STUDY OF CORRELATION BETWEEN METABOLIC SYNDROME AND KIDNEY FUNCTION TESTS

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2024

DECLARATION

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Ivon Francis Lewis

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List of Abbreviations

1. **ATP - Adenosine Triphosphate**
2. **BMI - Body Mass Index**
3. **CVD - Cardiovascular Disease**
4. **CKD -Chronic Kidney Disease**
5. **FBS-Fasting Blood Sugar**
6. **FLP-Fasting Lipid Profile**
7. **GK - Glycerol Kinase**
8. **GLDH - Glutamate Dehydrogenase**
9. **GPO - Glycerol Phosphate Oxidase**
10. **G6PDH - Glucose-6-Phosphate Dehydrogenase**
11. **HDL - High Density Lipoproteins**
12. **ICMR - Indian Council of Medical Research**
13. **IDF - International Diabetes Federation**
14. **LDL-C - Low Density Lipoproteins Cholesterol**
15. **MetS – Metabolic Syndrome**
16. **NCEP ATP III - National Cholesterol Education Program Adult Treatment Panel-III**
17. **NHANES - National Health and Nutrition Examination Survey**
18. **VLDL -Very Low-Density Lipoproteins**
19. **WC - Waist Circumference**
20. **WHR - Waist Hip Ratio**
21. **WHO - World Health Organization**

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INTRODUCTION

CHAPTER-I

Introduction

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INTRODUCTION

Metabolic syndrome (MetS) has become a significant health care problem worldwide, with its increase in prevalence (1).MetS interconnect with metabolic abnormalities, including hypertension, central obesity, insulin resistance, atherogenic dyslipidemia and closely associated with the development of various chronic conditions (2). Demographic, sociological, and psychological factors, play a crucial role in the development of chronic kidney disease and increase prevalence of metabolic syndrome (1).

Metabolic Syndrome (MetS) is a cluster of interrelated metabolic abnormalities that increase an individual's risk of cardiovascular diseases and type 2 diabetes mellitus (T2DM). The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) defines MetS as the presence of any three of the following five criteria:

- Waist circumference ≥102 cm (40 inches) in men or ≥88 cm (35 inches) in women.
- Serum triglycerides ≥150 mg/dL or drug treatment for elevated triglycerides.
- Serum HDL-C <40 mg/dL in men or <50 mg/dL in women or drug treatment for low HDL-C.
- Blood pressure ≥130/85 mmHg or drug treatment for hypertension.
- Fasting blood glucose ≥100 mg/dL or drug treatment for elevated blood glucose.

MetS is characterized by insulin resistance and pro-inflammatory states, contributing to the development of atherosclerosis and subsequent cardiovascular events. This condition is also associated with a higher risk of developing chronic kidney disease.

The prevalence of MetS varies significantly across different populations and is influenced by factors such as age, sex, ethnicity, and lifestyle. According to the International Diabetes Federation (IDF), approximately one-quarter of the world's adult population has MetS. The prevalence is higher in urbanized and economically developed regions due to sedentary lifestyles, unhealthy diets, and increasing rates of obesity.For instance, in the United States, the National Health and Nutrition

Examination Survey (NHANES) reported that about 34% of adults have MetS. In Europe, prevalence rates range from 20% to 30%.

In India, rapid economic development and urbanization, has a significant increase in the prevalence of MetS. The Indian population is particularly susceptible to MetS due to genetic predispositions and lifestyle factors. Studies have shown that the prevalence of MetS in urban areas of India is alarmingly high, ranging from 30% to 50%. The Indian Council of Medical Research (ICMR) study highlighted that MetS prevalence is higher in urban populations compared to rural populations, reflecting the impact of lifestyle changes associated with urbanization. Additionally, the prevalence is increasing among younger age groups, which is a concerning trend for future public health.

Metabolic Syndrome is a growing public health concern worldwide, with significant prevalence in both global and Indian populations. The association between MetS and renal dysfunction underscores the importance of early detection and monitoring of kidney health in these patients. Renal function tests are essential tools for identifying early renal impairment, guiding risk stratification, and implementing effective management strategies. Addressing renal dysfunction in MetS patients not only improves renal outcomes but also reduces the risk of cardiovascular diseases, thereby enhancing overall patient health and quality of life. This underscores the critical role of comprehensive management of MetS, including regular renal function testing, in mitigating the health risks associated with this multifaceted syndrome

Renal dysfunction, encompassing conditions such as CKD, is a common complication among individuals with MetS. Several pathophysiological mechanisms link MetS to renal dysfunction:

Insulin Resistance and Hyperglycemia: Insulin resistance and hyperglycemia lead to glomerular hyper filtration and increased intra glomerular pressure, which contribute to kidney damage over time.

Hypertension: Elevated blood pressure causes increased strain on the renal vasculature, leading to glomerulosclerosis and reduced renal function.

Dyslipidemia: Elevated triglycerides and low HDL-C levels contribute to atherosclerosis, affecting renal blood vessels and leading to ischemic injury.

Obesity: Central obesity promotes inflammation and oxidative stress, which exacerbate renal injury.

Pro-inflammatory and Pro-thrombotic States: MetS is associated with chronic low-grade inflammation and a pro-thrombotic state, both of which contribute to renal damage.

Studies have demonstrated a higher incidence of CKD among patients with MetS compared to those without MetS. The presence of MetS components, such as hypertension and hyperglycemia, significantly increases the risk of developing renal dysfunction. For instance, individuals with MetS are two to three times more likely to develop CKD compared to those without MetS.

Renal function tests are crucial for the early detection and monitoring of renal dysfunction in patients with MetS. Early identification of renal impairment allows for timely interventions to prevent the progression of CKD. Common renal function tests include Serum Creatinine, Serum Urea, Blood Urea Nitrogen (BUN), Glomerular Filtration Rate (GFR) and Urine Albumin-to-Creatinine Ratio (UACR).

One of the emerging areas of interest is the relationship between metabolic syndrome and renal function. Researchers have observed that patients with metabolic syndrome are at a higher risk of developing chronic kidney disease (1). The complex linkage between these two conditions is under investigation, with researchers exploring the various factors that contribute to this association. This study elucidates the impact of metabolic syndrome on renal function.

CHAPTER-II

REVIEW OF LITERATURE

CHAPTER-II

Review of Literature

REVIEW OF LITERATURE

Prevalence of Metabolic Syndrome

The prevalence of metabolic syndrome (MetS) is a significant public health concern both in India and globally. In India, studies indicate a high prevalence of MetS, with estimates ranging from 30.3% to 40.4% among urban populations, particularly among women and individuals with higher educational and occupational status (3). A multicentric study highlighted that the prevalence varied significantly across different regions, with northern India reporting rates as high as 28.6% and southern India reaching up to 45.9% (4). Furthermore, a systematic review and meta-analysis revealed that the overall prevalence of MetS in the Indian adult population is approximately 40%, which is notably higher than the 25% reported in Western populations (5,6).

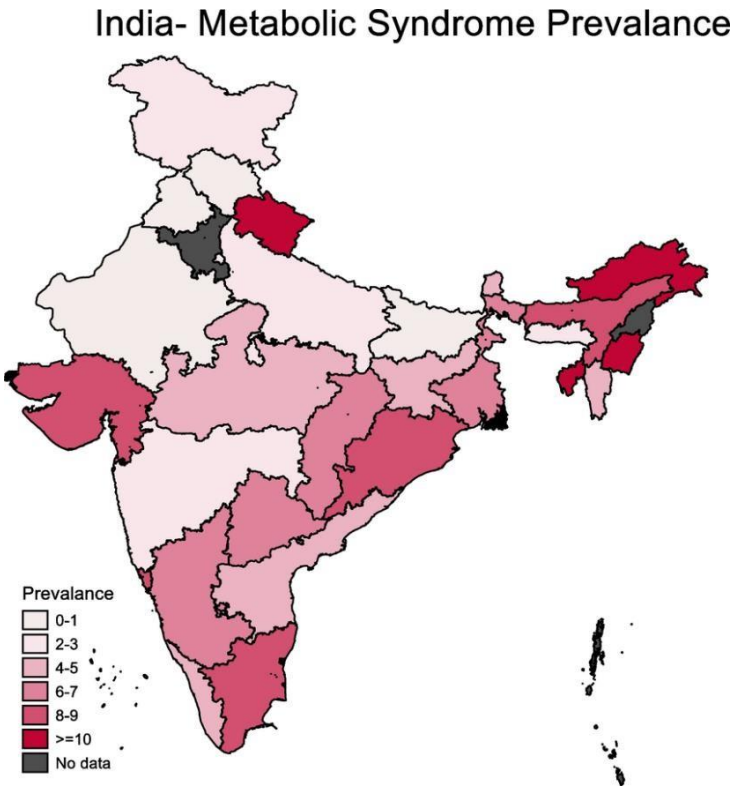

Fig 2.1.1: Prevalence of Metabolic Syndrome in India.(7)

The prevalence is maximum in Manipur (17%) and minimum in Punjab (less than 1%) which indicates wide interstate variability. The prevalence of metabolic syndrome was higher than the national-level prevalence in 15 states. More than 10%. prevalence reportedin four states - Uttarakhand, Arunachal Pradesh, Manipur and Tripura (7).

Globally, the prevalence of MetS is also alarming, with estimates suggesting that it affects about 25-50% of populations in urban areas of South Asia (8). The World Health Organization has recognized MetS as a growing epidemic, particularly in developing countries where urbanization and lifestyle changes contribute to rising obesity rates and sedentary behaviors (9). The prevalence of MetS varies by region, with studies indicating that it affects approximately 19.5-37.2% of men and 13.5-42.7% of women in Gulf countries (10), while in Asian populations, the prevalence is increasing despite lower obesity rates compared to Western populations (11).

The risk factors associated with MetS, including obesity, hypertension, dyslipidemia, and insulin resistance, are prevalent in both India and worldwide. For instance, in India, the urban population exhibits a higher incidence of these risk factors, with studies showing that obesity rates can reach as high as 68% and low HDL cholesterol levels at 81% (8). Similarly, globally, the increase in obesity and sedentary lifestyles has been linked to a rise in MetS, making it a critical area for public health intervention (9,12).

In conclusion, the prevalence of metabolic syndrome is a pressing issue in India, with significant regional variations and a higher occurrence compared to western countries. Globally, the trend mirrors that of India, with increasing rates of MetS attributed to lifestyle changes and urbanization. Addressing these challenges requires targeted public health strategies to mitigate the risk factors associated with MetS.

Pathophysiology of Metabolic Syndrome: The pathophysiology of metabolic syndrome (MetS) is complex and multifactorial, involving a constellation of metabolic

abnormalitiesthat significantly increase the risk of cardiovascular disease and type 2 diabetes. Central to the development of MetS is insulin resistance, characterized by the body's diminished ability to respond to insulin, leading to elevated blood glucose levels and increased fat accumulation (13) . Insulin resistance is often exacerbated by obesity, particularly central obesity, which is a key component of MetS. The accumulation of

visceral fat is associated with increased levels of free fatty acids, inflammatory cytokines, and adipokines, which contribute to the dysregulation of glucose and lipid metabolism (13,14).

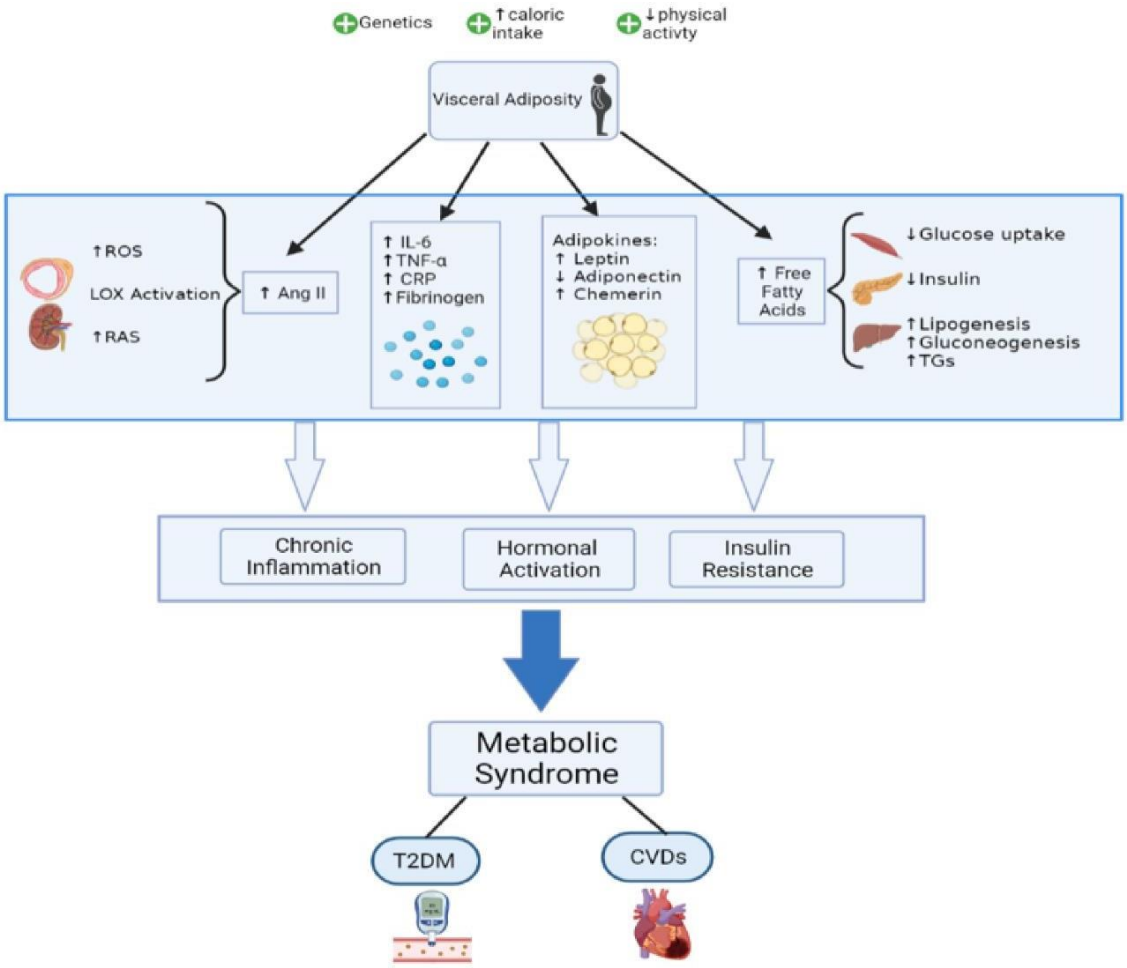

Fig 2.1.2: Pathophysiology of Metabolic Syndrome.

The components of MetS include hypertension, dyslipidemia (characterized by elevated triglycerides and low high-density lipoprotein cholesterol), and hyperglycemia (15). These metabolic abnormalities are interrelated; for instance, elevated triglycerides and low HDL cholesterol are often seen together, and both are influenced by insulin resistance (14). Furthermore, chronic low-grade inflammation plays a significant role in the pathogenesis of MetS. Inflammatory markers such as C-reactive protein (CRP) are frequently elevated in individuals with MetS, indicating a state of systemic inflammation that contributes to endothelial dysfunction and atherogenesis (13,15).

Genetic predisposition also plays a critical role in the development of MetS. Certain genetic factors may predispose individuals to obesity and insulin resistance, which in turn increases the risk of developing MetS (14). Additionally, lifestyle factors such as physical inactivity, poor dietary habits, and sedentary behavior further exacerbate the risk of MetS by promoting obesity and metabolic dysregulation (16,17).

The clinical implications of MetS are profound, as it is associated with an increased risk of cardiovascular diseases, including coronary artery disease and stroke, as well as type 2 diabetes (13,15). The recognition of MetS as a significant health issue has prompted research into potential therapeutic interventions, including lifestyle modifications, pharmacotherapy, and the modulation of dietary components to mitigate the inflammatory responses associated with MetS (13,14).

In summary, the pathophysiology of metabolic syndrome is characterized by a complex interplay of insulin resistance, obesity, inflammation, and genetic factors, leading to a cluster of metabolic abnormalities that significantly heighten the risk of serious health complications.

Causes of Metabolic Syndrome

Metabolic syndrome (MetS) is a multifaceted condition characterized by a cluster of metabolic abnormalities, including insulin resistance, obesity, dyslipidemia, and hypertension. The primary causative factor of MetS is insulin resistance, which leads to impaired glucose metabolism and increased fat accumulation, particularly visceral fat (18). This condition is often exacerbated by lifestyle factors such as physical inactivity and a diet high in saturated fats and carbohydrates, which contribute to obesity and metabolic dysregulation (18).

Obesity, especially central obesity, plays a crucial role in the development of MetS. The accumulation of visceral fat is associated with increased levels of free fatty acids and inflammatory cytokines, which further promote insulin resistance and metabolic dysfunction (18,19). Additionally, the presence of chronic low-grade inflammation is a significant contributor to the pathophysiology of MetS. Elevated levels of inflammatory markers, such as C-reactive protein (CRP), are often observed in individuals with MetS, indicating a systemic inflammatory response that can lead to endothelial dysfunction and cardiovascular complications (18,19).

Hormonal factors also influence the development of MetS. For instance, elevated aldosterone levels have been linked to increased adiposity and insulin resistance, suggesting a role for the renin-angiotensin-aldosterone system in the pathogenesis of MetS (20,21). Furthermore, the interplay between sex hormones and metabolic processes has been highlighted, with lower testosterone levels in men being associated with an increased risk of MetS (22).

