"STUDY OF MICROALBUMINURIA IN OBESE INDIVIDUALS AS AN EARLY INDICATOR OF NEPHROPATHY."

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ABSTRACT

Background: Obesity is becoming more widely recognized as risk factor for renal illness, although the precise prevalence of microalbuminuria, especially when other risk factors are taken into consideration, remains unknown. The current study is undertaken to correlate microalbuminuria with severity of obesity based on BMI.

Methods: A cross-sectional study on individuals attending R. L Jalappa Hospital from January 2020 to May 2021. With the approval of institutional ethics committee, patients who are meeting the inclusion criteria and exclusion criteria are recruited consecutively by convenient sampling till the necessary sample size is reached. Urinary albumin excretion, eGFR are considered as primary outcome of interest. BMI was considered as explanatory variable.

Results: A total of 109 individuals with a mean age of 37.78 ± 15.5 years with 42.20% males and 57.80% females are studied. The mean BMI is 27.38±2.04 kgm2. The mean ACR is 27.20±22.63.

Conclusions: Our study noted a weak positive correlation between BMI and ACR (r value=0.451, p value=<0.001).

Key words: BMI, obesity, microalbuminuria, albumin to creatinine ration, chronic renal failure, nephropathy

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LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
ABSI	A body shape index
ACR	Albumin-creatinine ratio
AGRP	Agouti-related protein
ALT	Alanine transaminase
BFP	Big fat positive
BMI	Body mass index
BSA	Body surface area
CCC	Cathodal closure contraction
CI	Confidence interval
CKD	Chronic kidney disease
CKD-EPI	Chronic Kidney Disease Epidemiology Collaboration formula
CP	Cerebral palsy
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
DTPA	Diethylenetriamine pentaacetic acid
eGFR	Estimated glomerular filtration rate
ESRD	End-stage renal disease
FBS	Fasting blood sugar
FSGS	Focal and segmental glomerulosclerosis
GBM	Glioblastoma multiforme
HTN	Hypertension
ICMR-INDIAB	Indian Council of Medical Research-India Diabetes
IQR	Interquartile range
LHA	Lateral hypothalamic area
MAU	Medical assessment unit
MC	Metacarpal bone
MCH	Mean cell hemoglobin
MDRD	Modification of the Diet in Renal Disease

MSH	Melanocyte-Stimulating hormone
NPY	Neuropeptide Y
OR	Operating room
ORG	Obesity-related glomerulopathy
PPBS	Post prandial blood sugar
PVN	Parenteral venous nutrition
RAAS	Renin-angiotensin-aldosterone system
RPF	Renal plasma flow
SBP	Systolic blood pressure
TDI	Tolerable daily intake
TGF-β	Transforming growth factor-β
TSH	Thyroid stimulating hormone
UACR	Urine Albumin-to-Creatinine Ratio
UAE	Urinary albumin excretion
USA	Unstable angina
VEGF	Vascular endothelial growth factor
VFA	Visceral fat area
WC	Waist circumference
WHO	World health organization
WHR	Waist-to-hip
WHtR	Waist-to-height ratio

INTRODUCTION

INTRODUCTION:

The prevalence of obesity has tremendously increased over the past half century, along with the rise in the prevalence of hypertension as well as diabetes.[1] Many posit that obesity also raises the risk for nephropathy through adverse effects on blood pressure, insulin resistance, inflammation, and dyslipidaemia, or even directly by altering systemic and glomerular haemodynamics.[2] The most common method for defining obesity is based on BMI (*i.e.*, a person's weight [kilograms] dividing that by the square of his or her height [meters]). The World Health Organisation (WHO) considers a BMI between 20 and 25 kg/m^2 as normal weight, a BMI between 25 and 30 kg/m^2 as overweight, and a BMI of more than 30 kg/m^2 as obese. It should be emphasized that population norms of BMI could be different based on ethnic and racial background (*i.e.*, the proportions of Asian people who are at a high risk of developing type 2 diabetes and CVD is substantial at a lower BMI).[3] Obesity classification according to the Asia-Pacific guidelines is categorized into four groups, namely underweight (<18.5 kg/m^2), normal weight (18.5–22.9 kg/m^2), overweight (23–24.9 kg/m^2), and obese (≥ 25 kg/m^2).[4]

Obesity has virtually tripled globally since 1975, according to statistics. In 2016, it was discovered that over 1.9 billion adults in the world, aged 18 years and above, were overweight. Among these over 650 million were obese. 39% of adults who were aged 18 years and over were determined to be overweight in 2016, and 13% were categorized obese.[5] The obesity prevalence in India varies according to age, geographical environment, gender, socio-economic status, etc. According to ICMR-INDIAB study conducted in 2015, the prevalence rate of obesity as well as central obesity varied from 11.8%- 31.3% and 16.9%-36.3% respectively.[6]

Because the obesity pandemic is spreading over the world, a better understanding of the various risk factors for obesity-related comorbidities is necessary. Microalbuminuria is

currently regarded as the most prominent risk factor for the increase in the morbidity as well as the mortality among the obese population..[7] Microalbuminuria is defined as the urinary albumin excretion of 30-300mg/g of creatinine. It is considered as the first sign of nephropathy. [8] Obese people experience compensatory hyperfiltration to meet the higher metabolic demands of their increasing body weight. The increase in the intraglomerular pressure also can cause damage to their kidneys and heighten the chances of them to develop chronic kidney disease over time. Another mechanism could be that the glomerular hyperfiltration occurring in obesity would probably be due to altered productions of various adipokines like leptin, adiponectin, resistin, etc. which will further lead to more albuminuria and abnormally high glomerular filtration rate.[9] It is still debatable whether is it the microalbuminuria in visceral obesity reflecting specific glomerular damage or is the marker of generalized endothelial cell dysfunction. In view of the known podocyte/endothelial cell interaction, presumably both explanations are true. [10]

