacteria. Release cytotoxic chemicals from granules	Most common leukocyte; lifespan of minutes to days
	Eosinophils 	165 (0–700)	Nucleus generally two-lobed; bright red-orange granules	Phagocytic cells; particularly effective with antigen- antibody complexes. Release antihistamines. Increase in allergies and parasitic infections	Lifespan of minutes to days
	Basophils 	44 (0–150)	Nucleus generally two-lobed but difficult to see due to presence of heavy, dense, dark purple granules	Promotes inflammation	Least common leukocyte; lifespan unknown
	Agranulocytes including lymphocytes and monocytes	2640 (1700–4950)	Lack abundant granules in cytoplasm; have a simple-shaped nucleus that may be indented	Body defenses	Group consists of two major cell types from different lineages
	Lymphocytes 	2185 (1500–4000)	Spherical cells with a single often large nucleus occupying much of the cell's volume; stains purple; seen in large (natural killer cells) and small (B and T cells) variants	Primarily specific (adaptive) immunity: T cells directly attack other cells (cellular immunity); B cells release antibodies (humoral immunity); natural killer cells are similar to T cells but nonspecific	Initial cells originate in bone marrow, but secondary production occurs in lymphatic tissue; several distinct subtypes; memory cells form after exposure to a pathogen and rapidly increase responses to subsequent exposure; lifespan of many years
	Monocytes 	455 (200–950)	Largest leukocyte with an indented or horseshoe-shaped nucleus	Very effective phagocytic cells engulfing pathogens or worn out cells; also serve as antigen-presenting cells (APCs) for other components of the immune system	Produced in red bone marrow; referred to as macrophages after leaving circulation
Platelets 		350,000 (150,000–500,000)	Cellular fragments surrounded by a plasma membrane and containing granules; purple stain	Hemostasis plus release growth factors for repair and healing of tissue	Formed from megakaryocytes that remain in the red bone marrow and shed platelets into circulation

The total blood volume is approximately about 5 litres. It mainly depends on height, weight and sex of the individual.[18] In the human body at tissue level it acts as transporter of oxygen, replenishes nutrients and helps in the removal of the waste products of metabolism. Loss of blood supply in a tissue causes ischemia and infarction, which could have fatal effects depending upon its location.

Blood volume is meticulously regulated by interaction of various organ systems. Multiple systems are involved in producing blood and regulating its volume. The renal system, specifically the kidney, is mainly responsible for maintenance of normal blood volume. The primary job of the kidney is filtration, re-absorption, and secretion. The main factor affecting blood volume regulation is the quantity of water and solute reabsorbed. More filtrate is reabsorbed when the effective blood volume falls, and less filtrate is reabsorbed when the blood volume rises.[19]

The erythropoietin from kidneys signals the bone marrow to produce red blood cells. Hence the kidney is solely responsible for both the regulation and partial production of blood volume.[19].

The cardiovascular system detects changes in the blood volume and maintains it through increasing or decreasing arterial pressure to ensure adequate perfusion to all the organs and tissues. The central nervous system also helps in regulating blood volume by interacting with major organ systems through sympathetic activity and ultimately showing its effect on the glomerulus and blood vessels.[20]

The physiology of red blood cells

In a healthy adult, red blood cells are produced daily at the rate of approximately 0.25 ml/kg with an average lifespan of about 120 days. In contrast to this the lifespan of the transfused RBC is approximately 50-60 days, which can be further reduced depending upon metabolic, biochemical and molecular changes associated with duration of storage of blood (storage lesion) [(21)] .

The storage of blood causes depletion of 2,3-diphosphoglycerate within few days to 1-2 weeks. Upon transfusion half of it is restored by 8 hours and completely reversed by 1 to 3 days which is significant in patients requiring massive transfusion.[(22)] The initial decrease in 2,3 DPG levels contributes to decreased life span.

FIGURE 2: THE GENESIS OF RED BLOOD CELLS AND FACTORS THAT AFFECT IT

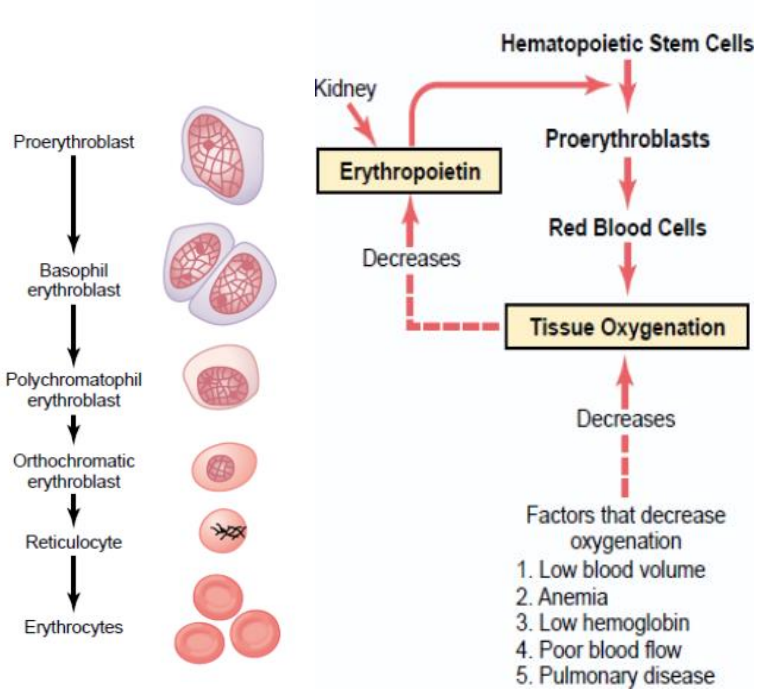
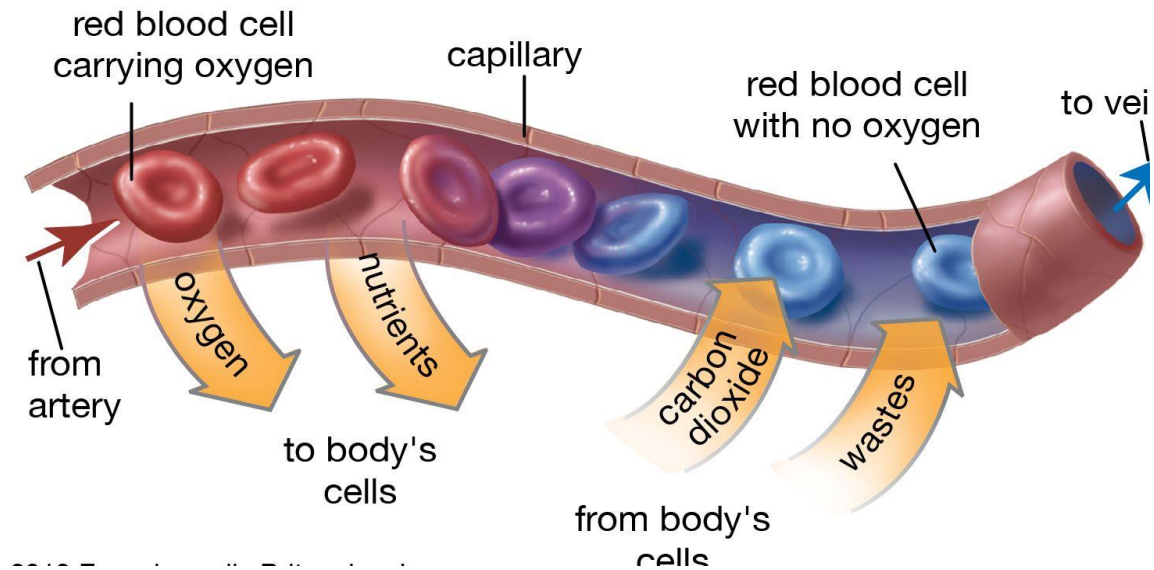


FIGURE 3: THE PHYSIOLOGY OF RED BLOOD CELLS



ERYTHROPOESIS:

Erythropoiesis is the process of development and maturation of red blood cells. All the cells that are present in the blood develop from a single progenitor stem cell. The first cell that is recognizable in the life of a red blood cell is the proerythroblast. As this cell starts to develop a nucleus is formed within it which is very small with basophilic cytoplasm filled with ribosomes. As the production of hemoglobin starts to begin within the cell the cytoplasm of the red blood cell starts to attract basic and eosin stains, called as the polychromatophilic erythroblast. [23,24]

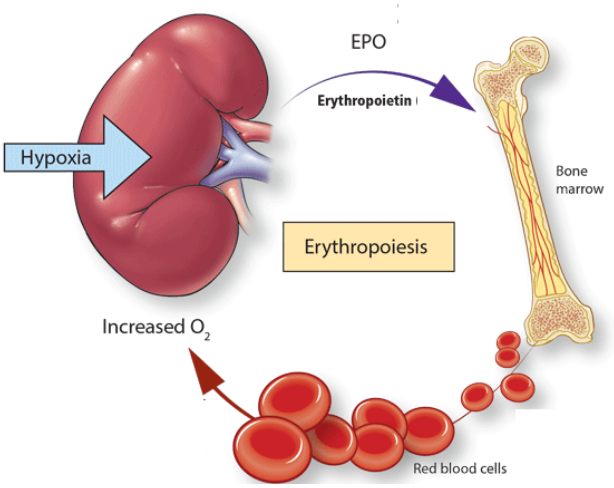
As the red blood cells further mature, the cytoplasm starts to become more eosinophilic, at this stage it is called as the orthochromatic erythroblast. [25, 26]

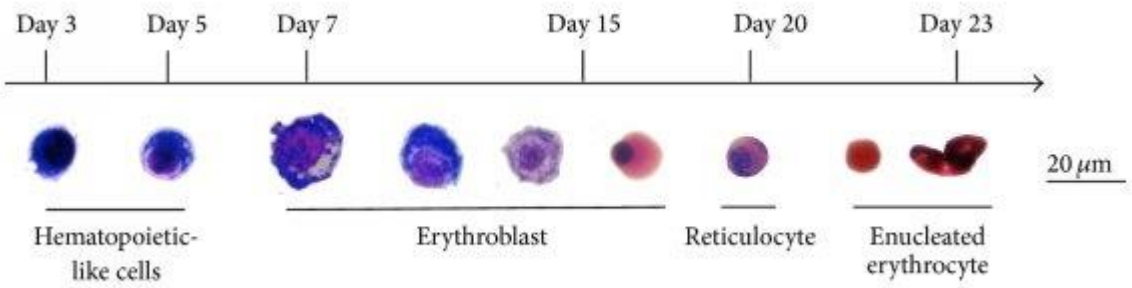
Following this stage, the nucleus is extruded, and it is called as the reticulocyte. It is at this stage the red blood cell enters the circulation. These cells are known to contain fragments and remnants of basophilic material including mitochondria and other organelles [27]. Once

the reticulocyte enters the blood stream, in order to mature it takes over 1 to 2 days. During the process the reticulocyte loses the basophilic cytoplasm of nucleus and becomes a mature red blood cell. Once the red blood cells become old and damaged, they are removed from the circulation constantly, mostly by the spleen. Following their degradation, the components of the red blood cells are recycled and used for new red blood cells formation. (27, 28)

The formation of erythrocytes also requires a constant supply of other substrates like the vitamins B12, folate and iron.[29]

FIGURE 4: ERYTHROPOESIS AT VARIOUS STAGES AND SITES





HAEMOGLOBIN:

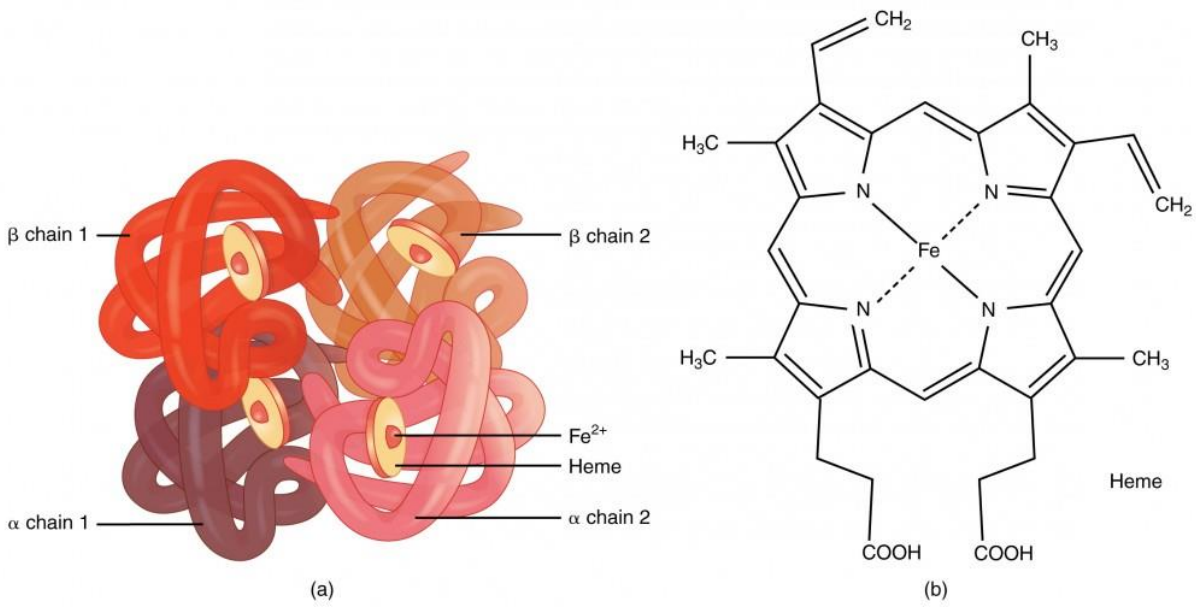
Hemoglobin is the oxygen carrying pigment of red blood cells. It carries oxygen from the lungs to the tissues and cells which plays a role in ensuring the oxidative metabolism within them. [30, 31]

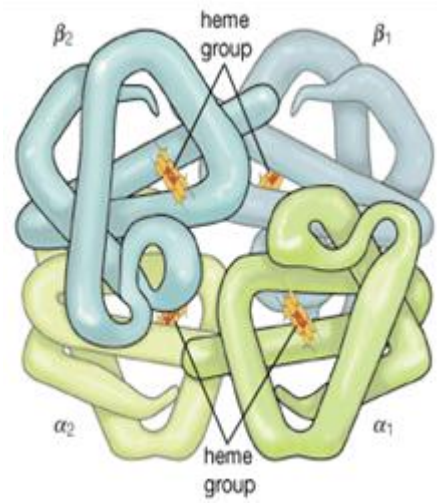
The structure of hemoglobin

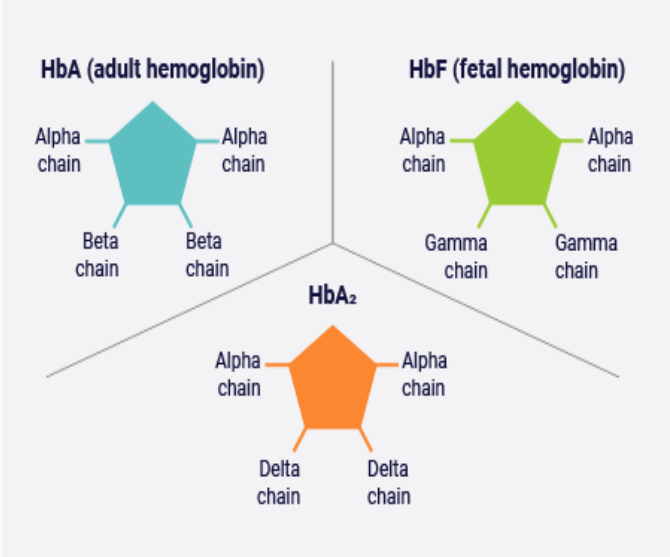
The hemoglobin is made up of haem and globin polypeptide chain tetramer. It is composed of a pair of alpha chains that are composed of 141 amino acids and a pair of beta chains that are made up of 146 amino acids. [31]

HbA is the main type in the adult hemoglobin with a structure of 2 α and 2 β chains. The human hemoglobin is encoded in a pair of tightly linked gene clusters. The alpha globin genes are located on the16th chromosome, and the beta genes are located on the 11th chromosome. [32]

FIGURE 5: HEMOGLOBIN CHAINS







HEMATOCRIT:

The hematocrit of the blood is defined as the ratio of the volume of the erythrocytes to the volume of the blood, also known as the packed cell volume (PCV).The term is derived from a Greek word(haima = blood; crit from the Greek krinein = to separate). Hematocrit is expressed in either percentage or ratio.[33,34]

The normal hematocrit (PCV) in a healthy adult is estimated to range between 40 to 48% and 60% in a new born (it takes approximately three months to reach adult level of hematocrit in newborn) . [33, 34]

Under normal circumstances there seems to be a linear relationship between the hematocrit (PCV) and the hemoglobin concentration, and it is possible to estimate the hematocrit by using the Hb, MCV and MCHC. [33, 34]

The formula that is commonly used to describe the relationship between hemoglobin MCV, MCHC and hematocrit follows: -

PCV (%) = (0.0485 × Hb (mmol/L) +0.0083) × 100

PCV (%) = MCV × RBC × 0.1

PCV (%) = Hb × 100 / MCHC

MCV (mean corpuscular volume) = average volume of red blood cells in a given sample and expressed in femtolitre.

MCHC (mean corpuscular hemoglobin concentration) is the average concentration of hemoglobin in a given volume of packed red blood cells which is expressed in grams per deciliter. The normal range for MCHC is generally between 32 and 36 g/dL. [33,34]

Low hematocrit

Indicates low number of circulating erythrocytes in blood, so there will be reduced capacity of hemoglobin to carry oxygen .[33-36]

Causes:

1) Over hydration
2) Haemorrhage
3) Chronic kidney disease
4) Haemolysis
5) Pernicious anaemia
6) Bone marrow failure
7) Auto immune diseases.

High hematocrit [33-36]

It indicates the higher level of circulating erythrocytes than normal or could be a reduction in the amount of plasma volume.

Causes:

1. Severe dehydration

2. Erythrocytosis

3. Polycythemia Vera

4. Hemochromatosis.

Alcohol

It has been noted that the consumption of alcohol in India is increasing and is because of the influence of various factors like:

- Changes that are seen in the socio economics status, [37-42]
- The alterations that have taken place in the social norms, [37-42]
- the various differences that are seen in the regional aspects, especially in the cultural background.[37-42]

In India, it is not only the branded alcohol that is increasing, but also there is an increase in the usage of illicit alcohol which makes it a very important issue in terms of the health aspect. [41]

Definitions regarding the different levels of alcohol consumption:

STANDARD DRINK:

The standard drink of alcohol refers to 12 ounces of beer (which is of 5% alcohol), five ounces of wine (which is of 12% alcohol) or 1.5 ounces of hard variety of liquor like whiskey, gin, vodka or tequila (which is of 40% alcohol). It also refers to an equilibrium of 14 gms of pure alcohol. Given that one fluid ounce equals to 30 millilitres. Thus, standard drink varies in volume based on the type of alcohol that is consumed. [43,44]

According to the amount/quantity of alcohol consumed, alcoholics are categorized as follows: [43, 44]

MODERATE ALCOHOLICS:

According to National Institute on Alcohol Abuse and Alcoholism (NIAAA) Moderate drinking is defined as up to two standard drinks per day for men and one standard drink per day for women [43,44]

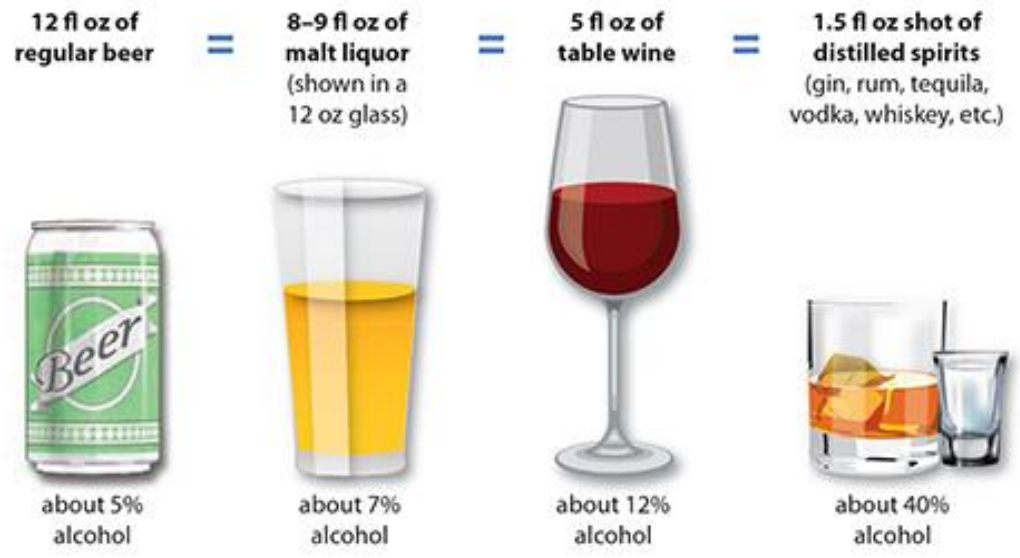
SEVERE ALCOHOLICS:

Heavy alcohol consumption is also referred to as hazardous drinking. By definition it refers to consumption of more than 14 standard drinks per week or more than four standard drinks in one setting for males and more than seven standard drinks per week or more than three standard drinks per in one sitting for females.[43,44]