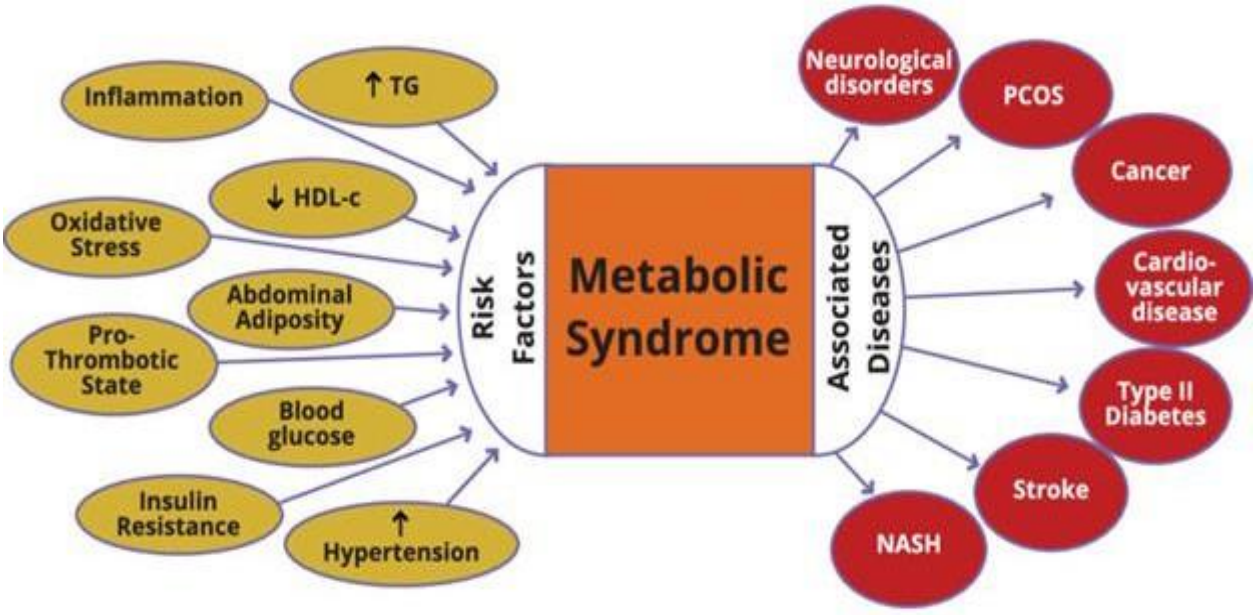

Fig 2.1.3: Risk Factors and Disease Associated with Metabolic Syndrome.

Genetic predisposition is another critical factor in the etiology of MetS. Genome-wide association studies have identified various genetic variants that increase susceptibility to obesity and insulin resistance, thereby contributing to the development of MetS (23). Additionally, certain conditions, such as polycystic ovary syndrome (PCOS), have been associated with MetS, particularly in women, highlighting the complex interplay between hormonal and metabolic factors (24).

The causes of metabolic syndrome are multifactorial, involving a combination of insulin resistance, obesity, inflammation, hormonal imbalances, and genetic predisposition. Addressing these underlying factors through lifestyle modifications and

pharmacological interventions is crucial for managing and preventing MetS and its associated health risks.

Diagnostic Criteria for Metabolic Syndrome

The diagnostic criteria for metabolic syndrome (MetS) have been established by several prominent organizations, including the National Cholesterol Education Program (NCEP),

the International Diabetes Federation (IDF), and the World Health Organization (WHO). Each of these organizations has proposed specific criteria that include a combination of metabolic abnormalities.

NCEP ATP III Criteria: According to the NCEP Adult Treatment Panel III, MetS is diagnosed when an individual exhibits at least three of the following five components:

- Abdominal obesity: Waist circumference ≥102 cm for men and ≥88 cm for women.

- Elevated triglycerides: Fasting triglycerides ≥150 mg/dL (1.7 mmol/L).

- Low HDL cholesterol: <40 mg/dL (1.03 mmol/L) for men and <50 mg/dL (1.29 mmol/L) for women.

- Hypertension: Blood pressure ≥130/85 mmHg or taking antihypertensive medication.

- Elevated fasting glucose: Fasting glucose ≥100 mg/dL (5.6 mmol/L) or diagnosed type 2 diabetes (25,26).

IDF Criteria: The IDF criteria emphasize abdominal obesity as a prerequisite for diagnosis. An individual is diagnosed with MetS if they have:

- Central obesity: Waist circumference ≥94 cm for men and ≥80 cm for women (specific ethnic cutoffs may apply).

- Plus, any two of the following:

- Elevated triglycerides: ≥150 mg/dL (1.7 mmol/L).

- Low HDL cholesterol: <40 mg/dL for men and <50 mg/dL for women.

- Hypertension: Systolic blood pressure ≥130 mmHg or diastolic ≥85 mmHg.

- Elevated fasting glucose: ≥100 mg/dL (5.6 mmol/L) or previously diagnosed type 2 diabetes (27,28).

WHO Criteria: The WHO criteria require evidence of insulin resistance (e.g., elevated fasting glucose or a history of diabetes) along with two of the following:

- Abdominal obesity: Waist circumference >94 cm for men and >80 cm for women.

- Elevated triglycerides: ≥150 mg/dL (1.7 mmol/L).

- Low HDL cholesterol: <35 mg/dL for men and <39 mg/dL for women.

- Hypertension: Blood pressure ≥140/90 mmHg or on antihypertensive treatment (29,30).

Variations in Criteria: Different populations may exhibit variations in the prevalence of MetS based on the criteria used. For instance, studies have shown that the prevalence of MetS can differ significantly when applying NCEP versus IDF criteria, highlighting the importance of context in diagnosis (31,32).

Table 2.1.1: Various Diagnostic Criteria for Metabolic Syndrome.

	IDF (Obesity + ≥ 2)	**AHA (≥ 3)**	**NCEP ATP III (≥ 3)**	**WHO (Insulin resistance / Diabetes + ≥ 2)**	**EGIR (Hyper-insulinemia + ≥ 2)**
Obesity	BMI ≥30 kg/m² or specific gender and ethnicity waist circumference cutoffs	Waist circumference for males >40 in, females >35 in	Waist circumference for males > 40 in, females > 35 in	Waist/hip ratio > 0.9 in males and > 0.85 in females or BMI ≥ 30 kg/m²	Waist circumference for males ≥ 94 cm, females ≥ 80 cm
Elevated Triglycerides	TG ≥150 mg/dL or treatment of this lipid abnormality	Fasting TG ≥150 mg/dL or treatment of this lipid abnormality	TG ≥150 mg/dL or treatment of this lipid abnormality	TG ≥150 mg/dL	TG ≥177 mg/dL
Decreased HDL	HDL <40 mg/dL in males and <50 mg/dL in females or specific treatment for this lipid abnormality	HDL <40 mg/dL in males and <50 mg/dL in females or treatment for this lipid abnormality	HDL <40 mg/dL in males and <50 mg/dL in females or treatment for this lipid abnormality	HDL <35 mg/dL in males and <39 mg/dL in females	HDL <39 mg/dL

Hypertension	SBP ≥130 or DBP ≥85 mm Hg or treatment of previously diagnosed hypertension	BP ≥130/85 mm Hg or taking medication for hypertension	SBP ≥130 or DBP ≥85 mm Hg or taking medication for hypertension	≥140/90 mm Hg	≥140/90 mm Hg or taking medication for hypertension
Hyper-glycemia	Fasting plasma glucose >100 mg/dL or previously diagnosed type 2 diabetes	Fasting glucose >100 mg/dL or taking medicine for high glucose	Fasting glucose >100 mg/dL or taking medicine for high glucose	Insulin resistance required	Insulin resistance required (plasma insulin >75th percentile)
Other				Urine albumin ≥20 µg/min or albumin ratio ≥30 mg/g	

Note: The table compares various diagnostic criteria for Metabolic Syndrome from different organizations such as IDF, AHA, NCEP ATP III, WHO, and EGIR.

The diagnosis of metabolic syndrome is based on a combination of clinical and laboratory criteria that assess abdominal obesity, lipid profiles, blood pressure, and glucose levels. The choice of criteria may depend on the population being studied and the specific health guidelines being followed.

Anthropometric Measurement in Metabolic Syndrome

Anthropometric measures play a crucial role in diagnosing and assessing metabolic syndrome (MetS), as they provide valuable insights into body composition and fat distribution, particularly central obesity. Central obesity, often assessed through waist circumference (WC), waist-to-hip ratio (WHR), and waist-to-height ratio (WHtR), is a significant predictor of MetS and its components (33,34).

Waist Circumference (WC): WC is a widely used anthropometric measure that reflects abdominal fat accumulation. It is a strong indicator of visceral fat, which is closely linked to insulin resistance and metabolic abnormalities (35). The International Diabetes Federation (IDF) and the National Cholesterol Education Program (NCEP) recommend specific cut-off values for WC to diagnose MetS: ≥94 cm for men and ≥80 cm for women (33). Studies have shown that increased WC correlates with higher risks of developing type 2 diabetes and cardiovascular diseases (36,37).

Waist-to-Hip Ratio (WHR): WHR is another important anthropometric measure that compares the circumference of the waist to that of the hips. It provides insight into fat distribution patterns, with higher ratios indicating greater central obesity and associated metabolic risks (34). WHR has been shown to be a reliable predictor of cardiovascular risk factors and is often used alongside WC to assess MetS (36,38).

Waist-to-Height Ratio (WHtR): WHtR is gaining recognition as a simple and effective measure for assessing central obesity. It is calculated by dividing waist circumference by height. Research suggests that WHtR may be a better predictor of metabolic syndrome than BMI or WC alone, as it accounts for height, which can influence fat distribution (34,38). A WHtR greater than 0.5 is often considered indicative of increased health risks associated with MetS (35).

Body Mass Index (BMI): While BMI is a commonly used measure of general obesity, it does not provide specific information about fat distribution. However, it remains a useful screening tool in conjunction with other anthropometric measures (39). Elevated BMI is associated with increased risk of MetS, but it is essential to consider it alongside WC and WHR for a comprehensive assessment (40,41).

Emerging Indices: Newer anthropometric indices, such as the Body Shape Index (ABSI), have been proposed to better assess the risk of MetS by incorporating both

waist circumference and height (42). These indices aim to provide a more nuanced understanding of body fat distribution and its implications for metabolic health.

The anthropometric measures, particularly waist circumference, waist-to-hip ratio, and waist-to-height ratio, are critical for diagnosing and assessing metabolic syndrome. They provide valuable information about central obesity, which is a key risk factor for the development of metabolic abnormalities and related health complications.

Obesity Vs Metabolic Syndrome

Central obesity is a critical component of metabolic syndrome (MetS) and plays a significant role in its pathophysiology. The accumulation of visceral fat, often measured by waist circumference, is closely associated with various metabolic abnormalities that characterize MetS, including insulin resistance, dyslipidemia, hypertension, and hyperglycemia (14,43,44).

The relationship between central obesity and MetS is well-documented. Central obesity is recognized as a primary driver of insulin resistance, which in turn leads to increased levels of triglycerides and decreased levels of high-density lipoprotein (HDL) cholesterol (37,45). This dyslipidemic profile, characterized by elevated triglycerides and low HDL cholesterol, contributes to the increased cardiovascular risk associated with MetS (43,46). Furthermore, studies have shown that central obesity is often the most prevalent component of MetS, particularly in certain populations, such as women and individuals with systemic lupus erythematosus (47,48).

The pathophysiological mechanisms linking central obesity to MetS involve chronic low-grade inflammation and hormonal dysregulation. Visceral adipose tissue secretes various pro-inflammatory cytokines and adipokines, which can lead to systemic inflammation and contribute to the development of insulin resistance (49). Additionally, hormonal changes associated with obesity, such as alterations in estrogen levels in postmenopausal women, can exacerbate the accumulation of abdominal fat and further increase the risk of MetS (46,49).

Moreover, the prevalence of central obesity has been rising globally, paralleling the increase in MetS cases. This trend underscores the importance of addressing lifestyle factors, such as diet and physical activity, to mitigate the risk of central obesity and its

associated metabolic consequences (44,45). Effective interventions targeting weight management and promoting physical activity can significantly reduce the incidence of MetS and improve overall metabolic health (14,50).

In summary, central obesity is a fundamental component of metabolic syndrome, serving as both a marker and a driver of the condition. Its association with insulin resistance, dyslipidemia, and systemic inflammation highlights the need for targeted interventions to address this critical risk factor in the prevention and management of MetS.

Insulin Resistance Vs Metabolic Syndrome

Insulin resistance is a central feature of metabolic syndrome (MetS) and plays a pivotal role in its pathogenesis. It is characterized by the body's diminished ability to respond to insulin, leading to impaired glucose uptake by tissues, particularly muscle and adipose tissue, and resulting in elevated blood glucose levels (51). This condition is often associated with obesity, particularly central obesity, which exacerbates insulin resistance through variousmechanisms, including increased free fatty acid release and inflammatory cytokine production from adipose tissue (52,53).

The relationship between insulin resistance and the components of MetS is well-established. Insulin resistance contributes to dyslipidemia, characterized by elevated triglycerides and reduced high-density lipoprotein (HDL) cholesterol levels, which are critical components of MetS (54,55). Furthermore, insulin resistance is linked to hypertension, as it promotes sodium retention and increases sympathetic nervous system activity, leading to elevated blood pressure (56). The presence of insulin resistance also predisposes individuals to type 2 diabetes, as the pancreas initially compensates for insulin resistance by increasing insulin secretion, which may eventually lead to beta-cell dysfunction and hyperglycemia (51,52).

Several biomarkers have been identified to assess insulin resistance, with the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) being one of the most commonly used (57). Elevated levels of insulin and glucose are indicative of insulin resistance and are often used in conjunction with other MetS criteria to establish a diagnosis (58). Research has shown that insulin resistance is prevalent in various populations, including children and adolescents, where it is associated with obesity and metabolic abnormalities (59,60).

Moreover, the pathophysiology of insulin resistance involves complex interactions between genetic, environmental, and lifestyle factors. For instance, high-fat diets have been shown to induce insulin resistance through mechanisms such as inflammation and oxidative stress (56,61). Additionally, chronic low-grade inflammation, often seen in individuals with obesity, further exacerbates insulin resistance and contributes to the development of MetS (62,63).

In conclusion, insulin resistance is a fundamental abnormality in metabolic syndrome, linking obesity, dyslipidemia, hypertension, and hyperglycemia. Understanding the mechanisms underlying insulin resistance and its role in MetS is crucial for developing effective prevention and treatment strategies aimed at improving insulin sensitivity and reducing the risk of associated complications.

Lipid Profile in Metabolic Syndrome

The lipid profile is a critical component in the assessment and diagnosis of metabolic syndrome (MetS), as dyslipidemia is one of the hallmark features of this condition.

Dyslipidemia in MetS is characterized by elevated triglycerides, low high-density lipoprotein cholesterol (HDL-C), and often increased low-density lipoprotein cholesterol (LDL-C) levels, which collectively contribute to an increased risk of cardiovascular disease (37,64,65)

Studies have consistently shown that individuals with MetS exhibit significant alterations in their lipid profiles. For instance, a correlation between central obesity and altered lipid profiles across various age groups, indicating that dyslipidemia is a prevalent risk factor for MetS (36). Similarly, hypertriglyceridemia and hypercholesterolemia are common in individuals with MetS, contributing to increased cardiovascular morbidity and mortality (64).

A characteristic feature of MetS is atherogenic dyslipidemia, which includes elevated levels of triglycerides and small, dense LDL particles, along with low levels of HDL-C.

This lipid profile is associated with an increased risk of atherosclerosis and cardiovascular events ,(65)

Research indicates that individuals with MetS often present with a lipid profile that includes increased triglycerides and decreased HDL-C, which are critical indicators of cardiovascular risk (65).

Obesity, particularly central obesity, is closely linked to dyslipidemia in MetS. Studies have shown that as body mass index (BMI) increases, there is a corresponding rise in triglyceride levels and a decrease in HDL-C levels (66,67). This relationship underscoresthe importance of weight management in mitigating the lipid abnormalities associated with MetS.

The presence of dyslipidemia in MetS necessitates careful monitoring and management to reduce cardiovascular risk. Interventions such as lifestyle modifications, including diet and exercise, as well as pharmacological treatments like statins, are often employed to improve lipid profiles and overall metabolic health (46,68). For example, the treatment with atorvastatin led to significant improvements in lipid profiles among patients with MetS (68).

Recent studies continue to explore the relationship between lipid profiles and MetS, including the role of thyroid function in lipid metabolism and its association with MetS

components (69,70). Understanding these relationships is crucial for developing targeted interventions to address dyslipidemia in patients with MetS..

The lipid profile is a vital aspect of metabolic syndrome, with dyslipidemia serving as a key indicator of increased cardiovascular risk. Effective management of lipid abnormalities through lifestyle changes and pharmacotherapy is essential for improving health outcomes in individuals with MetS.

Clinical Outcome in Metabolic Syndrome

The clinical outcomes associated with metabolic syndrome (MetS) are significant and multifaceted, impacting various aspects of health, including cardiovascular disease, cancer risk, and postoperative complications. The presence of MetS is linked to an

increased risk of developing serious health conditions, which underscores the importance of early detection and management.

Cardiovascular Disease: MetS is a well-established risk factor for cardiovascular diseases (CVD). The cluster of conditions that define MetS, including hypertension, dyslipidemia, and insulin resistance, contribute to the development of atherosclerosis and other cardiovascular complications (71). Studies have shown that individuals with MetS havea higher incidence of myocardial infarction, stroke, and heart failure compared to those without the syndrome (72). Furthermore, specific components of MetS, such as abdominal obesity and elevated triglycerides, have been identified as strong predictors of cardiovascular events (73).

Cancer Risk: There is a growing body of evidence suggesting that MetS is associated with an increased risk of certain cancers, particularly colorectal and breast cancers (74,75). The underlying mechanisms may involve insulin resistance, chronic inflammation, and hormonal changes that promote tumorigenesis. Patients with MetS are encouraged to undergo regular cancer screenings due to the heightened risk associated with the syndrome (74). Additionally, metabolic abnormalities linked to MetS may worsen clinical outcomes in cancer survivors, indicating a need for integrated management strategies (73).

