CORRELATION BETWEEN SERUM LIVER ENZYMES AND HYPERTENSION.

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IN

GENERAL MEDICINE

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ABBREVIATIONS

ABI - Ankle-Brachial Index

ACC - American College of Cardiology

ALP - Alkaline Phosphatase

ALT - Alanine Aminotransferase

AST - Aspartate Aminotransferase

BMI - Body Mass Index

CHF - Congestive Heart Failure

CO - Cardiac Output

DBP - Diastolic Blood Pressure

ECG - Electrocardiogram

eGFR - Estimated Glomerular Filtration Rate

ESC - European Society of Cardiology

ESR - Erythrocyte Sedimentation Rate

GGT - Gamma-Glutamyl Transferase

HbA1c - Hemoglobin A1c

HTN - Hypertension

IHD - Ischemic Heart Disease

LVH - Left Ventricular Hypertrophy

NAFLD - Non-Alcoholic Fatty Liver Disease

NSAIDs - Non-Steroidal Anti-Inflammatory Drugs

RAAS - Renin-Angiotensin-Aldosterone System

SBP - Systolic Blood Pressure

SVR - Systemic Vascular Resistance

WHO - World Health Organization

ABSTRACT

Background: The increasing prevalence of non-communicable diseases (NCDs), particularly hypertension, poses a global health challenge. Hypertension is a significant risk factor for cardiovascular diseases and stroke, with emerging evidence suggesting its association with liver dysfunction. Elevated liver enzymes such as ALT, AST, and ALP are potential markers of non-alcoholic fatty liver disease (NAFLD) and related conditions, which may contribute to hypertension's pathophysiology. This study aims to estimate serum liver enzyme levels in hypertensive and normotensive individuals and evaluate the correlation between serum liver enzymes and hypertension.

Materials and Methods: A hospital-based cross-sectional study was conducted at B.L.D.E (DU) Shri B.M. Patil Medical College with 140 participants, divided into hypertensive and normotensive groups. Liver enzyme levels were measured alongside other clinical parameters. Inclusion criteria included individuals aged ≥18 years with no history of hepatotoxic drug use or severe liver disease. Data were analyzed using SPSS v20, employing t-tests, Mann-Whitney U tests, and Chi-square tests to compare groups. A p-value <0.05 was considered statistically significant.

Results: The mean age of participants was 55.66±11.1 years, with a majority being male. Routine blood parameters did not differ significantly between

groups. However, hypertensive individuals exhibited significantly higher mean levels of ALT, AST, and ALP ($p<0.05$) compared to normotensive participants.

Conclusion: The study highlights a significant association between elevated liver enzymes and hypertension, suggesting liver dysfunction's potential role in hypertension's pathophysiology. Integrating liver enzyme assessments in hypertension management may provide insights into underlying mechanisms and identify individuals at risk for liver-related complications. Further studies are warranted to validate these findings and their clinical implications.

Keywords: Hypertension, Liver Enzymes, ALT, AST, ALP, Non-communicable Diseases, NAFLD

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INTRODUCTION

In the past, the world has faced challenges as complex as those now ascribed by a trio of threats: First, the undernourished and the unfinished agenda of infections; second, the increasing global burden of non-communicable diseases; and third, the complications arising from globalisation itself, like the ill-effects of climate changes.[1] Before the antibiotic era, communicable diseases had a dominant role, but with the advent of new efficient antibiotics, communicable diseases are now no more a big problem.[2]

Since there is an increase in the prevalence of diseases such as stroke, cardiovascular diseases, hypertension, diabetes, and cancer, non-communicable diseases are now projected as a global health crisis. The world health organisation's global status report (2010) states that non-communicable diseases are the leading cause of worldwide deaths contributing to 60%. [3] In India, the situation is bleak. In 2005, total mortality of 53% and 44% of daily adjusted life years lost was attributed to non-communicable diseases. By 2030, the total mortality by non-communicable diseases is projected to be 67% in India.[1,4]

The alarming rise in the magnitude of non-communicable diseases demands urgent attention. Recently, the world health organisation identified six risk factors for non-communicable diseases for death and those six risk factor sare hypertension; impaired glucose tolerance; tobacco usage; dyslipidemia; lack of physical activity and obesity.[1] Of the above-said risk factors, hypertension is responsible for 13% of total deaths in the world, followed by tobacco usage (9%); impaired glucose tolerance (6%); physical inactivity (6%), and obesity (5%).[5] Among the non-communicable diseases, hypertension claims a number of first because of the following reasons; the most common chronic condition, a significant risk factor for heart disease and stroke, accounts for most drug prescriptions, and throughout the world, it is the number one attributable risk for deaths. Hypertension is one of the main components of a crucial metabolic syndrome. Metabolic syndrome, if present in an individual, increases the risk of cardiovascular diseases.

The liver, which is a vital organ, has numerous functions such as synthesis, storage, degradation and biotransformation of bio-molecules in the human body.[6] The association of the development of hypertension with liver dysfunction is being increasingly recognised.[7,8]

The liver enzymes - alanine and aspartate aminotransferase (ALT and AST), γ-glutamyl transferase (GGT), and alkaline phosphatase (ALP) are being widely used as markers of liver function. The elevated levels of ALT, AST, and GGT point out to excess fat deposition in the liver, a condition termed non-alcoholic fatty liver disease (NAFLD).[9] These enzymes have been considered to have substantial clinical and epidemiological importance as convenient, reliable markers of NAFLD and related liver dysfunction. Some epidemiological studies have shown an association between ALT and GGT with metabolic syndrome, cardiovascular diseases and type 2-diabetes. In previous studies, cardiovascular diseases have been shown to be the leading causes of death in NAFLD, with higher rates coinciding with increased liver-related mortality throughout follow-up investigations.[10–12]

Since hypertension happens to be a significant risk factor for both cardiovascular diseases as well as stroke, there should be a well-devised approach to knowing the factors involved in the pathogenesis and preventing hypertension. Hence, it is necessary to study the levels of liver enzymes in hypertensive people to know the correlation between serum

liver enzymes and blood pressure for future intervention to control the elevation in blood pressure even after initiation of pharmacological therapy.[13]

REVIEW OF LITERATURE

Hypertension refers to the force of blood pressing against the walls of the systemic arteries.[14] It is categorized into two types:

"Primary (Essential) Hypertension: This type is diagnosed when a person's blood pressure consistently exceeds 140 mmHg systolic and 90 mmHg diastolic, without any identifiable underlying cause. Approximately 90–95% of hypertension cases fall into this category."

"Secondary Hypertension: This occurs when blood pressure surpasses 140 mmHg systolic and 90 mmHg diastolic due to a known underlying cause. It accounts for about 5–10% of hypertension cases."

Hypertension has become a significant public health concern among the global adult population, contributing to approximately 9.4 million deaths annually. It doubles the risk of developing cardiovascular conditions such as coronary heart disease, congestive heart failure, ischemic and hemorrhagic strokes, kidney failure, peripheral arterial disease, and hypertensive retinopathy.[15]

Hypertension (HTN) is identified by systolic blood pressure (SBP) readings of 130 mmHg or higher and/or diastolic blood pressure (DBP) exceeding 80 mmHg. Characterized by a persistent increase in arterial pressure, it is one of the most common chronic medical conditions worldwide.[16]

Hypertension has been a major focus of medical research over the past century, as it is a key comorbidity contributing to the development of stroke, myocardial infarction, kidney failure, heart failure, and other serious health conditions.

There is agreement that persistent blood pressure readings of 140/90mmHg or above should be treated with the conventional therapeutic aim of 130/80mmHg or less. The definition and classifications of hypertension have been developing throughout time.

Etiology[17]

Essential hypertension, also known as idiopathic hypertension, is the most prevalent form of the condition. It has long been suggested that high salt intake increases the risk of developing hypertension. One key factor linked

to the onset of essential hypertension is the individual's genetic predisposition to salt sensitivity. Approximately 50–60% of individuals with essential hypertension exhibit salt sensitivity, making them more prone to elevated blood pressure. [18–20]

Epidemiology[17]

Hypertension affects over one billion people globally, impacting up to 45% of the adult population. Its prevalence spans all socioeconomic and income groups and increases with age, affecting as much as 60% of individuals over 60 years old. A 2010 global health survey published in *The Lancet*, which analyzed patient data from 67 countries, identified hypertension as the leading cause of mortality and disability-adjusted life years since 1990.

Projections suggest that by 2025, the number of people living with hypertension could rise to 1.5 billion, reflecting an increase of 15–20%.[21]

Pathophysiology

Hypertension develops through several interconnected mechanisms, including increased salt absorption, which leads to fluid retention and

volume expansion, dysregulation of the renin-angiotensin-aldosterone system (RAAS), and heightened activity of the sympathetic nervous system. These factors collectively contribute to elevated total peripheral resistance and afterload, ultimately resulting in the onset of hypertension.

Primary hypertension

"Maintenance of arterial blood pressure is necessary for organ perfusion. In general, the arterial blood pressure is determined by the following equation:" [22]

"Blood pressure (BP) = Cardiac output (CO) x Systemic vascular resistance (SVR)"

Blood pressure adjusts to environmental changes to ensure adequate organ perfusion under diverse conditions. Its regulation is primarily influenced by three key factors: the sympathetic nervous system, the renin-angiotensin-aldosterone system (RAAS), and plasma volume, which is largely controlled by kidney function.

Primary hypertension (previously known as "essential" hypertension) is poorly understood, although it is most likely caused by a combination of hereditary and environmental variables that have several compounding effects on cardiovascular and renal structure and function. Some of these variables are described more below.

Risk factors for essential hypertension

Although the exact etiology of primary hypertension remains unclear, a number of risk factors are strongly and independently associated with its development, including:

- Age
- Family history
- Obesity
- Reduced nephrons number
- Race

- High sodium diet
- Excessive alcohol consumption
- Physical inactivity

Secondary or contributing causes for hypertension

A variety of medical problems, both common and uncommon, can raise blood pressure and contribute to secondary hypertension. These causes, which in many cases combine with risk factors for primary hypertension, are important impediments to obtaining optimal blood pressure control.