One of the basic indicators of nephropathy is microalbuminuria detected by presence of small amounts of albumin in urine (between 30–300 mg/24 h). It shows abnormally increased permeability for albumin in renal glomerular structure indicated morphological and structural changes. Microalbuminuria is not only a sign of increased risk of developing kidney disease, but also a sign of cardiovascular disease risk in patients with diabetes and high blood pressure.[11] Weisinger JR et al, gave the first description of focal segmental glomerulosclerosis as a specific renal complication of morbid obesity.[12] Men who have more visceral adipose tissue, as determined by computed tomography, have a higher frequency of albuminuria.[13] The BMI-independent link between abdominal obesity and poorer renal outcomes is also documented in regard to mortality in patients with ESRD [14] which was seen and it shows that visceral adiposity plays a direct role. In general, obesity and poorer renal outcomes were found to be associated even after controlling for potential mediators of

obesity's metabolic and cardiovascular effects, like diabetes mellitus and hypertension (high blood pressures) , implying that obesity may affect kidney function through mechanisms unrelated to any of these complications.[15]

An observational study reported a strong association between obesity and microalbuminuria. Microalbuminuria was highly prevalent among obese subjects.[16] A cross-sectional study done on consecutive obese people who were normotensive and nondiabetic with a BMI (body mass index) of 30-35 kg/m^2 as well as very obese people with BMI of more than 35 kg/m^2 showed that microalbuminuria was present more commonly in individuals who were very obese than those belonging to the obese group (24.0% versus 9.9%, $P = 0.043$) in an univariate analysis.[7] In another cross-sectional study, it was reported that younger, class 1 obese patients had a higher urinary albumin excretion, estimated glomerular filtration rate(eGFR), and three times higher microalbuminuria prevalence, even in absence of diabetes and hypertension, with a correlation between anthropometry and eGFR as compared with nonobese individuals.[17]

Most studies have focused on body mass index(BMI) for identification of obesity, but some studies suggested waist as an appropriate indicator to predict kidney disease.[18] However further research is essential to evaluate the link between both albuminuria and obesity especially in non-diabetics without high blood pressure to prevent more serious diseases by screening obese patients for microalbuminuria.

Need of the study

Obesity is becoming more widely recognised as a contributor to renal disease. Proteinuria as well as microalbuminuria have been linked to obesity. Obesity raises the probability of developing key additional chronic kidney disease (CKD) risk factors, such as diabetes mellitus and hypertension, and it is thought to have a direct impact on the development of both CKD as well as end-stage renal disease(ESRD). Indian obese population are known to having a different biochemical phenotype than other races. There is a paucity of Indian studies linking obesity and nephropathy, in absence of diabetes and hypertension. Since there is lack of information on microalbuminuria in an otherwise healthy obese Indian adults and whether these obese adults deserve targeted identification and clinical intervention, this study would serve as a tool to help in the decision making for intervention in nephropathy.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

1) To estimate the urinary albumin excretion in obese individuals.
2) To calculate the estimated glomerular filtration rate (eGFR) using MDRD formula in these patients
3) To correlate microalbuminuria with severity of obesity based on BMI.

REVIEW OF

LITERATURE

REVIEW OF LITERATURE:

Obesity

Obesity can be defined as a medical condition where excess body fat is accumulated to an extent that it may have adverse effect on health, reduced life expectancy and/ or increased health problems.[19] [20] In the young adult normal levels of body fat are considered to be 12–20 % body weight in males and 20–30 % bodyweight in females, while levels of > 25 % body weight in males and > 33 % body weight in females can be considered obese.[21]

Table 1: Body fat levels of men and women.

Body fat %			
Parameter	**Normal**	**Overweight**	**Obese**
Men	15-22	>22	>25
Women	18-32	>32	>35

Table: Body fat levels of men and women [21]

However, as individuals age there is an accretion of fat at the expense of lean body mass, so that at older ages the percentage of the body that is fat is considerably higher, even in individuals who do not gain weight.[22] Numerous publications based on the recommendations of expert committees have struggled with developing working definitions of weight status. These recommendations have evolved from weight-for-height standards to sex-specific population-dependent references. The most recent transition is a movement toward a single body mass index (BMI; in kg/m^2) standard that is applicable to all adults.[23]

Body mass index, or BMI, is a statistical index that uses a person's weight and height (in metres) to create a measure of body fat in both males and females of any age. BMI = weight (in kg)/ height2 (in m2) is computed by taking a person's weight (in kilogrammes) and dividing it by their height (in metres squared). The individual's BMI number is then calculated using this equation. Instead of utilising the basic height vs. weight charts, the

National Institute of Health (NIH) now utilises a person's BMI to determine whether he or she is underweight, normal weight, overweight, or obese. Both the National Institutes of Health (NIH) and the World Health Organization (WHO) now use these BMI classifications for White race, Hispanic races, and Black race people. However, these cutoffs understate the risk of obesity in Asian and South Asian populations, resulting in modest differences in classification.[24]

Table 2: Obesity classification according to WHO and Asia-Pacific guidelines [4]

Parameter	WHO (BMI)	Asia-Pacific (BMI)
Underweight	<18.5	<18.5
Normal	18.5-24.9	18.5-22.9
Overweight	25-29.9	23-24.9
Obese	>=30	>=25

Epidemiology

The worldwide prevalence of both overweight as well as obesity has doubled since 1980 to an extent that nearly a third of the world's population is now classified as overweight or obese. Obesity rates have increased in all ages and both sexes irrespective of geographical locality, ethnicity or socioeconomic status, although the obesity prevalence is generally greater in older persons and women. This trend was similar across various regions and countries, although absolute prevalence rates of overweight and obesity varied widely. For some developed countries, the prevalence rates of obesity seem to have levelled off in recent years.[25] The 2017 global nutrition report showed that 2 billion adults are overweight/obese and 41 million children are overweight worldwide.[26] Obesity prevalence increased in all regions from 2008 to 2016 with the highest rates observed among men in high-income Western countries and among women in Central Asia, the Middle Eastern Countries and North Africa. According to a study that reported national survey data from 2000 to 2018 in

the USA, obesity prevalence has increased to over 42% among adults and the severe obesity prevalance (BMI ≥40 kg/m2) doubled to 9.2% over the study period. [27]

In India, between the year 1998 and 2016, incidence of overweight and obesity increased among men and women, in both urban as well as rural areas. Between 1998 and 2016, the percentage of women in metropolitan areas without a high school diploma who were overweight or obese grew from about 15% to roughly 32%. Whereas the prevalence among men from urban areas with higher education increased from approximately 26% to around 34% between the year 2005 and the year 2016. In rural areas, a more similar rise in the prevalence of both overweight as well as obesity was found among all the individuals across the study period, irrespective of their socio-economic position. Among women from rural areas with higher education, overweight/obesity increased from 16% to around 25% between the year 1998 and 2016, while the prevalence among women from rural areas with no education also showed an increase from 4% to around 14%.[28] The obesity prevalence among reproductive-age women was 5.1% in India as per a cross-sectional study from the 2015–16 National Family Health Survey. [29]