Postoperative Complications: MetS has been shown to increase the risk of postoperative complications in various surgical settings. For instance, patients undergoing hepatectomy with MetS have been reported to experience higher rates of surgical site infections, respiratory failure, and acute renal failure compared to those without the syndrome (72). Similar findings have been observed in orthopedic procedures, where MetS is associated with increased adverse outcomes (76). These complications highlight the importance of optimizing the management of MetS prior to surgical interventions.

Diabetes and Metabolic Disorders: The presence of MetS significantly raises the risk of developing type 2 diabetes mellitus. Insulin resistance, a hallmark of MetS, leads to impaired glucose metabolism and can progress to diabetes if not addressed (71). This

progression not only affects metabolic health but also increases the risk of cardiovascular complications and other chronic diseases (71).

Mortality Risk: Studies indicate that MetS is associated with an increased risk of all-cause mortality, particularly due to cardiovascular diseases and diabetes-related complications (75). The clustering of risk factors inherent in MetS exacerbates health risks, making it a critical target for public health interventions aimed at reducing morbidity and mortality (71).

The clinical outcomes associated with metabolic syndrome are extensive and include increased risks of cardiovascular disease, certain cancers, postoperative complications, and diabetes. Effective management of MetS through lifestyle modifications and medical interventions is essential to mitigate these risks and improve overall health outcomes.

Renal Impairment in Metabolic Syndrome

Renal dysfunction is a significant concern in individuals with metabolic syndrome (MetS), as the interplay between metabolic abnormalities and kidney health can lead to chronic kidney disease (CKD) and other renal complications. The relationship between MetS and renal dysfunction is multifaceted, involving various mechanisms, including insulin resistance, hypertension, and dyslipidemia.

Insulin Resistance and Renal Function: Insulin resistance, a hallmark of MetS, has been shown to adversely affect renal function. Elevated insulin levels can lead to increased renal blood flow and hyperfiltration, which may initially preserve glomerular filtration rate (GFR) but eventually contribute to nephron damage and CKD (77). The study supports the notion that metabolic syndrome can accelerate the loss of kidney function through mechanisms such as hyperfiltration and increased renal blood flow due to elevated insulin levels (77).

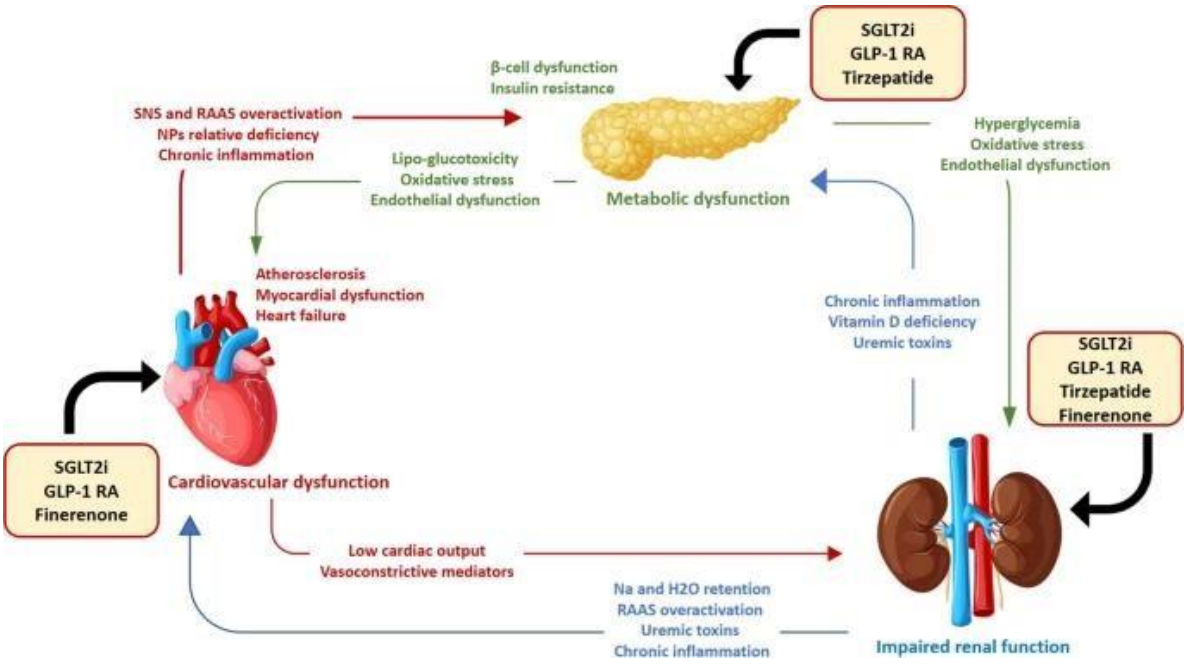

Fig:2.1.4 Association of Cardio-Renal-Metabolic Components.

Hypertension: Hypertension is a critical component of MetS and is a well-known risk factor for renal dysfunction. The increased vascular resistance associated with hypertension can lead to glomerular damage and decreased renal perfusion over time (77). Studies have demonstrated that individuals with MetS are at a higher risk of developing hypertension, which in turn exacerbates renal injury and accelerates the progression of CKD (78).

Dyslipidemia: Dyslipidemia, characterized by elevated triglycerides and low HDL cholesterol, is another key feature of MetS that impacts renal health. Dyslipidemia can lead to the accumulation of lipids in renal tissues, contributing to inflammation and fibrosis, which impair kidney function (79). The study highlights that subjects with metabolic syndrome are at increased risk of developing CKD and diminished renal function, emphasizing the importance of monitoring lipid profiles in these patients (79).

Microalbuminuria: Microalbuminuria is often an early indicator of renal dysfunction in patients with MetS. It reflects increased permeability of the glomerular filtration barrier and is associated with a higher risk of developing CKD (80). The presence of

microalbuminuria in individuals with MetS can indicate ongoing renal damage and necessitates further evaluation and management to prevent progression to more severe renal impairment (80).

Chronic Kidney Disease (CKD): The prevalence of CKD is significantly higher in individuals with MetS. Research indicates that metabolic syndrome is an independent predictor of CKD, with studies showing that a substantial proportion of patients with CKD also meet the criteria for MetS (78,81). The mechanisms linking MetS to CKD include the combined effects of insulin resistance, hypertension, and dyslipidemia, which collectively contribute to renal injury and functional decline (78).

Clinical Implications: The recognition of renal dysfunction in the context of MetS underscores the importance of early screening and intervention. Regular monitoring of renal function, including serum creatinine and urine albumin levels, is essential for individuals with MetS to detect early signs of renal impairment (77). Lifestyle modifications, such as weight management, dietary changes, and pharmacotherapy to control blood pressure and lipid levels, are critical in mitigating the risk of renal dysfunction in this population (78,79).

In conclusion, renal dysfunction is a significant complication of metabolic syndrome, driven by the interplay of insulin resistance, hypertension, and dyslipidemia. Early detection and management of renal abnormalities in individuals with MetS are crucial for preventing the progression to chronic kidney disease and improving overall health outcomes.

CHAPTER-III

AIM AND OBJECTIVES

CHAPTER-III

Aim and Objectives

AIM AND OBJECTIVES

3.1.1 REASON FOR CHOOSING THE TOPIC

The present study has identified the metabolic syndrome persons with factors 3,4 and 5 according to NCEP-ATP III criteria. The biochemical parameters for the renal function were estimated. The correlation of metabolic syndrome and kidney functions was done. Estimation of eGFR and urine albumin creatinine ratio values can indicate the development of renal dysfunction.

The study related to number of components of metabolic syndrome and correlation of kidney dysfunction among three groups with factors 3,4 and 5 is studied less till date.

3.1.2 RESEARCH PROBLEM

1) To determine the prevalence of metabolic syndrome in the study population (According to NCEP-ATP III criteria)

2) To compare the variation in renal function among three groups with factors 3, 4 and 5 identified according to NCEP-ATP III criteria.

3.1.3 AIM:

The present study aims to correlate the parameters of metabolic syndrome and kidney function tests.

Study Group-A	**Study Group-B**	**Study Group-C**
Metabolic syndrome persons with 3 components Based on the NCEP ATP III guidelines	Metabolic syndrome persons with 4 components Based on the NCEP ATP III guidelines	Metabolic syndrome persons with 5 components Based on the NCEP ATP III guidelines

CHAPTER-IV
METHODOLOGY

CHAPTER-IV

Methodology

METHODOLOGY

This investigative study is an observational descriptive study conducted at Dr. D.Y. Patil Medical College Hospital, Maharashtra. The research received ethical approval from the Institutional Ethical Committee (IEC) at Dr. D.Y. Patil Medical College Hospital. The study screened 1,156 participants out of those 400 individuals were found to be metabolic syndrome as follows.

Table 4.1.1 – NCEP ATP III Criteria for Metabolic Syndrome.

Sl. No	Criteria	Report
		At least three or more of the following
1)	Fasting blood sugar	More than 100mg%
2)	Blood pressure	More than 130/85 mm Hg
3)	Triglycerides	More than 150 mg %
4)	HDL cholesterol (males)	Less than40 mg %
	HDL cholesterol (females)	Less than 50 mg%
5)	Obesity (males)	More than 102cm
	Obesity (females)	More than 88 cm

These 400 subjects were included in the study and further divided into three group based on the NCEP ATP III guidelines by ICMR. Individuals with three factors positive were specifically labeled as "***A***", four factors were denoted as "***B***" and five factors were classified as "***C***"

Table 4.1.2– Categorization of Study Participants.

"A"	With 3 out 5 criteria for MetS
"B".	With 4 out 5 criteria for MetS
"C"	With all 5 criteria for MetS

Inclusion Criteria: In the study, individuals diagnosed with metabolic syndrome aged between 30-65 years were considered for inclusion and study includes urban population.

Exclusion Criteria: Individuals with a history of Hypothyroidism, Pregnancy, Ascites, Malignancies, Steroids, Familial dyslipidemia and Secondary hypertension were not included in the study.

A comprehensive proforma was completed, capturing the individual's name, age, gender, and anthropometric measurement.

Sample Size Determination:

It was determined that the prevalence rate of metabolic syndrome in India stands at 30% This statistical finding was derived with a confidence level of 95% and a margin of error of 5%. The calculation of the required sample size was performed utilizing the following Slovin's Formula.

$$\mathbf{n = \frac{N}{1+Ne^2}} \quad \text{- ---------- (4.1)}$$

Where:

n denotes the required sample size

N symbolizes the population size

e signifies the estimated margin of error (e = 0.05)

$$\boldsymbol{n = \frac{100000}{1 + 100000 * 0.05^2}}$$

$$\mathbf{n \sim \frac{100000}{251}}$$

$$\mathbf{n \sim 398.4}$$

$$\mathbf{n=400}$$

Upon application of the formula with the respective values, the estimated sample size was determined to be 400.

Anthropometric Measurements

The study involved taking precise anthropometric measurements comprising Blood pressure in mmHg, Waist Circumference (WC) and Hip circumference in centimeters. Subsequently, the Waist hip ratio was derived using the formula: Waist Circumference divided by hip circumference as per WHO guideline.

$$\mathbf{WHR} = \frac{\mathbf{Waist\ Circuference\ (cm)}}{\mathbf{Hip\ circuference\ (cm)}} \quad \text{----------(4.2)}$$

BLOOD SAMPLE COLLECTION

Instructions to the Patients

The study subjects were instructed to adhere to an overnight fasting regimen before collecting biological samples.

Blood Collection Procedure

Specifically, 2 mL of blood samples were drawn into a fluoride tube and 3ml into clot tube. Subsequently, the clot tubes were centrifugated at 3500 rpm for approximately 15 minutes at room temperature to facilitate the isolation of serum samples.

BIOCHEMICAL PARAMETERS

Blood Tests Performed

Plasma Fasting Blood Glucose (FBS)

Serum Urea,

Serum Creatinine,

Serum Uric Acid,

Serum Fasting Lipid Profile (FLP).

Urine Tests Performed

Urine Albumin

Urine Creatinine were analyzed using the semi-automatic analyzer Erba CHEM 5X.

Fasting Blood Glucose:

Method: Hexokinase Method

The hexokinase catalyzes the phosphorylation of glucose in the presence of ATP and magnesium ions, producing glucose-6-phosphate and ADP. Subsequently, glucose-6-phosphate dehydrogenase (G6P-DH) facilitates the oxidation of glucose-6-phosphate to gluconate-6-phosphate, by the reduction of NAD+ to NADH. The resultant increase in NADH concentration is directly proportional to the glucose concentration in the sample, which is quantifiable by measuring the absorbance at 340 nm.

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 10 µl of distilled water added to the B test tube, and then, 10 µl of standard glucose solution was added to the S test tube, and 10 µl of serum sample to T test tube. And then, 1 ml of Glucozyme working reagent was added to all the three test tubes. The mixture was gently mixed and incubated for 15 minutes at room temperature. Following incubation, the absorbance of each sample was measured at 515 nm.

Fasting Lipid Profile - Total Cholesterol:

Method: Enzymatic Cholesterol Esterase (CHE) method

The enzyme cholesterol esterase hydrolyzes cholesterol esters and forms free cholesterol and fatty acid molecules. In the presence of cholesterol oxidase this free cholesterol gets oxidized and cholest-4 ene-3 one and peroxide is liberated. In the presence of phenol and peroxidase the indicator quinoneimine is formed from hydrogen peroxide and 4-aminoantipyrine. The intensity of this colored complex is measured and is directly proportional to the cholesterol concentration present in the sample. The intensity is measured at 540nm.

2 Cholesterol esters + 2 H_2O → 2 Cholesterol + 2 Fatty acids

Oxidation of Cholesterol: 2 Cholesterol + 2 O_2 → 2 Cholestene-3-one + 2 H_2O_2

Quinoneimine Dye: 2 H_2O_2 + 4-Aminoantipyrine + Phenol → Quinoneimine + 4 H_2O

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 10 µl of distilled water added to the B test tube, and then, 10 µl of standard solution was added to the S test tube, and 10 µl of serum sample was added to the T test tube. Then, 1 ml of enzyme reagent was added to all the three test tubes. Now the mixture was gently mixed and incubated for 15 minutes at room temperature. The absorbance of standard and test was measured against the reagent blank at 540 nm. The reference range for serum cholesterol is 150-200 mg/dL.

Fasting Lipid Profile - Serum Triglycerides

Method: Enzymatic Lipase method

Triglycerides are hydrolyzed to glycerol and free fatty acids by lipases. In the presence of Adenosine Triphosphate (ATP) and glycerol kinase (GK), glycerol converts to glycerol 3-phosphate and Adenosine diphosphate (ADP). It is then oxidized by Glycerol Phosphate Oxidase (GPO) to yield hydrogen peroxide. Peroxidases catalyze the conversion of hydrogen peroxide, 4 – aminoantipyrine (4AAP) and 4-chlorophenol to produce a Purplish Brown coloured dye. The coloured complex measured 546 nm.

Triglycerides + 3 H_2O → Glycerol + 3 Fatty acids

Glycerol + ATP → Glycerol-3-phosphate + ADP

Glycerol-3-phosphate + O_2 → Dihydroxyacetone phosphate + H_2O_2

H_2O_2 + 4-AAP + MADB → Blue Dye + OH- + 3 H_2O

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 10 µl of distilled water added to the B test tube, and then, 10 µl of standard solution was added to the S test tube, and 10 µl of serum sample was added to the T test tube. Followed by the addition of 1 ml of enzyme reagent. After thorough mixing, tubes were incubated at ambient temperature for 15 minutes at 546 nm. Serum triglyceride levels typically range from 0 to 150 mg/dL.

Fasting Lipid Profile High-Density Lipoprotein (HDL)

Method: Enzymatic Lipase method

The enzymatic assay reaction commences with HDL-C, cholestenone, and hydrogen peroxide (H_2O_2), where HDL-C combines with cholestenone and H_2O_2 to initiate the reaction cascade. Its further catalyzed by the addition of 4-aminoantipyrine and N, N-bis (4-sulfobutyl)-3,5-dimethylaniline, disodium salt (HSDA), resulting in the production of the characteristic purple-blue pigment.

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 10 µl of distilled water added to the B test tube, and then, 6 µl of standard solution was added to the S test tube, and 6 µl of serum sample was added to the T test tube. Subsequently, 450 µl of Enzyme reagent (R1) was added to tubes S and T, followed by 150 µl of Enzyme reagent (R2) to ensure precise enzymatic activity. After careful mixing by gentle swirling, the test tubes were incubated at room temperature for 5 minutes at 546 nm. The reference range for serum HDL-C typically falls between 40 to 45 mg/dL, reflecting normal lipid metabolism within this interval.

Fasting Lipid Profile – Very Low-Density Lipoprotein (VLDL)

Method: Friedewald Formula (82)

The Friedewald Formula, is a widely utilized method for estimating Very Low-Density Lipoprotein (VLDL) cholesterol levels in a fasting lipid profile. In adults, the reference range for VLDL cholesterol is less than 30 mg/dL.

Calculation:

$$\mathbf{VLDL} = \frac{\mathbf{TGL}}{\mathbf{5}} \text{ - ----------(4.3)}$$

Fasting Lipid Profile – Low-Density Lipoprotein (LDL)

Method: Friedewald Formula (82)

The Friedewald Formula,, is a widely utilized method for estimating Low-Density Lipoprotein (LDL) cholesterol levels in a fasting lipid profile. The formula calculates LDL cholesterol using the following equation (Eqn. 4.4). In adults, the reference range for LDL cholesterol is typically considered to be less than 130 mg/dL.

Calculation:

$$\mathbf{LDL} = \mathbf{Total\ Cholesterol} - (\mathbf{HDL} + \mathbf{VLDL}) \text{----------} \mathbf{(4.4)}$$

Triglycerides/ HDL ratio:

Introduction:

The Triglycerides/HDL ratio is calculated dividing serum triglyceride by serum high-density lipoprotein measured by Fasting Lipid Profile (FLP) (83).