Major causes are

- Oral contraceptives
- Corticosteroids
- NSAIDs
- Eruthropoietin

- Sodium containing antacids
- Tyrosing kinase inhibitors
- Primary aldosteronism
- Obstructive sleep apnea
- Renovascular hypertension
- Cushings syndrome
- Pheochromocytoma

Evaluation

To diagnose hypertension, the ACC recommends obtaining at least two blood pressure measurements during two separate office visits. The ESC/ESH suggests taking three office blood pressure readings at intervals of 1–2 minutes, with additional readings if the first two differ by more than 10 mmHg. The average of the last two measurements is then used to determine blood pressure.

Both organizations emphasize the importance of accurate measurement and classifying patients into higher stages or grades of hypertension for effective management. Proper technique is crucial, including ensuring the patient is seated calmly for at least 5 minutes before measurement. Additionally, the blood pressure cuff should cover 80% of the arm circumference, as cuffs that are too large or too small can lead to inaccurate readings, either underestimating or overestimating blood pressure.Ambulatory blood pressure monitoring is the most accurate way to detect hypertension and may also help identify people with disguised hypertension and the white coat effect.

The evaluation consists of looking for signs of end-organ damage and consists of the following,

- 12 lead ECG
- Fundoscopy to look for retinopathy/ maculopathy
- Blood workup including ESR, complete blood count, eGFR, creatinine, electrolytes, thyroid profile, HbA1c, blood cholesterol levels, and serum uric acid

- Imaging including carotid doppler ultrasound, echocardiography and brain imaging
- Urine albumin - creatinine ratio
- Ankle-brachial pressure index - ABI

Association of hypertension with other condition;

Obesity:

Obesity significantly increases the likelihood of developing hypertension, particularly when it involves abdominal or visceral fat, as seen in metabolic syndrome. This, in turn, heightens the risk of ischemic heart disease (IHD). Data from the Framingham Study revealed that the incidence of hypertension was 46% higher in overweight men and 75% higher in overweight women—defined by a body mass index (BMI) of 25.0 to 29.9—compared to individuals with a normal BMI.[23]

Physical activity:

Regular aerobic exercise can help prevent hypertension and reduce blood pressure in people who are already hypertensive. The connection might

entail the reversal of age-related reductions in endothelium-dependent vasodilation.[23]

Alcohol consumption:

Excessive alcohol consumption, defined as more than two servings per day or binge drinking, can elevate blood pressure and increase arterial stiffness. High doses of alcohol exert a pressor effect by raising cardiac output and heart rate, likely due to heightened sympathetic nerve activity. Additionally, alcohol alters cell membrane properties, potentially increasing calcium influx by inhibiting sodium transport.

Smoking:

Cigarette smoking leads to an increase in blood pressure, primarily due to nicotine-triggered release of norepinephrine from adrenergic nerve terminals. Additionally, smoking causes a significant and acute reduction in radial artery compliance, regardless of the blood pressure elevation, further contributing to cardiovascular risk.

Hyperuricemia:

Hyperuricemia is seen in 25 - 50% of people with untreated primary hypertension, which is approximately 5 times the frequency reported in normotensive people. Hyperuricemia is caused by a reduction in renal blood flow, which is thought to be caused by nephrosclerosis.[24]

Hypercholesterolemia:

Because it affects endothelium-dependent vasodilation, hypercholesterolemia usually coexists with hypertension. Lipid lowering treatment improves nitric oxide bioavailability, decreases arterial stiffness, and lowers blood pressure.[25]

Mechanism of hypertension

Most cases of hypertension have no identifiable or specific cause, which is why the condition is referred to as primary (or essential) hypertension. Chronic hypertension arises as a response to an increase in cardiac output or peripheral resistance, often due to abnormalities in the factors that regulate these two forces. These underlying issues can involve various factors affecting cardiac output and peripheral resistance, and their interactions may differ in nature and severity among patients, potentially

triggering the condition.

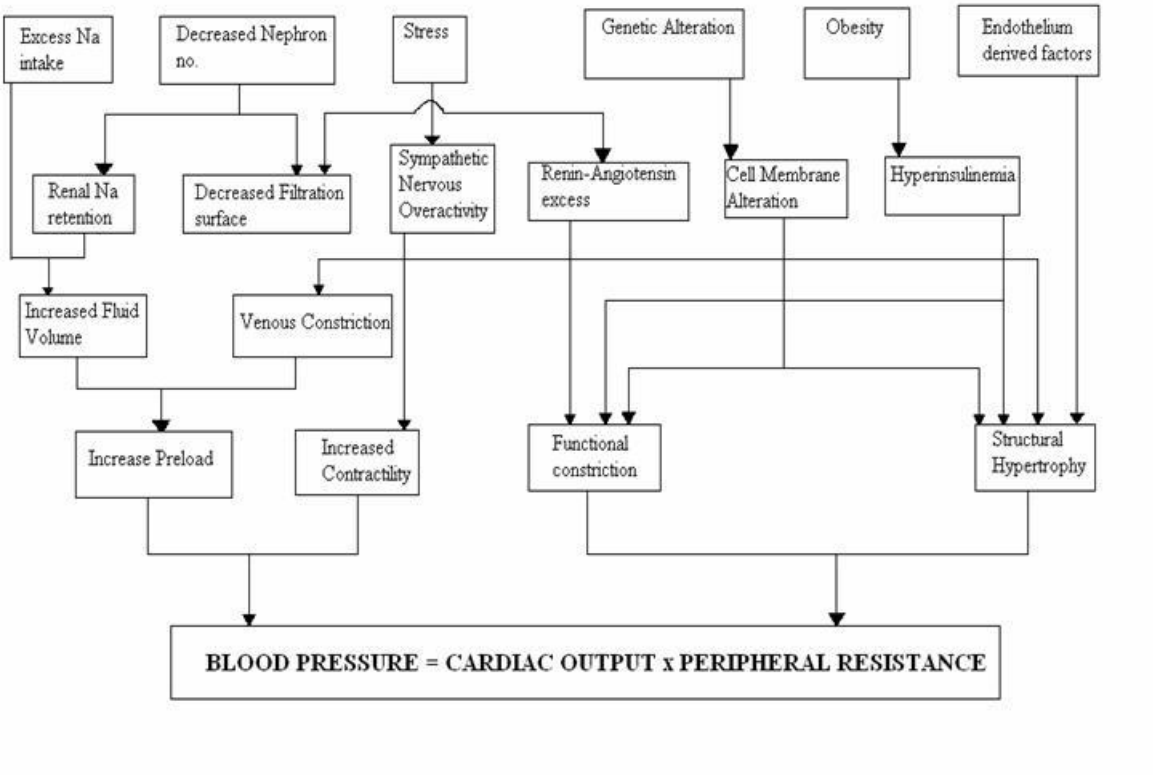

Figure 1: Factors involved in control of blood pressure

If left untreated, approximately 50% of individuals with hypertension die from coronary heart disease or congestive heart failure, about 33% from stroke, and 10-15% from renal failure. Those with rapidly progressing hypertension or diabetes, especially if proteinuria or other signs of nephropathy develop, are at a higher risk of dying from kidney failure.

Hypertension increases the strain on the left ventricular myocardium, leading to stiffness, hypertrophy, and accelerating the development of atherosclerosis in the coronary arteries. Early functional changes in hypertension are typically seen as left ventricular diastolic dysfunction, characterized by a reduced E/A ratio and a prolonged isovolemic relaxation time.[26,27]

Left Ventricular Hypertrophy

Hypertrophy can occur as a response to increased afterload due to elevated systemic vascular resistance. Left ventricular hypertrophy (LVH) is linked to several dysfunctions, including reduced coronary vasodilatory capacity, impaired left ventricular wall mechanics, and an abnormal diastolic filling pattern of the left ventricle.

In cases of congestive heart failure, LVH-related changes in both systolic and diastolic function can progress to heart failure. In the Framingham cohort, a 20 mm Hg increase in systolic blood pressure was associated with a 56% higher risk of congestive heart failure (CHF).

Individuals with hypertension often struggle to increase their end-diastolic volume under stress due to impaired left ventricular relaxation and compliance. This results in a sequence of events, where left ventricular end-diastolic pressure rises, followed by increased left atrial pressure, leading to pulmonary edema.[28,29]

Liver Enzymes and Their Importance: Association with Hypertension

Liver enzymes are biomolecules produced by liver cells that play essential roles in maintaining metabolic processes, detoxification, and overall homeostasis in the human body. These enzymes are critical for the synthesis, metabolism, and degradation of proteins, fats, and carbohydrates, as well as the regulation of biochemical reactions. When liver cells are damaged or stressed, these enzymes can leak into the bloodstream, serving as vital indicators of liver function and health. Among the most commonly assessed liver enzymes are alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP). These enzymes provide invaluable insights into liver health and can also reflect systemic conditions, including hypertension (HTN).[9]

Key Liver Enzymes and Their Functions

1. **Alanine Aminotransferase (ALT):** ALT is a transaminase enzyme primarily found in the liver. It catalyzes the transfer of an amino group from alanine to α-ketoglutarate, a critical step in amino acid metabolism. Elevated levels of ALT in the blood are often associated with liver cell injury or conditions such as fatty liver disease, hepatitis, or drug-induced liver damage. ALT is considered a sensitive marker of hepatocellular damage due to its liver-specific nature.[30]
2. **Aspartate Aminotransferase (AST):** Like ALT, AST is involved in amino acid metabolism and is found in the liver, heart, muscles, and other tissues. While it is less specific to the liver, elevated AST levels often indicate liver damage or systemic conditions such as myocardial infarction or muscle injury. A high AST/ALT ratio may point to advanced liver disease or alcoholic liver disease.[31]
3. **Gamma-Glutamyl Transferase (GGT):** GGT is an enzyme involved in the transfer of gamma-glutamyl functional groups, which are crucial for the metabolism of glutathione and other amino acids. GGT levels are highly sensitive to bile duct obstruction and alcohol

consumption. Elevated GGT is also associated with oxidative stress, cardiovascular diseases, and metabolic syndrome, linking it to conditions beyond liver-specific pathology.[32]

4. **Alkaline Phosphatase (ALP):** ALP is involved in dephosphorylation reactions and is present in the liver, bones, kidneys, and bile ducts. Elevated ALP levels are often linked to biliary obstruction, bone diseases, or certain cancers. In liver diseases, a significant increase in ALP may indicate cholestasis or liver infiltration.[33]

Importance of Liver Enzymes in Clinical Diagnostics

Liver enzymes are essential diagnostic markers used to evaluate liver health and function. Abnormal levels of these enzymes may indicate a wide range of liver diseases, including:

- Non-alcoholic fatty liver disease (NAFLD)
- Alcoholic liver disease
- Viral hepatitis (Hepatitis A, B, C, etc.)
- Drug-induced liver injury
- Cirrhosis or liver fibrosis

- Cholestatic disorders

Beyond liver-specific conditions, liver enzyme abnormalities often reflect systemic diseases, such as obesity, diabetes, and cardiovascular disorders, including hypertension.