BINGE DRINKING:

Binge drinking of alcohol refers to consumption of five or more than five standard drinks for men and four or more than four standard drinks for female within a span of two hours which results in a blood alcohol concentration of 0.08 grams per decilitre or greater. [43,44]

FIGURE 6 : STANDARD DRINK



Patterns of alcohol consumption at the global level:

The pattern of global alcohol consumption is influenced by number of practices, like the social norms, the cultural practises and the economic condition of the society and the financial independency of the individuals.[43,44] According to the survey done by the World Health organisation, they have stated that in the world at present, there are approximately 2.3 billion individuals who are drinkers currently. The global average alcohol consumption per capita is 6.2 litres of alcohol per year, with significant regional variations.[45] per capita alcohol consumption is highest in European countries and lowest in Middle Eastern countries due to religious beliefs. [45]

It is noted that globally the average consumption of alcohol is slightly higher in the Western countries as compared to the eastern population.[45]

EUROPEAN COUNTRIES:

In Europe studies have shown that there is highest level of consumption of alcohol and countries like Russia, Germany and the United Kingdom have a leading role to play in these statistics. This is because of the cultural traditions and the social acceptances of alcohol that have a very significant role in achieving these patterns. [45,46,47]

In the recent years, however, there has been a decline in some of the Western European countries in the patterns of alcohol consumption largely due to the alterations in the policies that have made and higher taxes that have been imposed on alcohol and stricter advertising regulations and the health awareness among the individuals [46,47,48]

UNITED STATES:

In countries like America, especially the Northern American region there is higher consumption of net alcohol levels, especially in the young. There is also a greater incidence of binge drinking among the young Americans. This is possibly because of the regional and cultural practises that contribute in a very significant way to the use of alcohol.(46)

In the Latin American regions cultural and social practises that influence the intake of alcohol especially in countries like Brazil and Mexico, that report very high levels of consumption of alcohol. (46,47)

AFRICA AND ASIA:

The African countries, as compared to the Americans and Europeans, have a much lesser degree of alcohol consumption. (48) In the Asian coordinates, there's a mixed picture with respect to the consumption of alcohol, (48,49) In countries Like South Korea and Japan, having very high consumption rates, as compared to countries like s while India and Indonesia have lower consumption rates. This is probably because of the mixed influence of religion and the cultural practise that are prevalent in that region. (51)

THE ALCOHOL CONSUMPTION PATTERN IN INDIAN SUBCONTINENT :

India being a very vast country there is a disparity in the statistics with respect to consumption of alcohol in various regions. (53-57)

- **Gender difference:** overall, the consumption of alcohol is much more in males as compared to females. According as per the National Family Health Survey – 5, that

approximately 19% of males between the ages above 15 years who consume alcohol, as compared to just 1% above 15 years in females who consume alcohol. This huge difference in the pattern of alcohol consumption in between the genders is because of the societal norms and cultural practises that discourages females from drinking.[57] But there is a growing tendency among women, particularly in cities, where drinking by women is becoming more socially acceptable and decreased social stigma. Though the alcohol consumption rate among women is increased, the alcohol abuse is less among them.[53-56]

- **AGE:** Men in the age group of 40–64 years had the greatest consumption rates, closely followed by those in the 15–39 age range.

- **High Consumption States:** In India, there are certain states like Karnataka, Tamil Nadu, Andhra Pradesh, Telangana in the South along with the north eastern states have reported high incidence of alcohol consumption .Approximately the five southern states namely, Andra Pradesh, Telangana, Kerala, Karnataka and Tamil Nadu itself account for approximal 45% of all the alcohol that is sold and consumed in India. [56,57,60,61]

- **Health Implication:** The health implications of alcohol consumption are profound. Excessive drinking is associated with over 200 diseases and injury conditions, including liver cirrhosis, various cancers, cardiovascular diseases, and mental health disorders. [62] The Global Burden of Disease Study 2016 attributes approximately 2.8 million deaths annually to alcohol use. [63]

- **Economic Costs:** Alcoholism not only shows impact on health but also causes a huge economic cost for the society by including the healthcare costs, law enforcement cost and by loss of productivity that occurs because of alcohol related problems. [59] A study published in The Lancet estimated that the economic burden of alcohol-related harm exceeds 1% of GDP in high-income countries.

- **Rising Trends:** Also, alcohol consumption in India has increased notably over the past three decades. There is a rise in the prevalence of alcohol use among men by 5.63% and among women by 5.24% from 1990 to 2017.

FACTORS DRIVING THE TRENDS:

Several factors contribute to the rising alcohol consumption in India:

1. **Economic Growth**: Increasing disposable incomes and urbanization have led to higher alcohol consumption, particularly in urban areas with greater accessibility and social acceptance.
2. **Cultural Shifts:** Changing social norms, especially among young professionals, have normalized alcohol consumption in social settings like restaurants, pubs, and fine dining establishments.
3. **Psychological Factors:** Co-occurring mental health disorders such as depression, anxiety, or trauma can increase the alcohol use.
4. **Environmental Factors:** Socioeconomic status, peer influence, cultural norms, and accessibility to alcohol can contribute.
5. **Policy and Regulation:** some states have imposed ban on alcohol sales (e.g., Bihar, Gujarat) to decrease the consumption of alcohol while few others have

liberalized regulations to boost revenue, reflecting a complex regulatory landscape.[60]

6. **Youth Consumption**: A concerning trend is the high rate of alcohol consumption among youth under 25, despite legal restrictions. Reports indicate that over 88% of this age group consumes or purchases alcohol illegally. According to reports, more than 88% of people in this age bracket buy or consume alcohol illegally.[42]
7. **Illicit Alcohol:** Despite the rise in the use of branded alcohol, the prevalence of illicit alcohol, is around 40% of the total alcohol consumed in India which poses a severe health risk. The consumption of illicit alcohol is often associated with methanol poisoning, leading to fatalities.[42]

Alcohol addiction, also known as alcoholism or alcohol use disorder (AUD), is a chronic condition characterized by compulsive alcohol consumption despite negative consequences. It is considered a complex disorder influenced by genetic, environmental, and psychological factors.[83,84]

Incidence and Prevalence: The incidence and prevalence of alcohol use disorder vary globally but are significant in many parts of the world. According to the World Health Organization (WHO), alcohol contributes to more than 3 million deaths annually and is a leading risk factor for premature mortality and disability.[85] Almost 1 in every 20 deaths are related to alcohol.

The problems associated with alcohol abuse in India

It is shown that alcohol abuse is leading to number of adverse outcomes, both on the health, the economic stages and on the social relations of the individual .It is also noted

that people who are involved in the abuse of alcohol have a higher rate of crime rate in terms of violent crimes, vehicular accidents, and crimes related to mental health.[65]

One of the most common health issues that is noted in individuals with alcohol abuse is alcoholic liver disease which range from fatty liver, alcoholic hepatitis to cirrhosis of liver and alcoholic encephalopathy.[66,67]

THE FATE OF ALCOHOL WITHIN THE HUMAN BODY:

Alcohol metabolism involves several steps primarily occurring in the liver. Here's a simplified breakdown:

Absorption: Alcohol is absorbed into the bloodstream primarily through the stomach and small intestine.

Distribution: Once enters the bloodstream, alcohol travels to various organs and tissues, affecting the central nervous system and other bodily functions.[68]

Metabolism: The liver is the primary site of alcohol metabolism. Here, alcohol undergoes two main metabolic pathways:

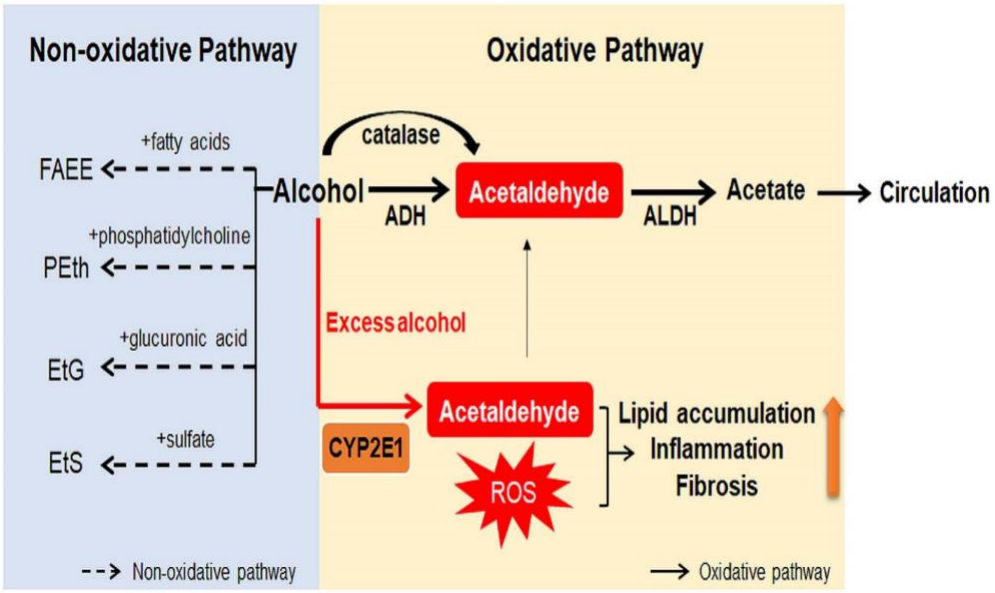
Alcohol Dehydrogenase (ADH) Pathway: This pathway converts alcohol into acetaldehyde using the primary enzyme system alcohol dehydrogenase. Acetaldehyde is a potential toxic substance that can cause various negative systemic effects.

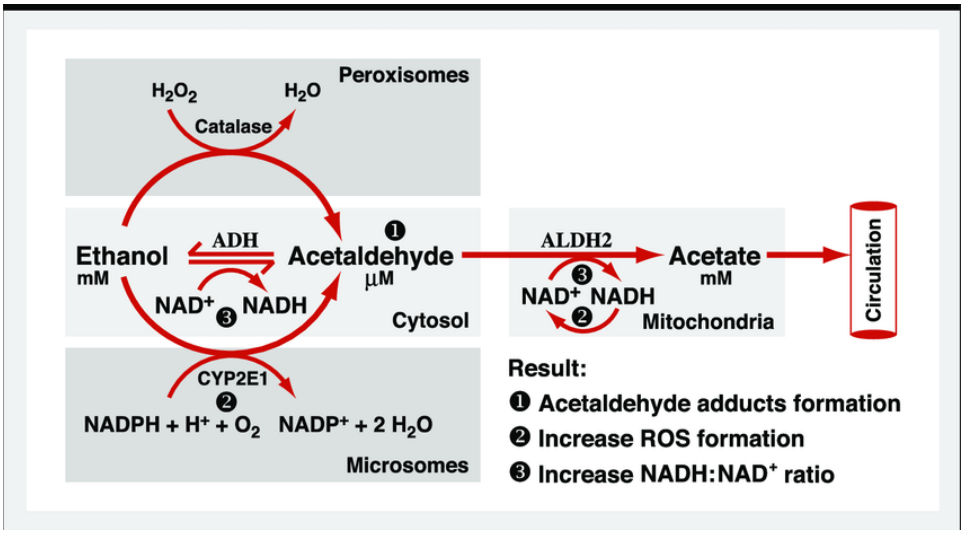
Aldehyde Dehydrogenase (ALDH) Pathway: Acetaldehyde is further metabolized into acetate by the enzyme aldehyde dehydrogenase. Acetate is then broken down into carbon dioxide and water, which can be eliminated from the body.[69]

Role of cytochrome P450: Alcohol metabolism is regulated by the cytochrome P450 2E1 (CYP2E1). Its activity is up regulated in the chronic excessive alcohol use.[70]

Elimination: The acetate that is formed is further broken metabolised into carbon dioxide and water which is eliminated from the body. The elimination of carbon dioxide and water takes place either through sweat urine or the respiratory system [69,70]

FIGURE 7: METABOLISM OF ALCOHOL





HEPATOTOXIC EFFECTS OF ACETALDEHYDE:

The pathogenesis of the hepatotoxic effects of acetaldehyde, a metabolite of alcohol, involves various mechanisms that contribute to liver damage and disease progression, Overall, the hepatotoxic effects of acetaldehyde contribute to the development of alcoholic liver diseases such as alcoholic hepatitis, fatty liver disease, and cirrhosis.[(70,71)]

Understanding the mechanisms underlying acetaldehyde-induced liver damage is crucial for the development of effective therapeutic interventions for alcohol-related liver diseases. ((70-77)

1. Formation of adducts: Acetaldehyde forms adducts with proteins and small molecules in liver cells. These impurities interfere with normal cellular processes and contribute to cellular damage.[(73)]

2. **Immune response:** The formation of these adducts triggers an immune response in the host, leading to inflammation and possibly autoimmune reactions in the liver.[(74)]

3. **Mitochondrial dysfunction:** Acetaldehyde prevents the transport of the important antioxidant glutathione into the mitochondria. This disease impairs the ability of liver cells to neutralize reactive oxygen species (ROS) and leads to oxidative stress-induced damage.[(75)]

4. **TNF-mediated hepatocyte death:** acetaldehyde-induced mitochondrial dysfunction and oxidative stress promote activation of tumor necrosis factor (TNF), a proinflammatory cytokine. TNF-mediated signaling pathways lead to hepatocyte (liver cell) death.[(76)]

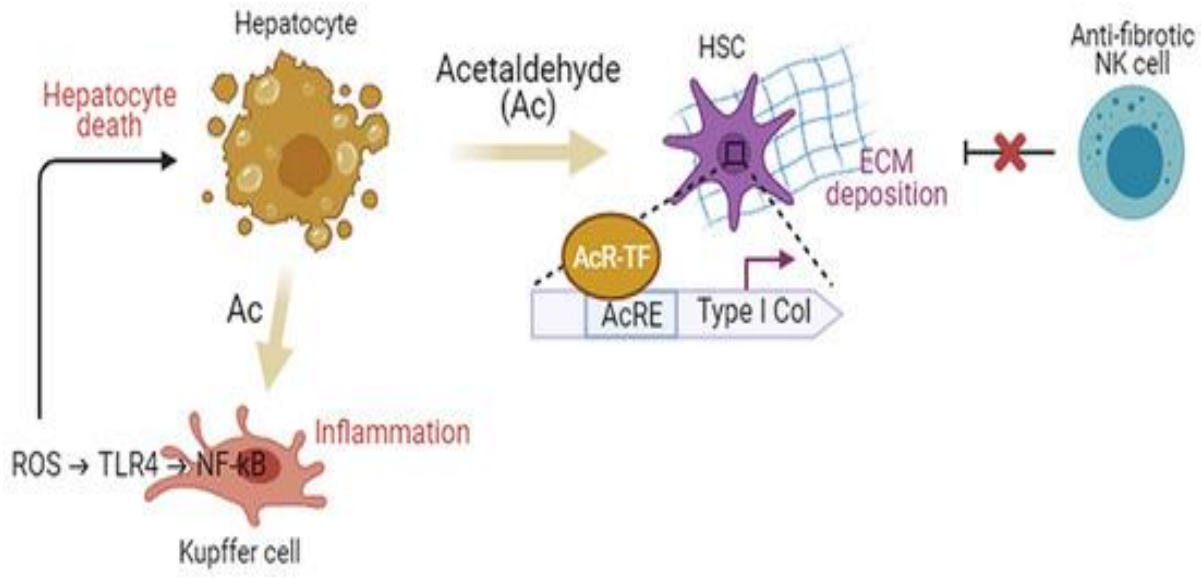
5. **Altered redox state and metabolic disorders**: oxidative stress caused by acetaldehyde, changes the redox state of liver cells and disrupts normal carbohydrate and lipid

metabolism. This disorder causes fat to accumulate in the liver cells, leading to the development of fatty liver disease.[77,79]

6. **Decreased ATP production**: Mitochondrial dysfunction caused by acetaldehyde reduces the production of adenosine triphosphate (ATP), the cell's main energy source. This reduction in ATP supply further impairs liver cell function and viability.[78]

7. **Increases collagen synthesis**: by stimulating the stellatae cells and collagen gene expression, responsible for increased collagen production.[78]

FIGURE 8: EFFECT OF ACETALDEHYDE ON THE LIVER



Alcoholic liver disease:

Alcoholic liver disease (ALD) encompasses a spectrum of liver pathologies caused by excessive alcohol consumption. It represents a significant global health burden, contributing to morbidity and mortality worldwide. ALD progresses through several stages, ranging from fatty liver (steatosis) to alcoholic hepatitis, fibrosis, cirrhosis, and ultimately, hepatocellular carcinoma (HCC). Understanding the pathophysiology, clinical manifestations, diagnosis, and management of ALD is crucial for healthcare professionals to mitigate its impact. Alcohol use disorders account for 4% of the global burden of disease. Alcoholic liver disease represents a significant public health concern with substantial morbidity and mortality. Its complex pathophysiology and diverse clinical manifestations necessitate a comprehensive approach to diagnosis and management. Efforts to raise awareness, implement effective screening strategies, and provide access to evidence-based interventions are essential in decreasing the burden of ALD and improving patient outcomes. [80-82,92]

Risk factors and disease modifiers for alcoholic liver disease:

1. **Genetics**: Certain genetic predispositions increase the risk.
    - Genetic polymorphisms in enzymes involved in alcohol metabolism, such as ADH and CYP2E1, influence susceptibility to ALD.[87]
    - In susceptible individuals who have a genetic defect, lysine replaces glutamine in ALDH, resulting in low or no ALDH activity and increased acetaldehyde levels.[87,92]
2. **Family history**: Family history strongly predisposes to alcoholic liver disease.[4,5,62]

3. **Gender** : women's relative risk of alcohol-related liver disease was significantly higher than that of men for each amount of alcohol.[4,5,88]

4. **Nutrition**:

- High consumption of unsaturated fats contributes to alcohol-induced liver damage. [89,92]
- High alcohol consumption, which causes deficiencies of micronutrients such as zinc, also contributes to the development and progression of alcohol-related liver damage[90]

5.Obesity - Obesity is also an independent risk factor for the development and progression of alcohol-induced hepatitis and cirrhosis.[4,5,91]

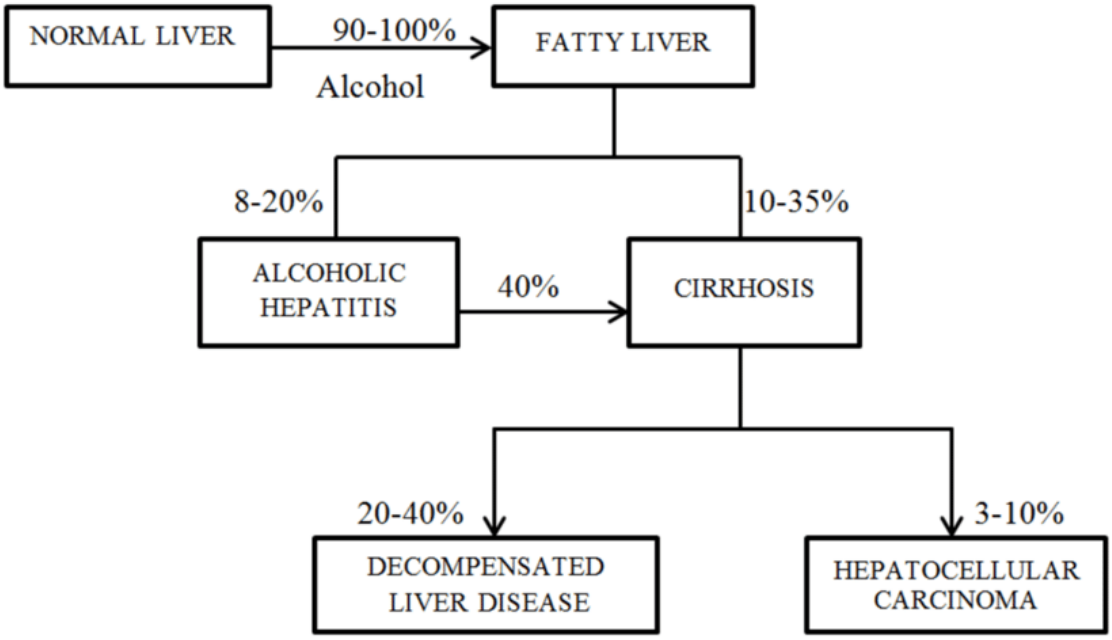
6. H**eavy smoking**. smoking increases the risk and rate of progression of alcoholic liver disease to cirrhosis and HCC.[4,5,62]