Calculation:

$$\mathbf{Triglycerides} = \frac{\mathbf{TGL}\ (\frac{\mathbf{mg}}{\mathbf{dL}})}{\mathbf{HDL}\ (\frac{\mathbf{mg}}{\mathbf{dL}})} \text{----------} \mathbf{(4.5)}$$

Serum Urea

Method: GLDH Method

In the presence of water and urease, urea is hydrolyzed and produces ammonia and carbon dioxide. In the presence of glutamate-dehydrogenase (GLDH) the ammonia from this reaction combines with 2-oxoglytarate and NADH and yields glutamate and NAD+. The test has been optimized so that the glutamate-dehydrogenase is the rate limiting enzyme. The urea concentration within the given time intervals is proportional the decrease in the absorbance. This test is preferably designed for analyzer application.

$$\text{Urea} + H_2O \xrightarrow{\text{Urease}} NH_3 + CO_2$$

$$\alpha\text{-KG} + NH_3 + \text{NADH} \xrightarrow{\text{GLDH}} \text{L-glutamate} + NAD^+$$

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 10 µl of distilled water added to the B test tube, and then, 10 µl of standard solution was added to the S test tube, and 10 µl of serum sample to the T test tube. Subsequently,

800 µl of Enzyme reagent (R1) was added to tubes S and T, followed by 200 µl of Enzyme reagent (R2) to ensure precise enzymatic activity. After careful mixing by gentle swirling, the test tubes are incubated at room temperature for 5 minutes at 340 nm. The reference range for serum urea typically falls between 19 to 45 mg/dL, reflecting normal urea within this interval.

Serum and Urine Creatinine

Method: Modified Jaffe Kinetic Method

Creatinine forms a colored orange-red complex in an alkaline picrate solution. The difference in absorbance at fixed times during conversion is proportional to the concentration of creatinine in the sample.

Creatinine + Picric acid → Creatinine picrate complex

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 50 µl of distilled water added to the B test tube, and then, 50 µl of standard solution was added to the S test tube, and 50 µl of serum/urine sample was added to the T test tube. Subsequently, 1000 µl of Enzyme reagent (R1) was added to tubes S and T, followed by 250 µl of Enzyme reagent (R2) to ensure precise enzymatic activity. After careful mixing by gentle swirling, the test tubes were incubated at room temperature for 5 minutes at 505 nm. The reference range for serum urea typically falls between 0.6 to 1.1 mg/dL for female and 0.7 to 1.3 mg/dL for male, reflecting normal creatinine levels within this interval.

Serum Uric Acid

Method: Uricase-POD

Uric Acid is oxidized to allantoin by uricase with the production of H_2O_2. The peroxide reacts with 4-aminoantipyrine (4-AAP) and TOOP in the presence of peroxidase to yield a quionemine dye.

$$\text{Uric} + O_2 + H_2O \xrightarrow{\text{Uricase}} \text{Allantoin} + H_2O_2 + CO_2$$

$$\text{TOOS} + 4\text{AAP} + 2\,H_2O_2 \xrightarrow{\text{Peroxidase}} \text{Quinoeimine dye} + 4H_2O$$

Three test tubes were prepared and labeled as blank (B), standard (S), and test (T). 10 µl of distilled water added to the B test tube, and then, 10 µl of standard solution was added to the S test tube, and 10 µl of serum sample to the T test tube. Subsequently, 800 µl of Enzyme reagent (R1) was added to tubes S and T, followed by 200 µl of Enzyme reagent (R2) to ensure precise enzymatic activity. After careful mixing by gentle swirling, the test tubes were incubated at room temperature for 5 minutes at 546 nm. The reference range for serum urea typically falls between 2.6 to 6.0 mg/dL for female and 3.5 to 7.2 mg/dL for male, reflecting normal creatinine levels within this interval.

Urine Albumin

Method: Turbidimetric immunoassay

Albumin test is a turbidimetric immunoassay for the detection of albumin in urine and is based on the principle of agglutination reaction. The test specimen is mixed with the active buffer (R1) and Antiserum reagent (R2) and allowed to react. Presence of albumin in the test specimen forms an insoluble complex producing a turbidity, which is measured at wavelength 340 nm.

Three test tubes were prepared and labeled as control (C), standard (S), and test (T). 24 µl of control added to the C test tube, and then, 24 µl of standard solution was added to the S test tube, and 24 µl of serum sample was added to the T test tube. Subsequently, 800 µl of Buffer reagent (R1) was added to tubes S and T, followed by 200 µl of antiserum (R2). After careful mixing by gentle swirling, the test tubes were incubated at room temperature for 5 minutes at 340 nm. The reference range for urine albumin typically falls below 25 mg/L.

Urine Albumin: Creatinine Ratio

The Urine Albumin: Creatinine ratio can be calculated dividing urine albumin by urine creatinine measured which help us to assess renal function (84).

Calculation:

$$\boldsymbol{Urine\ Albumin}:\boldsymbol{Creatinine\ Ratio}\ (\boldsymbol{UACR}) = \frac{\boldsymbol{Urine\ Albumin}\ (\frac{\boldsymbol{mg}}{\boldsymbol{L}})}{\boldsymbol{Urine\ Creatinine}\ (\frac{\boldsymbol{mg}}{\boldsymbol{L}})}$$

4.6 Statistical Analysis:

The collected data was statistically analyzed. The SPSS software, version 20, was applied for the statistical analysis. Categorical data is shown using percentages. The other data were expressed using the mean and standard deviation (SD). To determine whether there was a significant difference between the groups, a student's "t" test was employed. The Pearson correlation analysis is used to examine the significant association between different parameters. In the case of statistical significance, a p value of less than 0.05 was used.

CHAPTER-V

RESULTS

CHAPTER – V

Results

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RESULTS

A total of 1156 individuals were screened and 400 participants were identified as metabolic syndrome patients, based on National Cholesterol Education Program Adult Treatment Panel-III (NCEP ATP-III), with the prevalence of 34.6% in Fig.5.1.1

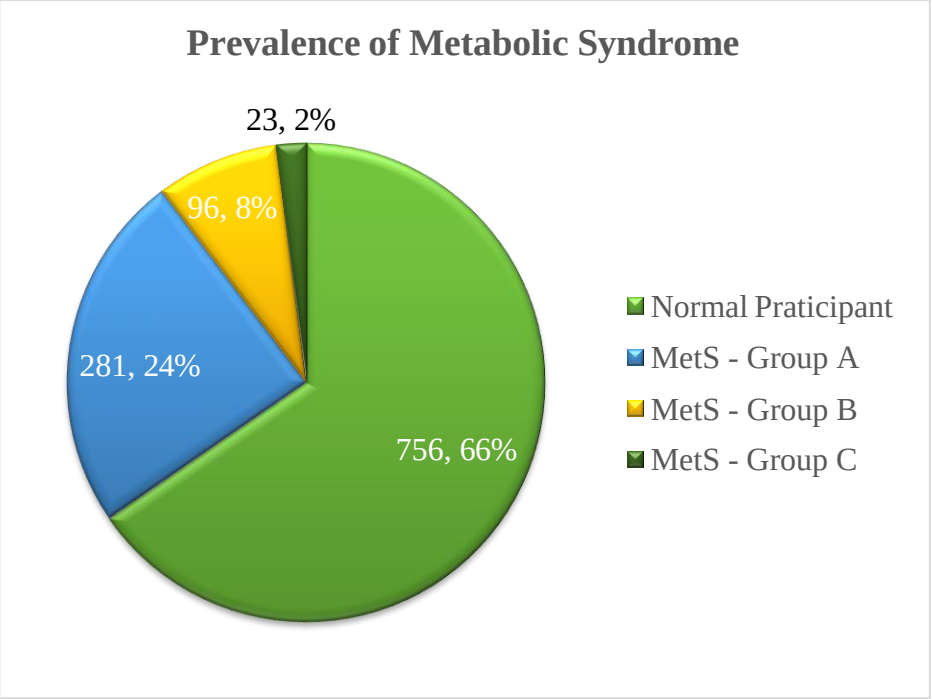

Fig 5.1.1: Prevalence of Metabolic Syndrome (Group A – 3/5 Criteria of MetS; Group B – 4/5 Criteria of MetS and Group C – all 5 Criteria of MetS) .

281 participants were identified as metabolic syndrome with three factors positive and labeled as Group "A" with a mean age of 52.21 ± 7.25, 96 participants were identified as metabolic syndrome with four factors positive and labeled as Group **"B"** with a mean age of 53.86 ± 6.97 and 23 participants were identified as metabolic syndrome with all five factors positive and labeled as Group "C" with a mean age of 54.73 ± 5.7. To determine the significant value among the groups we have performed one-way ANOVA, but it didn't show any significant difference between the group, $F(2, 397) = 2.87$, $p = 0.058$.

Table 5.1.1: Age Distribution among Metabolic Syndrome Groups A,B and C.

Age	Group A	Group B	Group C	χ2 value	df	P value
31-40	4	2	1	1.22	6	**0.97**
41-50	93	32	7			
51-60	151	51	12			
>60	33	11	3			

In Group "A", males were 67% (n=189) and the females were 33% (n=92). In Group "B", males were 84% (n=81) and the females were 16% (n=15). In Group "C", males were 65% (n=15) and the females were 35% (n=8) Fig 5.1.2.. To examine the relationship between gender and metabolic syndrome among groups, chi square test was performed. There was significant difference value with $X^2 = 10.66$, $p = 0.004$. In Group A, when compared with male, females were more likely to be MetS. In Group B, when compared with female, males were more likely to be MetS. And in Group C both males and females are equal likely hood to be MetS.

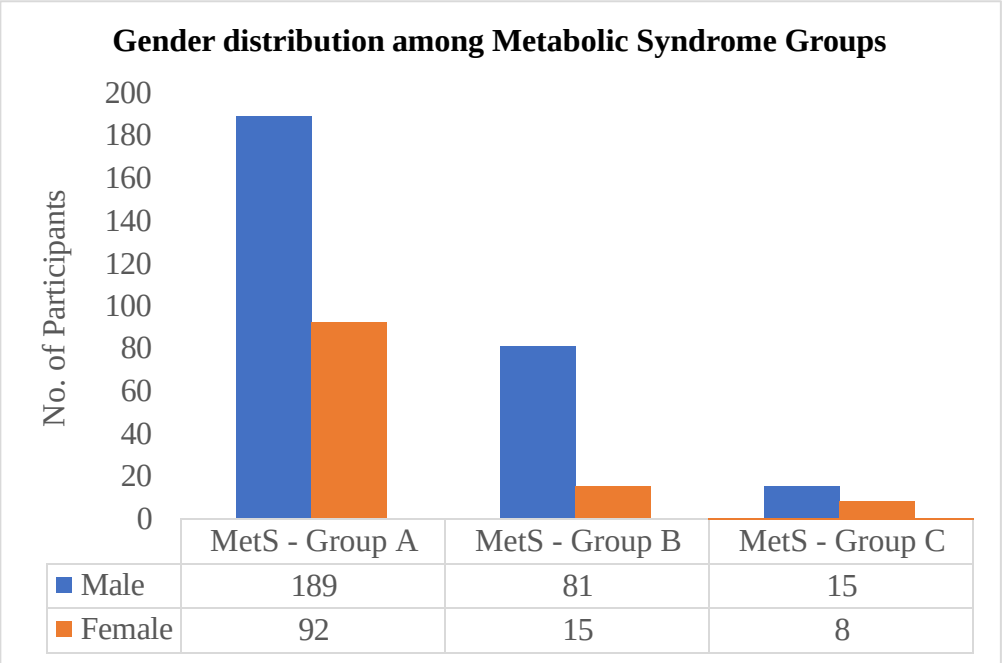

Fig 5.1.2: Gender Distribution among Metabolic Syndrome Groups.

Comparison of Anthropometric Measurement among Groups.

Waist circumference was compared among groups using one-way ANOVA. Results showed a significant difference in waist circumference among the groups, F (2, 397) = 9.448, p< 0.001. Post hoc analysis revealed, Group A and B have significant value, Group A has low waist circumference when compare with B, t = -4.291, p < 0.001. Whereas Group A and C, Group B and C didn't show any significant value.

Waist Hip Ratio (WHR) of Group A, B, and C is depicted in table 5.1.2. The mean WHR of Group A is 0.85 ± 0.06,Group B is 0.87 ± 0.05, and Group C is 0.91 ± 0.06. In comparison between the groups using one-way ANOVA, the waist hip ratio was found to be significant, F(2, 397) = 8.247, p = 0.001.

Table 5.1.2 Demographic Characteristics and Anthropometric Measurementsamong Groups A, B, and C.

Parameter	**Group A** **Mean** ± SD **n=281**	**Group B** **Mean** ± SD **n=96**	**Group C** **Mean** ± SD **n=23**	**p value**
Age in Yrs	52.21 ± 7.25	53.86 ± 6.97	54.73 ± 5.7	0.058
Male, n (%)	189 (67%)	81 (84%)	15 (65%)	0.004#
Female, n (%)	92 (33%)	15 (16 %)	08 (35%)	
Waist circumference (in cm)	101.59 ± 7.27	105.65 ± 6.79	103.93 ± 10.78	<0.001
Waist Hip Ratio	0.85 ± 0.06	0.87 ± 0.05	0.91 ± 0.06	0.001
Systolic Blood Pressure (mm Hg)	147.55 ± 7.85	148.11 ± 6.6	150 ± 8.45	0.158NS
Diastolic Blood Pressure (mm Hg)	84.83 ± 5.09	87.03 ± 4.63	83.33 ± 4.88	0.001

Note – p value 0.01 is significant, p value < 0.001 is highly significant.
[#] Chi square analysis was performed.

Group A - With 3 out 5 criteria for MetS; Group B - With 4 out 5 criteria for MetS; Group C - With all 5 criteria for MetS, NS- Not significant

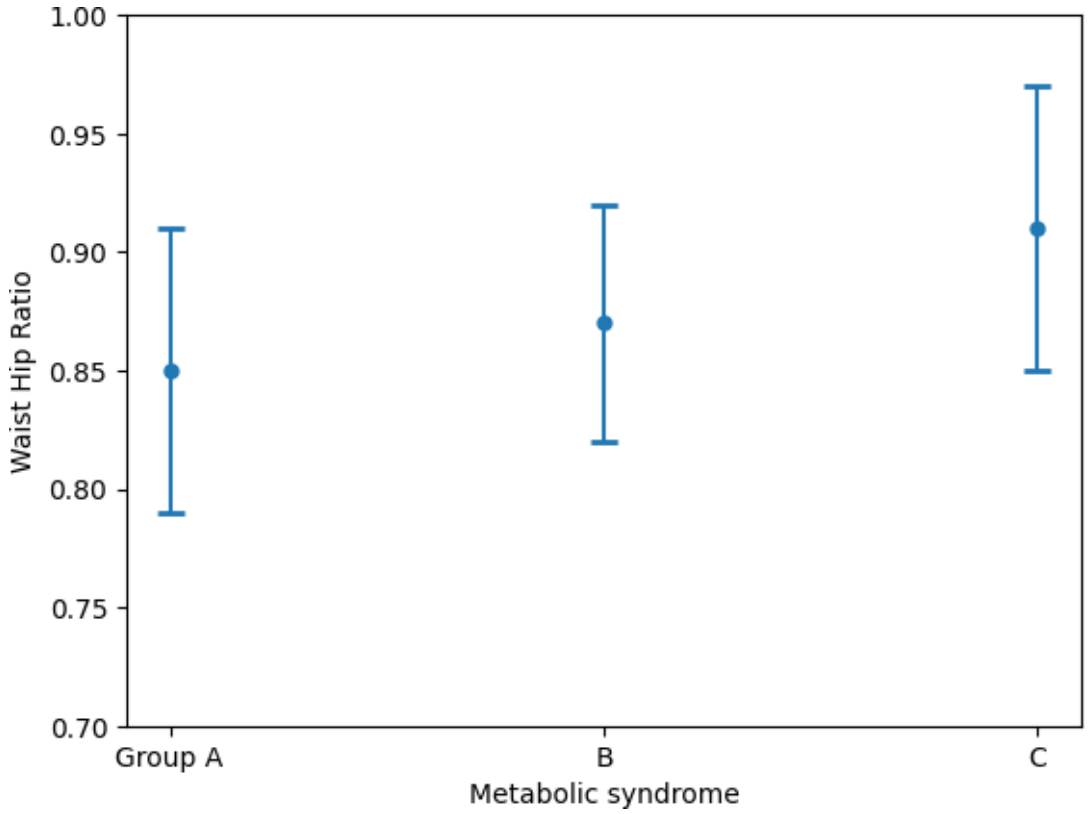

Fig 5.1.3: **Comparison of Waist Hip Ratio among Groups A, B, and C.**

Blood Pressure both systolic and diastolic are shown in table 5.1,2. The mean systolic blood pressure of Group A is 147.58 ± 7.67, Group B is 148.42 ± 6.57 and Group C is 150.42 ± 8.06. One way ANOVA was performed to identify significant between groups, but systolic blood pressure indicates no significant value, $F(2, 397) = 1.853$, $p < 0.158$. The mean diastolic blood pressure of Group A is 84.84 ± 5.01, Group B is 87.16 ± 4.53 and group C is 82.92 ± 4.64. There was significant value between groups. Post hoc analysis showed the group A had significant value with Group B. Group B and Group C had significant value. But, between group A and C it is not significant.

Comparison of Fasting Blood Glucose and Lipid Profile among Groups

One way ANOVA was conducted to compare the mean fasting glucose level among groups. The mean of group A is 123.47 ± 39.39, group B is 145.73 ± 50.63and group C is 185.0 ± 12.08. A significant value was found among the groups with $F(2, 397) = 16.908$, $p < 0.001$. Post hoc analysis showed between group A and B it is significant, It is significant as well between group B and C, But between group A and C it is not significant.