Association Between Liver Enzymes and Hypertension

Hypertension is a major risk factor for cardiovascular diseases and is increasingly recognized as a condition closely tied to metabolic and systemic disorders, including liver dysfunction. Evidence suggests that elevated liver enzymes, particularly ALT, AST, GGT, and ALP, are associated with the development and progression of hypertension.

Elevated ALT and AST in Hypertension:

Studies have shown that elevated ALT and AST levels are positively correlated with high blood pressure. This relationship may stem from the underlying metabolic disturbances that are common in hypertensive individuals, such as insulin resistance, systemic inflammation, and oxidative stress. In particular, ALT and AST are frequently elevated in

patients with NAFLD, a condition strongly linked to metabolic syndrome and hypertension.[34,35]

In hypertensive individuals, elevated ALT and AST levels may indicate subclinical liver dysfunction or NAFLD. The liver's role in lipid metabolism and systemic inflammation could exacerbate vascular dysfunction, contributing to elevated blood pressure. The independent association between elevated ALT and AST and hypertension suggests that liver health plays a significant role in vascular regulation.

Gamma-Glutamyl Transferase (GGT) and Hypertension:

GGT has emerged as a reliable biomarker for oxidative stress and cardiovascular risk. Elevated GGT levels are frequently observed in hypertensive patients and have been linked to the pathogenesis of hypertension through mechanisms such as:

- Increased oxidative stress leading to vascular endothelial dysfunction
- Enhanced systemic inflammation
- Dysregulation of glutathione metabolism

Several studies have demonstrated a dose-response relationship between GGT levels and hypertension risk. Furthermore, GGT is a strong predictor of cardiovascular morbidity and mortality, underscoring its importance in assessing hypertension-related complications.[36–38]

Alkaline Phosphatase (ALP) and Hypertension:

ALP is less specific to the liver but remains a critical marker in hypertensive individuals. Elevated ALP levels are associated with arterial stiffness, endothelial dysfunction, and systemic inflammation—key contributors to hypertension. High ALP levels have also been linked to an increased risk of cardiovascular diseases, suggesting that ALP may serve as an early indicator of vascular damage in hypertensive patients.

Potential Mechanisms Linking Liver Enzymes to Hypertension

1. **Oxidative Stress:** Liver enzymes, particularly GGT, are involved in oxidative stress pathways. Increased oxidative stress can damage vascular endothelium, leading to impaired vasodilation and elevated blood pressure.

2. **Systemic Inflammation:** Elevated liver enzymes often reflect systemic inflammation, which can contribute to vascular stiffness and increased peripheral resistance, both of which are hallmarks of hypertension.
3. **Metabolic Dysregulation:** Liver enzymes are often elevated in metabolic syndrome and insulin resistance, conditions that frequently coexist with hypertension. Dysregulated glucose and lipid metabolism can exacerbate hypertensive processes.
4. **Endothelial Dysfunction:** Abnormal liver enzyme levels may signal impaired endothelial function, a precursor to atherosclerosis and hypertension.[39,40]

Clinical Implications and Future Directions

Monitoring liver enzyme levels in hypertensive patients provides a dual benefit: evaluating liver health and assessing cardiovascular risk. Elevated liver enzymes in hypertensive individuals should prompt further investigation for underlying conditions, such as NAFLD or metabolic syndrome, which may require targeted interventions.

The integration of liver enzyme monitoring into hypertension management could lead to earlier detection of systemic complications, enabling timely therapeutic interventions. Moreover, reducing liver enzyme levels through lifestyle modifications (e.g., weight loss, dietary changes, and physical activity) or pharmacological treatments may help mitigate hypertension-related risks.

Conclusion

Liver enzymes, including ALT, AST, GGT, and ALP, are not only vital markers of liver health but also provide insights into systemic conditions like hypertension. Their association with oxidative stress, systemic inflammation, and metabolic dysregulation underscores their relevance in vascular health. Elevated liver enzyme levels in hypertensive individuals highlight the interplay between liver function and cardiovascular risk, emphasizing the importance of comprehensive monitoring and management. Future research should explore the mechanistic pathways linking liver dysfunction and hypertension, paving the way for integrated approaches to prevent and manage these interrelated conditions.

Various articles discussing the liver enzyme levels in hypertension;

In a study conducted by McCallum L et al., (2015) to assess the blood pressure control and the liver enzyme changes. Serum total bilirubin and alanine transaminase were significantly negatively associated with all-cause and cardiovascular mortality, while alkaline phosphatase and γ-glutamyl transpeptidase showed positive associations, and aspartate transaminase demonstrated a U-shaped relationship. Serum bilirubin improved continuous net reclassification for 25-year and 35-year cardiovascular mortality by 8% to 26%, while all liver markers combined enhanced reclassification by 19% to 47% compared to the reference model. Among hypertensive patients, liver enzymes and bilirubin within four standard deviations of the mean exhibited independent effects on mortality and blood pressure control. These findings highlight potential mechanisms linking liver markers to blood pressure and cardiovascular risk but provide limited support for their use in clinical risk stratification.[41]

In a study conducted by Rahman S et al., (2020) to assess the serum liver enzymes and hypertension association. The hypertensive group exhibited significantly higher mean concentrations of serum ALT, AST, and GGT

compared to the normotensive group ($p < 0.01$, $p < 0.01$, and $p < 0.001$, respectively). Elevated liver enzymes were more prevalent in the hypertensive group (49.2%) than in the normotensive group (38.1%), with a significantly higher prevalence of elevated ALT, AST, and GGT ($p < 0.01$, $p < 0.01$, and $p < 0.001$, respectively). A clear trend of increased liver enzyme levels was observed with rising blood pressure, and serum ALT and GGT demonstrated an independent association with hypertension. These findings highlight a higher prevalence of elevated liver enzymes among hypertensive individuals, with increased serum ALT and GGT activities positively linked to hypertension in Bangladeshi adults.[8]

In a study conducted by Ahmed R et al., (2021) to assess the liver enzymes in patients with hypertension. This retrospective observational study included 180 patients, with 33% being normotensive and 67% hypertensive, all having type 2 diabetes mellitus (T2DM). While no statistically significant differences were observed between study variables in the two groups, elevated levels of GGT, ALT, and AST were noted in normotensive T2DM patients compared to their hypertensive counterparts. Abnormal liver function was more prevalent among patients with uncontrolled diabetes compared to those with good glycemic control

($p<0.05$). The study concludes that elevated liver enzymes, including bilirubin, GGT, SGOT, and SGPT, are common among T2DM patients, irrespective of hypertensive status, emphasizing the importance of liver function monitoring in diabetic management.[42]

In a study conducted by Aberg F et al., (2022) to assess the association of arterial hypertension and liver enzymes. In fully adjusted Cox regression models, both measured systolic blood pressure and clinically defined hypertension (HTA) were associated with liver-related outcomes. Polygenic risk scores (PRSs) for systolic and diastolic blood pressure also showed significant associations with liver. Among individuals in the highest quintile of systolic blood pressure PRS, initiating antihypertensive medication was linked to reduced rates of liver-related outcomes. These findings indicate that both hypertension and genetic predisposition to elevated blood pressure are associated with adverse liver outcomes. The initiation of antihypertensive medication appears to mitigate this risk in individuals with a high genetic predisposition to HTA, underscoring the importance of timely blood pressure management in reducing liver-related complications.[43]

In a study conducted by Khalili P et al., (2022) to assess the relation between serum liver enzymes and hypertension. Among 9,930 participants (mean age: 49.94 ± 9.56 years, 46.56% men), higher levels of ALT, GGT, and ALP were associated with significantly increased odds of abnormal blood pressure. After adjusting for confounding variables, only elevated ALP remained independently significant for both males and. In participants with normal ALT, AST, GGT, and ALP levels, a dose-response relationship was observed between increasing blood pressure and enzyme levels in both genders. Notably, elevated ALP was the only liver enzyme significantly linked to the odds of stage 1 and stage 2 hypertension across both sexes. Elevated serum ALP activity is positively associated with higher odds of hypertension in males and females, suggesting it may serve as an early indicator of hypertension.[44]

In a study conducted by Fard M et al., (2022) to assess the association between serum liver enzyme and hypertension. The study included 8,267 participants, with 3,664 analyzed for γ-glutamyl transpeptidase (GGT) levels following propensity score matching (PSM). Multivariate Cox proportional hazards modeling revealed that elevated GGT levels were associated with a higher risk of hypertension. This relationship remained

positive and significant after PSM analysis. The 5-year incidence rates of hypertension were 1.27 and 0.81 per person-year for men and women, respectively. GGT demonstrated the highest accuracy among liver enzymes for predicting hypertension, with an AUROC of 0.7837. These findings suggest that GGT could serve as a valuable biomarker for the early detection of hypertension, highlighting the importance of monitoring its levels for preventive care.[45]

In a study conducted by Sakboonyarat B et al., (2023) to assess the association of raised blood pressure with elevated serum liver enzymes. In a study of active-duty RTA personnel, elevated serum AST and ALT levels were positively correlated with raised blood pressure (BP). Among males with hypertension (HT), the β coefficients for log-transformed AST and ALT were 0.13 (95% CI: 0.12–0.13) and 0.11 (95% CI: 0.11–0.12), respectively, while in females, the coefficients were 0.03 (95% CI: 0.02–0.04) and 0.07 (95% CI: 0.05–0.08). Hypertension was independently associated with higher odds of elevated AST and ALT in males, and elevated AST in females. These findings suggest that hypertension is linked to elevated AST and ALT levels, with the association influenced by sex differences. Monitoring liver enzymes may provide additional insights

into the relationship between BP and liver health, highlighting potential sex-specific implications for hypertension management.[46]