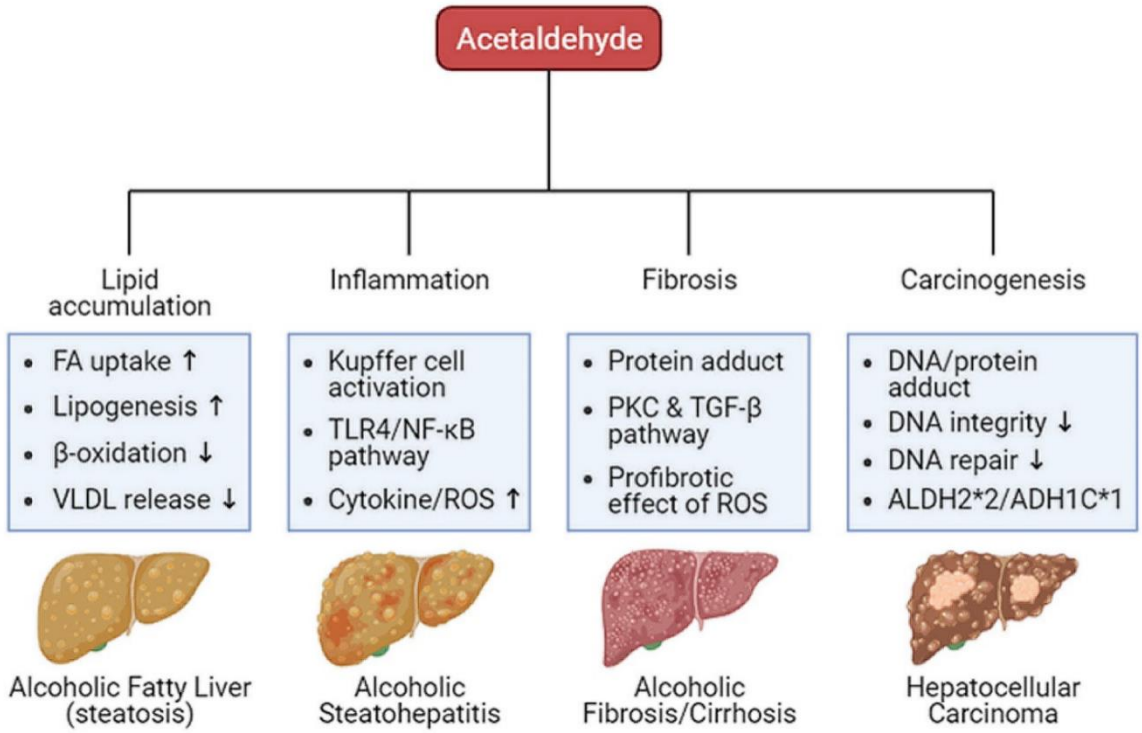
7 **Infections**: Infections related to HCV, hepatitis B and alcohol consumption further increase the progression of the disease.[4,5,62]

PATHOGENESIS:

chronic consumption of alcohol disrupts hepatic fat metabolism, leading to accumulation of triglycerides in hepatocytes, resulting in fatty liver. Acetaldehyde-induced oxidative stress and inflammation promote hepatocellular injury and apoptosis. Activation of hepatic stellate cells triggers fibrogenesis leading to liver fibrosis and eventually cirrhosis. In addition, alcohol abuse damages the intestinal barrier, allowing bacterial translocation and endotoxin release, further exacerbating liver inflammation and damage.[80-84]

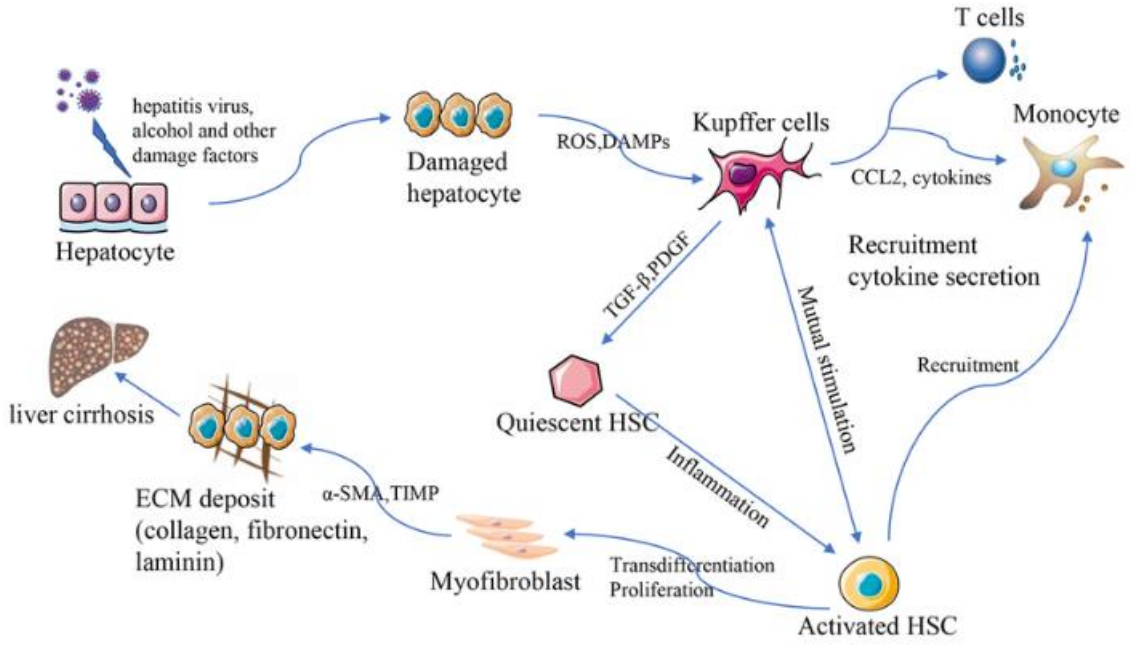
FIGURE 9: SPECTRUM OF ALCOHOLIC LIVER DISEASE:





The initiation of cirrhosis of liver is by the activation of the stellate cells that are present in the parenchyma of the liver which further activate the myofibroblast which under the influence of the transforming growth factor leads to the deposition of the collagen in the space of Disse leading to fibrosis eventually.[93,94] In the initial stages there will be only inflammation and at this stage if the trigger is identified and removed the injury is reversible. However, when there is repeated exposure to the toxin there will be recurrent apoptosis, undergoing fibrosis and regeneration process which leads to eventually cirrhosis of liver with nodule formation causing irregularity on surface of liver.[93] while inflammation of liver(hepatitis) and certain amount of fibrosis of liver are reversible, cirrhosis of liver is irreversible. [93]

FIGURE 10: PATHOGENESIS OF LIVER CIRRHOSIS



Pathogenesis of liver cirrhosis. Liver fibrosis is initiated by hepatic injury and the subsequent imbalance of ECM synthesis and degradation mediated by activated HSCs. Cirrhosis is the most advanced stage of liver fibrosis. ECM, extracellular matrix; HSCs, hepatic stellate cells; TIMP, tissue inhibitors of metalloproteinase; PDGF, platelet-derived growth factor; TGF-β, transforming growth factor-β

HISTOPATHOLOGY:

The mandatory features of classical microscopic triad to diagnose alcoholic liver disease include characteristic morphological distortions like bridging fiber septa, nodules within the parenchyma of the liver and disruption of the normal architecture of the liver. [95]

FIGURE 11: THE CLASSICAL MICROSCOPIC TRIAD

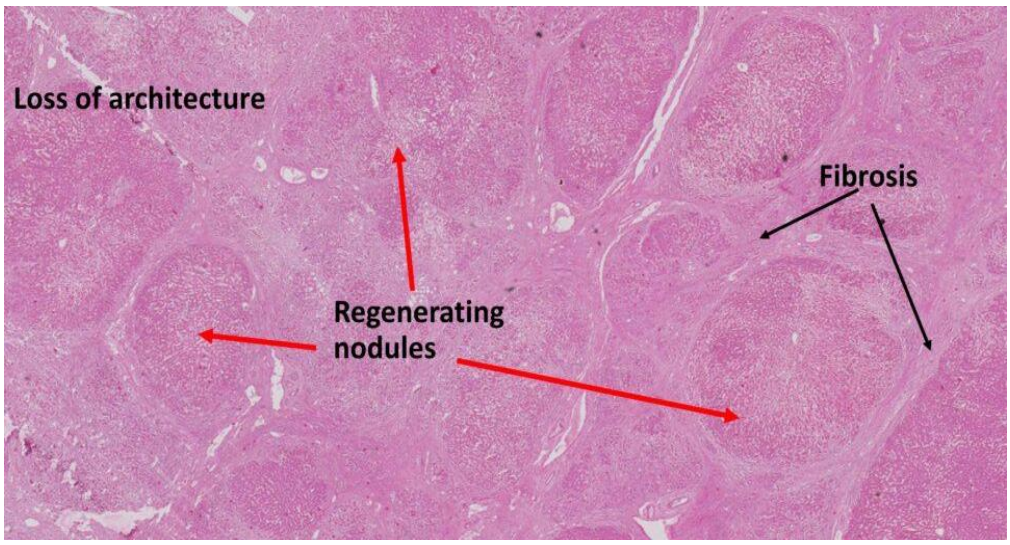
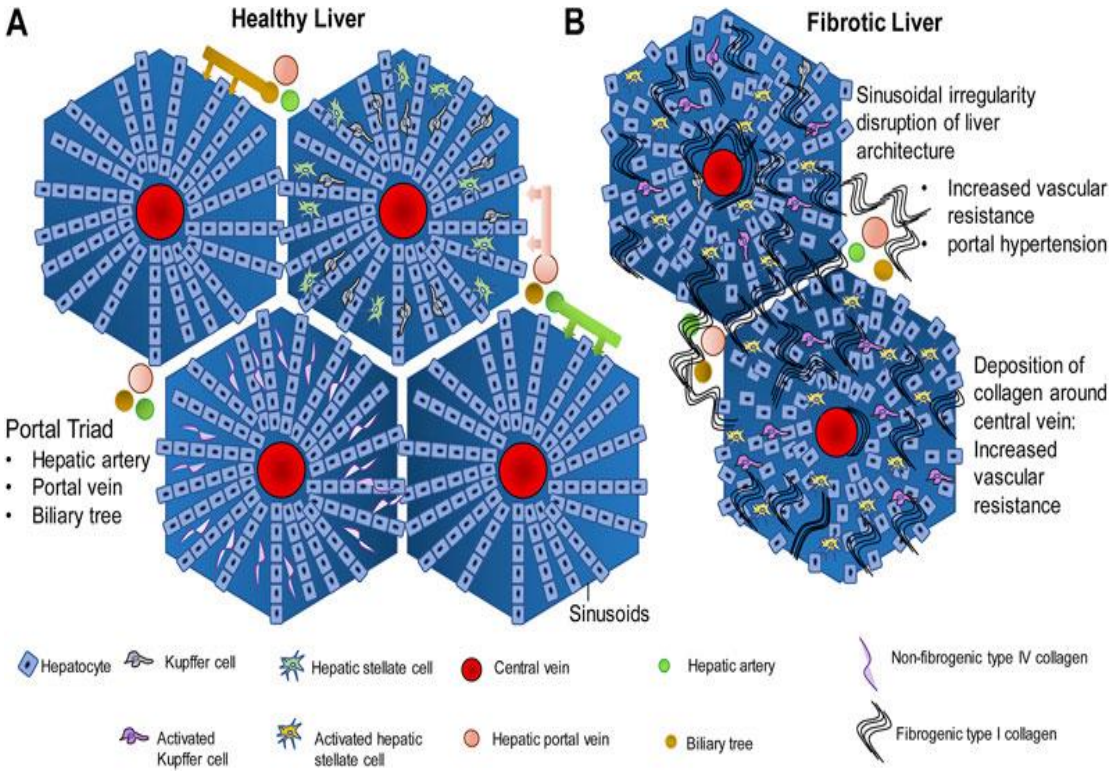
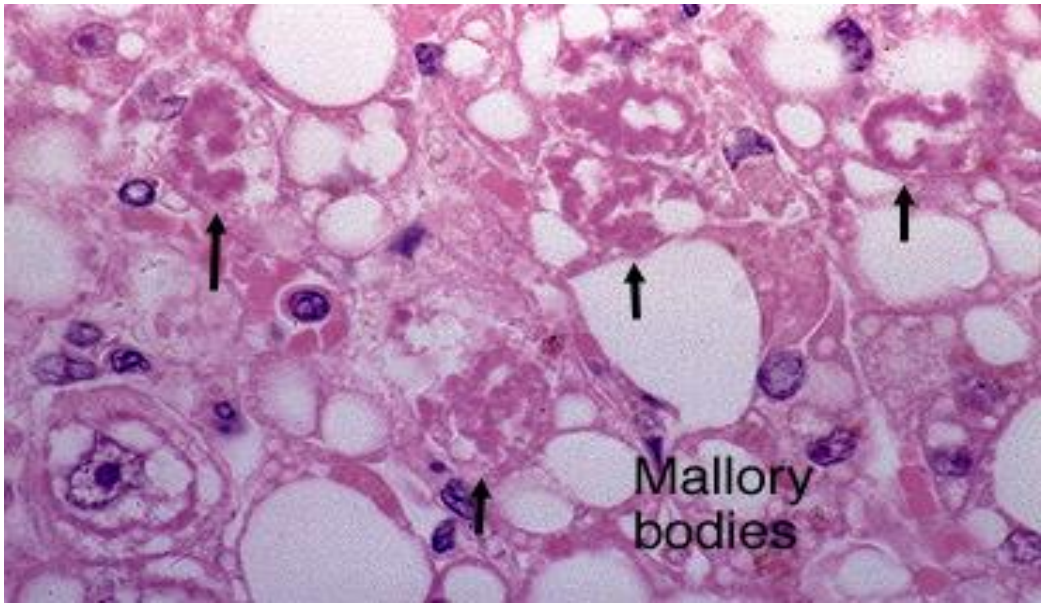


FIGURE 12: THE HEALTHY LIVER AND FIBROTIC LIVER CHANGES



Classic histologic features of alcoholic hepatitis include ballooning degeneration of hepatocytes, alcoholic hyaline (Mallory or Mallory-Denk bodies) in affected hepatocytes and surrounding neutrophilic infiltration with sinusoidal fibrosis[95]

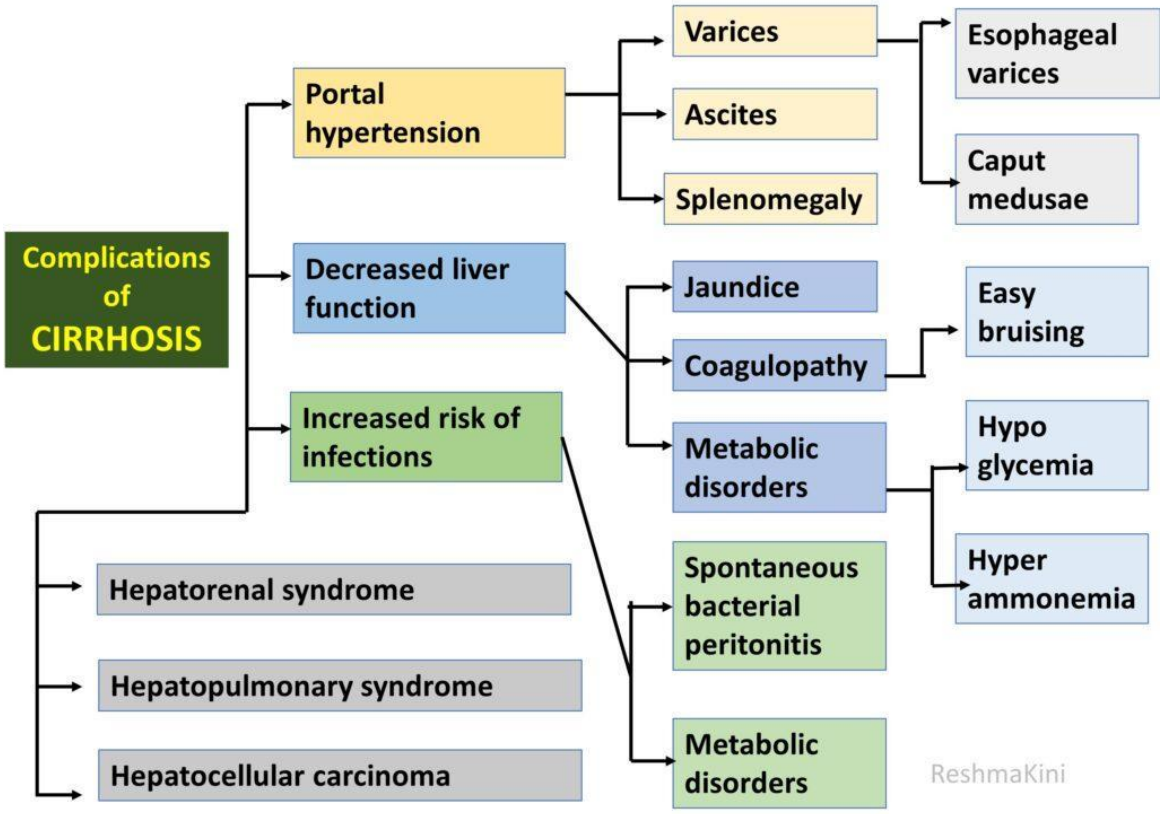
FIGURE 13: BALLOON DEGENERATION OF HEPATOCYTES WITH MALLORY-DENK BODIES



THE COMPLICATIONS OF CIRRHOSIS OF LIVER:

It is an excepted fact that, when the liver fails almost all systems of the body will be affected. This is because the liver being an organ of prime importance to the body, and it is involved in a number of functions. The major complications of cirrhosis of the liver are [96]

FIGURE 14: COMPLICATIONS OF CIRRHOSIS OF LIVER



One of the primary complications of cirrhosis is portal hypertension, which occurs when there is increased resistance to blood flow through the portal vein because of an increase in intrahepatic pressure secondary to fibrosis and cirrhosis. The development of portal hypertension leads to the formation of collateral blood vessels and results in esophageal varices, gastric varices and portosystemic shunts. These vessels are prone to rupture, causing life-threatening gastrointestinal bleeding[96]

EFFECT OF ALCOHOL ON VARIOUS BLOOD CELL COMPONEMTS

Alcohol consumption can have profound effects on various cells in the bloodstream, impacting their function and potentially leading to systemic consequences. Understanding these effects is crucial for comprehending the hematological manifestations of alcohol abuse.[7]

1. Alcohol and Red Blood Cells (RBC):

A. MACROCYTOSIS:

Alcohol abuse can cause macrocytosis, which is characterized by an increased medium corpuscular erythrocyte volume (MCV) seen more often. It is observed in chronic alcoholics due to impaired DNA synthesis of erythropoietic cells.[29] Chronic alcohol consumption can interfere with the absorption of folate and vitamin B12, which are necessary for erythropoiesis. It can also be caused by the direct toxic effect of alcohol on the bone marrow, and it is reversible once there is abstinence of alcohol for 2 to 4 months[7]

B. ANEMIA:

Anemia in alcoholics can be because of [97-100]:

- Nutritional deficiency – Folic acid and iron deficiency.[29]
- Structurally abnormal RBCs can undergo premature or accelerated destruction (hemolysis).
- Direct toxic of effect of alcohol by suppression of bone marrow
- Chronic gastrointestinal bleeding as a result of varices, hemorrhoids.
- Hypersplenism resulting in RBC destruction.

- Hepatic dysfunction.
- Altered coagulation profile.

2. Alcohol and White Blood Cells (WBC):

Chronic alcohol consumption suppresses the immune system, causing leukopenia, a decrease in the total number of white blood cells. This effect may be due to a direct toxic effect of alcohol on the bone marrow or damage to granulopoiesis. Conversely, acute alcohol consumption can cause a transient increase in white blood cells, especially neutrophils, due to stress-induced release of cortisol and catecholamines.[97] Alcohol abuse is associated with an increased risk of infection due to a weakened immune system, which predisposes people to bacterial, viral and fungal infections. [7,97]

3. Alcohol and platelets:

Chronic alcohol consumption can cause thrombocytopenia, a decrease in the number of platelets that can be caused by alcohol-induced bone marrow suppression or splenic sequestration. Chronic Alcoholism can impair platelet function and coagulation pathways, which promotes a prothrombotic state and increases the risk of bleeding. [7,97,99]

4. Hematopoietic Stem Cells (HSCs):

Chronic exposure to alcohol can disrupt hematopoiesis by altering the microenvironment of the bone marrow and impairing hematopoietic stem cell (HSC) function. This can lead to irregular production of blood cells, which contributes to the hematological abnormalities seen in alcoholics.[7,97]

5. Endothelial cells:

Chronic alcohol consumption can damage the endothelial cells lining blood vessels, causing endothelial dysfunction and impaired vasodilation. This effect can contribute to the development of hypertension, atherosclerosis and cardiovascular disease associated with alcohol abuse. Alcohol and its effect on red blood cell indices.[7,97]

Alcohol consumption significantly effects red blood cell (RBC) indices, which are important markers used in the evaluation of various hematological conditions. Here is how alcohol affects these indices:

1. Mean Blood Cell Volume (MCV):

MCV means the average volume of individual red blood cells and is measured in femtoliters(fl). Chronic alcohol consumption is often associated with an increase in MCV known as macrocytosis. Macrocytosis in alcoholics is mainly due to damage to DNA synthesis during erythropoiesis due to a lack of folate and vitamin B12, important co-factors in nucleotide synthesis. High MCV levels are often observed in the early stages of alcohol abuse and may be an early sign of alcohol-related hematological abnormalities. .[29,97-100]

2. Mean Corpuscular Hemoglobin (MCH) and Mean Corpuscular Hemoglobin Concentration (MCHC):

MCH represents the average amount of hemoglobin per red blood cell, while MCHC represents the concentration of hemoglobin in the red blood cells.[97-100] Chronic alcohol consumption usually does not significantly change MCH or MCHC values, since these parameters are mainly influenced by

hemoglobin concentration which is relatively unaffected by alcohol consumption.