Table 5.1.3 Comparison of FBS and Lipid Profile among Groups A, B, and C.

Parameter	**Group A** **Mean** ± SD **n=281**	**Group B** **Mean** ± SD **n=96**	**Group C** **Mean** ± SD **n=23**	**p value**
Fasting Blood Glucose (mg/dl)	123.4 ± 39.39.	145.73 ± 50.63	185.3 ± 12.08	<0.001
Total Cholesterol (mg/dl)	197.38 ± 34.23	195.4 ± 40.3	187.3 ± 8.07	0.28
Triglycerides (mg/dl)	202.14 ± 84.08	206.08 ± 38.59	240.73 ± 64.67	0.049
High Density Lipoprotein (mg/dl)	50.95 ± 4.93	50.87 ± 5.63	40.67 ± 8.07	0.001
Low Density Lipoprotein (mg/dl)	104.64 ± 34.24	104.87 ± 32.11	93.33 ± 17.20	0.078
Very Low Density Lipoprotein (mg/dl)	41.79 ± 9.85	39.67 ± 9.12	53.33 ± 8.12	0.050

Note – p value 0.01 is significant, p value < 0.001 is highly significant.
Group A - With 3 out 5 criteria for MetS; Group B - With 4 out 5 criteria for MetS; Group C - With all 5 criteria for MetS.

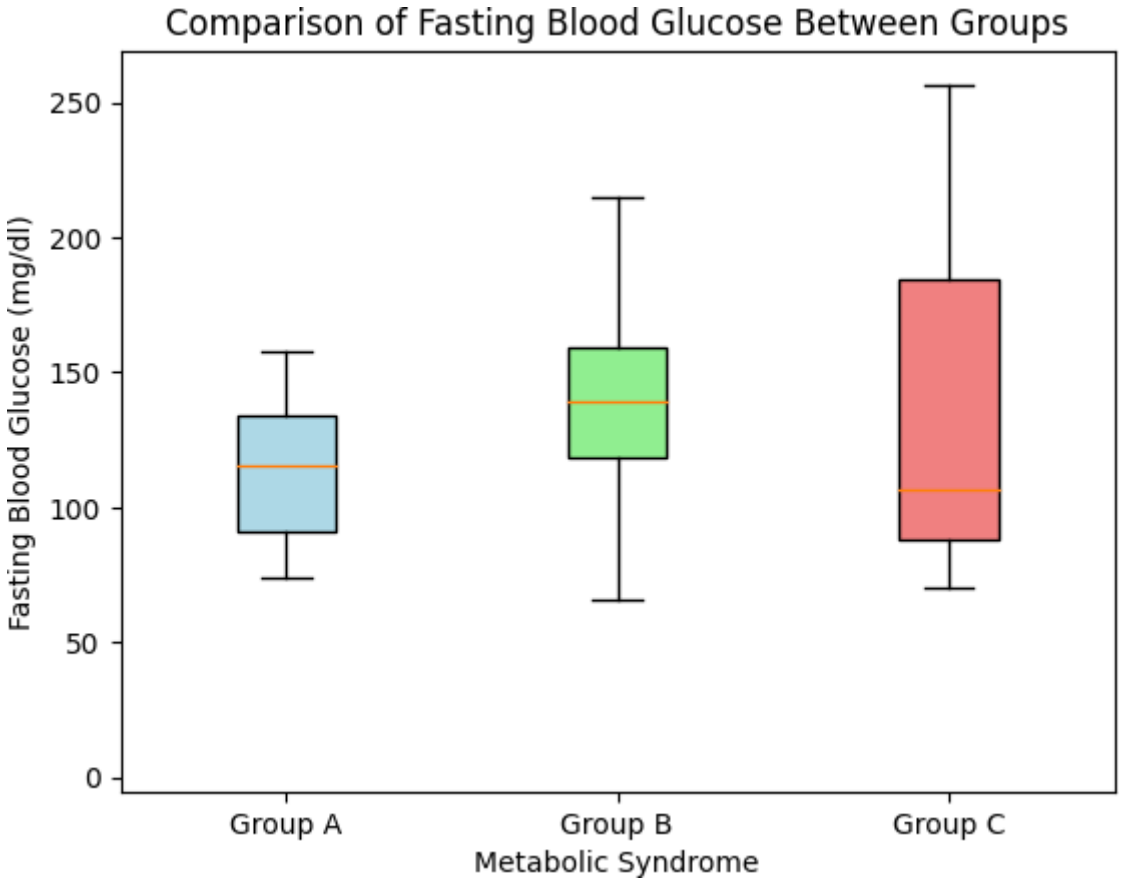

Fig 5.1.4: **Comparison of Fasting Blood Glucose among Groups A, B, and C.**

One way ANOVA was conducted to compare the mean Total Cholesterol level among groups. The mean of Group A is 197.38 ± 34.23, Group B is 195.4 ± 40.3and Group C is 187.3 ± 8.07. One-way ANOVA was used, the Total Cholesterol was not found to be significant, $F(2, 397) = 2.645$, $p = 0.072$.

One way ANOVA was conducted to compare the mean triglyceride level among groups. The mean of Group A is 202.14 ± 84.08, Group B is 206.08 ± 38.59and Group C is 240.73 ± 64.67. In comparison of groups one-way ANOVA was used, the triglyceride was found to be significant, $F(2, 397) = 3.041$, $p = 0.049$. Post hoc analysis showed there is no significant between groups.

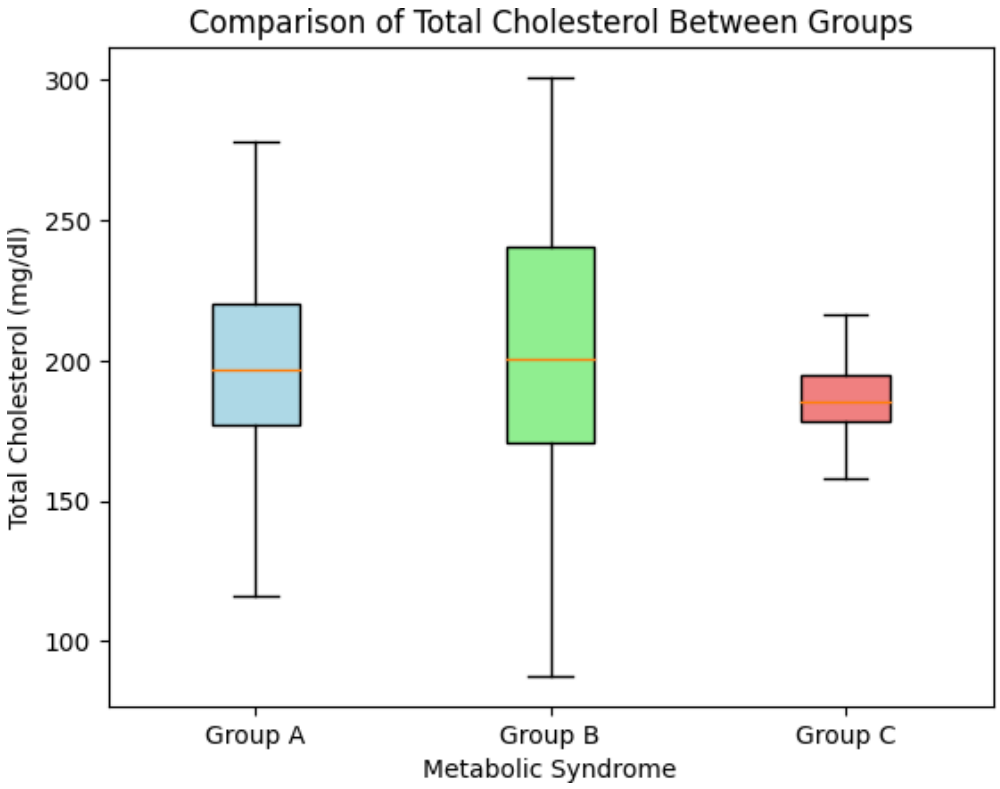

Fig 5.1.5:Comparison of Total Cholesterol among Groups A, B, and C.

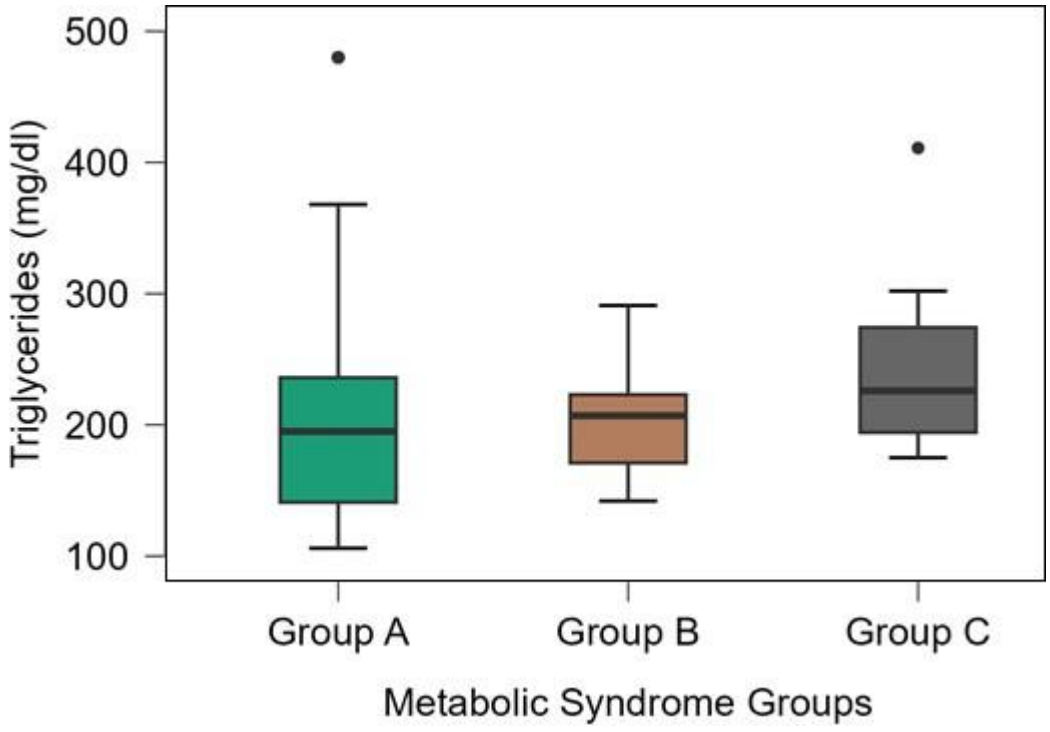

Fig 5.1.6: Comparison of Triglyceride among Groups A, B, and C.

One -way ANOVA was conducted to compare the groups mean levels of high-density lipoprotein. Group B's mean is 50.87 ± 5.63, Group C's mean is 40.67 ± 8.07, while Group A's mean is 50.95 ± 4.93. Between the groups, there was a significant difference, F (2, 397) = 45.963, $p < 0.001$. The results of the post-hoc study indicated a substantial relationship between groups A and B, A and C, and B and C.

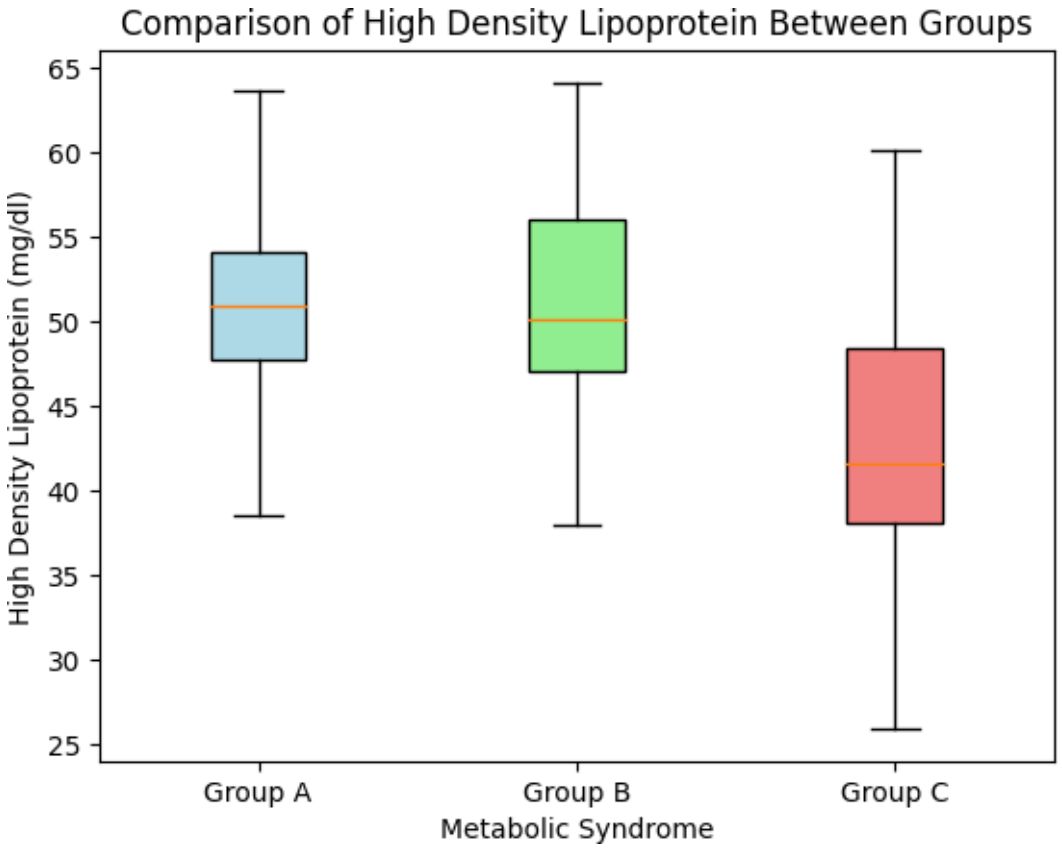

Fig 5.1.7: Comparison of High-Density Lipoprotein among Groups A, B, and C.

To compare the groups' mean levels of low density lipoprotein and very low density lipoprotein, one way ANOVA was used. Mean of low-density lipoprotein and very low density lipoprotein Group A is 104.64 ± 34.24 and 41.79 ± 9.85, Group B is 104.87 ± 32.11 and 39.67 ± 9.12, while Group C is 93.33 ± 17.20 and 53.33 ± 8.12 respectively. Between the groups, Low density lipoprotein was no significant difference, F (2, 397) = 2.565, p = 0.078, on the other hand very low-density lipoprotein showed mild significant difference, F (2, 397) = 3.026, p = 0.050. The results of the post-hoc analysis of low-density lipoprotein

and very low-density lipoprotein indicated no significant between groups A and B, A and C, and B and C.