In a study conducted by Baeradeh N et al., (2023) to assess the relation between liver enzymes and incidence of hypertension. This study examined the link between liver enzymes and hypertension risk in a cohort of 7,710 individuals aged 40–70 years. Elevated levels of alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) were significantly associated with an increased risk of hypertension, even after adjusting for potential confounding factors. Subgroup analyses also identified a notable relationship between ALP concentrations and high blood pressure within specific age ranges. These findings highlight the potential of monitoring liver enzymes—particularly ALT, GGT, and ALP—as predictive markers for hypertension risk, enabling early identification of individuals who may benefit from targeted preventive measures. This underscores the broader role of liver function monitoring in the prevention and management of hypertension.[47]

In a study conducted by Faramarzi E et al., (2024) to assess the relation between liver enzymes and hypertension. Among 14,184 participants, 5.7% had pre-hypertension (pre-HTN), and 39.6% had hypertension (HTN). In

adjusted models, AST levels of 19–23 IU/L were associated with a higher risk of pre-HTN. A dose-response relationship was observed with ALT, showing the highest odds of pre-HTN in the third tertile (OR: 1.34; 95% CI: 1.09–1.63), and GGT also exhibited elevated odds in the third tertile (OR: 1.25; 95% CI: 1.03–1.52). Similarly, the odds of HTN increased with higher levels of AST, ALT, ALP, and GGT, with the third tertile yielding the highest ORs. Among these enzymes, GGT was most strongly associated with HTN. These findings demonstrate that elevated levels of AST, ALT, ALP, and GGT are independently associated with pre-HTN (excluding ALP) and HTN, suggesting their potential utility as predictors for these conditions. Monitoring liver enzymes may enable primary care providers to identify at-risk individuals and implement timely interventions.[48]

AIMS & OBJECTIVES

The study's objective is to estimate the serum liver enzyme levels in randomly selected patients and to study the correlation of serum liver enzyme levels and hypertension.

MATERIAL & METHOD

Source of data: The information for the study was collected from OPD and IPD patients in B.L.D.E (DU) Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapura-583106, Karnataka.

Study design: Hospital-Basedcross-Sectional Study

Sample Size:

Using G*Power version 3.1.9.4 software for sample size calculation, The correlation between ALT and SBP (r=0.157, p=0.049), this study requires a total sample size of 140, so to achieve a power of 99% for detecting a difference in Means: **Exact -** Correlation: Bivariate normal model with 1% level of significance.

Inclusion criteria:

- Aged above 18 years.
- Normotensive patients.
- Hypertensive patients – both newly diagnosed and known cases of essential hypertension.

Exclusion criteria:

- Patients with a history of hepatotoxic drug intake.
- Patients with diagnosed causes of secondary hypertension.
- Patients with severe chronic or acute evidence of liver diseases.

Method of collection of data:

A detailed pro forma was used to collect detailed history and to record the vital parameters, and measure the anthropometric indices. Body weight was recorded using a portable weighing machine and height was measured using a stadiometer, and blood pressure was recorded using a standardised mercury sphygmomanometer. The Institutional Ethical Committee approved the study. The subjects were explained the procedure, and informed consent was obtained. Detailed history was then be elicited from the subjects to exclude diabetes mellitus and renal causes to rule out the causes of secondary hypertension.

Weight was recorded in kilograms using a portable standard weighing machine and vertical height was measured in centimetres using a stadiometer and Quetelet's index was used to calculate body mass index (BMI): weight(in kg)/height(in m^2)

To begin with, the subjects were seated in a quiet room with a comfortable room temperature in an armed chair with the arm and back supported and the legs uncrossed. The mercury sphygmomanometer should be at his/her heart level. It is necessary that there should be abstinence from caffeine ingestion before 30 min of measurement of BP, and then using a standard sphygmomanometer having a cuff size of 25cm x 12.5cm, blood pressure was recorded two times by auscultatory method, and the mean value of the two measurements was taken for analysis. Blood was collected from the antecubital vein in front of the forearm after sterilising the skin with a sterilised cotton swab. About 5 ml of venous blood was drawn from each subject in a plain dry vacutainer tube using disposable syringes.

Investigations

1. Liver Function Tests
2. ECG
3. RBS
4. Echocardiography
5. Chest X-ray PA View

6. CBC

7. Serum Electrolytes

8. Serum Creatinine

9. Blood Urea

10. Urine Routine

The liver enzymes at elevated levels were defined as one or more measurements of:

	MEN	**WOMEN**
AST	>35 U/L	>31 U/L
ALT	>45 U/L	>34 U/L
ALP	>128 U/L	>98 U/L

Hypertension was defined as: **Resting SBP >120 mm hg and/or DBP >90 mm hg.**

STATISTICAL ANALYSIS

The data obtained is entered in a Microsoft Excel sheet, and statistical analyses are performed using a statistical package for the social sciences (SPSS) (Version 20). Results are presented as Mean, SD, counts, and percentages, and diagrams for normally distributed continuous variables between the two groups will be compared using an independent sample t-test. For not normally distributed variables, the Mann-Whitney U test is used, for Categorical variables between the two groups are compared using the Chi-square test/Fisher's exact test. If $p<0.05$ will be considered statistically significant. All statistics are performed in two-tailed

RESULTS

Present study included total of 140 patients fulfilling inclusion criteria and separated into two groups with hypertension and without hypertension. The overall mean of the patients included was s55.66±11.1yrs.

Table 1: Overall mean age of the participants

	N	Minimum	Maximum	Mean	SD
Age	140	36.0	80.0	55.66	11.1

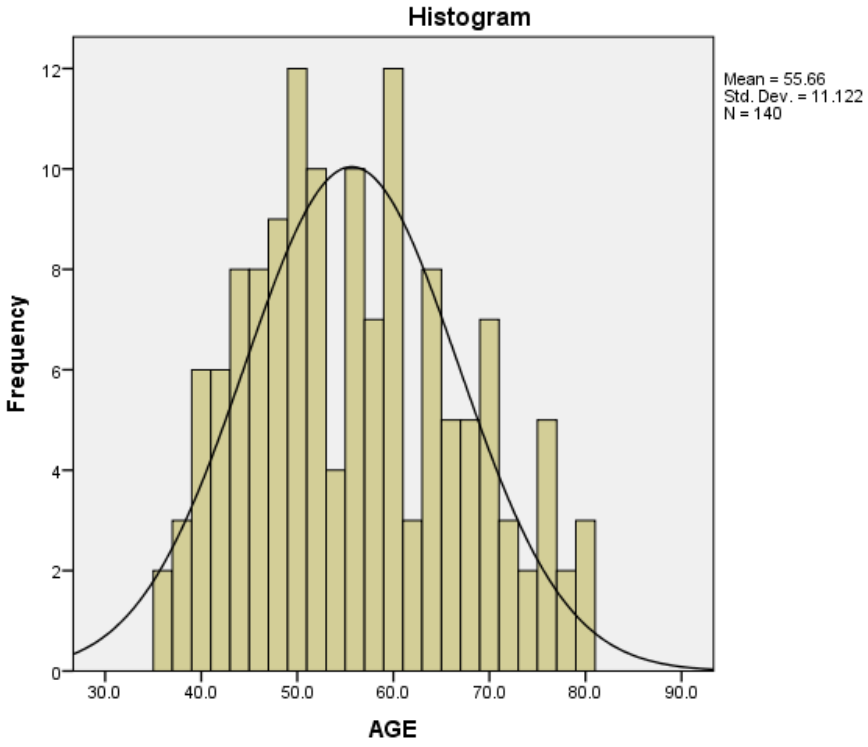

Figure 2: Histogram showing Overall mean age of the participants

Table 2: Comparison of mean age of patients between the groups

	Hypertensive		Normotensive		P-Value
	Mean	SD	Mean	SD	
Age	56.4	10.9	55.2	11.3	0.95

The mean age between the groups were comparable with no significant difference noted.

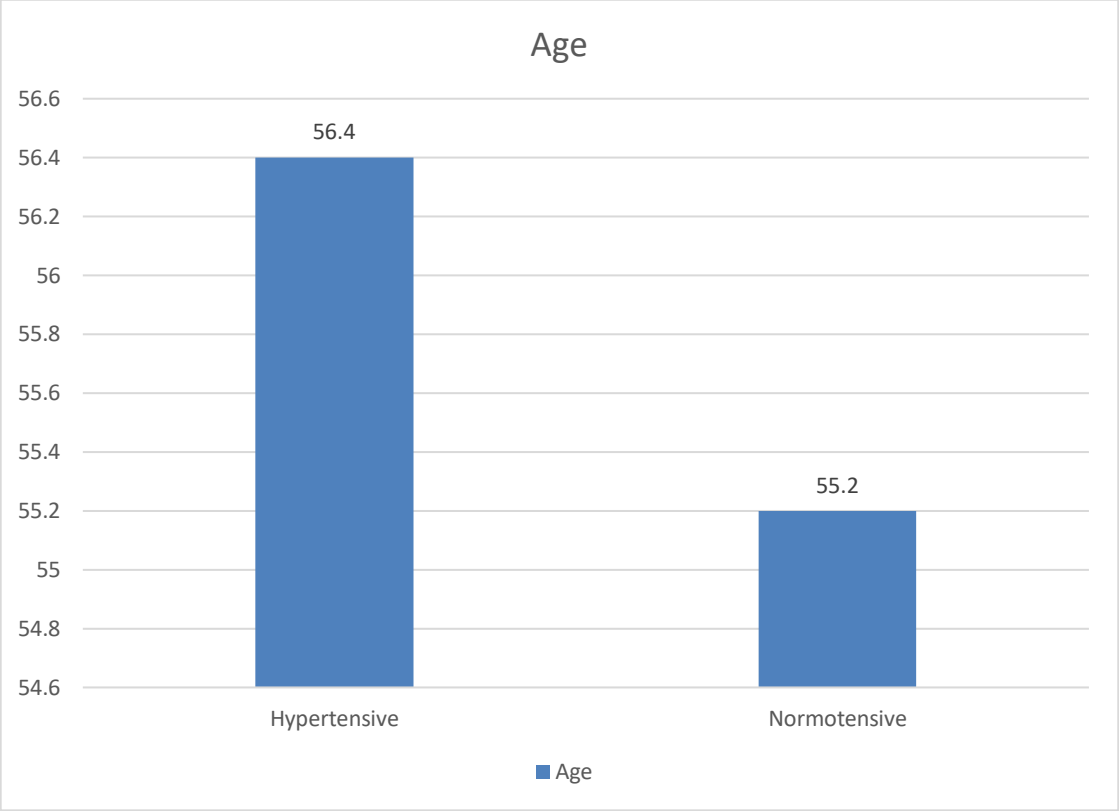

Figure 3: Comparison of mean age of patients between the groups

Table 3: Gender distribution between the groups

		Hypertensive		Normotensive	
		Count	N %	Count	N %
Sex	Female	15	28.3%	29	33.3%
	Male	38	71.7%	58	66.7%

Among the participants, majority were males compared to female. The distribution of gender between the groups were comparable with no significant difference.