3. Red blood cell distribution width (RDW):

RDW measures the variation in size of red blood cells (anisocytosis) and is expressed as a percentage. Larger RDW values indicate greater variability in red blood cells. Chronic alcohol abuse can cause an increase in RDW, reflecting the presence of both macrocytic and normocytic erythrocytes in the peripheral blood.[97-100]

Literature survey of the present study

Alcoholism is seen predominantly in low socioeconomic status. The accurate method for detection and monitoring of alcohol related problems is not available and there is no exact clinical finding or symptom in a patient history, that is sufficiently sensitive and specific to detect alcohol related problem in its early phase. The reasons for using biological laboratory markers are that they give objective information about alcohol consumption. Alcohol adversely affects the production and function of virtually all types of blood cells.[97-100]

Ballard H.S et al. alcohol is directly toxic to the bone marrow as well as to the mature cells in the blood stream.[97]The red cell features can be ascertained using hemoglobin, PCV, RBC count, MCV, MCHC, MCH and RDW. The MCV defines the size of the red blood cells and is in the range 87±7 fl or μm^3. The MCH measures the amount of hemoglobin per RBC which ranges from 29±2pg/ cell. The MCHC indicates hemoglobin content with cell volume, and it ranges between 34±2 g/dl. The RDW represents the coefficient of variation of the red blood cell volume distribution (size) and its normal value is between 13±1.5%.

Mean corpuscular volume (MCV), an index of red blood cell size, increases with excessive alcohol intake after 4 to 8 weeks. The sensitivity of MCV is too low to justify its use as a single indicator. However, it has higher specificity compared with other tests.[97]

Latvala et al conducted study on 144 adult patients with objective effect of alcohol consumption on complete blood cell counts, morphological review of peripheral blood and bone marrow, markers of liver status. There was statically significant difference in incidence of anemia, which was 51% in the alcohol abusers, as compared with 69% of the nonalcoholic ($p < 0.05$). Both mean cell volume of erythrocytes (macrocytosis; 67 vs. 18%; $p < 0.0001$) and mean cell hemoglobin (63 vs. 22%; $p < 0.0001$) were significantly elevated in the alcoholics than in the nonalcoholics.[98]

Elanchezhian et al conducted a cross-sectional study for a period of one year that Hb%, Mean RBC count, mean MCH, MCHC were normal among the non-alcoholic group and it started decreasing among moderate alcoholics and more so with severe alcoholics and a similar type of result was also seen with total count and platelet count and the difference was found to be statistically significant. MCV was found to be very high in severe alcoholics when compared to moderate and non-alcoholics. LFT, RFT and prothrombin time were found to be elevated among alcoholics group when compared to non-alcoholics and the difference was statistically significant.[99]

Das et al compared hematological parameter of patient with non-alcoholic fatty liver disease and alcoholic liver disease. Among patients with alcoholic liver disease hemoglobin, red blood cell and and platelet count were significantly reduced, while the mean corpuscular volume, mean corpuscular haemoglobin and prothrombin time expressed as an international normalized ratio (PT/INR) were significantly elevated.[100]

Akanni et al found PCV to be elevated among alcoholics, which he attributed to dehydration and hemoconcentration.(101)

Thomas E et al found RBC count, hemoglobin and hematocrit to be inversely proportional and MCV as well MCH to be directly proportional to alcohol consumption.[8] Difference in data from various studies and lack of studies from South India has prompted us to conduct this study.(102)

Dr. Dharmesh Gamit et al.(103) conducted a retrospective study for a period of 1 year. Total bilirubin, SGOT, SGPT, ALP were increased in Alcoholics as compared to non-alcoholics and it was statistically highly significant. There was fall in concentration of the total protein and albumin level in the alcoholics.(103)

Whitehead et al studied the effects of cigarette smoking and alcohol consumption on blood haemoglobin. They found that alcohol had no effect on the haemoglobin and PCV. They found a positive correlation between mean corpuscular volume and alcohol consumption.(104)

METHODS OF DATA COLLECTION

Sources of data :

Patients (moderate to severe alcoholics and nonalcoholics) admitted to the KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from 1st January 2023 to 31st December 2023 over a period of one year..

Study Design: A hospital based study.

Study Period: JANUARY 2023 TO DECEMBER 2023.

Sample Size:

n= 2S2(Z1-□+Z1-□) / d2

Where, Z1-□ = Z value for alpha level (1.96 at 5% □ error or 95 % confidence)

Z1-□ = Z value for beta level (1.282 at 10% □ error or 90% power)

S=pooled SD = (S1 + S2)/2

d - Margin of error = 1.09

Sample size n = 54 should be taken in each group (alcoholic and nonalcoholic group) but for the purpose of making calculations convenient 60 will be taken in each group.

Inclusion criteria:

1. Age > = 18 years.
2. All adult patients with history of moderate to severe alcohol consumption (more than 7 standard drinks per week for women and more than 14 standard drinks per week for men where 1 standard drink = 14 gms of pure alcohol = 360 ml of beer = 120 ml of wine = 45 ml of distilled spirits.)
3. 60 adults who are non-alcoholic taken as controls.

Exclusion criteria:

1. All patients who are less than 18 years.
2. Patients with other hepatic disorders.
3. Patients receiving hepatotoxic drugs.
4. Previous history of hematological malignancies.
5. Chronic illness such as tuberculosis.
6. Patients taking hematinics.
7. Patients with chronic kidney disease
8. Patients with bleeding disorders.

ETHICAL CONSIDERATION: the present study was approved by institutional committee of human ethics. All the patients participated in the study were explained about the risks and benefits of the study and informed consent was obtained.

SAMPLE METHOD: Hospital based study; all patients fulfilling the inclusion criteria were included in the study.

DATA COLLECTION TOOL: all the data of 120 patients that was collected was documented in the study proforma.

METHODOLOGY:

- A one year Hospital based cross sectional Study from January 2023 to December 2023 at KLE's Prabhakar Kore Hospital and Medical Research Centre, Belagavi.
- Patients above the age of 18 years of age fulfilling the inclusion and exclusion criteria will were included in the study.

- These subjects were categorized based on consumption of alcohol (more than 7 standard drinks per week for women and more than 14 standard drinks per week for men) or not.
- An informed consent was obtained from all the subjects.
- Blood was drawn for performing investigations to compare and analyze the hematological parameters among both the groups.

Statistical measures:

The information collected was entered in a master chart in Microsoft Excel sheet and was analyzed by SPSS version 27.00. For categorical variables descriptive statistics was used which consisted of mean, standard deviation and variance. For analyzing the qualitative data Chi square test, T test sensitivity and specificity was done. For comparision of continuous variables, ANOVA was applied and p value <0.05 was considered as a statistically significant.

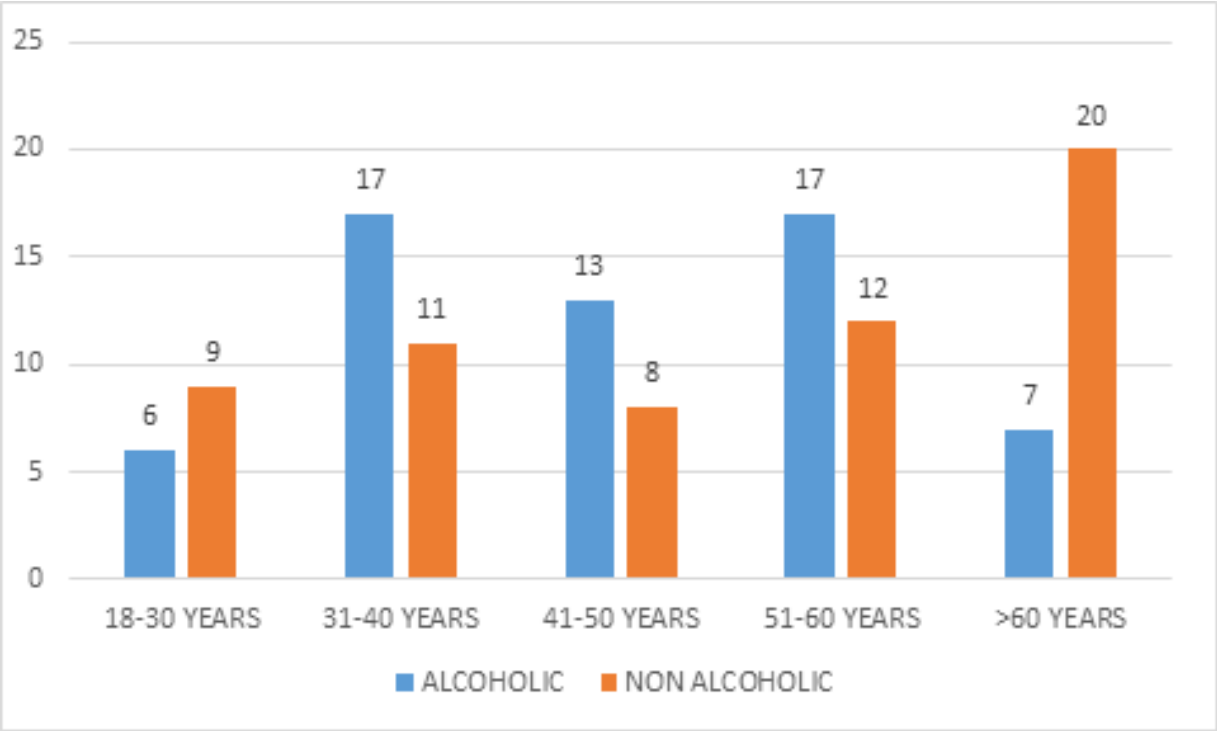
RESULTS

The present study was conducted in the department of General Medicine KLE's Dr. Prabhakar Kore hospital and MRC Belagavi from January 2023 to December 2023. A total of 120 cases were studied and findings observed are tabulated below:

TABLE 1: AGE WISE DISTRIBUTION OF PATIENTS:

AGE GROUP	**ALCOHOLIC**		**NON ALCOHOLIC**	
	No of patients	**% of patients**	**No of patients**	**% of patients**
18-30 YEARS	6	10. 00%	9	15. 00%
31-40 YEARS	17	28. 30%	11	18. 30%
41-50 YEARS	13	21. 70%	8	13. 30%
51-60 YEARS	17	28. 30%	12	20. 00%
>60 YEARS	7	11. 70%	20	33. 30%
TOTAL	**60**	**100%**	**60**	**100%**
MEAN ± SD	46. 82 ± 12. 94		51. 2 ± 18. 26	
P value	0.12			

GRAPH 1: AGE WISE DISTRIBUTION OF PATIENTS



In our present study among 120 patients the age ranged from 18-75years. The younger patient was 18 year old while the eldest was 75.

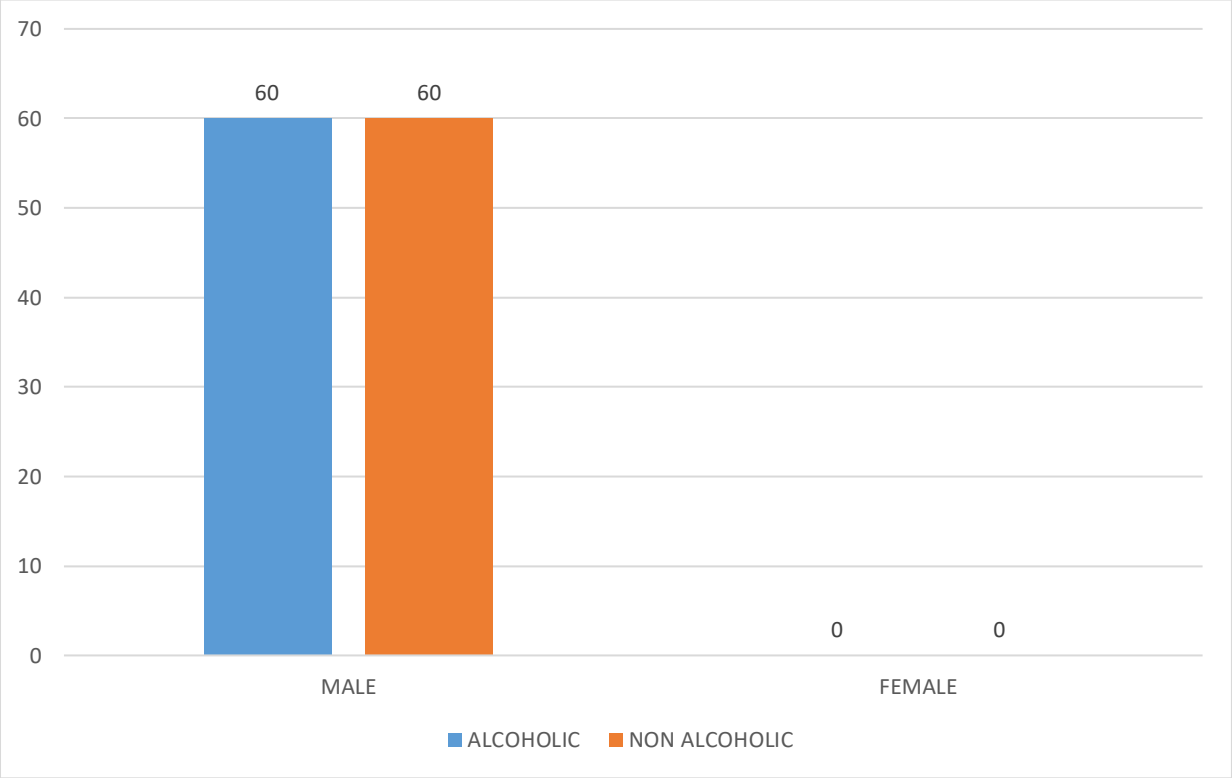
In alcoholic group we observed 17 (28. 30%) cases each in the age groups 31-40 years and 51-60 years, 13 (21. 70%) cases in the age group of 41-50 years, 7 (11.70%) in the age group of > 60 years and only 6 (10. 00%) cases in the age group of 18-30 years. A total of 60 patients were there in the alcoholic group.

In nonalcoholic group where there were 60 patients, 20 (33.30%) cases were in the age group > 60 years, 12 (20%) cases in the age group 51-60 years, 11 (18. 30%) cases in the age group 31-40 years, 9 (15. 00% %) cases in the age group 18-30 years and only 8 (13.30%) in 41-50 years. There was no statistically significant difference in the age between the groups with a p value more than 0. 05.

TABLE 2: GENDER WISE DISTRIBUTION OF PATIENTS:

GENDER		ALCOHOLIC	NON ALCOHOLIC	TOTAL
Number	MALE	60	60	120
	FEMALE	0	0	0
Percentage (%)	MALE	100. 00%	100. 00%	100. 00%
	FEMALE	0. 00%	0. 00%	0. 00%

GRAPH 2: GENDER WISE DISTRIBUTION OF PATIENTS



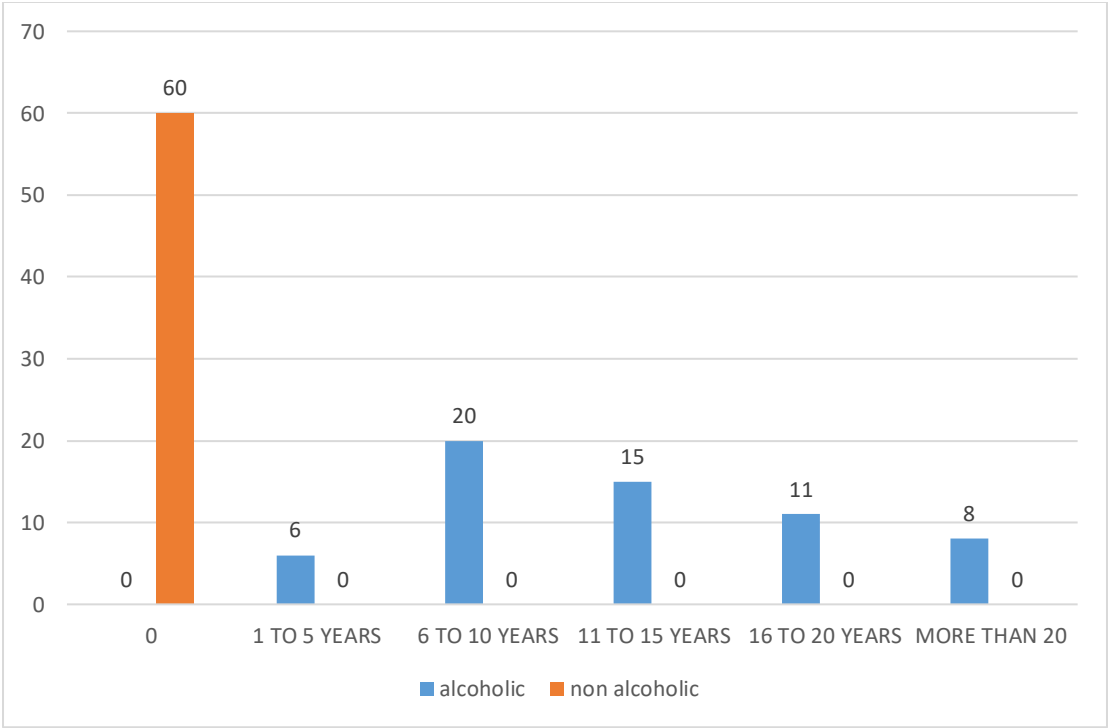
In our present study of 120 patients in both alcohol and non-alcohol group's only male patients were present, 60 in each group. There were no female patients in our study.

Inference: all 120 patients were male**.**

TABLE 3: DISTRIBUTION OF PATIENTS BASED ON DURATION OF ALCOHOL INGESTION (N=60)

Duration Of Alcohol Intake	Number of Alcoholics	% of Alcoholics
0	0	0. 00%
1 To 5 Years	6	10. 00%
6 To 10 Years	20	33. 33%
11 To 15 Years	15	25. 00%
16 To 20 Years	11	18. 33%
More Than 20	8	13. 33%
Total	60	100. 00%
MEAN ± SD	14. 48 ± 7. 57	

GRAPH 3: DISTRIBUTION OF PATIENTS BASED ON DURATION OF ALCOHOL INGESTION



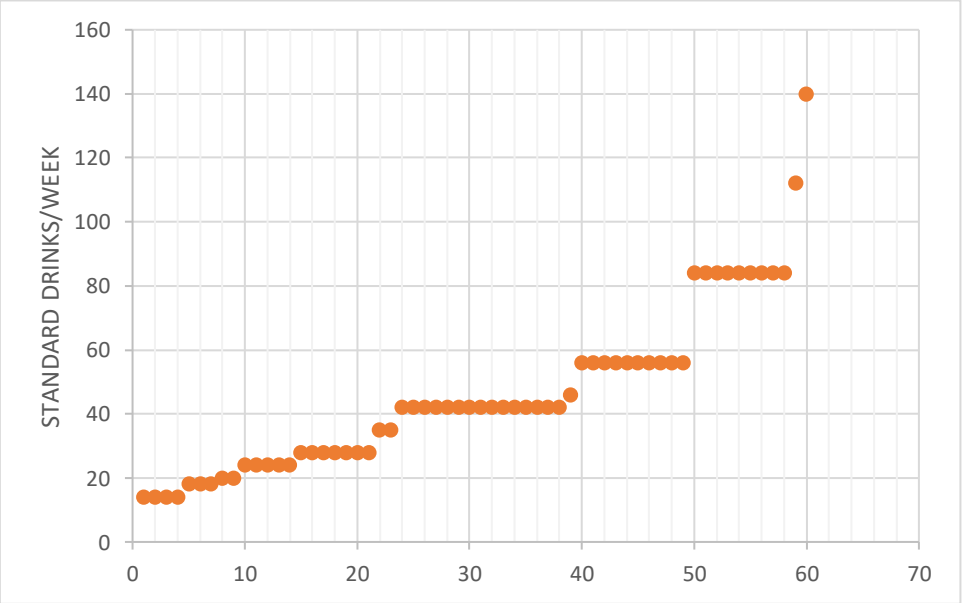
The above table depicts the duration of alcohol intake among the alcohol group (N=60). There were total of 60 patients who were alcoholic.

In our present study of alcoholics (N=60) there were 20 (33.33%) patients who are drinking for 5- 10 years, 15 (25.00%) patients for 10-15 years, 11 (18.33%) patients for 16-20 years and 8 (13.33%) patients for >20 years.