Indices for diagnosis

Anthropometric measures are simple, inexpensive, non-invasive tools to diagnosis obesity and to assess the risk of morbidity and mortality. The most widely used are body mass index or BMI, waist circumference (WC), waist-to-hip (WHR) and waist-to-height ratios, visceral fat area (VFA), body fat (BFP) and a new body shape index (ABSI).[30] BMI has been one of the most widely adopted weight-related anthropometric measures. [31] [32] Some central obesity indices like waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR) can outperform BMI. However, there is no found agreement on the usefulness of these mentioned measures because some studies state that they are strong predictors [33],

whereas others contradict this.[34] Waist circumference does not help distinguishing subcutaneous from visceral fat mass, but is widely accepted as a certain measure of fat distribution. Waist circumference and the waist-to-hip ratio (WHR) have also been used as other markers for abdominal obesity. [35] WHR is well studied but not as widely accepted measure as it was previously a decade ago. Visceral obesity is also linked with dyslipidemia and hypertension and abdominal visceral fat is associated strongly with cardiovascular risks. [36] [37] A Body Shape Index (ABSI), based on waist circumference (in metres), weight (in kilograms) and height (in metres), defined as WC / (BMI2/3 × height1/2), was proposed in 2012. The goal of ABSI is to predict diseases risks that cannot be readily captured by BMI.[38]

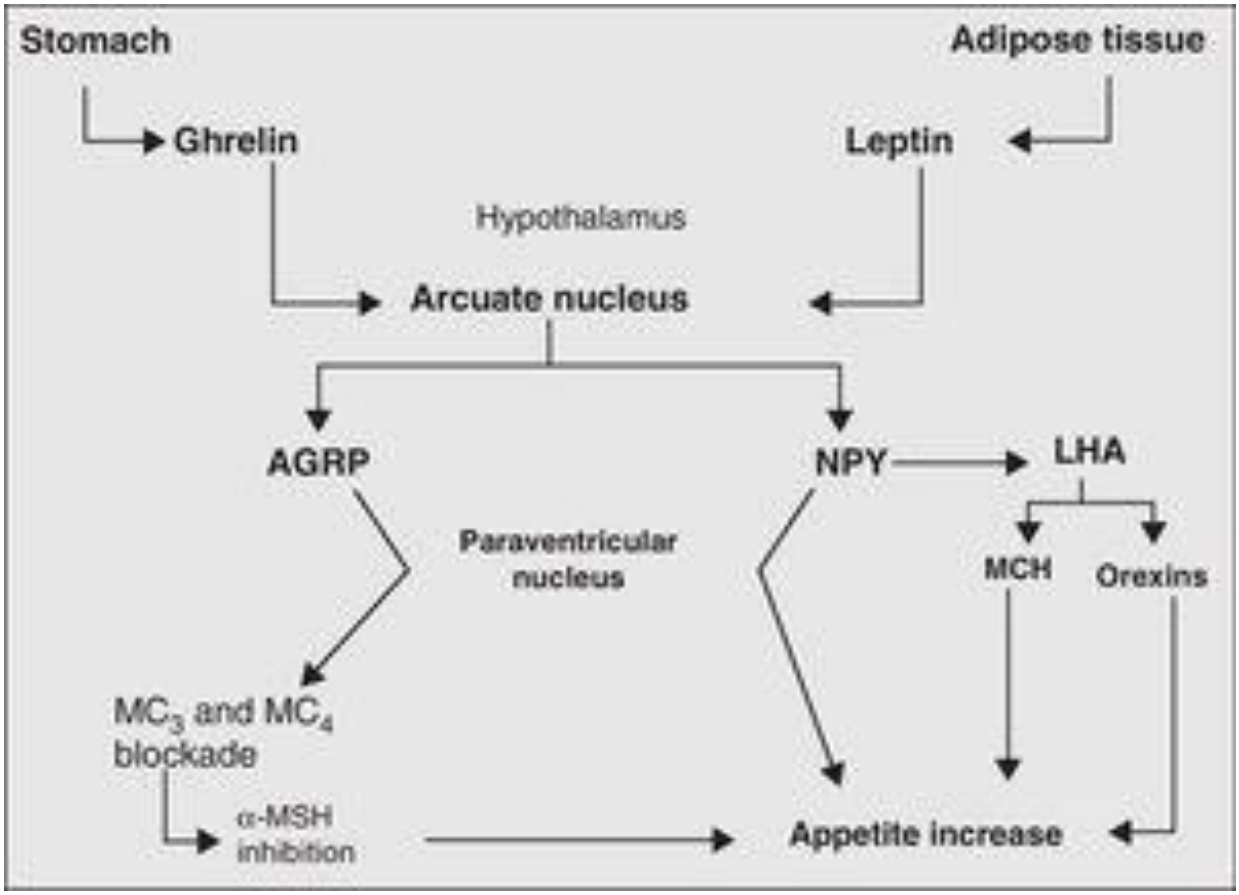
Etiology. Risk factors and Pathogenesis

Different scholars mention a lot of predisposing factors which vary depending on geography, social conditions, political and economic factors, and human genetics. In an aggregate, the commonest factors were sociodemographic, behavioral, genetic, and living in obesogenic environment. According to our current understandings of genetics and molecular biology, it helps us to understand that the etiology and pathogenesis of obesity is a complex phenomenon.9 In regard to this, the theory of sustained increase in the intake associated with a deficient energy expenditure is excessively simplistic, as obesity is found to have an origin which is very heterogeneous, and a variety of both various dietary and genetic factors are involved in its development.[39] [40]

Composition imbalances of intestinal microbiota have also found to be connected with the occurrence of both insulin resistance and increase in body weight.[41] Increase in the consumption of food of animal origin and carbonated beverages, which provide 20–30% of total daily energy intake and excess intake of fruit juice (more than 350mL/day) may promote obesity development in pre-school children, and even limit their growth.[42]