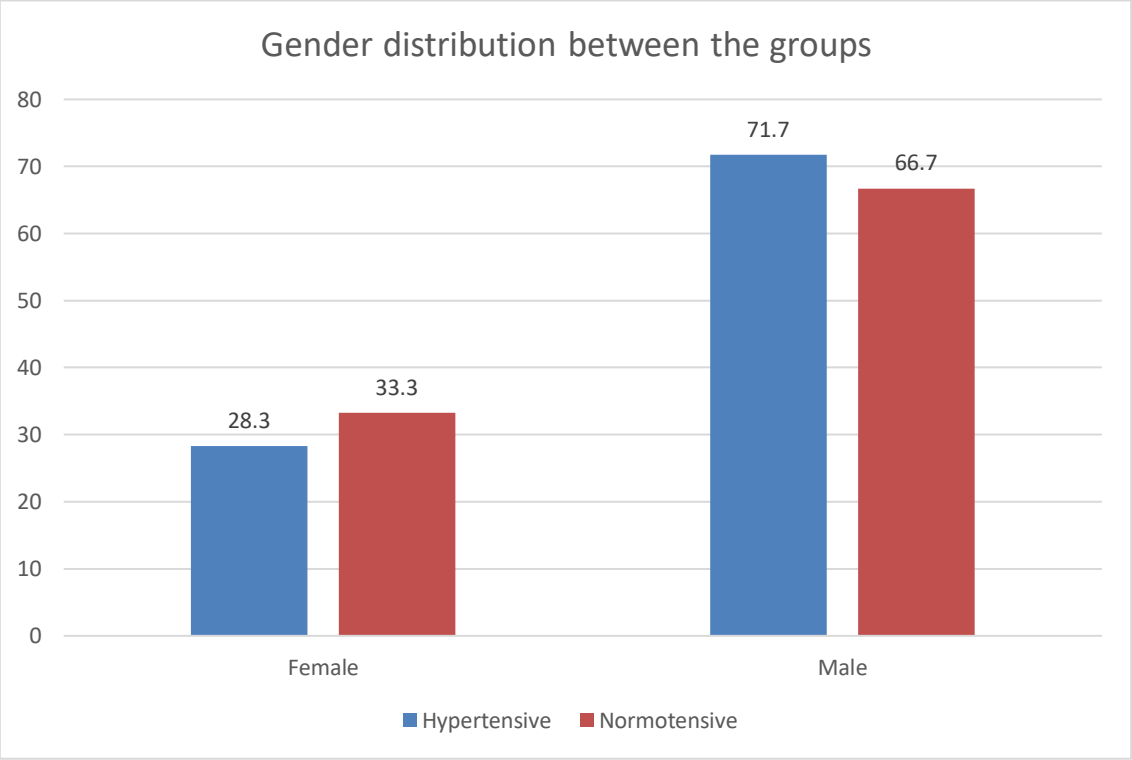

Figure 4: Gender distribution between the groups

Table 4: Comparison of blood parameters between the groups

	Hypertensive		Normotensive		P-Value
	Mean	SD	Mean	SD	

Haemoglobin	12.70	2.05	12.63	2.03	0.85
Creatinine	0.95	0.56	0.98	0.61	0.75
Sodium	139.75	7.91	139.49	7.92	0.85
Potassium	3.95	0.65	3.92	0.66	0.83
Total bilirubin	0.93	0.37	0.93	0.38	0.92
Total protein	5.39	0.62	5.46	0.62	0.54

On assessment of blood parameters, there is no significant difference noted in the mean haemoglobin, creatinine, serum sodium, potassium, total bilirubin and total protein levels between the groups.

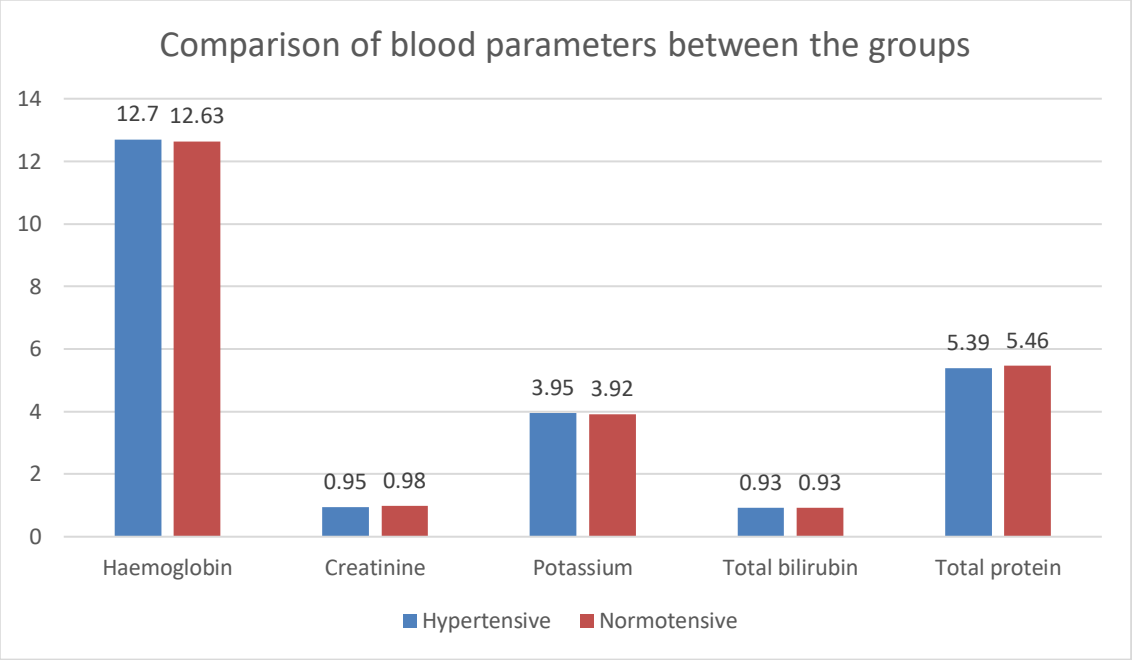

Figure 5: Comparison of blood parameters between the groups

Table 5: Comparison of the liver enzyme level between the groups

	Hypertensive		Normotensive		P-Value
	Mean	SD	Mean	SD	
ALT	42.3	32.4	26.7	22.9	0.05*
AST	41.1	34.4	31.8	28.9	0.05*

ALP	150.1	65.6	131.7	58.4	0.05*

On assessment of the liver enzymes, there was significant higher mean level of serum ALT, AST and ALP among the hypertensive patients compared to the normotensive individuals.($p<0.05$)

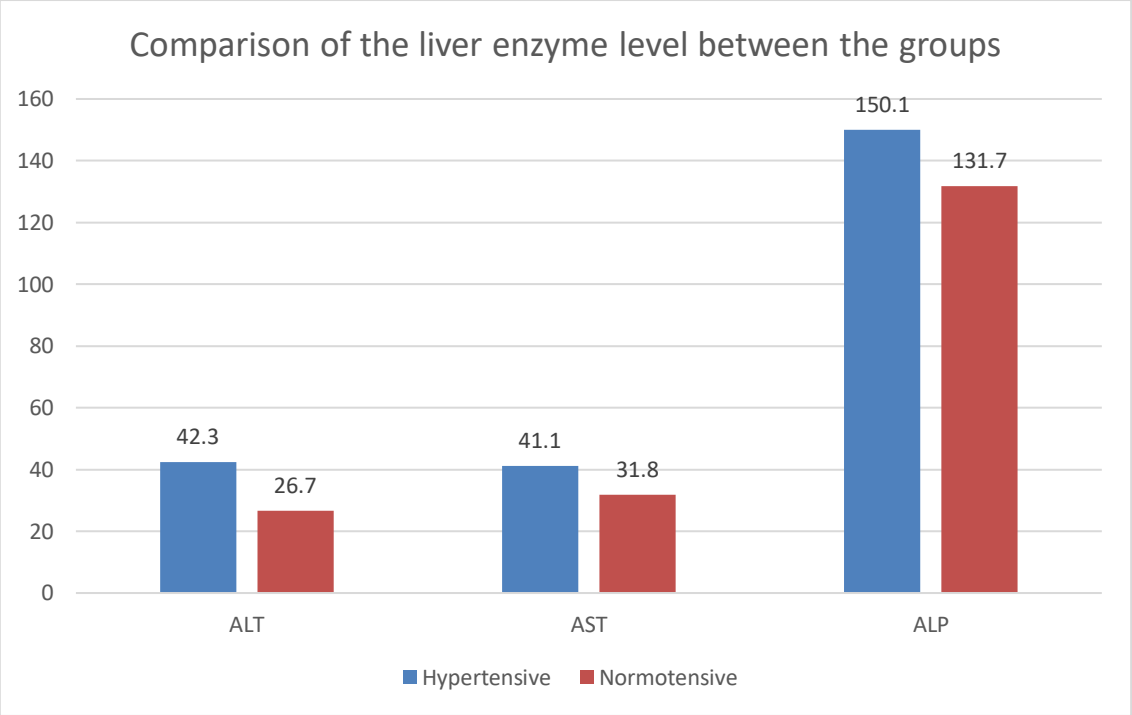

Figure 6: Comparison of the liver enzyme level between the groups

DISCUSSION

Hypertension is a leading global health concern, contributing significantly to cardiovascular morbidity and mortality. While its etiology is multifactorial, emerging evidence suggests that liver function may play a role in the development and progression of hypertension. Serum liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), are markers of liver health and metabolic processes. Elevated levels of these enzymes have been linked to systemic inflammation, oxidative stress, and metabolic syndrome, all of which are potential contributors to hypertension. This study aims to investigate the correlation between serum liver enzyme levels and hypertension, providing insights into their potential role as biomarkers or therapeutic targets in hypertensive individuals.