TABLE 4: DISTRIBUTION OF PATIENTS BASED ON ALCOHOL CONSUMPTION / WEEK:

AMOUNT	NUMBER OF PATIENS
< 14 STANDARD DRINKS /WEEK	0
≥ 14STANDARD DRINKS/WEEK	60
TOTAL	60 (100%)

GRAPH 4: DISTRIBUTION OF PATIENTS BASED ON ALCOHOL CONSUMPTION/WEEK

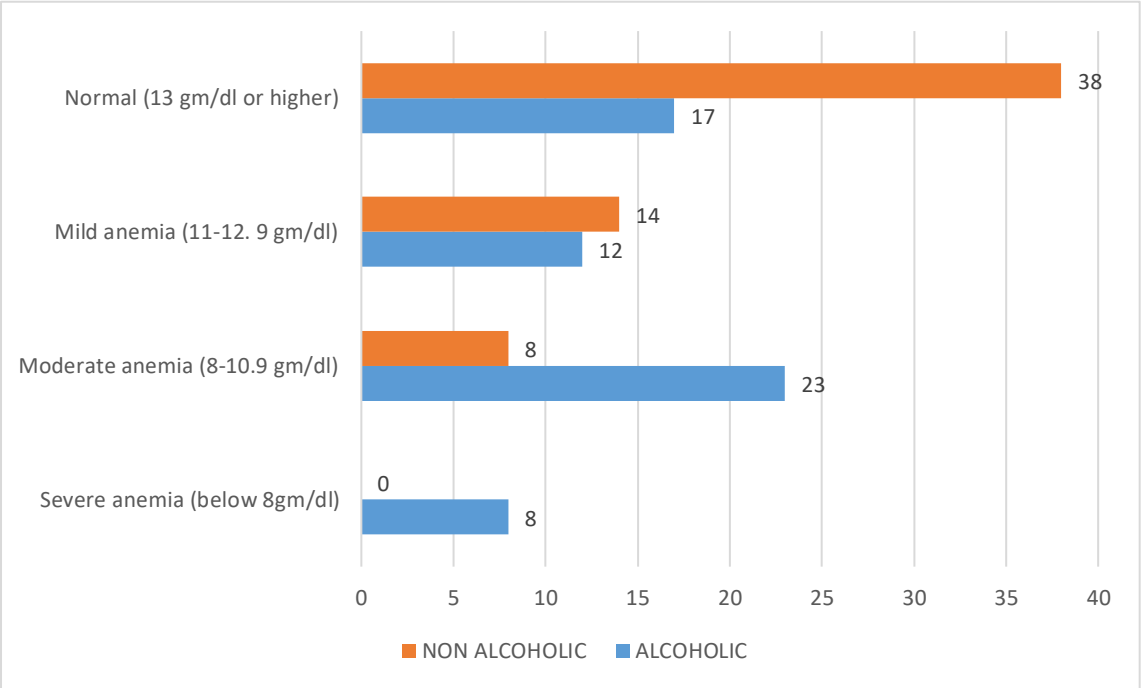


In our present study, all the 60 patients were severe alcoholics according to the National Institute of Alcohol Abuse and Alcoholism (NIAAA) who met the criteria of consuming ≥14 standard drinks/week.

TABLE 5: DISTRIBUTION OF PATIENTS BASED ON HEMOGLOBIN PERCENTAGE:

STUDY GROUP	WHO GRADING				Total
	Severe anemia (below 8gm/dl)	Moderate anemia (8-10.9 gm/dl)	Mild anemia (11-12. 9 gm/dl)	Normal (13 gm/dl or higher)	
ALCOHOLIC	8	23	12	17	60
	13. 3%	38. 3%	20. 0%	28. 3%	100. 0%
NON ALCOHOLIC	0	8	14	38	60
	0. 0%	13. 3%	23. 3%	63. 3%	100. 0%
TOTAL	8	31	26	55	120
	6. 7%	25. 8%	21. 7%	45. 8%	100. 0%

GRAPH 5: DISTRIBUTION OF PATIENTS BASED ON HEMOGLOBIN PERCENTAGE



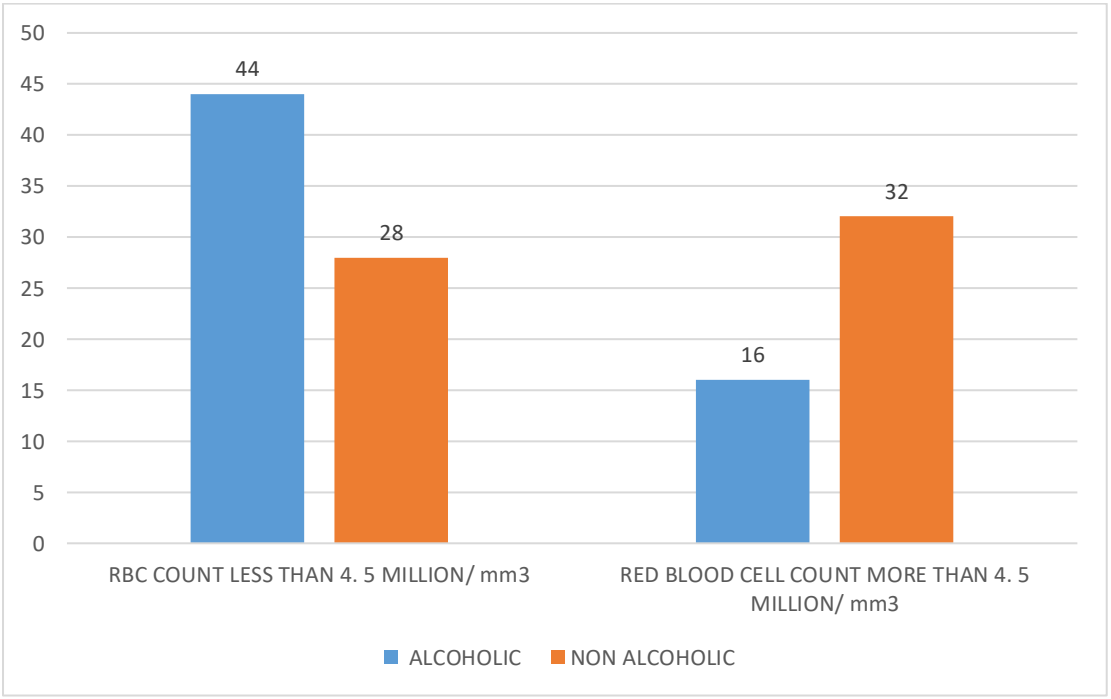
The above table depicts various haemoglobin percentages in both the groups of patients and same was categorized based on severity of anaemia.

In the alcoholic group, 17 (28. 3%) patients had normal haemoglobin, 12 (20. 0%) had mild anaemia, 23 (38. 3%) had moderate anaemia and only 8 (13. 3%) had severe anaemia. Whereas in the nonalcoholics group there were more number of patients who had normal haemoglobin i.e., 38 (63. 3%), while mild anaemia in 14 (23. 3%), moderate anaemia in 8 (13. 3%) and none had severe anaemia.

TABLE 6: DISTRIBUTION OF PATIENTS BASED ON RBC COUNT:

STUDY GROUP		RBC COUNT LESS THAN 4. 5 MILLION/ mm^3	RED BLOOD CELL COUNT MORE THAN 4. 5 MILLION/ mm^3	Total
Alcoholic	**Number**	44	16	60
	%	73. 3%	26. 7%	100. 0%
Non alcoholic	**number**	28	32	60
	%	46. 7%	53. 3%	100. 0%
TOTAL		72	48	120

GRAPH 6: DISTRIBUTION OF PATIENTS BASED ON RBC COUNT



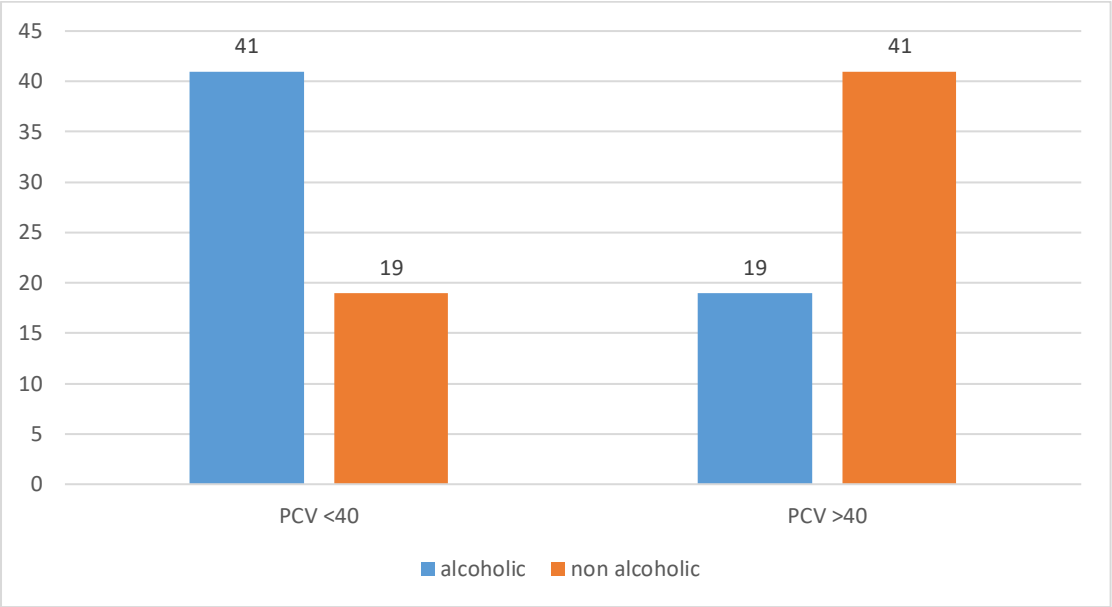
Similarly patients of both groups were categorized based up on the red blood cell (RBC) count. In alcoholics group, there were 44 (73. 3%) patients who had RBC count less than

4. 5 million/ mm^3 while remaining 16 (26. 7%) had a normal red blood cell count more than 4. 5 million/ mm^3. In nonalcoholics group, 28 (46. 7%) had RBC count less than 4. 5 million/ mm^3 while 32 (53. 3%) had a normal red blood cell count more than 4. 5 million/ mm^3.

TABLE 7: DISTRIBUTION OF PATIENTS BASED ON PACKED CELL VOLUME (PCV):

Study group		**PCV <40**	**PCV >40**	**Total**
alcoholic	**Number**	41	19	60
	%	68. 30%	31. 70%	100. 00%
Non alcoholic	**Number**	19	41	60
	%	31. 70%	68. 300%	100. 00%
Total		60	60	120

GRAPH 7: DISTRIBUTION OF PATIENTS BASED ON PACKED CELL VOLUME (PCV)

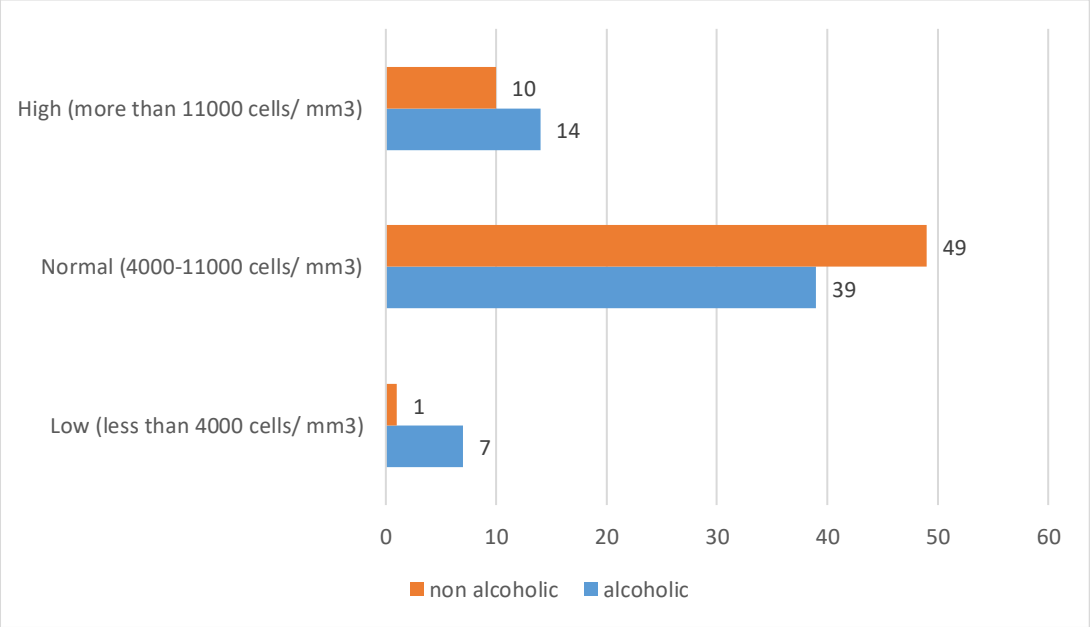


The above table no: 7 depicts distribution of patients based on packed cell volume (PCV). In the alcoholics group there were 41(68. 30%) patients whose PCV was below 40, while 19 (31. 70%) patients had above 40. In the nonalcoholics group 41 (68. 30%) had PCV above 40 and only 19 (31. 70%) patients had less than 40. There is a statistically significant difference between the groups with a p value <0. 001**.**

TABLE 8: DISTRIBUTION OF PATIENTS BASED ON TOTAL LEUCOCYTE COUNT (WBC):

Study group		Low (less than 4000 cells/ mm3)	Normal (4000-11000 cells/ mm3)	High (more than 11000 cells/ mm3)	Total
alcoholic	Number	7	39	14	60
	%	11. 70%	65. 00%	23. 30%	100. 00%
Non alcoholic	Number	1	49	10	60
	%	1. 70%	81. 70%	16. 70%	100. 00%
Total		8	88	24	120

GRAPH 8: DISTRIBUTION OF PATIENTS BASED ON TOTAL LEUCOCYTE COUNT (WBC)

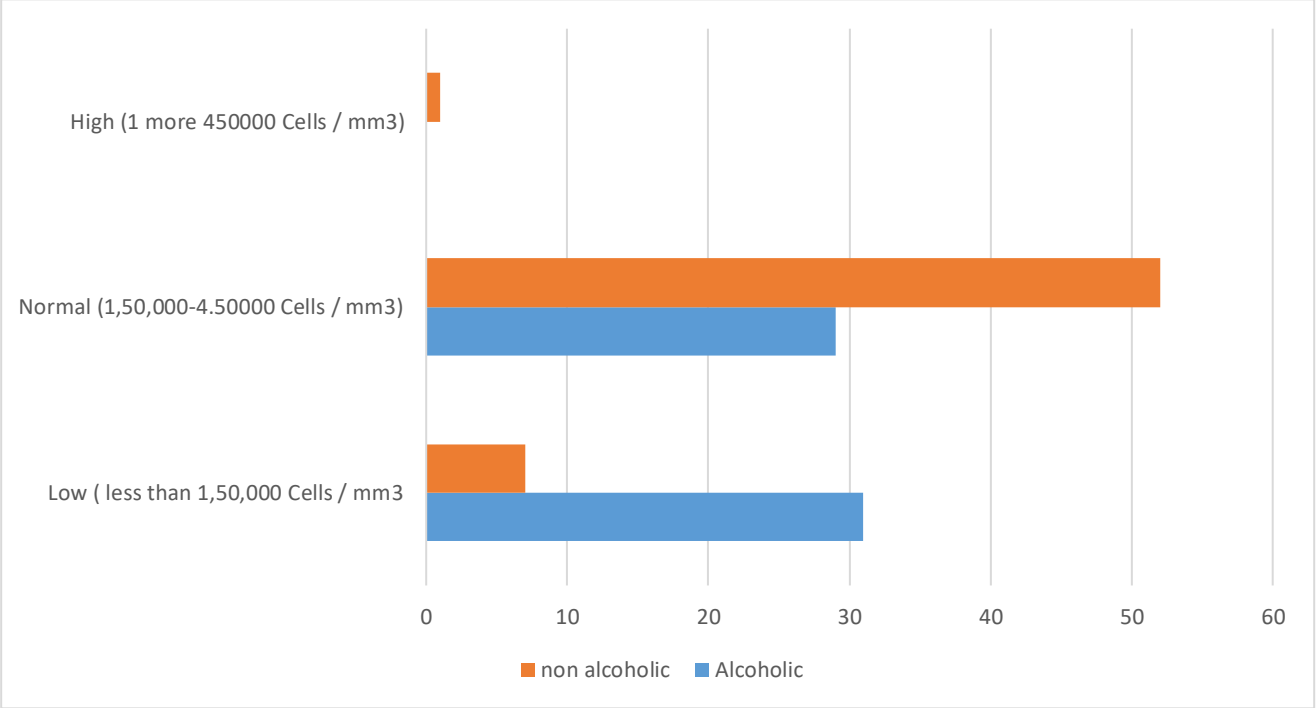


Similarly patients of both groups were further classified based upon their leucocyte count and observations made are depicted in the above given table no: 8.

TABLE 9: DISTRIBUTION OF PATIENTS BASED ON PLATELET COUNT:

STUDY GROUP		**Low (less than 1,50,000 Cells / mm3)**	**Normal (1,50,000-4,50,000 Cells / mm3)**	**High (more 4,50,000 Cells / mm3)**	**Total**
Alcoholic	**Number**	31	29	0	60
	%	51. 7%	48. 3%	0. 0%	100. 0%
Non alcoholic	**Number**	7	52	1	60
	%	11. 7%	86. 7%	1. 7%	100. 0%
Total		38	81	1	120

GRAPH 9: DISTRIBUTION OF PATIENTS BASED ON PLATELET COUNT



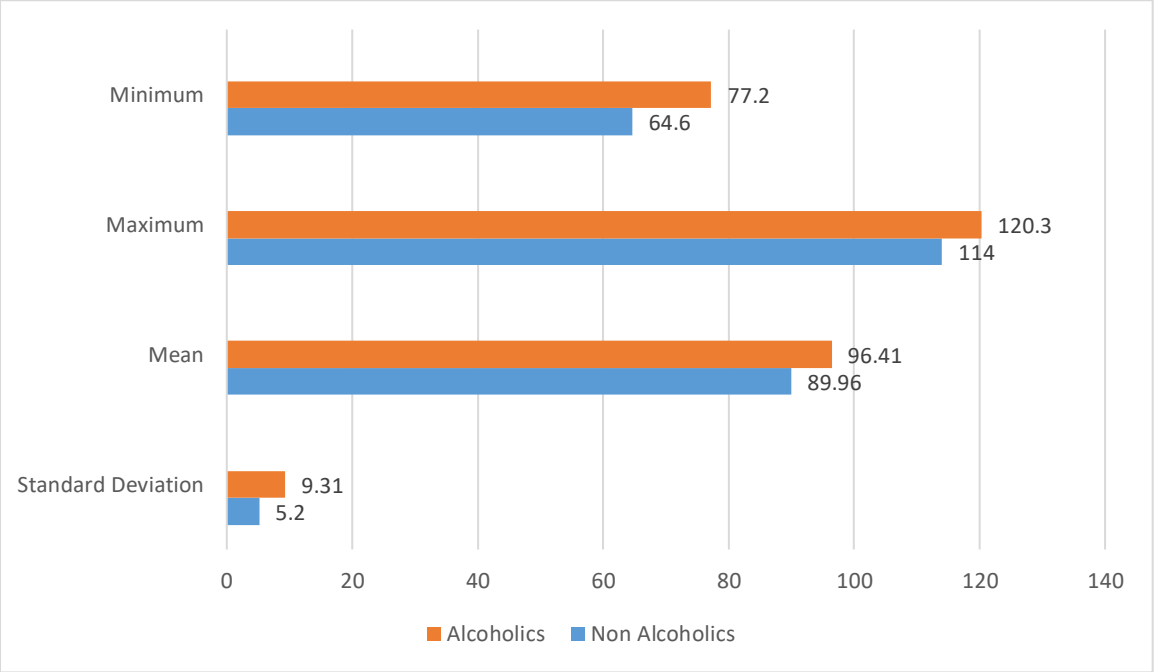
Similarly patients of both groups were distributed based upon their platelet count. In both alcoholic and non-alcoholic groups, the observations made are depicted as follows:

Majority of the patients in the alcoholic group had low platelet count i.e., 31 (51.70%), while remaining 29 patients had normal platelet count. In the nonalcoholic group majority patients 52 (86.7%) had a normal platelet count. There is a statistically significant difference between the groups with a p value 0.001.

TABLE 10: DISTRIBUTION OF PATIENTS BASED ON MEAN CORPUSCULAR VOLUME (MCV):

STUDY GROUP	Minimum	Maximum	Mean	Standard Deviation
alcoholic	77.2fl	120. 3 fl	96.41 fl	9.31fl
non alcoholic	64.6 fl	114 fl	89. 96 fl	5. 20 fl

GRAPH 10: DISTRIBUTION OF PATIENTS BASED ON MEAN CORPUSCULAR VOLUME (MCV)



The mean corpuscular volume in the both the groups is depicted in the above table no: 10. In our present study, the mean MCV was more in the alcoholic group i.e., 96. 41 fl $\pm$ 14. 78 fl when compared to the nonalcoholic group which is 89. 96 fl $\pm$ 15. 17 fl. The p value is statistically significant between both the groups (p = 0.038).