The central nervous system, the gastrointestinal system (GIT) including both the liver and the pancreas, and the adipocyte are also involved in the regulation of both expenditure of energy and as well as the intake.[43] The release of thyroid- simulating hormone is mediated by leptin, through the hypothalamus using sympathetic nervous system. [44] Body weight is also modulated by the noradrenergic receptors through leptin by stimulating both the alpha-1 and the beta-3 receptors, decreasing the intake and increasing the energy expenditure.[45] The amount of food that is ingested and selection of macronutrients is regulated by serotonin receptors. Receptor stimulation occurring at the hypothalamic level decreases the overall intake, and more specifically intake of fat.[46] Peptides like gastrin-releasing peptide, cholecystokinin, and bombes in decrease food intake. Insulin promotes glucose uptake and accumulation of lipids in the tissues there by exerting an anabolic effect.[47]

Figure: Main orexigenic mechanisms involved in regulation of appetite. Lateral hypothalamic area or LHA ; AGRP, agouti-related peptide; MCH, melanocyte concentrating hormone; MC, MSH receptors; sMSH, melanocyte-stimulating hormone; NPY, neuropeptide Y; PVN, paraventricular nucleus.[48]



The two efferent systems for body weight control, the endocrine system–including growth, thyroid, and gonadal hormones, glucocorticoids, and insulin–and the neurovegetative system, jointly contribute to regulating energy balance. An intake in energy which is greater than total energy expenditure will result inevitably in an increase in the adipose tissue, which in turn is always associated with lean mass and body weight increases, in whose control total energy expenditure plays a very significant role.[49]

Complications, Management, prevention

High BMI and accumulation of body fat mass are an important predictor for metabolic disorders. Obesity during pregnancy leads to adverse neonatal outcomes (skeletal muscle injury, respiratory distress syndrome, injury to peripheral nervous system, bacterial sepsis, convulsion, hypoglycemia). Obesity is also found to be associated with a range of comorbidities, including diabetes mellitus, hypertension, dyslipidemia, cardiovascular disease, obstructive sleep apnea, chronic obstructive pulmonary diseases, cancer, chronic disease morbidity and mortality, premature death, and atrial fibrillation. There is also a strong association between hypertension and WC, BMI, and waist hip ratio (WHR).[50]

Diet and exercise continue to be the mainstays for obesity management, an increasing number of patients require pharmacological support to achieve or maintain body weight reduction.

Core actions that have been proposed by WHO for prevention of obesity:[51]

- Reduce the commercial pressure on people (especiatially children) to consume producs high in energy
- Reducing the amount of fat, sugar, and salt in products
- Enable easier as well as cheaper access to healthy food
- Introduce various measures that help to improve food and physical activity in schools and the workplace to be increased
- Implement better urban design and transport policies, thereby promoting cycling and walking
- Creating opportunities for people in local environments helping them to be more active physically in their leisure time
- Encouraging health services to give advice on healthy diet and physical activity, and exclusive breast feeding to be promoted

Microalbuminuria

Microalbuminuria (MA) can be defined as the urine albumin above the normal range (currently defined as less than <30 mg/d) but below the range detectable with the conventional dipstick methodology (eg, >300 mg).[52] Microalbuminuria is defined as urinary albumin excretion of 30-300 mg/day, or 20-200 µg/min. It is considered as an earlier sign of vascular damage. It is a marker of general vascular dysfunction and is nowadays been considered as a predictor of worse outcomes for both kidney and heart patients. A very significant correlation is observed between blood pressure and microalbuminuria. A significantly higher frequency of microalbuminuria is associated even with a high normal blood pressure and this way could be a biomarker of increased risk of cardiovascular disease. It could be also be taken as an indicator of both insulin resistance and of increased risk of renal and cardiovascular diseases associated with metabolic syndrome. Renal involvement is a major pivotal development occurring in diabetes. Microalbuminuria is generally found to be the first clinical sign of renal dysfunction in diabetics. It is seen that risk of cardiovascular and renal involvement is increased even in the high normal range of microalbuminuria (below 30 mg/day). [53]

Albumin physiology and pathology

The most abundant circulating protein which is found in the plasma is albumin. In any healthy human patient albumin itself represents half of the total protein content (around 3.5 g/dL to 5 g/dL) of plasma in them. Albumin is synthesized by the hepatocytes in the liver and is excreted rapidly into the bloodstream at the rate of around 10 gm to 15 gm per day. The amount albumin that is stored in the liver is very little, as most of it gets excreted rapidly into the bloodstream. In humans, the serum albumin serves as a significant modulator of plasma oncotic pressure and also functions as a transporter of endogenous and exogenous

(i.e. drugs) ligands. Serum albumin can also be measured in clinical medicine via a standard serum laboratory testing, and this measure has been considered as a highly sensitive marker for the assessment of an individual patient's nutritional status.[54] Albumin serves various multiple functions being the most abundant plasma protein. It acts as a carrier of hormones, vitamins, metabolites and multiple drugs. It serves as an acid- base buffer, as an antioxidant and supports the oncotic pressure and volume of blood. The presence of albumin in urine is also considered to be the result of the balance between glomerular filtration and tubular reabsorption. Albuminuria has been accepted as an independent risk factor and a marker for renal as well as cardiovascular disease, and during the past decade, evidence has suggested that albumin itself may cause progression of renal disease.[55]

Etiology and risk factors

Microalbuminuria develops from a dysfunction in the glomerular basement membrane (GBM) to allow albumin to enter the urine. The enzyme N-deacetylase is necessary to form heparan sulfate, which is how the GBM derives its negative charge. Furthermore, inadequate control of blood sugars inhibits this enzyme, reducing GBM's negative charge and allowing excessive amounts of albumin to leak out.[56] Advanced glycosylation end-products can also chelate with the proteins of both the GBM and mesangial matrix, thereby neutralizing albumin's negative charge.[57] having comorbidities that cause endothelial damage is considered a risk factor. These include increased age, insulin resistance, dyslipidemia, obesity, hypertension, decreased physical activity, and smoking.[52]

Pathophysiology

Developing microalbuminuria arises when the GBM barrier, a complex sieve, leaks an increased amount of albumin. The proposed mechanism is a combination of glomerular size

enlargement, GBM thickening, mesangial expansion, and effacement of podocyte foot process. Microalbuminuria can also occur via tubular under-reabsorption.[58 59]