Present study included total of 140 patients fulfilling inclusion criteria and separated into two groups with hypertension and without hypertension. The overall mean of the patients included was 55.66±11.1yrs. The mean age between the groups were comparable with no significant difference noted. Among the participants, the majority were males compared to females. The

distribution of gender between the groups were comparable with no significant difference.

In similar, study by Khalili P et al., documented mean age of 49.94yrs with 46.56% were male patients.[44] Also in study by Rahman et al., majority of the participants were male compared to female with no significant difference in mean age between the groups.[8]

On assessment of blood parameters, there is no significant difference noted in the mean haemoglobin, creatinine, serum sodium, potassium, total bilirubin and total protein levels between the groups. On assessment of the liver enzymes, there was significant higher mean level of serum ALT, AST and ALP among the hypertensive patients compared to the normotensive individuals.($p<0.05$)

In concordance to present study findings, Rahman S et al., documented with hypertensive group exhibited significantly higher mean concentrations of serum ALT, AST, and GGT compared to the normotensive group ($p < 0.01$, $p < 0.01$, and $p < 0.001$, respectively). Elevated liver enzymes were more prevalent in the hypertensive group (49.2%) than in the normotensive group (38.1%), with a significantly higher prevalence of elevated ALT, AST, and GGT ($p < 0.01$, $p < 0.01$, and $p < 0.001$, respectively). A clear

trend of increased liver enzyme levels was observed with rising blood pressure, and serum ALT and GGT demonstrated an independent association with hypertension.[8]

Also in study by Khalili P et al., the higher levels of ALT, GGT, and ALP were associated with significantly increased odds of abnormal blood pressure. [44] Similarly Sakboonyarat B et al., found elevated serum AST and ALT levels were positively correlated with raised blood pressure (BP). Also, Hypertension was independently associated with higher odds of elevated AST and ALT in males, and elevated AST in females.[46] In line Baeradeh N et al., documented with Elevated levels of alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP) were significantly associated with an increased risk of hypertension, even after adjusting for potential confounding factors. Subgroup analyses also identified a notable relationship between ALP concentrations and high blood pressure within specific age ranges. [47]

Faramarzi E et al., study findings demonstrate that elevated levels of AST, ALT, ALP, and GGT are independently associated with pre-HTN (excluding ALP) and HTN, suggesting their potential utility as predictors for these conditions. Monitoring liver enzymes may enable primary care

providers to identify at-risk individuals and implement timely interventions.[48]

McCallum L et al., study liver enzymes and bilirubin in hypertensive patients was within four standard deviations of the mean exhibited independent effects on mortality and blood pressure control. These findings highlight potential mechanisms linking liver markers to blood pressure and cardiovascular risk but provide limited support for their use in clinical risk stratification.[41]

Recommendations

1. **Routine Monitoring of Liver Enzymes**: Considering the significant link between elevated levels of serum ALT, AST, and ALP and hypertension, it is advised to incorporate liver enzyme assessments into the routine screening and ongoing care of hypertensive patients.
2. **Integrated Care Approach**: Healthcare providers managing hypertension should adopt a collaborative approach, involving hepatologists or metabolic health specialists, to evaluate and address potential liver dysfunction or associated metabolic disorders when high liver enzyme levels are observed.

3. **Lifestyle Modifications**: Hypertensive patients with elevated liver enzyme levels should be encouraged to adopt healthier lifestyle choices, such as following a nutritious diet, engaging in regular exercise, maintaining a healthy weight, and avoiding harmful substances like excessive alcohol, to enhance liver function and overall cardiovascular health.
4. **Further Research**: Comprehensive, large-scale longitudinal studies are necessary to clarify the causal relationship between elevated liver enzymes and hypertension and to explore the underlying biological mechanisms connecting the two.
5. **Risk Stratification**: Including liver enzyme levels in hypertension risk assessment models may help identify patients at a higher likelihood of developing complications, enabling healthcare providers to tailor management strategies more effectively.
6. **Public Health Awareness**: Enhancing understanding of the relationship between liver health and hypertension among healthcare professionals and the general population is crucial for fostering early diagnosis and timely intervention.

SUMMARY

- Present study included total of 140 patients fulfilling inclusion criteria and separated into two groups with hypertension and without hypertension. The overall mean of the patients included was 55.66±11.1yrs.
- The mean age between the groups were comparable with no significant difference noted.
- Among the participants, majority were males compared to female. The distribution of gender between the groups were comparable with no significant difference.
- On assessment of blood parameters, there is no significant difference noted in the mean haemoglobin, creatinine, serum sodium, potassium, total bilirubin and total protein levels between the groups.
- On assessment of the liver enzymes, there was significant higher mean level of serum ALT, AST and ALP among the hypertensive patients compared to the normotensive individuals.($p<0.05$)

CONCLUSION

The findings of this study emphasize a noteworthy association between elevated serum liver enzyme levels and hypertension, suggesting that liver function may play a role in the pathophysiology of hypertension. While demographic and routine blood parameters showed no significant differences, the consistently higher levels of ALT, AST, and ALP in hypertensive individuals highlight the potential value of incorporating liver enzyme assessments into the clinical evaluation of patients with hypertension. This could aid in identifying individuals at risk of developing liver-related complications or uncovering underlying mechanisms linking liver health and hypertension. Further research is essential to confirm these findings and explore their clinical implications, but these results underscore the importance of a holistic approach to managing hypertension that considers liver function.

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ANNEXURE I

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 885/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "CORRELATION BETWEEN SERUM LIVER ENZYMES & HYPERTENSION".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.SHARAN BHARAT KONIN

NAME OF THE GUIDE: **DR.SHANKARAGOUDA S. PATIL, ASSOCIATE PROFESSOR, DEPT. OF GENERAL MEDICINE.**

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

ANNEXURE II

CONSENT FORM

BLDE DU'S SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR- 586103

TITLE OF THE PROJECT: **CORRELATION BETWEEN SERUM LIVER ENZYMES AND HYPERTENSION.**

PRINCIPAL INVESTIGATOR: Dr. SHARAN BHARAT KONIN

+91 9148243904

P.G. GUIDE NAME: Dr. SHANKARGOUDA S PATIL

ASSOCIATE PROFESSOR

DEPARTMENT OF GENERAL MEDICINE

All aspects of this consent form are explained to the patient in the language he/she understands.

INFORMED PART

PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

PROCEDURE:

I am aware that in addition to routine care received, the investigator will ask me a series of questions. I have been

asked to undergo the necessary investigations and treatment to help the investigator in this study.

RISK AND DISCOMFORTS:

I understand that I may experience pain and discomfort during the examination or treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings associated with the usual course of treatment.

BENEFITS:

I understand that my participation in this study will help the patient's survival and better outcomes.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to confidentiality and privacy regulations. Information of a sensitive personal nature will not be a part of the medical records but will be stored in the investigator's research file and identified only by code number. The code key connecting names to numbers will be kept in a separate location. Suppose the data are used for publication in the medical literature or for

teaching purposes. No name will be used in that case, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the pictures and videotapes and hear the audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study time.

Dr. SHARAN BHARAT KONIN is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the study, which might influence my continued participation.

If, during the study or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the hospital's social worker is available to talk with me. I will be given a copy of this consent form to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that Dr. SHARAN BHARAT KONIN may terminate

my participation in the study after she has explained the reasons for doing so and has helped arrange for my continued care by my physician or physical therapist if this is appropriate

INJURY STATEMENT:

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

In the patient's language, I explained the purpose of the research, the procedures required, and the possible risks and benefits to the best of my ability.

_____ _____

Dr. SHARAN BHARAT KONIN Date:

(Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. SHARAN BHARAT KONIN has explained the purpose of the research, the study procedures that I will undergo, the possible risks and discomforts, and the benefits that I may experience in my language. I have read and understand this consent form. Therefore, I agree to consent to participate as a subject in this research project.

_____ _____

Participant / Guardian Date

_____ _____

Witness to signature Date

ANNEXURE – III

CASE PROFORMA

B.L.D.E (DEEMED TO BE UNIVERSITY)

SHRI B M PATIL MEDICAL COLLEGE

VIJAYAPURA, KARNATAKA

SCHEME OF CASE TAKING

Informant :

NAME: CASE NO:

AGE: IP NO:

SEX: DOA:

RELIGION: DOD:

PAST OCCUPATION:

PRESENT OCCUPATION:

RESIDENCE:

CHIEF COMPLAINTS:

HISTORY OF PRESENT ILLNESS:

PAST HISTORY:

PERSONAL HISTORY:

FAMILY HISTORY:

TREATMENT HISTORY:

GENERAL PHYSICAL EXAMINATION

HEIGHT:

WEIGHT:

BODY MASS INDEX:

VITALS

PR:

BP:

RR:

TEMP:

HEAD-TO-TOE EXAMINATION:

SYSTEMIC EXAMINATION:

CENTRAL NERVOUS SYSTEM:

RESPIRATORY SYSTEM:

CARDIOVASCULAR SYSTEM:

PER ABDOMEN:

INVESTIGATIONS

1. HAEMATOLOGY –

1) HEMOGLOBIN	GM. %
2) TOTAL WBC COUNTS	CELLS/MM^3
3) DIFFERENTIAL COUNTS -	
NEUTROPHILS	%
LYMPHOCYTES	%
EOSINOPHILS	%
MONOCYTES	%