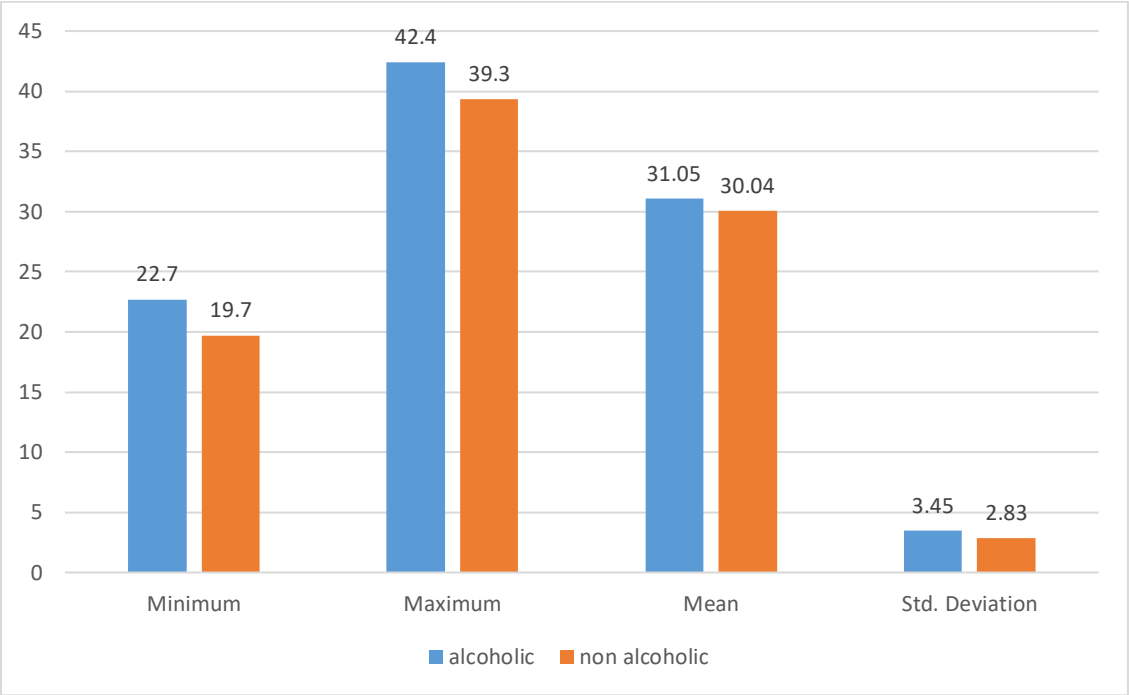
TABLE 11: DISTRIBUTION OF PATIENTS BASED ON MEAN CORPUSCULAR HEMOGLOBIN (MCH):

STUDY GROUP	Minimum	Maximum	Mean	Standard Deviation
alcoholic	22. 7pg	42. 4 pg	31. 05 pg	3. 45 pg
non alcoholic	19. 7 pg	39. 3 pg	30. 04 pg	2. 83 pg

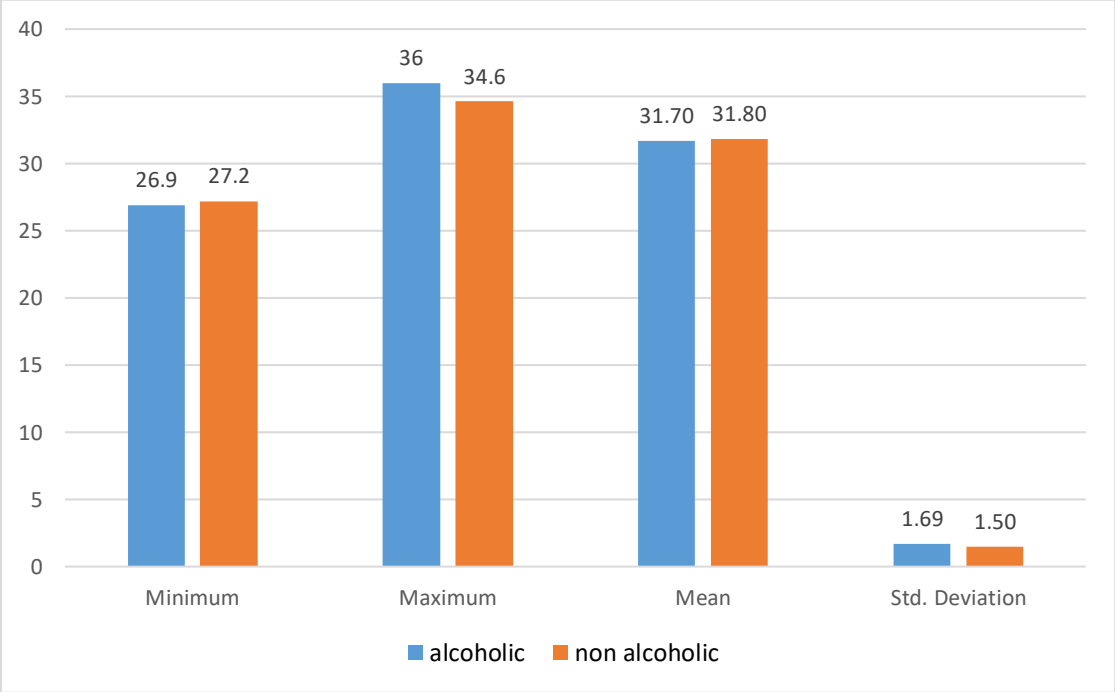
TABLE 12: DISTRIBUTION OF PATIENTS BASED ON MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC):

STUDY GROUP	Minimum	Maximum	Mean	Std. Deviation
alcoholic	26. 9 %	36 %	31. 70%	1. 69 %
non alcoholic	27. 2 %	34. 6 %	31. 80 %	1. 50 %

GRAPH 11: DISTRIBUTION OF PATIENTS BASED ON MEAN CORPUSCULAR HEMOGLOBIN (MCH)



GRAPH 12: DISTRIBUTION OF PATIENTS BASED ON MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (MCHC)

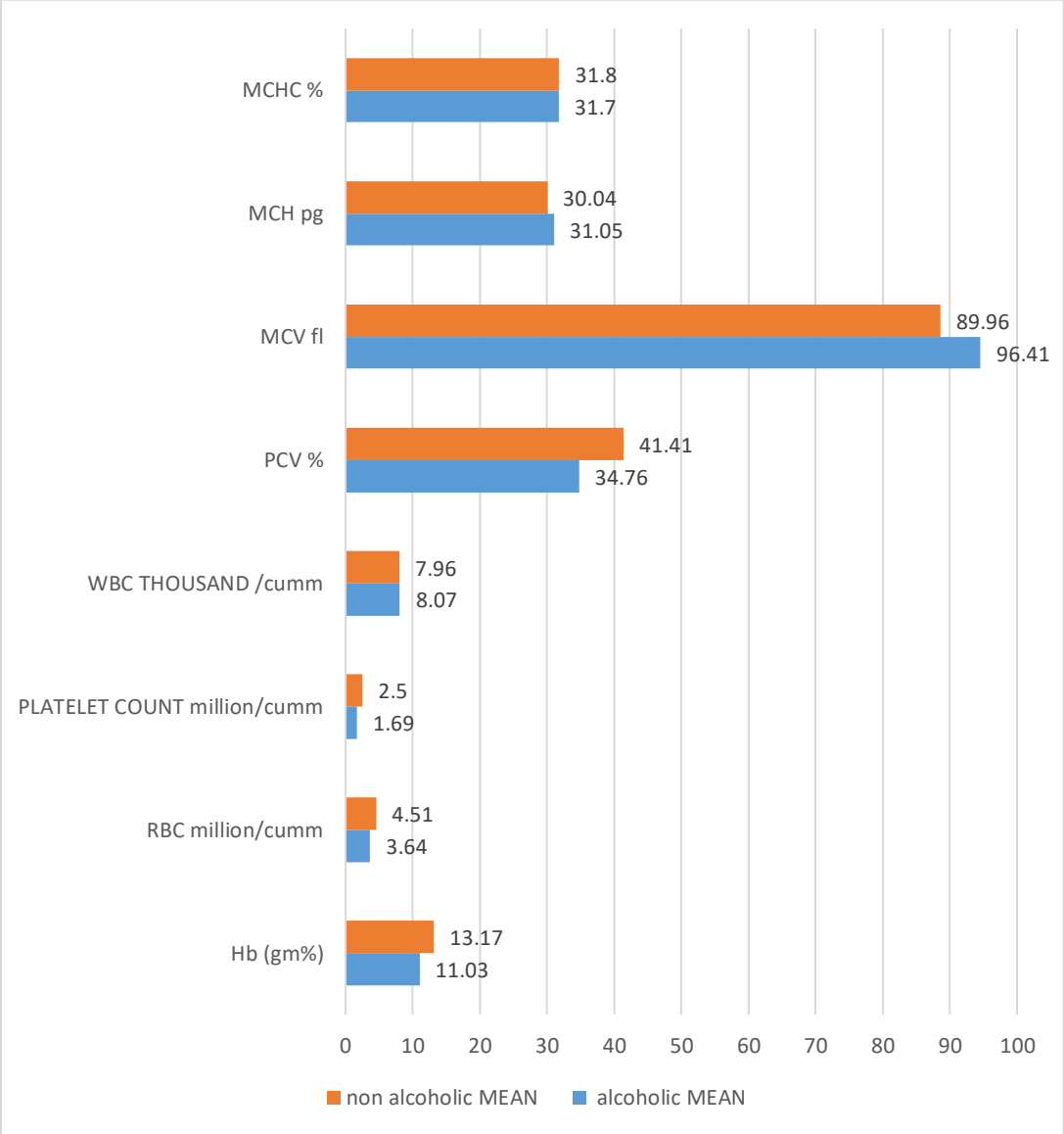


Similarly, MCH in table no: 11, MCHC in table no: 12 were compared between both the groups and p value was not statistically significant between the groups.

TABLE 13: DISTRIBUTION OF PATIENTS BASED ON HEMATOLOGICAL INDICES:

GROUP	**ALCOHOLIC**		**NON ALCOHOLIC**		**p value**	**Significance**
PARAMETERS	**MEAN**	**STDEV**	**MEAN**	**STDEV**		
Hb (gm %)	11. 03	2. 89	13. 17	1. 83	<0. 001	significant
RBC(million /cumm)	3. 64	1. 09	4. 51	0. 74	<0. 001	significant
WBC (cells/cumm)	8071. 67	3600. 24	7966. 42	2577. 53	0. 85	Not significant
PLATELET COUNT (million/cumm)	1.69	9.3	2.5	8.2	<0. 001	significant
PCV %	34. 76	9. 26	41. 41	5. 21	<0. 001	significant
MCV fl	94. 57	14. 66	88. 58	17. 31	0. 04	significant
MCH pg	31. 05	3. 42	30. 04	2. 83	0. 07	Not significant
MCHC %	31. 70	1. 69	31. 80	1. 50	0. 76	Not significant

GRAPH 13: DISTRIBUTION OF PATIENTS BASED ON HEMATOLOGICAL INDICES



The above table no: 13 and graph depicts all the RBC indices in both the groups. There was a statistically significant difference between the groups with a p value less than 0. 05 in terms of hemoglobin, red blood cell count, platelet count, packed cell volume and the mean corpuscular volume. There was no statistically significant difference between the groups with a p value >0.05 with respect to mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration and WBC Count.

TABLE 14: DISTRIBUTION OF PATIENTS BASED ON LIVER FUNCTION TESTS (LFT):

GROUP	ALCOHOLIC		NON ALCOHOLIC			
PARAMETERS	**MEAN**	**STDEV**	**MEAN**	**STDEV**	**p value**	**Significance**
T. BILIRUBIN mg/dl	4. 24	7. 25	1. 21	3. 68	0. 006882	significant
D. BILIRUBIN mg/dl	3. 02	5. 65	0. 39	0. 42	0. 000715	significant
I. BILIRUBIN mg/dl	1. 19	1. 87	0. 34	0. 20	0. 001062	significant
TOTAL PROTEIN	6. 31	0. 92	6. 77	0. 64	0. 002712	significant
ALBUMIN	3. 36	1. 09	3. 91	0. 56	0. 001965	significant
A/G RATIO	1. 13	0. 46	1. 48	0. 47	< 0. 001	significant
SGOT	80. 64	64. 13	32. 37	28. 18	< 0. 001	significant
SGPT	47. 30	43. 09	27. 30	19. 54	0. 003246	significant
ALP	124. 48	51. 99	100. 87	35. 00	0. 004927	significant

GRAPH 14: DISTRIBUTION OF PATIENTS BASED ON LIVER FUNCTION TESTS (LFT)

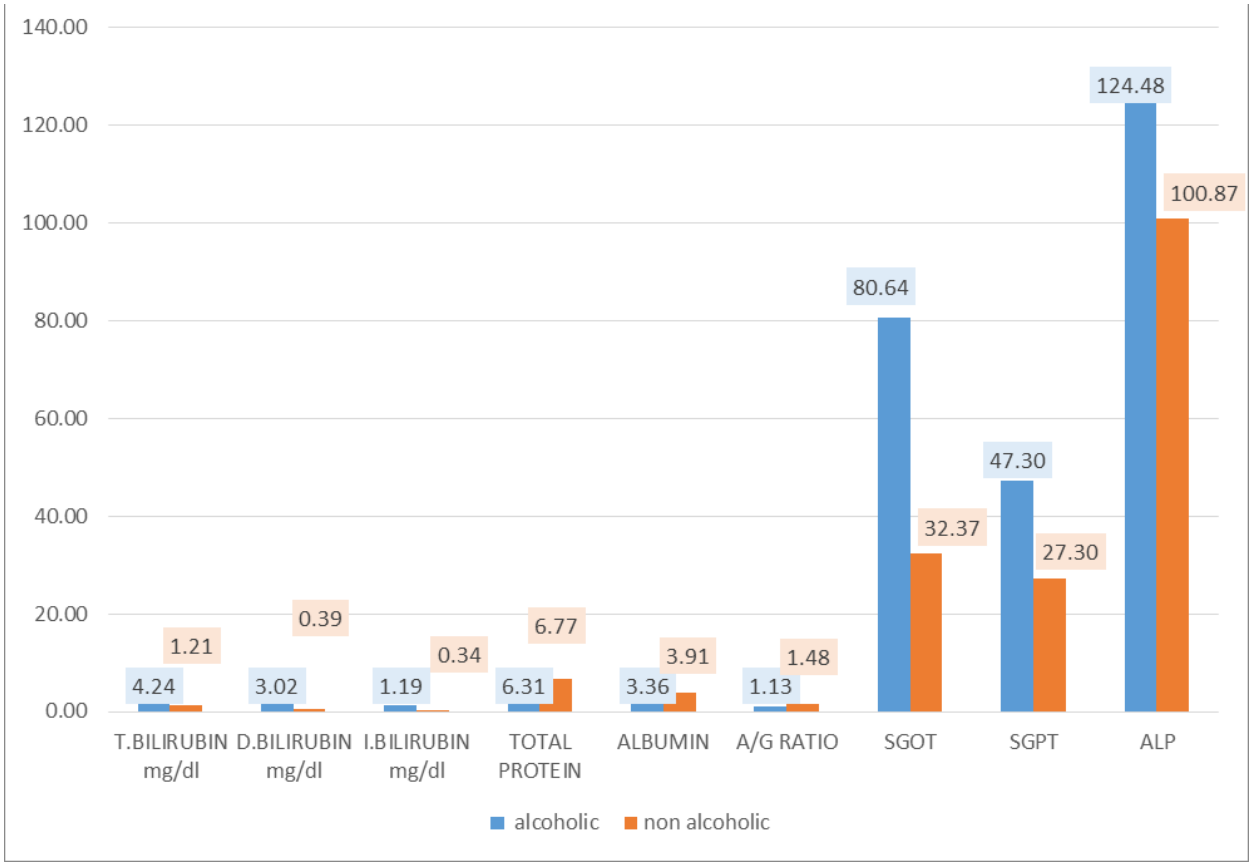
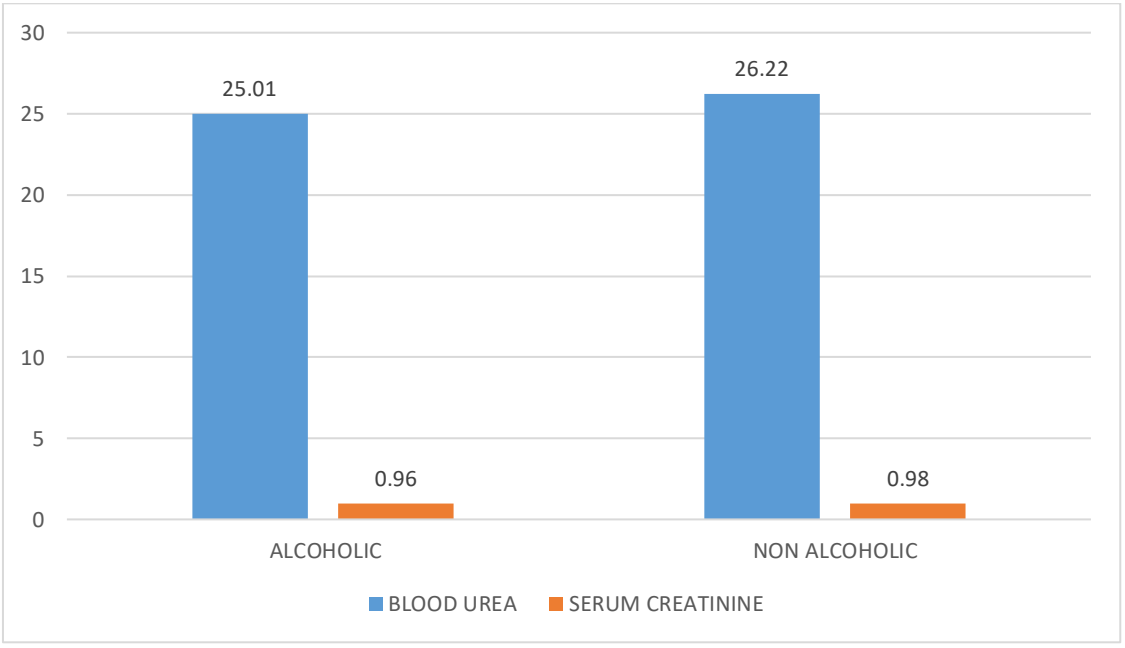


Table no: 14 depicts liver function tests in both the groups. We observed the abnormalities of liver function tests in the alcoholic group are statistically significant with respect to all parameters when compared to the nonalcoholics.

TABLE 15: DISTRIBUTION OF PATIENTS BASED ON RENAL FUNCTION TESTS:

RENAL FUNCTION TESTS		ALCOHOLIC	NON ALCOHOLIC	p value
BLOOD UREA	MEAN	25. 01 mg/dl	26. 22 mg/dl	0. 68
	SD	18. 65 mg/dl	13. 17 mg/dl	Non significant
SERUM CREATININE	MEAN	0. 96	0. 98	0. 91
	SD	0. 45	0. 49	Non significant

GRAPH 15: DISTRIBUTION OF PATIENTS BASED ON RENAL FUNCTION TESTS

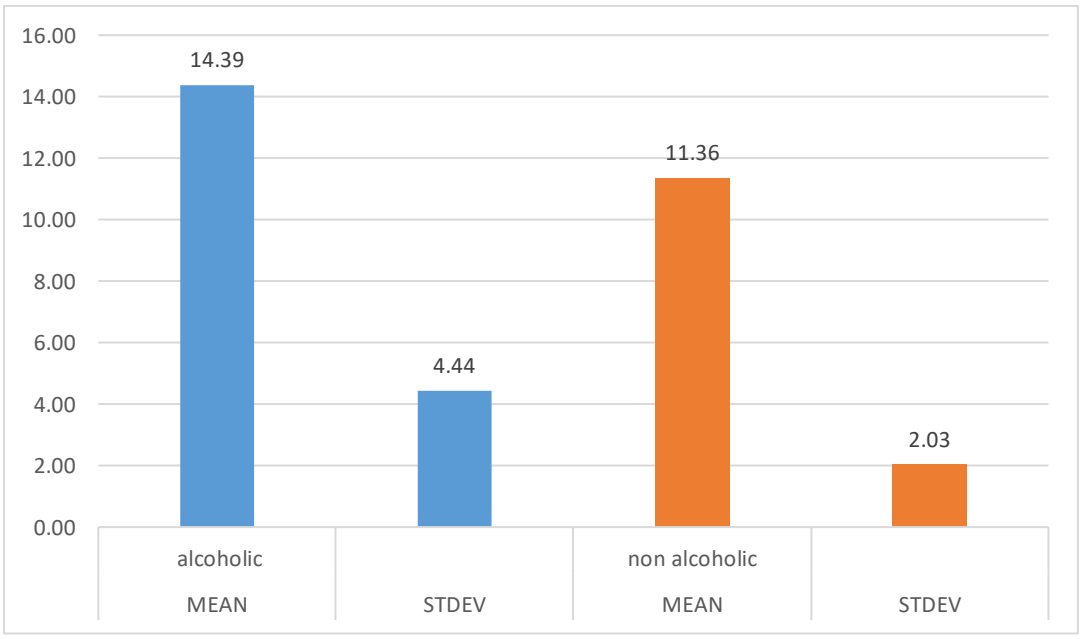


All patients were subjected to renal function tests i.e., blood urea and serum creatinine. The observations made are depicted in the above table no: 15 and there is no much statistically significant difference between the groups.

TABLE 16: DISTRIBUTION OF PATIENTS BASED ON PROTHROMBIN TIME (PT):

GROUP	alcoholic		non alcoholic		p value and significance
STATS	MEAN	STDEV	MEAN	STDEV	
P. TIME (sec)	14. 39	4. 44	11. 36	2. 03	<0. 001

GRAPH 16: DISTRIBUTION OF PATIENTS BASED ON PROTHROMBIN TIME (PT)



Similarly in our present study the patients were distributed based upon prothrombin time. Statistically significant difference with a p value <0.001 is observed between both the groups with mean prothrombin time of 14.39 ± 4. 44 seconds in the alcoholic group while it was 11.36 ± 2.03 seconds in the nonalcoholic group.