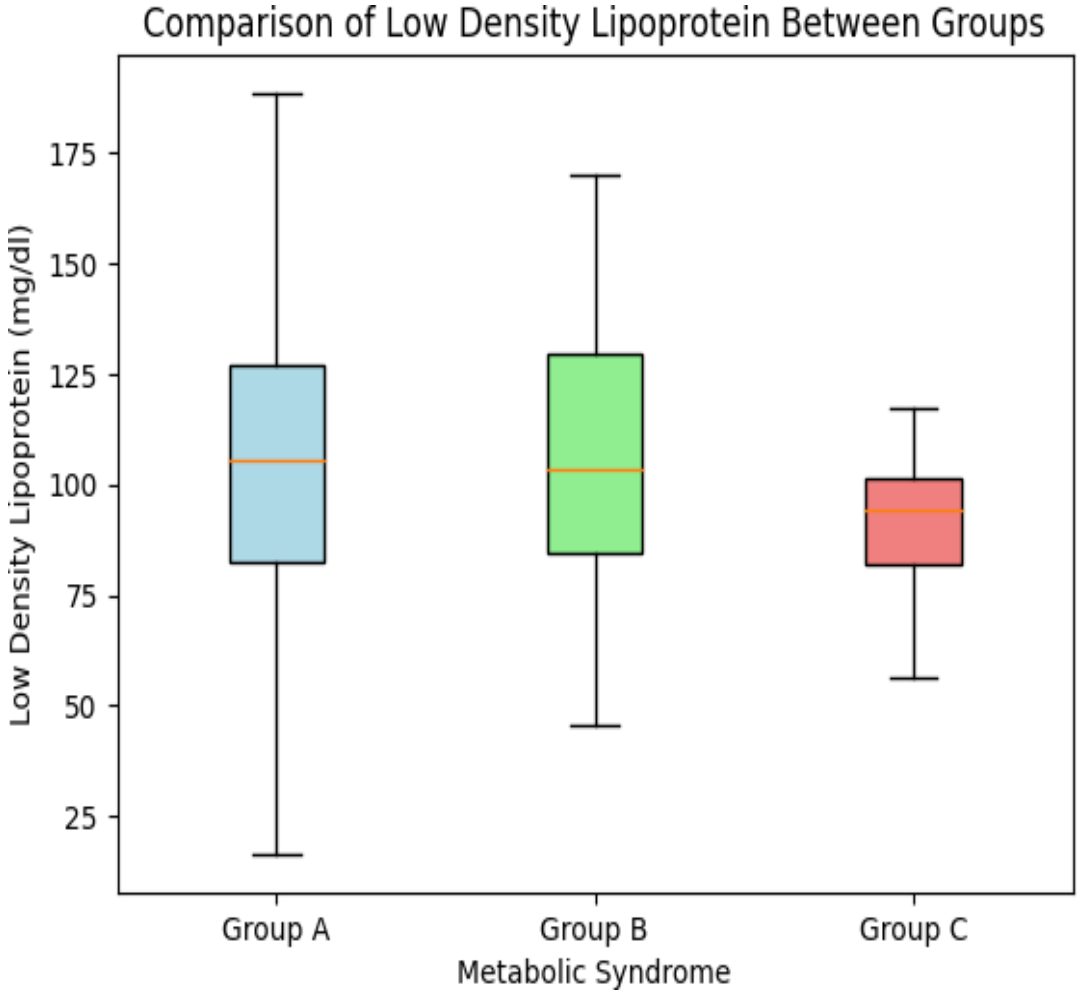

Fig 5.1,8: Comparison of Low-Density Lipoprotein among Groups

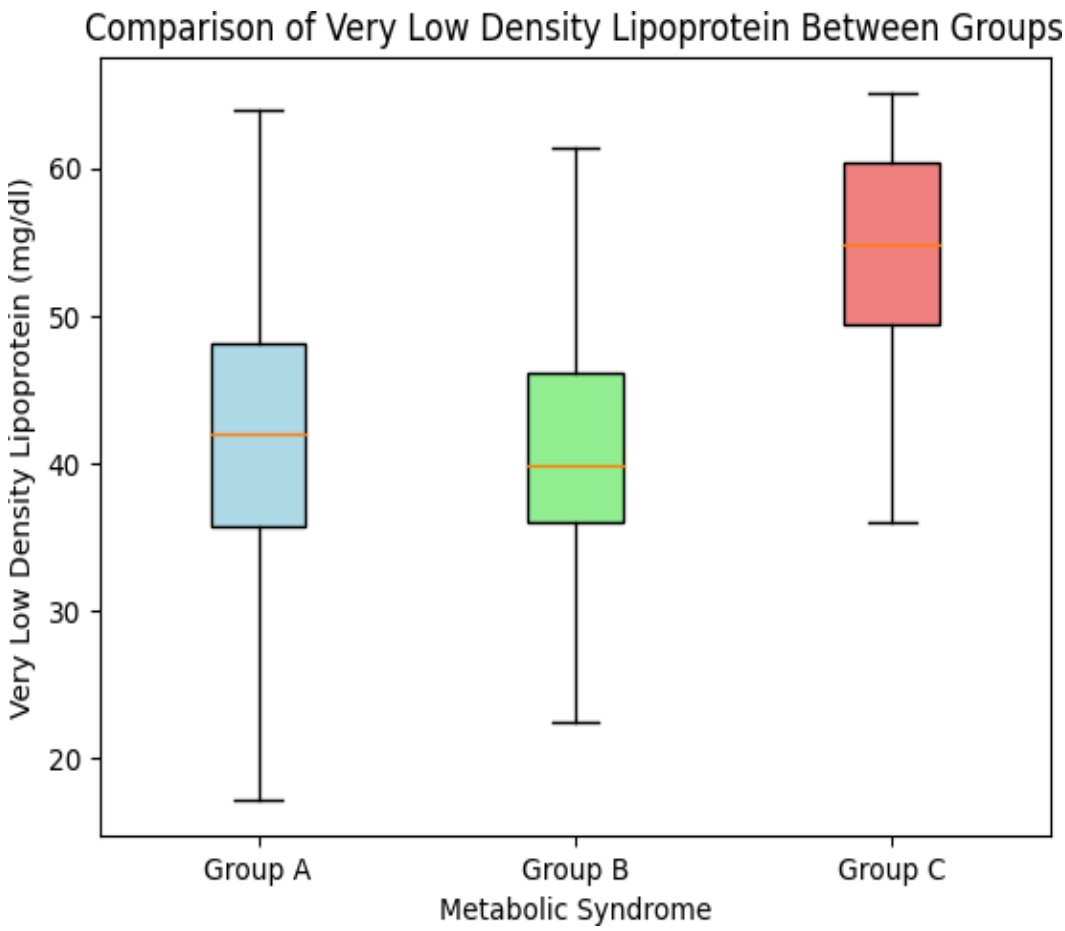

Fig 5.1.9: Comparison of Very Low-Density Lipoprotein among Groups A, B, and C.

Comparison of Renal Function Test among Groups

Mean serum Blood Urea Nitrogen levels among groups were compared by performing one way analysis. The mean serum blood urea nitrogen level of Group A is 12.80 ± 1.04, Group B is 12.29 ± 1.34 and Group C is 12.26 ± 1.47. Result indicates that there was a significant difference among the groups, $F(2, 397) = 8.091$, $p < 0.001$. Post hoc analysis revealed, Group A and B only showed significance.

Table 5.1.4 Comparison of Renal Function Tests among Groups A, B, and C.

Parameter	**Group A** **Mean** ± SD **n=281**	**Group B** **Mean** ± SD **n=96**	**Group C** **Mean** ± SD **n=23**	**p value**
Blood Urea Nitrogen (mg /dl)	12.80 ± 1.04	12.29 ± 1.34	12.26 ± 1.47	< 0.001
Serum Creatinine (mg/dl)	1.06 ± 0.12	1.01 ± 0.14	0.99 ± 0.23	0.001
Serum Urea (mg/dl)	29.03 ± 2.28	27.54 ± 3.16	28 ± 3.72	< 0.001
Serum Uric Acid (mg/dl)	5.65 ± 0.91	5.38 ± 1.7	5.55 ± 1.08	0.218

Note – p value 0.01 is significant, p value < 0.001 is highly significant.
Group a - With 3 out 5 criteria for MetS; Group b - With 4 out 5 criteria for MetS; Group c - With all 5 criteria for MetS.

One way ANOVA was conducted to compare the mean serum creatinine among groups. The mean of group A is 1.06 ± 0.12, group B is 1.01 ± 0.14and group C is 0.99 ± 0.23. A significant difference was found among the groups with $F(2, 397) = 8.112$, $p < 0.001$. Post hoc analysis showed there is significant between group A and B as well as group A and C, but there is no significant between group B and C.

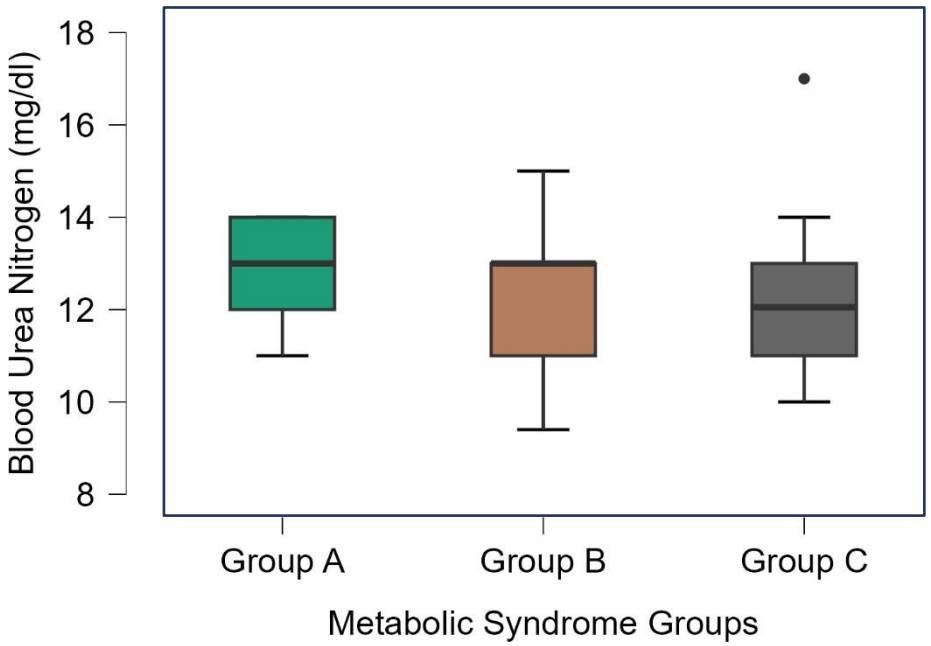

Fig 5.2.0: Comparison of Blood Urea Nitrogen among Groups A, B, and C.

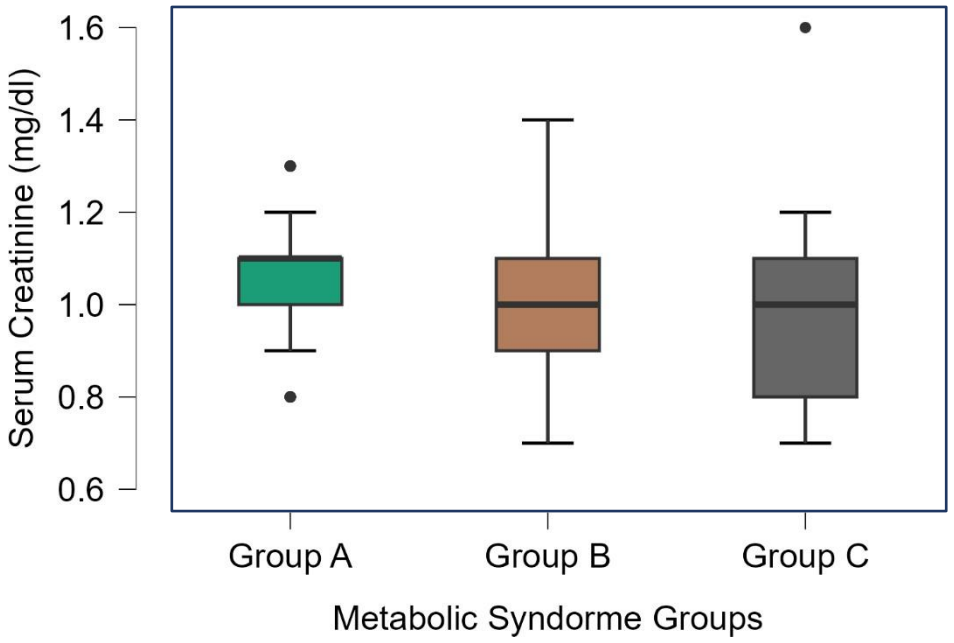

Fig.5.2.1 Comparison of Serum Creatinine among Groups A, B, and C.

One-way ANOVA was conducted to compare the groups' mean levels of serum urea. Group A's mean is 29.03 ± 2.28, Group B's mean is 27.54 ± 3.16, while Group C's mean is 28 ± 3.72. Between the groups, there was a significant difference, $F(2, 397) = 45.963$, $p < 0.001$. The results of the post-hoc study indicated a substantial relationship between groups A and B. Group A and C, and B and C didn't show relationship between them.

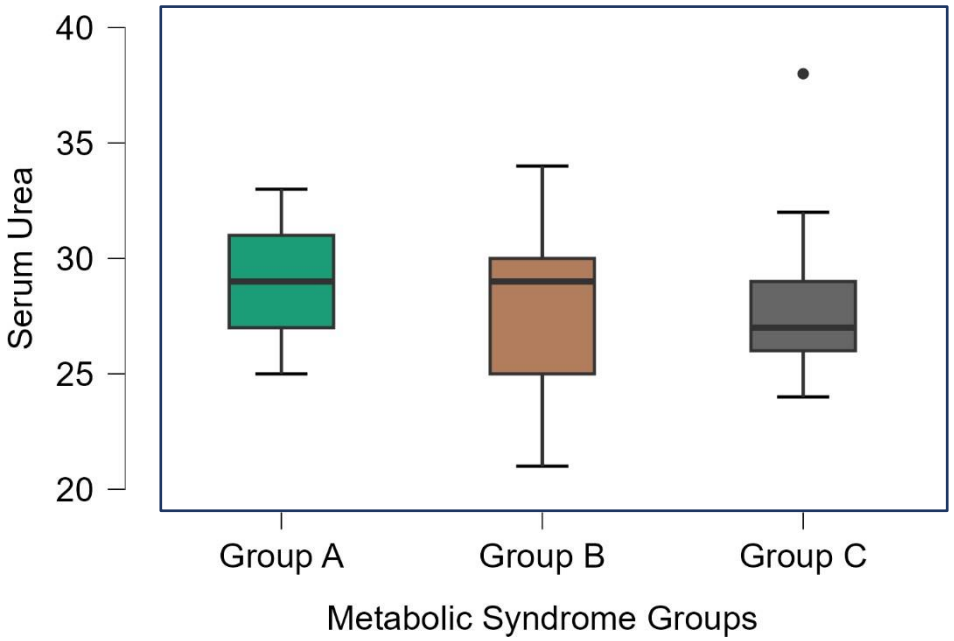

Fig.5.2.2 Comparison of Serum Urea among Groups A, B, and C.

One way ANOVA was conducted to compare the mean serum uric Acid level levels among groups. The mean uric acid levels of Group A is5.65 ± 0.91, Group B is 5.38 ± 1.7and Group C is 5.55 ± 1.08. In comparisonof groups one-way ANOVA was used, the uric acid was found have no significance among groups, $F(2, 397) = 1.529$, $p = 0.218$. Post hoc analysis also showed there is no significant between groups.

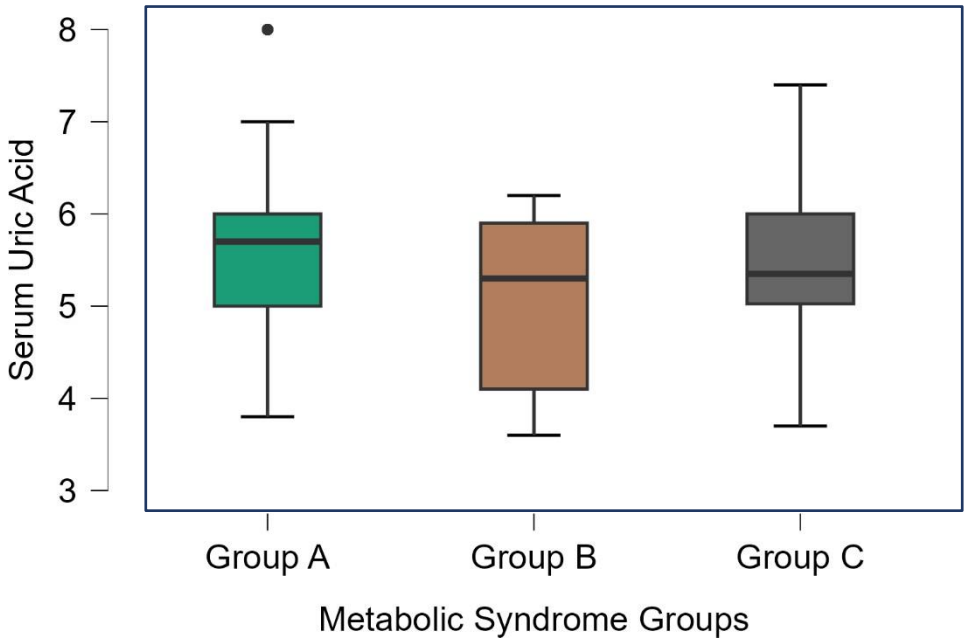

Fig.5.2.3 Comparison of Serum Uric Acid among Groups A, B, and C.

Comparison of Urinary Parameters among Groups

Table 5.1.5 Comparison of Renal Function tests among Groups A, B, and C.

Parameter	**Group A** **Mean** ± SD **n=281**	**Group B** **Mean** ± SD **n=96**	**Group C** **Mean** ± SD **n=23**	**p value**
Estimated Glomerular filtration rate (ml/min/1.73m^2)	72.59± 11.39	77.88 ± 12.14	75.33 ± 9.79	< 0.001
Urine Creatinine (mg/dl)	1.68 ± 0.94	2.28 ± 1.34	2.29 ± 0.99	< 0.001
Urine Albumin (mg/dl)	7.35 ± 3.44	9.59 ± 3.74	8.40 ± 4.29	< 0.001
Urine Albumin: Creatinine Ratio	4.81 ± 2.24	5.01 ± 2.39	3.84 ± 1.57	0.076

Note – p value 0.01 is significant, p value < 0.001 is highly significant.

One way ANOVA was conducted to compare the mean Estimated Glomerular filtration rate among groups. The mean of group A is 72.59± 11.39, group B is 77.88 ± 12.14and group C is 75.33 ± 9.79. A significant difference was found among the groups with F (2, 397) = 7.703, $p < 0.001$. Post hoc analysis showed there is significant between group A and C, but there is no significant between group A and C and group B and C.

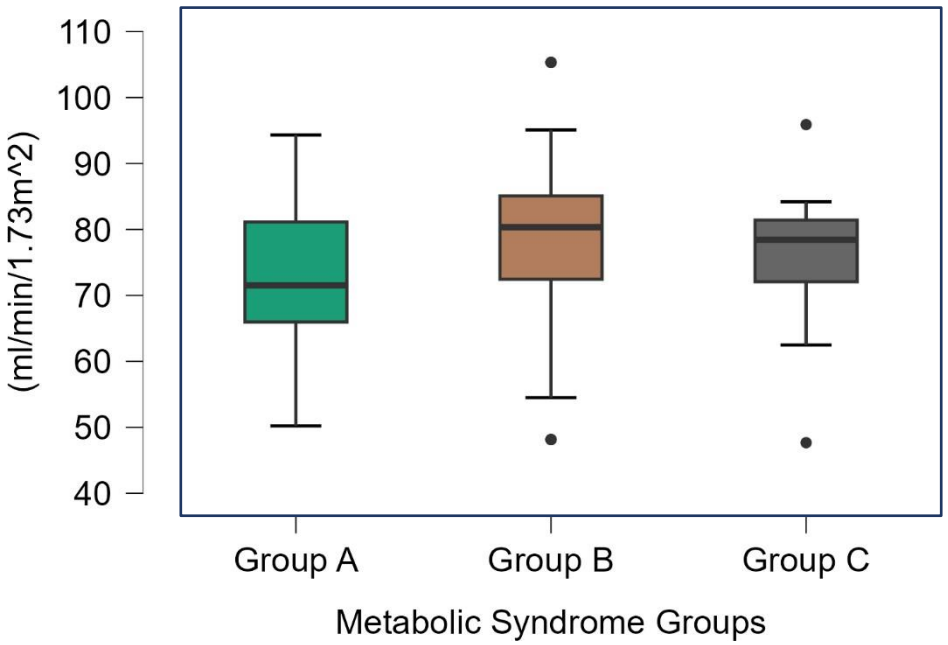

Fig. 5.2.4 Comparison of Estimated Glomerular Filtration among groups A, B, and C.