Dysregulated enzymatic metabolism of the extracellular matrix is the pathogenesis behind developing endothelial damage.[60 61] Thus, at vascular places, other than just the renal system, the albumin can either leak out of or enter the vessel wall. When this happens, albumin can stimulate inflammation, lipid accumulation, and atherosclerosis, which eventually could form fixed albuminuria and decreased kidney function.[59]

Diagnosis

The gold standard diagnostic test is a 24-hour urine collection as it has the lowest variability, but it is labor-intensive.[60 56] As described above, the urinary albumin/creatinine ratio corrects for urine concentration and volume but can vary through other factors. The urine spot collection, changes depending on the urine volume.[60 52] Other alternatives have undergone development, but they have similar sensitivities to the 24-hour collection. These are immunoturbidimetry, immunonephelometry, enzyme-linked immunosorbent assays, immunoassay with latex bodies, radial immunodiffusion, and fluroimmunoassay.[60 56]

Complications, Management, prevention

If microalbuminuria is present, aggressive measures should ensue with the ultimate goal of decreasing the risk of cardio-metabolic complications.[62] The first-line treatment is lifestyle modifications to control diabetes and hypertension. Although it seems trivial, this can save retinal function, prevent further kidney damage, decrease stroke risk, and decrease microvascular complications.[52]

Several experimental drugs show promising evidence but require further studies to evaluate if they decrease the long-term cardiovascular disease associated with microalbuminuria. For

one of the groups, the mechanism is by decreasing protein glycation. These medications are thiamine, ALT-711 (a cross-link breaker of advanced glycation), pimagedine (a second-generation inhibitor of advanced glycation). Additionally, ruboxistaurin, a protein kinase C-beta inhibitor, has been implicated in decreasing UAE.[63] Increase in the vascular endothelial growth factor (VEGF), which acts by regulating the vascular permeability and angiogenesis, has been linked with microalbuminuria in type 2 diabetic patients. Thus, VEGF inhibitors could be helpful in patients with microalbuminuria.[61] Glycosaminoglycans, such as sulodexide, can also decrease albuminuria.[60] Statins, have a well-documented effect in reducing the risk of cardiovascular disease, but its role in decreasing urinary albumin excretion is still controversial. Due to its cardioprotection, it merits inclusion in the treatment regimen.[60]

When considering the associated comorbidities with microalbuminuria, the treatment recommendations should also include weight loss, aspirin, and maintaining low-density lipoprotein cholesterol of less than 100.[63] Finally, the level of microalbuminuria can serve as an indicator of treatment response, more specifically in patients with hyperinsulinemia, insulin resistance, and hypertension. [62] [60]

Association between microalbuminuria and obesity

Obesity-driven glomerular hyperfiltration contributes to glomerulomegaly, namely, obesity-related glomerulopathy (ORG), which in turn can further result in the development of proteinuria, secondary focal and segmental glomerulosclerosis (FSGS), and progressive CKD.[64] ORG is defined pathologically as glomerulomegaly, with or without FSGS, occurring in individuals with a body mass index(BMI) ≥30 kg/ m2. [65] Its pathological characteristics include glomerular hypertrophy, FSGS or both, with clinical manifestations of proteinuria and other metabolic disorders.

Correlation of microalbuminuria with severity of obesity based on BMI

A descriptive, cross-sectional study showed that compared with non-obese people, patients with both central and peripheral obesity had a risk of a higher UACR, where as for those with only peripheral obesity or central obesity, there was no positive relationship. BMI ≥28.0 kg/m2 was diagnosed as peripheral obesity and even after adjusting for multiple factors, obesity itself remained an seperate risk factor for high UACR.[66]

A study on Chronic Renal Insufficiency Cohort examined the implications of obesity on the performances of estimated glomerular filtration rate(eGFR), the urine albumin-creatinine ratio (ACR), and excretory burden, the urinary creatinine excretion rate increased with BMI, resulting in a reciprocal underestimation bias of albuminuria assessment by ACR. In regression models adjusted for age and race, geometric mean creatininuria in women was 15% (95% confidence interval (CI) 9–21%), 23% (17–31%), 29% (22–37%), and 31% (24–39%) greater, respectively, for 25<=BMI<30, 30<=BMI<35, 35<=BMI<40, and >=40 kg/m2 versus 18.5<=BMI<25 kg/m2 (P<0.001 for all). In men, the corresponding increases were 18% (12–24%), 27% (20–34%), 34% (26–43%), and 34% (25–42%, P<0.001 for all).[67]

A study conducted on 50 apparently healthy, young obese (20–30 years) and middle aged obese adults (age 31–50 years) having a mean BMI of 30.12+/-3.36 for males and 34.63+/-7.49 for females showed a correlation which was significantly positive of BMI with microalbuminuria with r value 0.3228 and p value 0.02.[68] A cross-sectional study of 200 adults with BMI ≥25 kg/m2 reported a 11% microalbuminuria prevalence and it positively increased with increasing of BMI (P = 0.04).[11] In a cross-sectional analytical study, out of 47 patients who were class 1 obese, with BMI 25–29.99 kg/m2 14.9% had microalbuminuria.[17] In another cross-sectional study of general population aged ≥30 years where obesity was defined as body mass index ≥28.0 kg/m2 and central obesity was defined as waist-to-hip ratio ≥0.85 for females and ≥0.90 for males, it was noted that average levels

of BMI (25.4 *vs.* 24.4, *P*<0.001) and WHR (0.89 *vs.* 0.87, *P*<0.001) were higher in participants with elevated UACR than in participants with normal UACR. Both obesity (21.4% *vs.* 13.3%) and central obesity (64.1% *vs.* 48.5%) were more common in participants with elevated UACR than in those with normal UACR (all *P* values <0.001). It was observed that individuals with higher levels of BMI and WHR had higher risk of abnormal urine albumin.[69]