DISCUSSION

In our present study of 120 patients, 60 patients in each alcoholic group and nonalcoholic group were studied and subjected to hematological indices to see whether there is an influence of alcohol and same thing was compared to nonalcoholics.

In our study of 120 patients, the age ranged from 18-75years. The younger patient was 18 year old while the eldest was 75 year old.

In alcoholic group there were 17(28. 30%) patients in each age group of 31-40 years and 51-60 years, 13 (21. 70%) patients in the age group of 41-50 years, 7 (11.70%) patients in the age group of > 60 years and only 6 (10. 00%) patients in between the age group of 18-30 years. Where as in nonalcoholic group, 20 (33.30%) patients were in the age group of > 60 years , 12 (20%) patients in the age group of 51-60 years , 11 (18. 30%) patients in the age group of 31-40 years , 9 (15. 00% %) patients in the age group of 18-30 years and only 8 (13.30%) in the age group of 41-50 years.

Namrata Aggarwal et al.[105] had almost similar comparison in their study population with our present study related to age distribution of patients, where their mean age was 48.67 years.

Another study by T. Oduola et al.[106] studied in 200 patients, the mean age of their patients was 36.04 ± 11.28 years. This is in sharp contrast to our study. May be this difference could be because of more number of patients in their study which is 200 patients while 120 patients in our study.

When gender was taken into consideration, we found male predominance in our study. All 120 patients were male patients. No female patients were present in both the alcoholic and nonalcoholic groups.

Surinder Kumar Salwan et al.[107] had similar observations in the study population (N=90), where all the patients were male with no female patients in their study. Also a study by T. Oduola et al.[106] who had all male patients was similar to our study. This is in sharp contrast with D. Chalmers et al.[108] (N=219) who had 146 male patients and 73 female patients.

In our study of alcoholic groups, 20 (33.33%) patients were ingesting alcohol for more than 5- 10 years, 15 (25.00%) patients for more than 10-15 years, 11 (18.33%) patients for more than 15-20 years and 8 (13.33%) patients for more than 20 years. Thus majority of the patients (56.66%) in our alcoholic group are consuming alcohol for more than 10 years In our present study, all the 60 patients were severe alcoholics according to national institute of alcohol abuse and alcoholism (NIAAA) who met the criteria of consuming ≥14 standard drinks/week.

A study by Namrata Aggarwal et al.[105] in their study population, most of the patients were consuming alcohol for 11-20 years while remaining patients were consuming for less than 10 years. A study by D. Chalmers et al.[108] in their study population most of them are severe alcoholic for more than 10 years. Also in a study by T. Oduola et al.[106] 50% of them are severely alcoholic for more than 10 years.

Most of the studies have found more the duration and as well as amount (heavy/severe drinking) of alcohol consumption has got reflection on hemoglobin %, leucocyte count, platelet count, MCH, MCHC. In our study we observed effect on hematological indices like hemoglobin %, WBC count, platelet count, PCV and MCV.

The probable explanation for this is prolonged consumption of alcohol has direct or indirect effect on hematopoiesis who indulge in alcohol consumption for more number of years and heavy/severe drinking.

We categorized patients in both the groups (alcoholic and nonalcoholic) according to severity of anemia based on WHO classification as mild, moderate and severe anemia as shown in the above table no: 5. We observed 31 patients had moderate to severe anemia in alcoholic groups and 12 had mild anemia, while only 17 had normal hemoglobin levels. Whereas in the nonalcoholic group there were more number of patients who had normal hemoglobin (N=38) while 14 patients had mild anaemia, 8 patients had moderate anaemia and none had severe anaemia with a statistically significant difference between the groups with a p value < 0.001.

A study by Berad et al.[109] had similar observations who had moderate to severe anemia in the majority of their study population. A study by Surinder Kumar Salwan et al.[107] also observed moderate to severe anaemia in their study population in moderate to heavy drinkers.

In our present study in alcoholic groups, 44 (73. 3%) patients had RBC count below 4. 5 million/ mm^3 while remaining 16 (26. 7%) had above 4. 5 million/ mm^3. In nonalcoholics group, 28 (46. 7%) patients had RBC count below 4. 5 million/ mm^3 while 32 (53. 3%) patients had above 4. 5 million/ mm^3 which had statistical significance (p <0.001).

A study by Namrata Aggarwal et al.[105] also observed low RBC count in their study population of alcoholics when compared to nonalcoholic subjects. This reduction in RBC count is direct or indirect suppression of bone marrow / hematopoiesis leading to decreased RBC count in patients with prolonged and heavy drinking. A study by Berad et al.[109] also

observed reduced RBC count in their study population with prolonged and moderate to heavy drinkers and their observation had significant p value (p <0.001).

When we attempted comparison of Packed cell volume (PCV) in both the groups, in alcoholic group majority of them had PCV below 40 (N=41; 68. 30%) while remaining 19 (31. 70%) patients had above 40. Whereas In the nonalcoholic group 41 (68. 30%) had PCV above 40 and only 19 (31. 70%) patients had less than 40 with statistically significant difference between the groups with a p value 0. 001**.**

A study by Surinder Kumar Salwan et al.[107] observed the similar effection of PCV in their study population and has positive correlation in alcoholic group. A probable explanation for this is there is a direct effect of alcohol on hematopoiesis in these heavy/severe drinkers with prolonged drinking. It is essential to detect these changes early and treating them timely which may reflect on morbidity and mortality.

A study Berad et al.[109] is also of similar opinion in these chronic alcoholics. Doing psychiatric counseling for alcohol dependency and treating anemia timely to prevent further complications of hematological changes helps in preventing future complications of cirrhosis of liver, cardiac, renal, cerebellar degeneration, as well as other complications like neuropathies and pancreatitis.

In our present study, in alcoholic groups the leucocyte count was less than 4000 cells/ mm3 in 7 (11. 70%) patients, normal (4000-11000 cells/ mm3) in 39 (65. 00%) patients and 14 (23. 30%) patients had more than 11000 cells/ mm3.whereas in nonalcoholics only 1(1. 70%) patient had leucocyte count was less than 4000 cells/ mm3 while majority of them i.e., 49 (81. 70%) patients had normal count and 10 (16. 70%) patients had more than 11000 cells/ mm3.

This is in sharp contrast to a study by Elanchezhian et al.[99] who have found significant correlation in alcoholics (moderate to heavy drinkers) with statistically significant low WBC count in their study population.

The above WBC count >11,000 cells/ mm3 in alcoholic group (N=14) and 10 in non-alcoholic group, we would feel probably because of concurrent focus of infection which was not evident clinically when we enrolled these patients for study. Also, we are of opinion probable explanation for this is lacking. Most of the studies carried by different authors have not explained cause of leucocytosis in their study population and majority observed leucopenia in their study population.

When platelet count was taken into consideration, among alcoholics 31 (51.70%) patients had low platelet count (less than 1,50,000 Cells / mm3), while remaining 29 patients had normal platelet count (1,50,000-4,50,000 Cells / mm3) and none had high platelet count (more 4,50,000 Cells / mm3). In the nonalcoholics only 7 (11. 7%) patients had platelet count less than 1,50,000 Cells / mm3 while remaining 52 (86.7%) patients had platelet count 1,50,000-4,50,000 Cells/mm3 and only 1 (1. 7%) had platelet count more 4,50,000 Cells / mm3. There is a statistically significant difference between the groups with a p value <0.001. The probable explanation for high platelet count in this one patient we could not ascertain.

A study by Das S K et al.[100] also found platelet count abnormality (low platelet count) in their study population. Their observation was all patients who were chronic and heavy alcoholics had low platelet count and none had low platelet count in comparable group (nonalcoholic group).A study by O.Erhabor et al.[110] observed significantly low platelet count in patients of chronic alcoholics. This was significantly lower in heavy alcoholics when compared to nonalcoholics. The explanation for this observation is heavy alcohol

consumption causes hypocellularity of marrow leading to anemia, leucopenia, thrombocytopenia and their relative sequalae.

In our present study we attempted to compare the mean corpuscular volume in both the groups (alcoholics and nonalcoholics). In alcoholics MCV was elevated with mean MCV of 96.41 $\pm$ 9.31 fl as compared to nonalcoholic group which is 89. 06 $\pm$ 5.20 fl. This reflection is having a positive correlation in alcoholics with statistically significant p value <0.05 in our study.

A study by Elanchezhian et al.[99] found a very high MCV in their study population especially in chronic and severe alcoholics and there was a positive correlation of MCV being high in alcoholics group compared to nonalcoholics group. In a study by Berad et al.[109] almost 90% of the chronic and heavy alcoholics had macrocytosis (MCV between 100-110 femtolitre) in the alcoholic groups. Macrocytosis occurs even in patients with folic acid and vitamin B12 replenishment in absence of alcohol liver disease. The probable mechanism for this is not known. After a period of 2-6 months of abstinence from alcohol, the macrocytosis would disappear. A study by Surinder Kumar Salwan et al.[107] had a positive correlation of MCV with chronic alcoholics by elevation of MCV.

In a similar attempt for observation of MCH and MCHC together in both the groups (alcoholics and nonalcoholics), there was no much difference observed as far as MCH and MCHC are concerned in our study population as depicted in the above table no: 11 and table no :12.

A study by Surinder Kumar Salwan et al.[107] did not show any positive correlation for MCH in their study population of both alcoholics and nonalcoholics. Another study by T. Oduola et al.[106] also observed similar findings to our study. This is in sharp contrast to a study by Berad et al.[109] who observed in moderate to severe alcoholic subjects low hemoglobin %, mean MCH and mean MCHC as compared to nonalcoholic groups. Further

they observed the decreasing trend of MCH and MCHC in their alcoholic group and also observed normal WBC count in both the groups. The probable explanation for this mechanism is unclear.

Different hematological indices were depicted in the above table no : 13 with p value being significant for hemoglobin % , RBC count, platelet count , PCV and MCV. The other indices are not having significant p value as depicted in the above table no: 13.

This is in sharp contrast to a study by Berad et al.[109] who have found positive correlation with all the hematological indices.

As depicted in the above table no: 14 all the liver function tests have got positive correlation in alcoholic groups as compared to nonalcoholic groups in our study. This probably reflects chronic and severe/heavy alcohol drinking though mild abnormalities of SGOT and SGPT were observed in our study population.

A study by Dharmesh Gamit et al.[103] , who have found liver function abnormalities in their study population of alcoholics except for total protein not having significant correlation. A study by Surinder Kumar Salwan et al.[107] have found significant correlation with all the parameters of liver function tests in the alcoholic groups. The probable explanation for this is chronic and moderate to heavy drinking which has got direct deleterious effects on liver functions.

We subjected both the group patients (alcoholics and nonalcoholics) for renal function tests (serum urea and creatinine). In both the groups there was no comparable significance observed.

This is in sharp contrast to a study by Elanchezhian et al.[99] who have found abnormalities of blood urea and serum creatinine in severe alcoholics compared to nonalcoholics. They also observed mild to moderate elevation in mild to moderate alcoholics which was statistically significant in their study population. The renal functions are effected in patients

of chronic and heavy/severe drinkers which may be a direct effect of chronic and heavy drinking in these patients.

Similarly we subjected all our patients for prothrombin time estimation and we found statistically significant p value in alcoholic as compared to nonalcoholic group.
A study by O.Erhabor et al[110] observed hemostatic parameters like PT and APTT were significantly higher among the alcoholics when compared to the nonalcoholics in their study population. The probable explanation for this could be the direct negative effect of alcohol on hemostatic factors because of abnormality in synthesis of them by liver.

Finally, we found positive correlation with hemoglobin %, RBC count, platelet count, PCV, MCV, all parameters of liver function tests, prothrombin time in the alcoholic group with statistically significant p value. However our study did not show any significant positive correlation with WBC count, MCH, MCHC, blood urea and serum creatinine with the alcoholics. Alcoholic liver disease and adverse effects of alcoholism on health is well established fact but here through our present study we currently emphasize on the early changes of hematological parameters with the prolonged and heavy/severe consumption of alcohol which is on raising trend with the social and cultural shift especially among the youngsters.

SUMMARY

In our present study of 120 patients titled "**COMPARISION OH HEMATOLOGICAL PARAMETERS IN ALCOHOLICS AND NONALCOHOLICS "** during the study period from January 2023 to December 2023 carried out in the Department of General Medicine, on patients admitted to KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre.

The results of our study are summarized as below:

1. There were total 120 patients who were enrolled for the present study-60 alcoholic and 60 nonalcoholic.
2. All 120 patients were males with no female patients.
3. Majority of our patients consume alcohol for the duration more than 10 years (56.66%).
4. All the 60 alcoholic patients are severe alcoholics who consume ≥14 standard drinks/week.
5. Moderate to severe anemia is predominantly seen with the alcoholic group compared to nonalcoholic group.
6. Our study reflected significantly low RBC count, low platelet count and PCV in the alcoholic group with statistically significant p value being <0.01.
7. Macrocytosis was profoundly noticed with the alcoholic group with mean MCV of 96.41 fl which was statistically significant when compared to nonalcoholic group.

8. We did not observe any positive correlation with parameters like WBC count, MCH, MCHC, blood urea and creatinine.

9. All the parameters of liver function tests were significantly abnormal in the alcoholic groups reflecting the direct deleterious effects of prolonged and severe consumption of alcohol on liver health.

10. The prothrombin time was significantly elevated in the alcoholics group with p value being <0.01.

11. We observed that both duration and heavy drinking of alcohol was having positive correlation with parameters we took into consideration in this study.

CONCLUSION

- In our present study of 120 patients there were 60 patients in each group (alcoholics and non-alcoholics) for comparing haematological parameters in them. We hereby conclude :
- There is a positive correlation with the alcoholics group as far as haemoglobin %, RBC count, PCV, Platelet count, MCV, all the parameters of liver function tests and prothrombin time were concerned.
- There is no significant correlation with the parameters like WBC count, MCH, MCHC, blood urea and creatinine between the groups.
- These simple cost effective parameters would help us in detecting haematological changes early in chronic and severe alcoholics and helps us in directing them to counselling for alcohol dependence timely and properly to decrease the future complications like alcoholic liver disease , pancreatitis, cerebellar degeneration , neuropathy ,cardiac and other related diseases.
- This would also decrease the morbidity and mortality associated with alcoholism.
- We also feel with the rise in trends of alcohol consumption especially among the youngsters due to increased social acceptance, social drinking patterns converting into habituation/dependence and cultural shifts, as a doctors it is necessary for early detection, intervention and counselling the dependant alcoholics for abstinence/refrain from alcohol which has high impact mainly on decreasing alcohol related personal problems, professional problems, social problems and especially financial burden on the families of alcoholics.

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ANNEXURE I – INFORMED CONSENT FORM

Title Of Research Study:

"Comparison of hematological parameters in alcoholics and non-alcoholics"

Introduction and Purpose: -

Alcoholism is the leading social and health problem worldwide. So identifying the changes early and appropriate counseling and treatment would decrease the mortality and morbidity among the alcoholics.

Procedure:
If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood and urine samples for the necessary investigations.

-Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:
Taking part in this study is voluntary. You may choose not to take part in this study.
If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive standard treatment for patients with your condition.

Privacy and Confidentiality:
All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:
Does not apply to this research

Financial incentives for participation:
You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:
The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

Dr. HARSHA HEGDE

Chairman,

College Ethical Dissertation

Research Committee

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has Been read to me, this consent form and have had all the questions answered

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

Participant's name :……...

Signature / Left thumb impression:……...
of the participant

Name of the legally authorized :……...
representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

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

Date:

Place:

ANNEXURE II – PROFORMA

CASE NO	
NAME	
IP NO	
AGE	**YEARS**
SEX	**MALE FEMALE**
ADDRESS	
OCCUPATION	

Complaints at presentation	
Past history	
Personal history and habits	
1. Duration of alcohol intake **2. Quantity of alcohol intake** **3. Frequency**	
Treatment history	

Vitals :

Temperature	
Pulse	
Respiratory rate	
Blood pressure	

PHYSICAL EXAMINATION:

	Yes	**No**
Pallor		
Icterus		
Lymphadenopathy		
Cyanosis		
Clubbing		
Edema		

SYSTEMIC EXAMINATION:

C.V.S	
R.S.	
C.N.S	
PER ABDOMEN	

INVESTIGATIONS:

Hemoglobin		Platelet count		A/G ratio	
RBC		MCV		SGOT	
PCV		MCH		SGPT	
Total count		MCHC		ALP	
neutrophils		Total bilirubin		Blood Urea	

lymphocytes		Direct bilirubin		S.creatinine	
Basophils		Indirect bilirubin		prothrombin time	
eisinophils		Total proteins			
monocytes		Serum albumin			