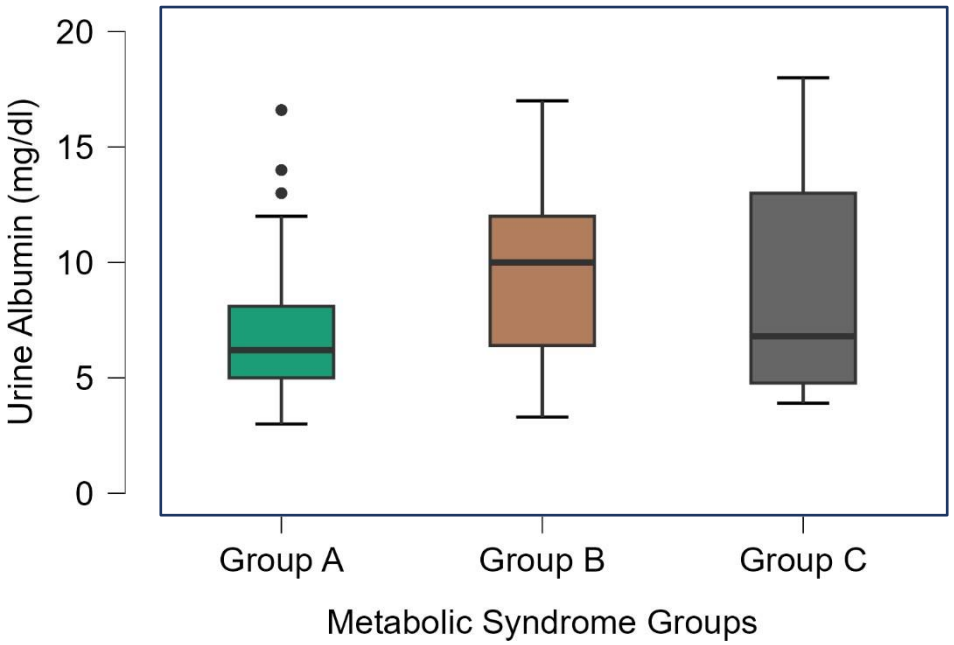

Fig.5.2.5 Comparison of Urine Albumin among Groups A, B, and C.

Mean serum Urine Albumin levels among groups were compared by performing one way analysis. The mean Urine Albumin level of Group A is 7.35 ± 3.44, Group B is 9.59 ± 3.74 and Group C is 8.40 ± 4.29. Result indicates that there was a significant difference among the groups, F (2, 397) = 14.325, p < 0.001. Post hoc analysis revealed, Group A and B only showed significance.

One-way ANOVA was conducted to compare the groups' mean levels of Urine Creatinine. Group A's mean is 1.68 ± 0.94, Group B's mean is 2.28 ± 1.34, while Group C's mean is 2.29 ± 0.99. Between the groups, there was a significant difference, F (2, 397) = 13.654, p < 0.001. The results of the post-doctoral study indicated a substantial relationship between groups A and B. Group A and C, and B and C didn't show relationship between them.

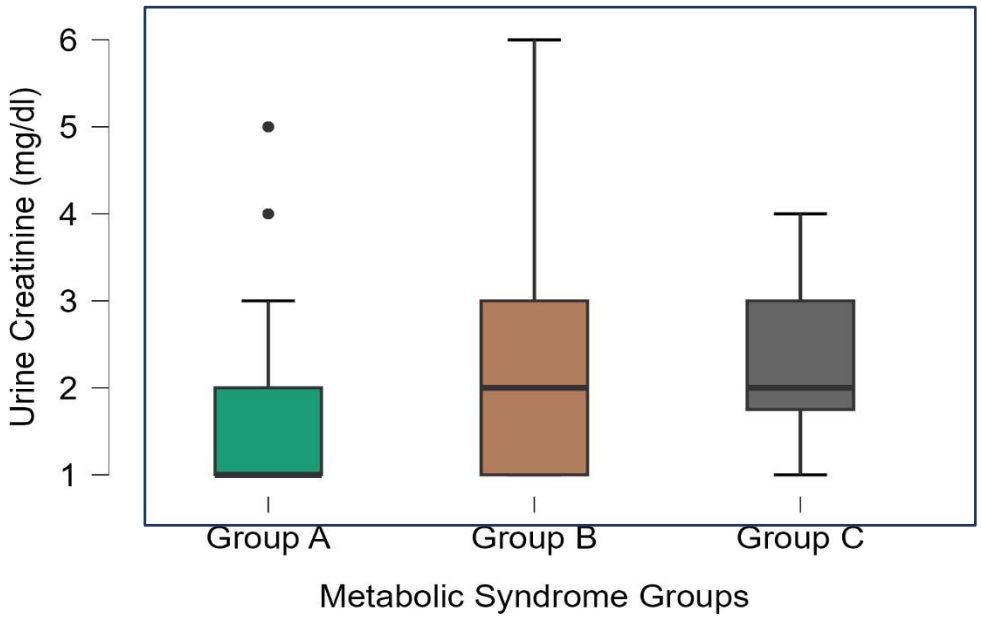

Fig.5.2.6 Comparison of Urine Creatinine among Groups A, B, and C.

One way ANOVA was conducted to compare the mean Urine albumin: creatinine ration among groups. The mean of group A is 4.81 ± 2.24, group B is 5.01 ± 2.39 and group C is 3.84 ± 1.57. No significant difference was found among the groups with F (2, 397) = 2.595, p = 0.076. Post hoc analysis did not show any significant difference between the groups.

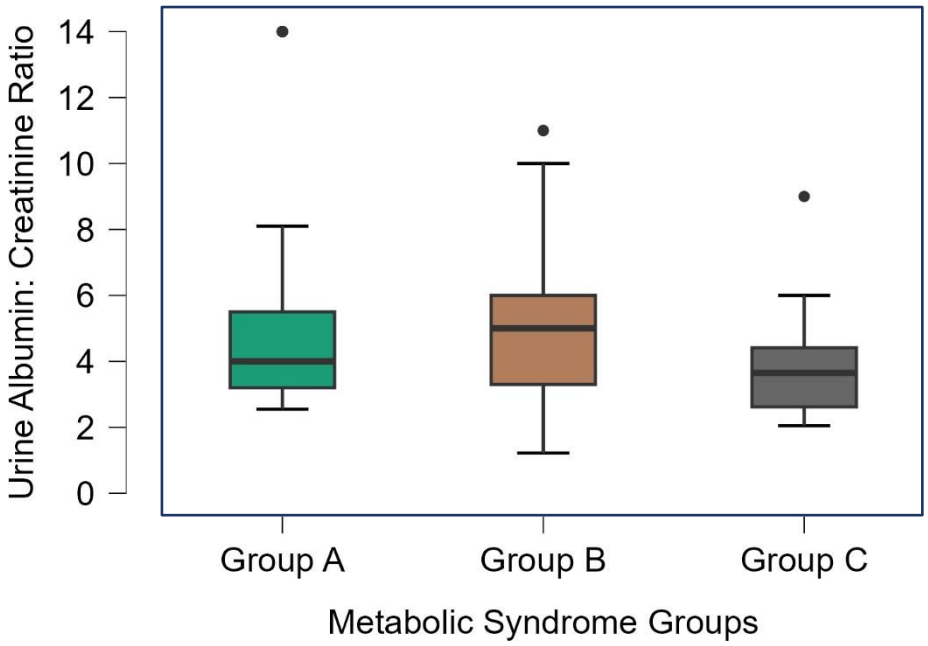

Fig.5.2.7 Comparison of Urine Albumin: Creatinine Ratio among Groups A, B, and C.

Association of Waist Circumference and Waist Hip Ratio with Anthropometric measurement and Biochemical Parameters

Present study aim to find association between waist circumference, waist hip ratio with anthropometric measure of blood pressure and various biochemical parameters. To assess the significance and direction of the association, a statistical analysis tool Pearson's correlation coefficient was employed.

Table. 5.1.6 Correlation of Waist Circumference and Waist Hip Ratio with Blood Pressure.

Variable	Waist Circumference		Waist Hip Ratio	
	Pearson's (r - Value)	p-value	Pearson's (r - Value)	p-value
Systolic Blood Pressure	0.312	< .001	-0.151	0.003
Diastolic Blood Pressure	-0.126	0.011	0.135	0.007

Positive correlation was found between waist circumference with systolic blood pressure with r = 0.312, p < .001.Weak negative correlation with diastolic blood pressure r = -0.126, p = .011. Waist hip ratio had a positive and weak negative correlation with systolic blood pressure (r = 0.381, p < .001) and diastolic blood pressure (r = -0.043, p = .39) respectively.

Table.5.1.7 Correlation of Waist Circumference and Waist Hip Ratio with Fasting Blood Glucose.

Variable	Waist Circumference		Waist Hip Ratio	
	Pearson's (r - Value)	p-value	Pearson's (r - Value)	p-value
Fasting Blood Glucose	0.208	< .001	0.038	0.445

Result showed fasting blood glucose had positive correlation with waist circumference, r = 0.208, p <0.001 and no significant correlation with waist hip ratio, r = 0.038, p = 0.445.

Table.5.1.8 Correlation of Waist Circumference and Waist Hip Ratio with Lipid Profile.

Variable	Waist Circumference		Waist Hip Ratio	
	Pearson's (r - Value)	p-value	Pearson's (r - Value)	p-value
Total Cholesterol	0.014	0.774	-0.251	<0.001
Triglyceride	0.12	0.016	-0.044	0.378
High Density Lipoprotein (HDL)	-0.286	< .001	-0.326	<0.001
Low Density Lipoprotein (LDL)	0.002	0.965	-0.233	<0.001
Very Low Density Lipoprotein (VLDL)	0.132	0.008	-0.016	0.754

Waist circumference showed a weak positive correlation with triglyceride (r = 0.120, p = 0.016) and very low density lipoprotein (r = 0.132, p = 0.008). But no significant correlation with total cholesterol and low density lipoprotein. With high density lipoprotein it showed a significant negative correlation.

Waist hip ratio showed negative correlation with total cholesterol (r = -0.251, p < .001), high density lipoprotein (r = -0.326, p < .001) and low density lipoprotein (r = -0.233, p < .001) as

depicted in the table.It didn’t show any significant correlation with lipid triglyceride(r = -0.044, p = 0.378) and VLDL (r = -0.016, p = 0.754).

Table 5.1.9: Correlation of Waist Circumference and Waist Hip Ratio with Renal Profile.

Variable	Waist Circumference		Waist Hip Ratio	
	Pearson's (r - Value)	p-value	Pearson's (r - Value)	p-value
Blood Urea Nitrogen	0.106	0.035	0.019	0.711
Serum Urea	0.107	0.032	0.029	0.656
Serum Creatinine	0.372	< .001	0.212	0.001
Serum Uric Acid	0.203	< .001	0.070	0.160

Waist circumference showed significant weak positive correlation with serum creatinine (r = 0.372, p < 0.001) and serum uric acid (r = 0.203, p < 0.001). But no correlation with Blood urea nitrogen and urea.

Serum creatinine alone had a significant weak positive correlation with waist hip ratio (r = 0.212. p < 0.001). Waist hip ratio was not significantly correlated with blood urea nitrogen (r = 0.019, p = 0.711), serum urea (r = 0.029, p = 0.656) and uric acid (r = 0.07, p = 0.16).

Table.5.2.0 Correlation of Waist Circumference and Waist Hip Ratio with Renal Profile.

Variable	Waist Circumference		Waist Hip Ratio	
	Pearson's (r - Value)	p-value	Pearson's (r - Value)	p-value
Urine Albumin	0.187	< .001	0.172	< .001
Urine Creatinine	0.117	0.02	0.128	0.011
Urine Albumin: Creatinine ratio	0.137	0.006	0.086	0.085
Estimated glomerular filtration rate	0.253	< .001	0.352	< .001

With waist circumference, estimated glomerular filtration rate showed a significant weak positive correlation ($r = 0.253$, $p < 0.001$). Other parameter urine albumin ($r = 0.187$, $p < 0.001$), urine creatinine ($r = 0.117$, $p = 0.02$) and urine albumin creatinine ratio ($r = 0.137$, $p = 0.006$) showed significant but there was no correlation. Similarly, estimated glomerular filtration rate alone showed positive correlation with waist hip ratio ($r = 0.352$, $p < 0.001$), other parameter such as urine albumin ($r = 0.172$, $p < 0.001$), urine creatinine ($r = 0.128$, $p = 0.011$) and urine albumin creatinine ratio ($r = 0.086$, $p = 0.085$) didn't show any correlation.

CHAPTER-VI
DISCUSSION

CHAPTER-VI

Discussion

DISCUSSION

Metabolic syndrome is a combination of pathophysiological conditions such as obesity, insulin resistance, hypertension, and dyslipidemia, which together increase the risk of cardiovascular disease and diabetes (85).Metabolic syndrome with global prevalence of 20 - 25 % in adult population, it increases the risk of causing Type 2 diabetes mellitus and cardiovascular diseases along with renal dysfunction (86). As India being the diabetic capital of the world, it has high prevalence of metabolic syndrome almost one-third of its adult population (87).

Present study showed overall of 34.6% of adults in study population were found to be metabolic syndrome. A study conducted in West Bengal reported a prevalence rate of 32.75% (88). In eastern part of India also recorded a similar prevalence rate of 33.5% metabolic syndrome (89). Study have documented increased prevalence of metabolic syndrome seen in female population (90). But in our study, we had a higher prevalence among males, indicating a potential shift in risk factors. Study done on Mexican Americans using APT III criteria for diagnosing metabolic syndrome documented a high prevalence 78% in male when compared with female (91).

One of the major risk factors for metabolic syndrome is central obesity (92). An anthropometric measurement which can be assessed using waist circumference and waist hip ratio (93). Waist hip ratio is the common indicator for deposition of abdominal fat and help in the evaluation of distribution of visceral fat in the body (94). Accumulation of abdominal fat will increase the risk of cardiovascular events in adults with metabolic syndrome (95). Studies have shown waist hip ratio is a predicator of metabolic syndrome in Indian population (96) In this study, we have evaluated waist hip ratio with renal dysfunction in metabolic syndrome and its correlation with various

renal parameters. Our reports suggested association between elevated waist hip ratio with impaired renal function in metabolic syndrome. A positive correlation between waist hip ratio and microalbumin in individual having central obesity is reported(97).

This study further emphasizes the importance of monitoring waist hip ratio as a potential indicator for renal health in patients with metabolic syndrome. A positive association between waist hip ratio and renal dysfunction in patient with metabolic syndrome is observed (98). Previous research indicate that increased waist hip ratio is associated with increase in inflammatory response, which can contribute to renal damage. A significant decrease in eGFR and increase in albuminuria in metabolic syndrome is reported(99).

. In metabolic syndrome, central obesity plays a vital role and itis the key factor for hyperlipidemia. Both central obesity and hyperlipidemia are the central axis in the pathogenesis of metabolic syndrome, This might lead to vicious cycle on the renal impairment. Several studies in the past have discussed the significant increase in the lipid profile in metabolic syndrome when compared with health control. A significant change in the lipid parameters among metabolic syndrome patients, who were categorized based on the NECP ATP III guidelines is highlighted. (100). In our study we have documented, there was significant different seen in triglyceride, high density lipoprotein and very low-density lipoprotein but no significant changes in total cholesterol and low-density lipoprotein among metabolic syndrome-sub-groups. A similar finding was reported along with notable variation in fasting blood glucose among metabolic syndrome subjects(101).

Urea, blood urea nitrogen, creatinine and uric acid levels were used to assess the renal dysfunction in metabolic syndrome. Our results indicated that this parameter showed significant difference among metabolic syndrome except uric acid. Increased levels of urea and blood urea nitrogen has direct impact on the renal function, suggesting a potential link between metabolic syndrome and kidney impairment. An increase in the level of blood urea nitrogen associated with reduce renal clearance in patient with metabolic syndrome is observed(102).High concentration of urea induces insulin resistance and oxidative stress which are potential risk factor for cardiovascular complication and renal impairment in metabolic syndrome(103). Even literatures have documented genetic factors associated with blood urea nitrogen and renal outcome(104). Accumulation of blood urea nitrogen and urea stimulate endothelin activity results in vascular dysfunction which help us to understand the cause of renal impairment in metabolic syndrome.

Serum creatinine is one routine biochemical marker used to assess the renal health. In metabolic syndrome also creatinine plays a vital role in evaluating renal function. Risk factors of metabolic syndrome include obesity, insulin resistance, hypertension and hyperlipidemia were known to be a potential risk factor for chronic kidney disease (105). Current study reported creatinine levels had significant difference among metabolic syndrome patients but the levels are remained in the normal range. Creatinine level within the normal range but has significant association with risk of metabolic syndrome is documented (106). Metabolic syndrome patient presenting with diabetes and albuminuria has high risk of developing CKD (99).