Pathophysiology for development of microalbuminuria in obesity

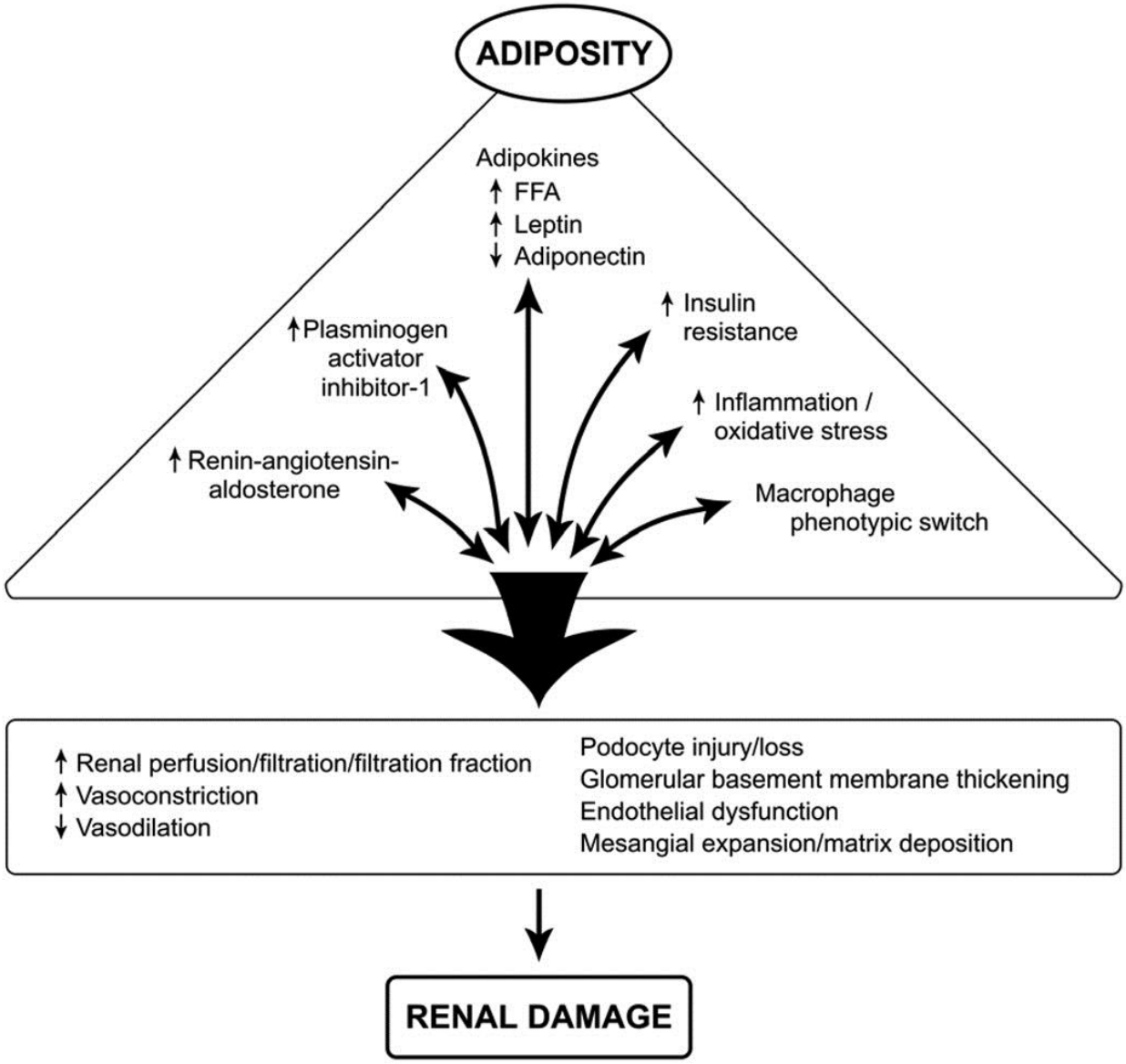
Obesity is a significant risk factor for the development of renal disease. It raises the likelihood of developing key risk factors for chronic kidney disease (CKD), like diabetes mellitus and/or hypertension, and it can also cause chronic kidney disease(CKD) and end-stage renal disease (ESRD). Obese people have a (presumably) compensatory mechanism in the form of hyperfiltration to meet the increased metabolic demands brought on by their greater body weight. The damage to the renal structure can also occur due to the increased intraglomerula pressure and further raise the risk of developing Chronic kidney disease in the long term. [15]

Hemodynamic changes, dysregulations of hormone response and abnormal lipid metabolism in the similar context of obesity are the main pathogenesis of ORG. Hemodynamic changes induced by obesity lead to a rise in multiple parameters such as renal plasma flow (RPF) and glomerular filtration rate (GFR) as well as compensated glomerulomegaly.[70] The renin-angiotensin-aldosterone system (RAAS) interacting with hemodynamic changes in case of obesity promotes the progression of ORG.[71] Moreover, dysregulation of hormone response and lipid ectopic accumulation directly or indirectly injure the morphology and function of renal cells, leading to a glomeruli decline and glomerulomegaly. [72]

Hyperleptinemia in obesity plays a part in the ORG development. Leptin promotes the expression of glomerular transforming growth factor-β1 and increases collagen type IV mRNA production, which enhances the accumulation of extracellular matrix and results in renal fibrosis and glomerulosclerosis.[73] Leptin also acts on renal tubules, promoting salt reabsorption and filtration, consequently leading to glomerulosclerosis via TGF-β and subsequent renal dysfunction. [74]

Adiponectin maintains podocyte integrity via binding to its type 1 receptor and signaling pathway controlled by 5′AMP-activated protein kinase. Increased BMI reduces adiponectin secretion, leaving the glomerular barrier in a vulnerable condition. Furthermore, excessive caloric intake promotes fetuin-A expression, which attenuates adiponectin production and reduces the activation of energy receptors in liver cells and podocytes, resulting in podocyte reduction and the proteinuria development.[75]

Figure 1: Mechanisms of obesity related renal disease. Adipose secretes a large number of mediators with impact on renal function and structure, culminating in renal damage.[76]



Estimated GFR in obesity

Assessment of glomerular filtration rate(GFR) is a crucial step in the evaluation of kidney function or failure. Ideally, GFR should be measured directly using substances that are freely filtered by the kidneys but neither secreted nor reabsorbed, such as inulin, iohexol or iothalamate. However, these compounds are used only in research settings because they are not always available, they are costly, and their use is time consuming. Therefore, in most

clinical situations, GFR is estimated from formulae which include the Modification of the Diet in Renal Disease (MDRD) formula, the Chronic Kidney Disease Epidemiology Collaboration formula (CKD-EPI) and the Cockcroft Gault formula. The latter is an estimation of creatinine clearance which itself is an approximation of GFR. The use of these equations (Cockcroft-Gault and MDRD) to estimate kidney function has been recommended by the National Kidney Foundation. [77]