Basophils	%
Platelet count	
ESR	At the end of 1st hour

2. BIOCHEMISTRY:

- **RENAL FUNCTION TEST**

CREATININE	MG/DL
UREA	MG/DL
SODIUM	MEQ/L
POTASSIUM	MEQ/L

- **LIVER FUNCTION TESTS**

TOTAL BILIRUBIN	MG/DL
DIRECT BILIRUBIN	MG/DL
INDIRECT BILIRUBIN	MG/DL
SERUM PROTEIN	MG/DL
SERUM ALBUMIN	MG/DL
SGOT (AST)	IU/L
SGPT (ALT)	IU/L

3. ECG:

CONCLUSION:

DATE:-

SIGNATURE:-

MASTERCHART

SL.NO	NAME	AGE	SEX	IP NO.	BP	STATUS	TOTAL BIL	TOTAL PR(	ALT	AST	ALP	CREATINI	SODIUM	POTASSIU	HB
1	LAXMAN	62	MALE	16628	150/90	HYPERTEN	1	6	42	134	260	1.1	133	3.6	11.2
2	JAGADISH	46	MALE	16564	140/90	HYPERTEN	0.4	5.5	84	66	152	0.6	136	5.5	13.6
3	RACHAYYA	46	MALE	43638	154/90	HYPERTEN	1.2	4.8	38	69	77	1	144	4.2	10.3
4	SANTOSH	43	MALE	328029	160/86	HYPERTEN	0.8	5.2	29	27	84	0.2	140	3.8	12.2
5	SADASHIV	67	MALE	49361	130/80	HYPERTEN	1.1	6.3	66	18	184	1.5	138	4	15.6
6	SURESH	54	MALE	251258	140/80	HYPERTEN	0.7	4.9	18	20	210	0.7	131	3	13.3
7	BHIMAPPA	50	MALE	270621	150/100	HYPERTEN	0.5	5.2	92	16	192	1.6	128	3.4	11.8
8	JEETKUMA	68	MALE	270616	120/90	HYPERTEN	0.8	5.9	22	16	174	0.7	150	3.8	12
9	SHANKRAI	80	MALE	253559	138/90	HYPERTEN	1.4	5	19	24	96	0.4	141	4.3	14.1
10	KAREPPA	68	MALE	391811	144/96	HYPERTEN	0.3	4	76	20	110	0.1	137	3.5	14.9
11	SUBHASH	59	MALE	275874	160/92	HYPERTEN	0.9	5.7	21	19	122	1	139	4.1	15.6
12	KASHINAT	55	MALE	121366	158/100	HYPERTEN	1.1	4.7	17	20	104	0.5	126	3.9	13.4
13	AKASH	66	MALE	266935	180/100	HYPERTEN	1.3	5.1	96	17	244	1.3	132	4.8	12.5
14	PARASHUI	72	MALE	258365	140/88	HYPERTEN	0.5	5.2	17	26	110	0.9	137	5	11.7
15	BHIMRAO	63	MALE	111944	150/70	HYPERTEN	1.6	5.8	26	18	94	1	146	4.1	10.5
16	ANIL	49	MALE	256750	140/90	HYPERTEN	1.4	6.1	90	16	106	0.6	144	3.8	11
17	MALLEGAI	55	MALE	230052	110/80	HYPERTEN	1	6.6	23	92	130	0.8	137	4.4	10.8
18	UMAR FAI	38	MALE	270916	150/98	HYPERTEN	0.4	5.9	19	104	110	1.1	142	3.5	9.8
19	PANCHAY	51	MALE	260468	148/90	HYPERTEN	1.2	5.3	16	96	111	0.2	150	2.9	15.9
20	IRANNA	69	MALE	254762	160/100	HYPERTEN	0.8	6	24	20	260	0.9	162	3	8.7
21	PARASHUI	74	MALE	126884	158/100	HYPERTEN	1.1	5.5	78	19	252	0.7	154	3.6	10.4
22	SHARANAI	80	MALE	272385	140/88	HYPERTEN	0.7	4.8	21	16	106	1.5	133	5.5	13.2
23	SHANTIVE	57	MALE	224021	140/90	HYPERTEN	0.5	5.2	91	84	94	2	138	4.2	14.7
24	SACHIN	64	MALE	259116	150/100	HYPERTEN	0.8	6.3	25	110	102	1.7	140	3.8	13.3
25	SHANTAPF	40	MALE	278927	170/90	HYPERTEN	1.4	4.9	18	81	111	3.1	139	4	14.9
26	BHIMANG	76	MALE	276672	146/94	HYPERTEN	0.3	5.2	19	86	122	1.4	133	3	15.9
27	AMBRISH	49	MALE	278418	200/100	HYPERTEN	0.9	5.9	14	97	304	0.8	136	3.4	10.6
28	RAGHAVEI	66	MALE	271991	158/100	HYPERTEN	1.1	5	15	90	100	0.3	144	3.8	11.2
29	DEVALUBA	70	FEMALE	328993	148/90	HYPERTEN	1.3	4	24	84	89	0.8	140	4.3	13.6
30	KASTURIB/	71	FEMALE	319523	152/88	HYPERTEN	0.5	5.7	96	20	114	1.3	138	3.5	10.3
31	LAXMI BAI	60	FEMALE	328812	160/90	HYPERTEN	1.6	4.7	14	23	208	1.1	131	4.1	12.2
32	KALAVATI	52	FEMALE	263808	152/88	HYPERTEN	1.4	5.1	76	82	178	0.6	128	3.9	15.6
33	LAXMIBAI	43	FEMALE	261416	150/90	HYPERTEN	1	5.2	92	12	110	1	150	4.8	13.3
34	KORABAI	51	FEMALE	141441	140/90	HYPERTEN	0.4	5.8	18	16	222	0.2	141	5	11.8
35	SANDHYA	56	FEMALE	273855	154/90	HYPERTEN	1.2	6.1	85	20	194	1.5	137	4.1	12
36	ROOPA BU	49	FEMALE	169791	160/86	HYPERTEN	0.8	6.6	23	78	256	0.7	139	3.8	14.1
37	SAVITHRI I	61	FEMALE	216953	130/80	HYPERTEN	1.1	5.9	91	66	99	1.6	126	4.4	14.9
38	ASHWINI I	40	FEMALE	222088	140/80	HYPERTEN	0.7	5.3	80	20	220	0.7	132	3.5	15.6
39	RUKMINI F	52	FEMALE	245066	150/100	HYPERTEN	0.5	6	21	19	263	0.4	137	2.9	13.4
40	SHANUBA	66	FEMALE	247449	120/90	HYPERTEN	0.8	5.5	122	15	300	0.1	146	3	12.5
41	GEETA JAI	60	FEMALE	250421	138/90	HYPERTEN	1.4	4.8	64	21	95	1	144	3.6	11.7
42	MAHADEV	44	FEMALE	250918	144/96	HYPERTEN	0.3	5.2	28	19	92	0.5	137	5.5	10.5
43	ASHA KALI	48	FEMALE	261648	160/92	HYPERTEN	0.9	6.3	10	14	98	1.3	142	4.2	11
44	RAMACHA	45	MALE	313449	158/100	HYPERTEN	1.1	4.9	11	73	103	0.9	150	3.8	10.8
45	ANIL KUM	46	MALE	170930	180/100	HYPERTEN	1.3	5.2	15	13	124	1	162	4	9.8
46	SHANKARA	43	MALE	130652	140/88	HYPERTEN	0.5	5.9	13	8	232	0.6	154	3	15.9
47	GURUPAD	50	MALE	181532	150/70	HYPERTEN	1.6	5	18	22	112	0.8	133	3.4	8.7
48	SHRISHAIL	59	MALE	176849	140/90	HYPERTEN	1.4	4	70	17	90	1.1	138	3.8	10.4
49	BHIMRAO	55	MALE	135693	110/80	HYPERTEN	1	5.7	20	18	99	0.2	140	4.3	13.2
50	PRABHU Y	44	MALE	190434	150/98	HYPERTEN	0.4	4.7	16	21	222	0.9	139	3.5	14.7
51	SIDARAYA	49	MALE	184245	148/90	HYPERTEN	1.2	5.1	84	56	111	0.7	133	4.1	13.3
52	SIDDU NA	53	MALE	198526	160/100	HYPERTEN	0.8	5.2	15	8	100	1.5	136	3.9	14.9
53	BASANGO	56	MALE	220406	158/100	HYPERTEN	1.1	5.8	19	11	102	2	144	4.8	15.9
54	MALLIKAR	47	MALE	225199	110/70	NORMOTE	0.7	6.1	67	17	108	1.7	140	5	10.6
55	RAMESH F	73	MALE	227946	120/80	NORMOTE	0.5	6.6	20	88	155	3.1	138	4.1	11.2
56	RAFIQ BEF	69	MALE	217808	100/60	NORMOTE	0.8	5.9	10	22	93	1.4	131	3.8	13.6
57	RAVATAPF	75	MALE	419934	120/90	NORMOTE	1.4	5.3	16	14	95	0.8	128	4.4	10.3
58	LAXMAN J	41	MALE	231747	90/60	NORMOTE	0.3	6	18	20	93	0.3	150	3.5	12.2
59	IRANNA BI	70	MALE	245084	106/80	NORMOTE	0.9	5.5	84	94	117	0.8	141	2.9	15.6
60	MALIKSAB	68	MALE	255410	112/88	NORMOTE	1.1	4.8	22	21	180	1.3	137	3	13.3
61	VILAS AWA	76	MALE	204956	98/50	NORMOTE	1.3	5.2	9	25	110	1.1	139	3.6	11.8
62	PARASHUI	45	MALE	261703	100/60	NORMOTE	0.5	6.3	16	17	94	0.6	126	5.5	12
63	ADAVAYYA	58	MALE	340990	120/90	NORMOTE	1.6	4.9	75	79	109	1	132	4.2	14.1
64	LINGESH M	48	MALE	280754	130/70	NORMOTE	1.4	5.2	10	21	117	0.2	137	3.8	14.9
65	SHIVANGC	56	MALE	279708	128/80	NORMOTE	1	5.9	94	27	118	1.5	146	4	15.6
66	SHARANBA	62	MALE	289522	106/96	NORMOTE	0.4	5	14	18	120	0.7	144	3	13.4
67	MANJUNA	51	MALE	270757	100/72	NORMOTE	1.2	4	20	12	115	1.6	137	3.4	12.5
68	MALLAPPA	58	MALE	21335	120/98	NORMOTE	0.8	5.7	17	98	106	0.7	142	3.8	11.7
69	BASAPPA	48	MALE	305947	124/84	NORMOTE	1.1	4.7	80	14	99	0.4	150	4.3	10.5