ANNEXURE III - MASTER CHART

S . NO	IP NUMBER	AGE	duration (years)	SD/week	Hb (gm%)	RBC million/cumm	PCV %	TC	PLATELET	MCV fl	MCH pg	MCHC %	T.BILIRUBIN mg/dl	D.BILIRUBIN mg/dl	I.BILIRUBIN mg/dl	TOTAL PROTEIN	ALBUMIN	A/G RATIO	SGOT	SGPT	ALP	BLOOD UREA	S.CREATININE	P.TIME (sec)
1	1177977	58	10	21	15.5	4.81	45.7	8200	189000	94.9	32.3	33.9	1.63	0.47	1.16	6.6	3.6	1.2	168	107	179	15	0.63	13.7
2	1193895	43	7	14	13.3	3.77	39.9	6900	206000	96.3	31.8	30.4	2.21	1.71	0.5	5.5	3.5	1.8	204	143	147	19	1.07	13.9
3	1194307	46	15	21	8.3	2.58	27	5000	25000	78.5	26.2	30	9.11	3.92	5.19	5.5	2.8	1	37	18	237	25	0.79	25.7
4	1197834	74	20	18	11.8	4.61	35.6	12300	244000	77.2	25.6	30.3	1.33	1.21	0.12	5.6	2.8	1	22	21	91	20	0.9	11.1
5	1199480	49	20	24	9.9	3.41	27.9	9500	146000	99	35.5	35.5	0.21	0.11	0.1	5	2.2	0.8	55	34	127	25	2.38	12
6	10006661	61	12	21	12.3	4.88	40.4	8700	179000	82.8	25.2	30.4	0.72	0.029	0.043	6.6	4.4	2	27	43	79	27.3	0.99	11.4
7	10006125	41	15	18	9.1	2.4	30.4	7700	76000	114	37.5	32.9	8.07	4.52	3.55	5.5	2.7	1	45	29	217	14.3	0.65	25
8	10008139	57	25	56	8.9	2.48	30.2	9300	143000	113	35.9	31.6	3.13	2.23	0.9	6.4	2.3	0.5	68	24	166	15.6	1.13	18.7
9	10006963	39	10	42	14.1	3.89	41.2	11300	241000	99	31.2	32.3	15.5	10.8	4.7	8.2	3.1	0.6	167	87	147	35.7	0.53	12.3
10	10005316	60	10	21	6	2.64	22.3	5800	205000	84.5	22.7	26.9	0.14	0.06	0.08	6.5	3.7	1.3	21	19	56	21.4	0.96	12
11	10006286	54	20	18	9.37	3.1	30.6	4400	92000	98.7	30	30.4	0.36	0.2	0.16	4.8	1.8	0.6	53	16	126	23.7	1.1	15.5
12	1168172	35	10	56	5.8	2.68	21.5	2800	296000	94.9	32.7	34.5	1.49	0.79	0.7	5.9	2.3	1.5	40	43	117	16.5	0.8	13
13	10007930	28	3	24	13.8	4.5	40.7	9200	328000	96.5	31.3	32.4	18.4	13.2	5.2	7.6	4.3	1.3	146	116	173	10.5	0.73	19
14	10006437	52	35	42	15.6	4.92	49.8	8500	341000	98.1	32.3	32.2	0.94	0.38	0.56	6.4	3.9	1.6	24	12	89	27.6	0.9	12
15	10002781	35	10	42	12.5	4.8	46.2	11300	243000	88.4	30.4	30.2	2.1	0.8	1.3	6.7	3.8	1.3	74.2	28.3	103	16.3	0.8	13
16	10007607	31	5	84	14.6	4.3	49.2	6800	280000	98.7	32.5	32.8	0.2	0.1	0.1	5.9	2.3	0.6	27.3	27.9	110	11.5	0.7	12
17	10007529	27	12	42	5.9	1.68	18.9	6200	80000	106.2	35.1	33	4.33	4.03	0.3	7.5	2.1	0.4	37	19	194	43.4	0.6	22
18	10008500	57	30	56	16.6	5.7	49.9	11000	147000	99.7	32.2	30.7	1.22	0.39	0.83	7	4.3	1.6	19	21	169	19.9	0.7	12
19	10008562	38	10	42	9.1	3.15	32.1	5600	422000	101.4	33.3	31.4	0.4	0.2	0.19	5.5	2.2	0.7	9	15	154	10.5	0.76	12
20	10006895	60	20	21	11.2	3.95	34.4	4500	129000	87.1	28.5	32.7	2.16	1.14	1.02	7.6	3.9	1.1	26	12	140	6.96	0.89	11
21	10007825	55	20	42	9.9	2.84	31.4	3400	131000	110.6	34	31.5	1.17	0.68	0.47	5.6	2.4	0.8	56	26	114	12	0.8	11
22	10007974	40	8	112	11.4	4.34	35	14000	155000	80.5	26.4	32.7	3.01	2.61	0.36	6.9	3	0.8	129	41	157	10.3	0.67	15.6
23	10008738	24	5	24	12.8	4.08	46.3	7800	213000	80.3	32.4	31.9	1.22	0.41	0.8	7.2	4.3	1.5	46	76	93	22.4	0.8	10
24	10008812	52	15	56	13.9	4.83	43.4	5200	100000	89.2	28.8	32	1.87	0.8	0.98	7.4	4.6	1	116	102	92	16.3	0.4	11.2
25	10008925	30	10	56	15	4.7	43.9	3900	170000	92.2	31.5	34.2	0.48	0.32	0.16	6.8	3.8	1.3	145	85	121	10	0.87	11.5
26	10016495	38	20	42	10.3	3.23	31	12000	110000	95.8	32	33.4	17.4	13.2	4.2	6	2.1	0.5	235	68	120	11.6	0.5	14
27	10018530	59	10	35	10.7	4.58	37.6	16000	271000	82.1	23.4	28.5	0.66	0.25	0.41	7.6	4.2	1.2	5	30	75	22	0.97	12
28	10031078	56	8	14	13.2	4.7	39.8	7200	270000	94.4	31	32.2	0.3	0.1	0.2	6.7	4.2	1.7	78	49	88	9.7	1	11
29	10028900	76	20	24	11.5	3.91	37.1	3700	199000	94.9	29.4	36	0.59	0.34	0.25	6.5	4	1.6	13	12	82	15.8	0.92	11.5
30	10029965	44	10	42	10.3	2.75	33.5	4000	63000	92.8	29.4	32	3.35	2.27	1.08	5.9	2.6	0.8	45	17	107	20.4	0.7	18
31	10029196	53	8	14	8.1	2.96	30.4	1400	83000	92	30	30.2	3.34	1.82	1.52	4.9	2.4	1	115	34	103	77.1	0.9	14
32	10029993	68	30	84	7	2.02	21.2	8000	89000	93	30.9	30.8	2.66	1.88	0.78	5.1	2.3	0.8	82	37	84	91.7	1.09	13
33	10029968	52	12	42	14.6	4.92	42	9200	353000	92.6	30.4	31.3	0.18	0.1	0.08	6.8	4.3	1.7	43	71	54	15.4	0.8	10.6
34	10028438	32	10	42	11	3.61	35.1	11000	320000	97.3	30.5	31.3	0.46	0.14	0.32	7.2	3.8	1.1	17	16	127	21.9	1.5	11.2
35	10026839	42	5	21	13.8	4.54	43.6	10000	72000	96	30.4	31.7	1.62	0.65	0.97	6	3.2	1.1	54	24	28	37	1	14.4
36	10028542	31	13	42	12.3	4.39	39.5	4900	111000	90.1	28	31.1	1	0.4	0.57	6.9	4.2	1.6	271	255	86	19.1	1.05	11.4
37	10028754	48	5	21	12.4	4.62	39.4	9200	208000	89.1	29	30.3	0.45	0.23	0.22	5.6	3.6	1.8	30	22	64	23.7	1.48	10.5
38	10028879	37	10	84	9.1	2.7	32.3	14000	90000	91	30	30.3	37.1	25.6	11.5	5.9	2.8	0.9	15	36	56	25.2	0.9	32.5
39	10032983	55	15	24	15.6	5.73	48.3	14000	232000	96.5	30.7	31.8	0.62	0.25	0.37	6.5	4.1	1.7	16	18	69	42.1	0.9	11.9
40	10033586	40	10	84	9.4	2.81	31.4	4300	53000	111.7	33.5	29.9	2.79	2.02	0.77	6.8	2.7	0.7	73	46	131	15.6	0.93	19.6
41	10032659	54	10	24	14.6	4.86	43.6	11000	287000	93.6	30	32.1	1.02	0.55	0.47	7.5	4.4	1.4	22	20	92	43.1	1.03	10.4
42	10034612	35	8	84	5.6	1.5	20	10000	170000	113.5	36.1	31.8	5.1	4.63	0.47	5.8	1.9	0.5	63	35	98	6.9	0.5	26.3
43	10036177	42	15	42	16.9	6.34	47.4	8000	140000	89.6	26.7	29.8	1.04	0.5	0.54	5.8	3.6	1.6	32	34	100	17.3	1.17	14
44	10036187	55	15	42	8.6	3	29.2	10000	164000	85.2	28.8	33.7	3.5	2.9	0.5	6.4	4.2	0.5	104	66	219	24.9	0.85	17.6
45	10035925	38	15	42	7.5	2.02	28.4	14000	415000	92.6	27.5	29.7	29.6	25.8	3.8	5.6	2.9	1.1	110	20	81	72.8	3.05	16.4
46	10037048	28	4	84	14.8	4.46	44.2	6500	81000	91.2	33.1	33.4	0.65	0.29	0.36	7.1	4.1	1.4	96	176	144	10	0.67	13
47	10040223	43	20	42	10	2.36	28.4	5800	77000	120.3	42.4	35.2	1.53	1.25	0.28	4.9	8.9	1.4	166	104	300	38.4	1	13.2
48	10039465	47	20	56	13	4.39	41.5	10500	92000	94.5	29.6	31.3	0.55	0.31	0.24	8.5	3.5	0.7	36	25	75	37.3	1.52	14.4
49	10040442	60	20	56	9.6	3.04	32.9	9700	86000	108.2	31.6	29.2	4.4	3.57	0.63	5.4	2.2	0.7	171	48	221	89.6	2.51	17.8
50	10040990	41	15	56	7.3	2.1	21.1	3800	71000	106	31.1	29.9	8.63	6.43	2.2	6.4	3.3	1.1	256	53	103	53.6	0.7	13.1
51	10045392	70	30	35	9.6	3.1	3.03	5500	159000	99.1	30	31.1	1.25	0.64	0.6	5.8	3.6	0.6	68	13	164	15.2	1.03	14
52	10048309	30	10	35	12.3	3.85	37.5	4800	130000	96	31	33	0.31	0.14	0.17	7.1	4.4	1.6	47	23	143	14.2	0.83	12
53	10048221	36	10	42	10	3.72	32	3100	51000	86.3	27	31.3	0.74	0.37	0.37	7.8	3.8	1	146	37	113	11.1	0.53	12
54	10048536	45	15	42	12.7	3.9	40.9	9400	85000	90.5	29	31.1	4.46	2.92	1.54	4.9	2	0.7	84	25	169	43.3	1.3	14.1
55	10048908	48	20	56	8.5	2.85	32.1	6200	147000	102.3	32.1	31.4	7.7	6.94	0.76	7.2	2.3	0.5	144	43	224	10	0.8	14.9
56	10047788	40	12	56	6.9	1.87	15.2	4300	92000	107.8	32.6	30.3	1.16	0.47	0.69	5.5	4	2.5	144	46	104	15.2	0.7	11.3
57	10047197	40	12	84	8.5	2.36	30.3	20000	180000	104.7	36	34.4	24.5	20.8	3.7	5	2.6	0.9	114	26	144	15.6	0.9	20
58	10047820	36	15	42	10.1	3.4	34.4	9000	110000	112.7	36	31.9	1.18	0.75	0.43	6	3.9	1.9	45	25	68	26	0.8	12.1
59	10037251	75	30	42	9.2	3.2	31.1	7500	210000	113.1	35.1	32.1	1	0.5	0.5	6	3.1	0.9	68	60	112	16.4	0.8	12.1
60	10040258	69	30	56	10.1	3.4	36.4	7000	114000	106.1	33.3	31.9	1.89	1.19	0.7	5.1	2.4	1	69	59	123	15.1	0.9	12.4
61	10035863	60	Non Alcoholic	Non Alcoholic	13	4.21	40.1	8800	82000	92.6	30.8	33.3	0.29	0.16	0.13	7.9	4.1	1.5	16	29	80	25.5	0.7	11
62	10036134	43	Non Alcoholic	Non Alcoholic	14.7	4.62	44.1	9400	249000	93.4	31.9	34.1	0.45	0.19	0.26	6.2	4	1.8	25	27	147	25.8	0.97	9.6
63	10034514	59	Non Alcoholic	Non Alcoholic	12.7	4.47	38	5200	286000	93.5	29.4	31.4	0.26	0.18	0.08	5.1	2.5	1	36	25	80	19.8	1.11	11
64	10029593	56	Non Alcoholic	Non Alcoholic	13.2	4.8	42.6	12000	293000	88.6	27.4	31	0.76	0.46	0.3	6.8	4.1	1.1	16	18	90	20.3	0.7	11.4
65	10027852	73	Non Alcoholic	Non Alcoholic	12.3	3.87	39.4	5800	173000	91	31.8	31.2	0.35	0.08	0.27	6.4	3.6	1.3	30	25	66	18.8	1.07	11.5
66	10028401	64	Non Alcoholic	Non Alcoholic	13.9	4.57	43.8	6000	203000	89.9	28.5	31.7	0.67	0.32	0.35	6.5	4.1	1.7	16	19	56	25.3	0.86	15.7
67	10028210	42	Non Alcoholic	Non Alcoholic	13.3	4.8	43.7	8800	215000	89.5	27.3	30.4	0.35	0.15	0.2	6.4	4	1.7	35	26	99	16.5	0.65	10.4
68	10027578	20	Non Alcoholic	Non Alcoholic	13.6	4.36	40.4	7600	328000	92.6	31.3	32.8	0.42	0.18	0.24	6.7	4	1.5	20	10	140	15.1	0.8	12.3
69	10029280	55	Non Alcoholic	Non Alcoholic	13.8	4.48	40.2	10000	420000	90.6	30.3	32.1	0.61	0.24	0.34	6.9	4.2	1.6	16	19	109	21.6	0.79	11
70	10030052	24	Non Alcoholic	Non Alcoholic	15.5	5.34	47.1	7400	220000	90.1	30.2	30.3	1.18	0.5	0.68	7.3	4.5	1.6	15	10	53	17.8	0.89	11.5
71	10029758	75	Non Alcoholic	Non Alcoholic	9.1	4.63	33.2	10700	480000	64.6	19.7	30.4	0.58	0.38	0.2	6.5	3.6	1	37	21	108	25.3	0.7	11.4
72	10012713	28	Non Alcoholic	Non Alcoholic	16.7	5.55	48	5300	352000	91	31.9	31.9	0.74	0.14	0.63	8.2	4.6	1.3	40	30	86	29.1	0.8	11.1
73	10031833	68	Non Alcoholic	Non Alcoholic	13.1	4.65	38	5600	114000	77.5	28.2	32.2	0.4	0.17	0.23	6.4	3.5	1.2	42	46	111	18	0.62	11.4
74	10032646	70	Non Alcoholic	Non Alcoholic	10.9	3.54	38.4	5400	221000	90	30.8	30	0.23	0.13	0.1	6.6	3.8	1.4	14	13	126	43	1.27	11
75	10035020	52	Non Alcoholic	Non Alcoholic	10.7	4.07	39.1	8900	137000	94.5	31.2	32	1.05	0.53	0.52	8.3	4.4	1.1	88	65	149	18	0.6	11.15
76	10033484	74	Non Alcoholic	Non Alcoholic	13.2	4.35	41.4	5200	177000	97	31	32	0.55	0.27	0.28	6.4	3.9	1.6	13	10	99	24	0.9	10.7
77	10039377	50	Non Alcoholic	Non Alcoholic	13.4	5.13	46.1	5700	214000	89.7	26.1	29.1	0.6	0.3	0.3	6.8	4.1	1.1	18	20	89	29	0.8	11
78	10039365	65	Non Alcoholic	Non Alcoholic	12.5	4.43	40.7	7800	304000	81.9	28.2	30.7	0.67	0.24	0.43	6.1	3.7	1.5	23	18	96	31.7	1.5	12.2
79	10039356	62	Non Alcoholic	Non Alcoholic	12.8	3.89	40.1	6500	340000	92.7	29	31.2	0.55	0.22	0.33	7	4.2	1.4	76	60	111	39.1	1.4	10.7
80	10036947	24	Non Alcoholic	Non Alcoholic	9.7	2.79	31.3	5200	143000	101	34.8	31.4	1.06	0.85	0.21	5.2	2.9	1.3	40	32	120	14.8	0.66	11
81	10035353	53	Non Alcoholic	Non Alcoholic	11.6	3.99	35.8	9500	268000	89.9	29.1	32.4	0.43	0.22	0.21	6.5	4.3	2	34	40	134	23.8	1	11
82	10039260	20	Non Alcoholic	Non Alcoholic	15.8	4.79	46.2	4600	237000	96.6	33	34.1	0.4	0.1	0.3	7.1	4.5	1.7	32	22	120	16.2	8	12.2
83	10039347	37	Non Alcoholic	Non Alcoholic	12.7	5.05	43.4	8100	321000	85.9	25.1	29.3	29	0.16	0.13	6.5	3.5	1.2	25	26	119	13.4	0.92	11
84	10039345	63	Non Alcoholic	Non Alcoholic	12.3	4.85	45.2	9700	274000	73.2	25.4	27.2	0.36	0.18	0.18	5.6	3.6	1.8	202	15	85	84.1	1.03	11
85	10040633	33	Non Alcoholic	Non Alcoholic	14.5	4.67	44.6	7400	319000	89	30	31.1	0.54	0.34	0.2	6.1	4.4	1.7	30	23	98	16.4	0.8	11
86	10040352	23	Non Alcoholic	Non Alcoholic	15.4	5.49	46.1	7000	208000	92.2	30.1	31	0.62	0.3	0.32	7.5	4.9	1.9	25	26	80	19.8	1.07	11.2
87	10041651	52	Non Alcoholic	Non Alcoholic	13.3	4.16	45	6000	304000	90.1	29.9	31.1	0.58	0.38	0.2	7	3.5	1	23	33	86	26.6	0.5	11.6
88	10040196	35	Non Alcoholic	Non Alcoholic	12.6	4.15	42.1	4200	240000	91.1	29.4	31.3	0.65	0.43	0.22	6.8	3.9	2.8	27	18	90	24	0.7	11.1
89	10040426	56	Non Alcoholic	Non Alcoholic	12.1	4.11	42.7	7600	357000	89.9	27.4	30.3	0.45	0.25	0.2	6.7	3.4	3.3	29	34	89	30	0.7	11.5
90	10025896	47	Non Alcoholic	Non Alcoholic	13.4	4.4	40.9	8700	407000	90.6	30.3	30.3	0.69	0.43	0.26	7.1	4.3	2.8	40	35	99	30.7	0.8	11.7
91	1169174	35	Non Alcoholic	Non Alcoholic	13.9	4.54	43.4	5300	219000	92.1	30.5	32	1.74	1.32	0.42	6.5	3.5	1.2	29	43	51	42	0.85	15
92	1177634	60	Non Alcoholic	Non Alcoholic	14	5.86	40.6	6700	215000	79.2	31	31	0.5	0.16	0.34	6.8	4.1	2	20	18	68	23	0.86	0.14
93	1188172	75	Non Alcoholic	Non Alcoholic	14.4	4.85	40.8	11500	371000	95	30	30.4	1.47	0.87	0.6	6.7	4.1	1.6	19	15	65	18	0.54	11
94	1179875	39	Non Alcoholic	Non Alcoholic	14.5	4.2	43.8	12500	352000	82.7	27.3	33	0.86	0.32	0.54	7.7	4.3	1.3	32	139	122	23	0.84	12
95	1166519	45	Non Alcoholic	Non Alcoholic	12.4	4.8	37.5	10000	250000	78.1	25.7	32.9	0.83	0.61	0.22	6.5	3.5	1.1	18	20	192	20	0.3	12.2
96	1197360	82	Non Alcoholic	Non Alcoholic	13.7	4.62	39.7	8200	208000	86.6	29.6	34.5	0.68	0.29	0.39	7.6	4	1.1	22	13	93	28	0.85	11
97	1197812	72	Non Alcoholic	Non Alcoholic	14.5	4.66	41.9	6300	180000	89.3	31.1	34.6	0.99	0.37	0.62	6.7	3.7	1.3	17	27	73	20	0.81	11.1
98	1199195	56	Non Alcoholic	Non Alcoholic	15.6	6.37	52.2	10500	175000	81.9	24.5	29.9	0.59	0.3	0.29	7	4.5	1.8	30	26	120	21	1.05	11.9
99	10005544	55	Non Alcoholic	Non Alcoholic	13.1	3.97	39.7	8600	407000	90	33	33	0.54	0.3	0.24	6.8	4	1.4	32	28	80	22.1	0.89	10.7
100	10008207	74	Non Alcoholic	Non Alcoholic	14.1	4.53	41.6	7900	213000	90.4	31.1	33.8	0.59	0.29	0.3	6.6	3.5	1.1	26	13	68	19.5	0.76	10.6
101	10008383	85	Non Alcoholic	Non Alcoholic	14.2	4.73	41.9	8800	193000	88.5	30	33.9	0.72	0.33	0.39	7	3.8	1.2	17	10	233	20.5	1.08	10.8
102	10007692	63	Non Alcoholic	Non Alcoholic	13.8	4.8	41.2	14000	193000	88.2	32	32.3	1.33	0.56	0.77	7.4	4.4	1.5	21	22	70	37	1.2	12
103	10008290	56	Non Alcoholic	Non Alcoholic	14.3	4.49	42.8	8600	235000	95.4	31.8	33.23	0.3	0.13	0.17	6.9	4.4	1.8	21	18	102	22	0.67	10.3
104	10006998	82	Non Alcoholic	Non Alcoholic	14.2	5.01	44.5	10300	210000	91.5	31.2	31.4	1.14	0.48	0.66	6.3	3.4	1.2	20	16	100	11.7	1.1	11
105	10008173	35	Non Alcoholic	Non Alcoholic	11.2	4.14	42.3	6700	268000	89.9	32.2	31.8	1.06	0.52	0.54	7.7	5.1	2	24	17	59	48	1.01	11
106	10006482	18	Non Alcoholic	Non Alcoholic	16.7	5.69	55.8	10500	331000	79.5	30.5	31.1	0.56	0.31	0.25	7.5	4.8	1.8	16	14	104	11.9	0.83	11
107	10007458	31	Non Alcoholic	Non Alcoholic	15.1	4.69	50.3	10700	145000	83.9	31	31.4	2.2	0.98	1.22	6.3	3.8	1.5	25	25	144	19.3	0.71	13.1
108	1184266	63	Non Alcoholic	Non Alcoholic	10.8	3.6	33.6	6500	219000	93.2	30.1	32.3	0.56	0.29	0.27	6.1	3.2	1.1	30	21	138	27	0.83	11
109	10002103	38	Non Alcoholic	Non Alcoholic	12.3	3.78	39.2	4400	360000	104.5	32.8	31.4	0.45	0.19	0.26	7.6	4.3	1.3	14	18	99	15.8	0.75	11
110	1118156	72	Non Alcoholic	Non Alcoholic	14.2	4.48	43.6	9780	225000	96.8	32.5	30.9	0.6	0.2	0.4	6.6	3.4	1	26	25	87	26	0.8	14
111	10008415	45	Non Alcoholic	Non Alcoholic	11.2	4.15	37.6	15000	264000	95.5	32.1	32	0.36	0.18	0.18	6.1	3.8	1.7	20	23	85	51.3	0.76	10.5
112	10007856	46	Non Alcoholic	Non Alcoholic	14.5	4.92	44.2	8900	298000	89.8	29.5	32.8	1.13	0.46	0.67	7.2	4.2	1.4	48	45	75	23.2	1.02	11
113	10008435	76	Non Alcoholic	Non Alcoholic	10.2	4.69	43.3	9700	218000	95	30	33	0.42	0.1	0.32	6.3	3.2	1	26	30	130	70.7	0.86	11.3
114	10007297	33	Non Alcoholic	Non Alcoholic	11.9	4.11	34.7	8400	164000	84.4	28.9	34.2	0.61	0.2	0.41	5.8	3	1.1	111	72	100	16.5	0.92	14
115	10008388	76	Non Alcoholic	Non Alcoholic	8.5	2.15	24.6	3300	125000	114	39.3	34.5	1.4	0.76	0.64	6.7	3.4	1	29	17	87	40.8	1.02	12
116	10007558	27	Non Alcoholic	Non Alcoholic	13.3	4.78	39.6	7800	278000	82.7	27.8	33.6	0.7	0.29	0.41	7.3	4	1.2	34	21	61	28.1	0.85	10
117	10029520	33	Non Alcoholic	Non Alcoholic	15.8	5.97	46.2	7800	276000	89	30	33	0.71	0.36	0.35	7.8	4.8	1.6	21	14	112	18.3	0.78	10
118	10025187	40	Non Alcoholic	Non Alcoholic	8.3	2.39	25.9	8700	132000	109	35.3	32.3	3.13	3.07	0.06	6.1	2	0.5	59	29	193	12.1	0.77	18.2
119	10035016	47	Non Alcoholic	Non Alcoholic	14.4	5.06	43.8	7600	207000	90.4	31	30.1	0.57	0.34	0.23	6.7	4.3	1.8	26	47	55	43.8	0.92	10.4
120	10036138	30	Non Alcoholic	Non Alcoholic	13.1	4.32	40.4	8200	230000	91.1	31	30.2	0.43	0.2	0.23	7.1	3.9	1.2	26	17	71	29.3	0.81	12.8