Uric Acid is end product metabolite of purine metabolism. It has significant role in the evaluation of renal function and cardiovascular diseases. Estimation of uric acid levels

in metabolic syndrome patients help clinicians to assess both renal and cardiac health. Hyperuricemia is often associated with gout, nephrolithiasis and metabolic syndrome. Present study didn't show any significant difference in the uric acid level in metabolic syndrome patients and levels are found to be with the normal range. However, in the past, literatures have documented strong association between uric acid with the risk factor of metabolic syndrome such as central obesity, hyperlipidemia, and hypertension and insulin resistance. Hyperuricemia in metabolic syndrome may result in decreased excretion of uric acid and cause endothelial dysfunction which lead to renal damage and cardiovascular disorders(107). Increased uric acid level is associated with renal and cardiac outcome in non-metabolic syndrome patients (108). Studies indicate that uric acid can be used in predicting pre metabolic syndrome (109). Our result showed a positive correlation between waist hip ratio and uric acid levels. A positive correlation between uric acid level , BMI and waist hip ratio is reported. (110)

Present study was focused on the renal impairment in metabolic syndrome. One of the novel markers to assess the renal function is estimated glomerular filtration rate (eGFR)(111). Result showed there was decreased eGFR in metabolic syndrome when compare with the reference range. Renal impairment and metabolic syndrome share common pathophysiological mechanisms that includes central obesity, hypertension, hyperlipidemia and insulin resistance(105,). Risk of developing decreased eGFR in metabolic syndrome patient increases by an odds of 1.76 and the association of blood pressure with decreased eGFR in metabolic syndrome is documented (112). The association of dyslipidemia with decrease eGFR in metabolic syndrome and eGFR is an independent marker for renal impairment in metabolic syndrome(113). Along with eGFR, urine albumin, urine creatinine and urine albumin creatinine ratio also were

analyzed in this study. These parameters also found to have significant association with renal impairment. Both urine albumin and creatinine are well established marker in the assessment of renal function. Urine albumin will help to assess the renal injury and prognosis of the CKD(105). Urine creatinine will be useful to identify the percentage of renal function(114). Urine albumin: creatinine ratio asses both renal and cardiovascular functions (115). The present study shows a significant increase in the excretion of urine albumin and urine creatinine in metabolic syndrome subjects. But their ratio was found no significant difference among metabolic syndrome. Studies in past document indicate that an increased excretion of urine albumin and creatinine in various populations and can be used for evaluation of renal function (116). These parameters are associated with major pathophysiology of metabolic syndrome. The urine albumin creatinine ratio can be used to assess the micro vascular function (115).

In summary, the data we analyzed and from other findings, we conclude that monitoring these urinary biomarkers is crucial for early detection and management of renal impairment in individuals with metabolic syndrome.

CHAPTER-VII

CONCLUSIONS

CHAPTER-VII

Conclusions

CONCLUSIONS

Our study indicates the association between waist hip ratio and renal impairment in metabolic syndrome persons**.** A significant association between biochemical parameter of fasting blood glucose, lipid profile and renal profile including urinary parameters among metabolic syndrome patients is indicated.

Overall, our study highlights the critical role of these renal markers in the pathophysiology of metabolic syndrome. Monitoring eGFR, Serum urea and creatinine along with urinary albumin creatinine ratio could provide valuable insights for early diagnosis, risk stratification, and management of individuals at risk of renal impairment in metabolic syndrome.

CHAPTER-VIII

REFERENCES

CHAPTER-VIII

References

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CHAPTER-IX
RECOMMENDATIONS

CHAPTER – IX

Recommendations

Recommendations

- A significant association exists between elevated waist hip ratio and impaired renal function in metabolic syndrome patients.

- Our study further emphasizes the importance of monitoring waist hip ratio as a potential indicator for renal health in patients with metabolic syndrome.

- Increased levels of urea and blood urea nitrogen has direct impact on the renal function.

- Our results showed decreased eGFR in metabolic syndrome when compared with reference range.

- Both urine albumin and urine creatinine are well established marker in the assessment of renal function.

- However, research work in this area with more sample size is required.

- Limitation - A large number of participants for screening is required in specific regions.

ANNEXURES

ANNEXURE- II

LIST OF PUBLICATIONS

1) Original Article

Title- 'Identifying the Association of Prediabetes and Cardiovascular Diseases with Metabolic Syndrome Patients in India'

Authors name- Ivon Lewis[1], Dr. A.A. Joshi*[2], Dr. Shimpa Sharma[3], Prashanth Kumar Goudappala[4]

Journal name - Journal of Dutta Meghe Institute of Medical Sciences

University www.journaldmims.com(IP:182.65.93.121) (2023:18:30-3)

2) Original Article

Title – 'Exploring the Relationship Between Metabolic Syndrome and Renal Dysfunction in a Hospital -Based Indian Cohort'

Authors Name: - Ivon Lewis[1], Dr. A.A. Joshi*[2] Dr. Shimpa Sharma[3,] Dr. Asha kumari[4] Dr. Preethika A[5], Dr. Prashant kumar Goudappala[6]

Journal name- African Journal of Biomedical Research

ISSN:1119-5096 Date- September 2024

ORAL PRESENTATION

No.	Conference	State/National/International	Date	Organized by
1	**BMSeCON 2023**	**International**	**14/12/2023**	**Aarupadai Veedu Medical College, Puducherry, India**

POSTER PRESENTATION

No.	conference	State/national/international	Date	Organized by
1	**International Conference on Radiation Biology**	**International**	**24/11/2023**	**K.S.Hegde Medical Academy, Mangaluru, Karnataka**

CME

1) Techno Cognito Physiologia, conducted by Physiology Department, FMMC, Mangalore on 24/10/2024.

2) Cardiopulmonary Resuscitation, conducted by Department of Anesthesiology, KIMS, Karwar on 01/09/2018

3) Laboratory Errors in Medical Practice, conducted by Biochemistry Department, KIMS, Karwar on 25/11/2017.

NITTE | K S HEGDE MEDICAL ACADEMY

ICRB 2023

INTERNATIONAL CONFERENCE ON RADIATION BIOLOGY
(UNDER THE AUSPICES OF INDIAN SOCIETY OF RADIATION BIOLOGY)

CERTIFICATE OF PARTICIPATION

This is to certify that

MR. IVON FRANCIS LEWIS

has participated as a Poster Presenter / ~~Oral Presenter~~
on 23rd Nov to 25th Nov 2023
organized by K S Hegde Medical Academy, Nitte (Deemed to be University).

Dr. Madhu Bala	Dr. P S Prakash	Organizing Secretary
President, ISRB	Convener	ICRB - 2023
	ICRB - 2023	

VINAYAKA MISSION'S RESEARCH FOUNDATION
(Deemed to be University under section 3 of the UGC Act 1956)

AVMC
AARUPADAI VEEDU MEDICAL COLLEGE

BMSeCON 2023

4th International e-conference on

"Beyond Boundaries: Exploring Excellence in Basic Medical Sciences"

Tamil Nadu Medical Council
6 Credit Hours

Certificate of Appreciation

This certificate is presented to

Mr Ivon Francis Lewis

for making an Oral Presentation (Ph.D) for his/her paper titled "**Metabolic Syndrome-Early Diagnostic Biomarker**" in **BMSeCON-2023**, organized by the Departments of Anatomy, Biochemistry, Physiology, and Center for Biomedical Research, AVMCH, Puducherry, India held from 13th to 15th December, 2023.

Dr. M. Manju	Dr. Ilankathir S	Dr. Gopinath M	Dr. Rakesh Sehgal
Organizing Secretary	Organizing Chairperson	Organizing Chairperson	Dean, AVMC

Father Muller Medical College

(A Unit of Father Muller Charitable Institutions)
Kankanady, Mangalore-575002

DEPARTMENT OF PHYSIOLOGY

Certificate

TECHKNOW COGNITO PHYSIOLOGIA

Decoding Physiology of Learning in Digital Era

This is to certify that

Mr. Ivon Francis Lewis,

Assistant Professor, G R Medical College & Research Center,
has attended the CME conducted at
Father Muller Medical College, Mangalore on 24th October 2024.

Dr Neetha Shastry	Dr Jnaneshwara P Shenoy	Dr Antony Sylvan Dsouza	Rev Fr. Ajith B Menezes
Organising Secretary	Organising Chairperson	Dean, FMMC	Administrator, FMMC

CME CERTIFICATE

Government of Karnataka

Karwar Institute of Medical Sciences, Karwar

(An Autonomous Institution)

Department of Biochemistry

CME On

"Laboratory Errors in Medical Practice"

This is to certify that Mr. IVON FRANCIS LEWIS bearing Reg. No. _____

registered with Karnataka Medical Council, Address KaIMS Karwar has participated as DELEGATE in CME held on 26th December 2017. Karnataka Medical Council has granted TWO credit hours

Vide Letter No. : KMC/CME/609/2017, dated : 25-11-2017.

Dr. Poornima R. T.	Dr. B. N. Malawadi	Dr. Kanchi Prahlad V.	Dr. Shivanand M. Dodamani
Organizing Chairperson	Organizing Secretary	Vice-President KMC, CME Accreditation Committee	Dean & Director, KaIMS, Karwar

Government of Karnataka

Karwar Institute of Medical Sciences, Karwar

(DEPARTMENT OF ANAESTHESIOLOGY)

CME CERTIFICATE

This is to certify that Mr. Ivon Francis Lewis, address KRIMS, Karwar has participated as delegate in the "CARDIO PULMONARY RESUSCITATION" CME held on 29.09.2018. Karnataka Medical Council has granted 2 credit hours vide letter no. K.M.C./C.M.E./37/2018 dated 04/09/2018.

Dr. Manjunath T. Bhat	Dr. Bharath Kumar Hegde	Dr. Kaveri Prahlad V.	Dr. Shivanand M. Dodamani
Org. Chairperson	Org. Secretary	K.M.C. Vice-President	Dean & Director

D. Y. PATIL MEDICAL COLLEGE, KOLHAPUR

Constituent College of D.Y.Patil Education Society Deemed University, Kolhapur

NAAC Accrediated 'A' Grade

Dr. Rakesh Kumar Sharma
Dean & Professor (Obst. & Gyn.)

Padmshree Dr. D. Y. Patil
Founder President

Dr. Sanjay D. Patil
President

Outward No. DMCK/85/2017

Date :
14 MAR 2017

INSTITUTIONAL ETHICS COMMITTEE, D. Y. PATIL MEDICAL COLLEGE, KOLHAPUR.

This is to certify that the research project titled,

"A Study of Correlation Between Metabolic Syndrome with Kidney Function Tests."

Submitted by : **Dr. Lewis Ivon Francis**

Under the supervision of appointed Guide (if any): **Dr. A. A. Joshi**

Has been studied by the Institutional Ethics Committee (IEC) at its meeting held on **14/03/2017** and granted approval for the study with due effect with the following caveats:

1. If you desire any change in the protocol or standard recording document at any time, please submit the same to the IEC for information and approval before the change is implemented.
2. All serious and/or unexpected adverse events due to the drug/procedures tested in the study must be informed to the IEC within 24 hours and steps for appropriate treatment must be immediately instituted.
3. In case of injury/disability/death of any participant attributable to the drug/procedure under study, all compensation is to be made by the sponsor of the study.
4. The Chief investigator/Researcher must inform the IEC immediately if the study is terminated earlier than planned with the reasons for the same.
5. The final results of the study must be communicated to the IEC within 3 months of the completion of data collection.
6. The researcher must take all precautions to safeguard the rights, safety, dignity and wellbeing of the participants in the study.
7. The researcher must be up to date about all information regarding the risk/benefit ratio of any drug/procedure being used and any new information must be conveyed to the IEC immediately. The IEC reserves the right to change a decision on the project in the light of any new knowledge.
8. Before publishing the results of the study, the researcher must take permission from the Dean of the Institution.
9. Annual progress report should be submitted for all sponsored projects to the committee.
10. Unethical conduct of research in non-sponsored projects will result in withdrawal of the ethics approval and negation of all data collected till that date.

Dr. Mrs. Shimpa R. Sharma
(Member Secretary, IEC)

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(DRAFT COPY)

Original Article

Identifying the Association of Prediabetes and Cardiovascular Diseases with Metabolic Syndrome Patients in India

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Abstract

Background: Increased urbanization decreases physical activity which promotes many life-threatening diseases among people in developing countries like India. Metabolic syndrome increases the health burden and mortality rate among middle-aged people. Early diagnosis of the risk factor associated with this pathological disorder can be a help to overcome this life-threatening pathological disorder by delaying disease progression. **Materials and Methods:** A total of 400 patients with metabolic syndrome are identified. Patient blood samples were collected and processed. Biochemical parameters include fasting blood sugar and lipid profiles which include TC, HDL, LDL very-LDL (VLDL), and triglycerides were analyzed using an autoanalyzer. Altered fasting blood sugar level and lipid profile are used to diagnose the status of diabetes and cardiovascular disorder. **Results:** The fasting blood sugar levels of the patients were in the range of prediabetic condition and so the patients are more prevalent to diabetes mellitus. An altered lipid profile ratio with a decreased level of good cholesterol and increased level of bad cholesterol was also observed in association with prediabetic condition. **Conclusion:** The altered levels of these biochemical parameters in metabolic syndrome patients gives the idea about its association of prediabetes with cardiovascular disorders among the Indian population.

Keywords: Cardiovascular diseases, diabetes, lipid profile, metabolic syndrome

Introduction

Metabolic syndrome is a cluster of metabolic disorders which affects the normal pathological state by alteration in the metabolic levels of lipids and glucose with increased arterial blood pressure.[1] Abnormal in their physical and biochemical parameters alters the normal cellular pathophysiological functions, which increases mortality by its health burden. Approximately one-third of the adult global population can be affected by metabolic syndrome with varying definitions. Over the past few years, metabolic syndrome has close association with human life threatening diseases such as cardiovascular diseases, arterial hypertension worldwide. In India, both cities and towns have increased the prevalence of this life-threatening disease due to increased urbanization.[2] Although the association of metabolic syndrome with many cardiovascular diseases occur due to dyslipidemia and arterial hypertension. Lipids are one of the major biomolecules that provide essential energy and act as building blocks of the cell. Human cells preserve lipids in the form of neutral lipids for their physiological function. Deregulation in lipid metabolism increases the risk of many pathological diseases.[3]

Dyslipidemia is a well-known risk factor of coronary heart disease where increased low-density lipoprotein (LDL), total cholesterol (TC), triglycerides and decreased high-density lipoprotein (HDL) were noted.[4] In addition to dyslipidemia, hypertension is another chief risk factor for cardiovascular disease, which accounts for about 80% of total cardiovascular deaths.[5] This altered serum lipid ratio is closely associated with increased blood pressure and other diseases such as diabetes which is mediated by insulin resistance which affects

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Research Article

Exploring The Relationship Between Metabolic Syndrome And Renal Dysfunction In A Hospital-Based Indian Cohort

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ABSTRACT

Background: Metabolic Syndrome (MetS) is a cluster of conditions that increase the risk of cardiovascular disease and other health problems. This retrospective study aims to investigate the relationship between MetS components and renal function parameters in a hospital based cohort.

Methods: The National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) recommendations classified 300 people aged 35–65 as having or not having MetS. Renal function, lipids, and glucose were measured. Chi-square tests and one-way ANOVA were used to examine MetS and renal function.

Results: MetS was found in 40% of study participants. Age group significantly correlated with MetS status ($\chi2$ = 23.31, p = 0.001), with higher prevalence in older age groups. There was no significant correlation between gender and MetS status ($\chi2$ = 0.795, p = 0.373). FBS levels differed considerably across groups (p < 0.001), while HDL levels were negatively linked with MetS components (p < 0.001). Both serum creatinine and urine albumin levels were considerably increased in patients with more MetS components (p = 0.001 and p < 0.001, respectively), suggesting renal impairment.

Conclusion: This emphasises the importance of early MetS diagnosis and treatment to prevent kidney problems. Future research should focus longitudinal analyses to better understand MetS-renal health cause-and-effect links.

Keywords: Metabolic syndrome, MetS, renal function, NCEP ATP III, diabetes, cardiovascular disease.

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residing at .....

do hereby confirm that:

(i) I have been asked by the student/researcher of D. Y. Patil Medical College/Centre for Interdisciplinary Research/ D. Y. Patil College of Pharmacy/ D. Y. Patil College of Physiotherapy, Kolhapur whether I wish to participate in a study under the aegis of the Medical College.

(ii) The nature of the study being undertaken by the student/ researcher, as well as the extent of my participation in it, have been duly explained to me in a language that I understand;

(iii) The potential risks and consequences associated with this study have also been duly explained to me in a language that I understand;

(iv) I also understand that my participation in this study is only for the benefit of advancement in the field of medical research and that at no point in time is my participation being solicited for any pecuniary gain by the researcher or the D. Y. Patil Medical College/Centre for Interdisciplinary Research/ D. Y. Patil College of Pharmacy/ D. Y. Patil College of Physiotherapy, Kolhapur.

(v) I have also been explained that I am in no way obliged to participate in the study and that, once I have agreed to participate in the study, I am still free to withdraw from participation in the study at any point in time upon notifying the D. Y. Patil Medical College/Centre for Interdisciplinary Research/ D. Y. Patil College of Pharmacy/ D. Y. Patil College of Physiotherapy, Kolhapur in writing in the prescribed form without assigning any reason;

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(vii) I have been explained that any data collected out of my participation in the study will only be used for academic purposes and/or for further medical research;

(viii) I have also been reassured that any publication of the data collected during the course of the study or any publication of its conclusions, shall be done on a ‘no names’ basis and

shall under no circumstances reveal my personal identity. Any personal details likely to reveal my personal identity shall at all times remain confidential;

(ix) The contents and effect of this consent form have also been duly explained to me in a language that I understand;

By affixing my signature/thumb print hereto, I am therefore freely and voluntarily signifying my consent, intent and willingness to participate in the study of the student researcher for the purposes of the postgraduate dissertation under the egis of the D. Y. Patil Medical College/Centre for Interdisciplinary Research/ D. Y. Patil College of Pharmacy/ D. Y. Patil College of Physiotherapy, Kolhapur. I also certify that my right to privacy has not been infringed in any manner.

[SIGNATURE/THUMB PRINT OF PARTICIPANT]
DATE:

WITNESSED BY:
(1) NAME:

TITLE/CAPACITY:
SIGNATURE:
(2) NAME:

TITLE/CAPACITY:
SIGNATURE:

Demographic Parameter Form

Name		
Age		
Gender		
Address		
Occupation		
Education		
Marital status		
Contact number		
Whether the individual has history of following conditions. Please tick the appropriate option.		
Hypothyroidism	**Yes**	**No**
Ascites	**Yes**	**No**
Malignancies	**Yes**	**No**
Steroids,	**Yes**	**No**
Familial Dyslipidemia	**Yes**	**No**
Secondary Hypertension	**Yes**	**No**