70	RAJKUMAI	49	MALE	343019	108/92	NORMOTE	0.7	5.1	21	8	265	0.1	162	3.5	11
71	BASAVARA	60	MALE	340568	110/70	NORMOTE	0.5	5.2	24	19	94	1	154	4.1	10.8
72	LAKKAPPA	70	MALE	373909	120/80	NORMOTE	0.8	5.8	59	22	104	0.5	133	3.9	9.8
73	BASAVARA	64	MALE	352880	100/60	NORMOTE	1.4	6.1	16	14	107	1.3	138	4.8	15.9
74	SUNIL MA	69	MALE	226693	120/90	NORMOTE	0.3	6.6	19	101	91	0.9	140	5	8.7
75	SHIVASHA	58	MALE	375020	90/60	NORMOTE	0.9	5.9	20	12	100	1	139	4.1	10.4
76	BHAKTARA	56	MALE	357633	106/80	NORMOTE	1.1	5.3	13	10	126	0.6	133	3.8	13.2
77	SANGAPPA	67	MALE	385197	112/88	NORMOTE	1.3	6	12	9	248	0.8	136	4.4	14.7
78	VIJAYKUM	37	MALE	370757	98/50	NORMOTE	0.5	5.5	82	11	122	1.1	144	3.5	13.3
79	LAXMAN B	42	MALE	349122	100/60	NORMOTE	1.6	4.8	10	16	111	0.2	140	2.9	14.9
80	SHARANAI	43	MALE	348135	120/90	NORMOTE	1.4	5.2	22	65	98	0.9	138	3	15.9
81	KALYANAF	50	MALE	190028	130/70	NORMOTE	1	6.3	23	19	107	0.7	131	3.6	10.6
82	MAHANTE	41	MALE	15321	128/80	NORMOTE	0.4	4.9	10	24	108	1.5	128	5.5	11.2
83	SOMA MA	48	MALE	308343	106/96	NORMOTE	1.2	5.2	70	21	96	2	150	4.2	13.6
84	SAMBAJI M	39	MALE	303675	100/72	NORMOTE	0.8	5.9	19	80	95	1.7	141	3.8	10.3
85	SOMANIN	49	MALE	409955	120/98	NORMOTE	1.1	5	8	18	113	3.1	137	4	12.2
86	KAMALESH	44	MALE	310829	124/84	NORMOTE	0.7	4	11	14	304	1.4	139	3	15.6
87	RAJESH SA	51	MALE	273527	108/92	NORMOTE	0.5	5.7	86	71	106	0.8	126	3.4	13.3
88	BASAVARA	52	MALE	271171	110/70	NORMOTE	0.8	4.7	12	26	99	0.3	132	3.8	11.8
89	MALLAPPA	48	MALE	250729	120/80	NORMOTE	1.4	5.1	17	23	95	0.8	137	4.3	12
90	RAMESH D	42	MALE	136335	100/60	NORMOTE	0.3	5.2	20	15	110	1.3	146	3.5	14.1
91	PRAKASH I	40	MALE	329894	120/90	NORMOTE	0.9	5.8	21	18	92	1.1	144	4.1	14.9
92	DUGGA VE	64	MALE	327865	90/60	NORMOTE	1.1	6.1	24	11	115	0.6	137	3.9	15.6
93	AJAY BHIS	58	MALE	337776	106/80	NORMOTE	1.3	6.6	19	10	119	1	142	4.8	13.4
94	MAHANTE	57	MALE	281984	112/88	NORMOTE	0.5	5.9	74	85	121	0.2	150	5	12.5
95	RAJIV BAD	64	MALE	356264	98/50	NORMOTE	1.6	5.3	12	19	90	1.5	162	4.1	11.7
96	MALLAPPA	60	MALE	303035	100/60	NORMOTE	1.4	6	9	21	198	0.7	154	3.8	10.5
97	RAMAPPA	52	MALE	363486	120/90	NORMOTE	1	5.5	10	25	102	1.6	133	4.4	11
98	SHIVANAN	50	MALE	370007	130/70	NORMOTE	0.4	4.8	8	18	99	0.7	138	3.5	10.8
99	KALLANGC	58	MALE	349696	128/80	NORMOTE	1.2	5.2	11	9	92	0.4	140	2.9	9.8
100	SANGAPPA	46	MALE	381565	106/96	NORMOTE	0.8	6.3	22	22	97	0.1	139	3	15.9
101	ANAND SA	75	MALE	387160	100/72	NORMOTE	1.1	4.9	66	70	100	1	133	3.6	8.7
102	YAMANAP	60	MALE	382715	120/98	NORMOTE	0.7	5.2	14	21	116	0.5	136	5.5	10.4
103	VISHNU LA	47	MALE	395365	124/84	NORMOTE	0.5	5.9	22	18	166	1.3	144	4.2	13.2
104	DADU KHA	49	MALE	294761	108/92	NORMOTE	0.8	5	21	14	96	0.9	140	3.8	14.7
105	CHANDAP	63	MALE	405018	110/70	NORMOTE	1.4	4	17	22	104	1	138	4	13.3
106	SHANKRAI	59	MALE	406116	120/80	NORMOTE	0.3	5.7	50	19	111	0.6	131	3	14.9
107	RAVI KUM	66	MALE	409902	100/60	NORMOTE	0.9	4.7	18	10	100	0.8	128	3.4	15.9
108	BHIMAPPA	72	MALE	404950	120/90	NORMOTE	1.1	5.1	28	16	102	1.1	150	3.8	10.6
109	CHANDRA	80	MALE	418060	90/60	NORMOTE	1.3	5.2	23	120	98	0.2	141	4.3	11.2
110	MUTTANN	56	MALE	424972	106/80	NORMOTE	0.5	5.8	20	15	172	0.9	137	3.5	13.6
111	SANDEEP I	78	MALE	250836	112/88	NORMOTE	1.6	6.1	18	10	110	0.7	139	4.1	10.3
112	TANUJA LO	38	FEMALE	120577	98/50	NORMOTE	1.4	6.6	18	20	165	1.5	126	3.9	12.2
113	AMBIKA M	42	FEMALE	112231	100/60	NORMOTE	1	5.9	84	68	211	2	132	4.8	15.6
114	DEEPA DO	48	FEMALE	112229	120/90	NORMOTE	0.4	5.3	12	18	98	1.7	137	5	13.3
115	ARCHANA	52	FEMALE	57293	130/70	NORMOTE	1.2	6	14	12	100	3.1	146	4.1	11.8
116	BHAGYASH	40	FEMALE	55284	128/80	NORMOTE	0.8	5.5	16	15	243	1.4	144	3.8	12
117	ASHWINI (	56	FEMALE	49032	106/96	NORMOTE	1.1	4.8	11	88	102	0.8	137	4.4	14.1
118	CHANDUB	65	FEMALE	21493	100/72	NORMOTE	0.7	5.2	10	92	196	0.3	142	3.5	14.9
119	RESHMA D	53	FEMALE	15013	120/98	NORMOTE	0.5	6.3	19	21	96	0.8	150	2.9	15.6
120	KARISHMA	36	FEMALE	9106	124/84	NORMOTE	0.8	4.9	14	20	106	1.3	162	3	13.4
121	ROOPA SH	50	FEMALE	6535	108/92	NORMOTE	1.4	5.2	15	14	290	1.1	154	3.6	12.5
122	MEGHA M	47	FEMALE	3874	110/70	NORMOTE	0.3	5.9	21	74	220	0.6	133	5.5	11.7
123	KAVITHA E	51	FEMALE	390778	120/80	NORMOTE	0.9	5	18	25	353	1	138	4.2	10.5
124	RAKMAJI L	46	FEMALE	349160	100/60	NORMOTE	1.1	4	78	22	96	0.2	140	3.8	11
125	MANJULA	53	FEMALE	310434	120/90	NORMOTE	1.3	5.7	22	18	101	1.5	139	4	10.8
126	DRAKSHAY	60	FEMALE	152896	90/60	NORMOTE	0.5	4.7	25	69	182	0.7	133	3	9.8
127	BASAMMA	43	FEMALE	350428	106/80	NORMOTE	1.6	5.1	19	11	99	1.6	136	3.4	15.9
128	SHANTAM	59	FEMALE	351395	112/88	NORMOTE	1.4	5.2	14	18	105	0.7	144	3.8	8.7
129	SHANTABA	70	FEMALE	213936	98/50	NORMOTE	1	5.8	16	18	97	0.4	140	4.3	10.4
130	LAXMIBAI	75	FEMALE	224594	100/60	NORMOTE	0.4	6.1	10	16	264	0.1	138	3.5	13.2
131	SHANTAVV	63	FEMALE	256651	120/90	NORMOTE	1.2	6.6	21	10	96	1	131	4.1	14.7
132	SIDDAMM	59	FEMALE	10075	130/70	NORMOTE	0.8	5.9	13	89	302	0.5	128	3.9	13.3
133	TARABAI K	77	FEMALE	6960	128/80	NORMOTE	1.1	5.3	23	19	101	1.3	150	4.8	14.9
134	LALABI NIF	45	FEMALE	9052	106/96	NORMOTE	0.7	6	19	15	109	0.9	141	5	15.9
135	MADEVI W	59	FEMALE	6881	100/72	NORMOTE	0.5	5.5	17	18	99	1	137	4.1	10.6
136	SUMAN BA	36	FEMALE	7072	120/98	NORMOTE	0.8	4.8	15	92	97	0.6	139	3.8	11.2
137	SUSHMITA	40	FEMALE	6982	124/84	NORMOTE	1.4	5.2	16	21	205	0.8	126	4.4	13.6
138	NEELAMM	64	FEMALE	6169	108/92	NORMOTE	0.3	6.3	70	15	98	1.1	132	3.5	10.3
139	SAHEBI VA	56	FEMALE	6187	110/70	NORMOTE	0.9	4.9	18	12	100	0.2	137	2.9	12.2
140	ROOPA M(	42	FEMALE	5516	120/80	NORMOTE	1.1	5.2	20	77	196	0.9	146	3	15.6