There is a lack of consensus on the most appropriate method for estimation of GFR or CLcr in individuals with obesity. The controversy relates to the use of 2 body size descriptors that confound these equations. The Cockcroft-Gault equation relies on total body weight and so overestimates GFR in obese patients. The MDRD equation indexes GFR based on a normalized body surface area, that is, mL/min/1.73 m^2. Conversion of MDRD estimated GFR to non-normalized body surface area overestimates Glomerular filtration rate in patients with obesity.[78]

Albuminuria can be the first manifestation of kidney injury in ORG. Since a 24-hour urine collection is inconvenient to obtain, the albumin-to-creatinine ratio (ACR) is increasingly used in clinical practice as a test of similar validity. The degree of correlation between ACR and albuminuria in daily urine collection can be increased by computing the logarithm for ACR.[73]

MOST RELEVANT STUDIES:

Qin et al., (2021)[66] conducted a descriptive, cross-sectional study to find the relation between obesity and albuminuria in patients with no CKD. BMI ≥28.0 kg/m2 was diagnosed as peripheral obesity. WHR ≥0.9 for males as well as WHR ≥0.85 for females were used to diagnose central obesity. Participants with both, central and peripheral obesity, had a higher risk of elevated UACR, even after adjusting for multiple factors (OR: 1.14, 95% CI: 1.07 to

1.12, p<0.001). The study concluded that people with both central and peripheral obesity are prone to a high UACR.

Hemayati et al (2020)[11] conducted a cross-sectional study to measure urine albumin to creatinine ratio in obese patients without any diabetes mellitus and with normal blood pressure with BMI ≥25 kg/m^2. The prevalence of microalbuminuria was positively increased with increasing of BMI (P= 0.04). Moreover, microalbuminuria was significantly higher in people with waist to hip ratio (WHR) >1 (P=0.02). This study detected the association of microalbuminuria with BMI and WHR.

Kittiskulnam et al., (2020)[79] evaluated the performance of the reexpressed Modification of Diet in Renal Disease (MDRD), reexpressed MDRD with Thai racial factor, Thai estimated GFR (eGFR) as well as Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations among obese patients, defined as body mass index(BMI) ≥25 kg/m2 with the reference measured GFR (mGFR) determined by 99mTc-diethylene triamine penta-acetic acid (99mTc-DTPA) plasma clearance method. Among participants with Body mass index(BMI) of more than 35 kg/m2 (n = 48), the mean error of all equations was extremely wide and significantly higher for all equations compared with the lower BMI category. Also, the strength of agreement evaluated by TDI, CCC, and CP were low in the subset of patients with BMI ≥35 kg/m2. The study concluded that estimating equations generally underestimated the reference mGFR in subjects with obesity.

Rothberg et al., (2020)[80] reported **w**ith severe obesity, high fat-free mass and BSA result in low estimates of eGFR indexed to 1.73 m^2 BSA, especially when creatinine-based estimating equations are used. GFR indexed to actual BSA is approximately 50% higher. When eGFR is indexed to actual BSA, many subjects display evidence of renal hyperfiltration which improves with weight loss. In subjects with severe obesity undergoing medical weight loss,

estimating equations that use cystatin C and are indexed to actual BSA may provide a more accurate assessment of renal function.

Ray et al., (2020)[81] conducted an observational and cross-sectional study to evaluate the renal function by using serum urea, serum creatinine, eGFR, and proteinuria in normal, overweight, and obese adults to identify renal impairment. The obese subjects showed higher urea and creatinine levels compared to normal subjects. A significant negative relationship was noted in eGFR (MDRD) and BMI. The prevalence of proteinuria among subjects was 3%. A total of 3% prevalence of renal dysfunction was noted among adults and out of this 2% was found in obese adults. It was concluded that increasing BMI has a significant contributing factor for renal impairment in obese adults.

Xie et al., (2018) [82] investigated the relationship of BMI and waist circumference with the establishment of chronic kidney disease (CKD) in hypertensive patients Comparing with those with normal weight (BMI <24.0 kg/m), participants with obesity (BMI ≥28.0 kg/m) had an increased chance of the primary event (OR = 1.82; 95% CI 1.15-2.90) and rapid renal function decline (OR = 1.26; 95% CI 0.95-1.67). However, waist circumference had no obvious effect on the risk of the primary event (per 5 cm increment: OR = 0.94, 95% CI 0.85-1.04) or rapid renal function decline (OR = 0.96, 95% CI 0.90-1.03). Higher BMI, but not waist circumference, was associated significantly with a rise in the risk of CKD development in hypertensive patients with normal kidney function.

Bhatt et al., (2019)[17] conducted a cross-sectional study to assess the urinary albumin excretion (UAE), prevalence of MAU, and values of estimated glomerular filtration rate(eGFR) in obese nondiabetic and nonhypertensive patients. The study found that younger, class 1 obese patients had a higher UAE, eGFR, and three times higher MAU prevalence, even in absence of diabetes and hypertension, with a correlation between anthropometry and eGFR as compared with nonobese individuals.

Karthikeyan et al., (2019)[83] conducted a case control study on fifty obese cases and fifty non obese controls. Among the study participants, 32% and 4% had microalbuminuria in obese and non-obese group, respectively. Also, obese participants were 11.29 times at higher chances of having microalbuminuria when compared with the non-obese patients with significant p value (p=0.002). The study concluded that microalbuminuria can be utilized to predict the risk of complications in obese subjects in order to bring down the overall morbidity and mortality related to renal function.

Osman et al. (2018)[84] conducted a study to find whether obesity and its various indices has a detrimental effect on the kidney function as manifested by microalbuminuria. 120 young male adults divided into two groups, one with BMI between 18 and 25 and the obese group with BMI of more than 30 are studied. No significant difference in the incidence of microalbuminuria between the normal and the obese subjects was found. No correlation was found between the level of microalbuminuria and the various indices of obesity. The study concluded that obesity does not cause microalbuminuria in young adult obese males who are otherwise healthy.

Rosenstock et al., (2018)[85] obese patients who are undergoing bariatric surgery to determine the albuminuria and proteinuria prevalence in obese patients with and without any associated diabetes and Hypertension. Only the presence of diabetes was found to be associated with proteinuria and albuminuria on a multivariate analysis. Body mass index, age, and Hypertension were not predictive. The conclusion of the study was that even patients who neither had diabetes nor Hypertension, had much greater amounts of microalbuminuria and proteinuria than seen in the general US population, there by reflecting obesity itself causing adverse effect on renal physiology.

Seo et al., (2016) [8] aimed to check for the microalbuminuria prevalence according to body mass index (BMI) and abdominal obesity criteria in individuals aged 30 years or older with

no diabetes, hypertension, renal failure, or overt proteinuria. The microalbuminuria occurence was found to be 5.1%. The risk of microalbuminuria was significant only in the underweight group (odds ratio, 13.22; 95% confidence interval, 2.55–68.63; P=0.002) after adjusting for confounding factors, abdominal obesity was associated with microalbuminuria significantly. The study concluded that the occurence of microalbuminuria was associated with underweight in men and was not associated with waist circumference in either men or women.

Purohit et al., (2016)[68] studied healthy obese, young (group I) and middle aged (group II) adults for prevalence of MAU and prediabetes and study its association with Framingham risk score. The group I patients had 50 % cases of MAU and group II had 25 % patients with MAU. Group II 63.63 % pre-diabetics. The values of MAU obtained were correlated with age, gender, body mass index, systolic and diastolic blood pressure, FBS, waist to hip ratio using Pearson's Coefficient ($p < 0.05$). The 10 year CVD risk calculated using FRS in subjects with MAU was higher as compared to those without MAU. The study concluded that Indian, young and middle aged obese adults to be at a risk of prediabetes, MAU and CV risk warranting their routine screening for better clinical outcomes.

Bouquegneau et al., (2016)[86] analysed and assessed data from patients with a body mass index (BMI) higher than 30 kg m−2 who underwent a measurement for glomerular filtration rate. eGFR calculation was done using the CKD-EPI and Modification of Diet in Renal Disease (MDRD) equations, which was de-indexed by BSA, and the CG equation, either by using the ABW, AIBW or lean body weight (LBW) for the weight variable and comparing it with measured Glomerular filtration rate, which was expressed in ml min−1. This de-indexed MDRD equation appeared to be the most suitable in in obese patients for estimating the non-indexed GFR for the purpose of drug dosage adaptation.

Fouad et al., (2016)[87] conducted a study to verify the prevalence of obesity and the associated chances of developing CKD among 3000 Egyptian students. The prevalence of CKD among subjects with BMI >25 was 6.5%, almost all of them had BMI >35. They found that ACR and eGFR rose progressively with increasing BMI.

Minoo et al., (2015)[7] sought to establish whether the severity of obesity is related with the existence of renal damage while microalbuminuria acts, itself as individual factor, unbiased of other risk factors such as diabetes mellitus and hypertension in a cross- sectional study. Between obese and very obese people, there were no significant variations in serum creatinine, urine albumin concentration, or UACR. The study revealed that the presence of increased urine albumin excretion and microalbuminuria in patients with extreme obesity cannot be predicted as compared to those with a lesser obesity status.

Ren et al. (2015) [88] looked into the relationship involving obesity measures and albuminuria in Chinese population in a population-based cross-sectional study in 8600 subjects aged 40 years or older from a community in Guangzhou. Pearson's correlation analysis and multivariate linear regression analysis revealed that body mass index (BMI), waist circumference and body fat content were significantly correlated with ACR (all $P<0.01$). Prevalence of low-grade albuminuria and increased urinary albumin excretion gradually increased across the BMI, waist circumference and body fat content quartiles (all P for trend<0.0001). The study concluded that obesity measures are associated with urinary albumin excretion in middle-aged and elderly Chinese.

Du et al., (2014)[69] conducted a cross-sectional study to find the association of microalbuminuria with interaction between obesity as well as central obesity in general population aged ≥30 years. Obesity was defined as body mass index ≥28.0 kg/m2 and central obesity was defined as waist-to-hip ratio ≥0.85 for females and ≥0.90 for males. After controlling for potential covariates, participants with both obesity as well as central obesity

have significantly increased risk for elevated UACR (OR=1.82 $P<0.001$) compared to those with neither. Additive interaction analysis indicated that about 43.9% of the risk of elevated UACR in participants with both obesity as well as central obesity was attributed to the interaction between obesity and central obesity (the attributable proportion because of the interaction: 0.439; 95% CI: 0.110–0.768). The multipliable interactive effect between obesity and central obesity on elevated UACR was not found significant (OR=1.82, $P=0.078$). The study concluded that microalbuminuria was significantly associated with the interaction between obesity and central obesity.

Fotheringham et al., (2014)[67] examined the effect of obesity on the performances of estimated glomerular-filtration rate (eGFR), the urine albumin:creatinine ratio (ACR), and excretory burden in 3611 participants of the Chronic Renal Insufficiency Cohort. Urine creatinine excretion significantly increased with body mass index (BMI) (34 and 31% greater at 40 kg/m2 or more versus the normal of 18.5–25 kg/m2) in men and women, respectively, such that patients with a normal BMI and an ACR of 30 mg/g had the same 24-h albuminuria as severely obese patients with ACR 23 mg/g. The bias of eGFR (referenced to body surface area-indexed iothalamate (i-)GFR) had a U-shaped relationship to obesity in men but progressively increased in women. Nevertheless, obesityassociated body surface area increases were accompanied by a greater absolute (non-indexed) iGFR for a given eGFR, particularly in men. Thus, for a given ACR and eGFR, obese individuals have greater albuminuria, absolute GFR, and excretory burden.

Arreola-Guerra et al., (2014)[89] studied the performance of the CKD-EPI and MDRD formulae for estimating glomerular-filtration rate (GFR) in patients of Hispanic origin with normal renal function and these results were compared with the gold standard (GFR measured by Tc99DTPA). They concluded that in healthy Mexican adults, the CKD-EPI formula is a better predictor of the mGFR than the MDRD-IDMS formula. BMI is

significantly associated with the performance of the CKD-EPI formula and is better in those with a BMI greater than 25kg/m^2. Both formulae overestimate mGFR.

Pavan et al., (2011)[16] conducted an observational study to evaluate the connection of obesity defined as per Asia-Pacific guidelines with microalbuminuria which is an early marker of kidney disease in adults. There was a strong connection between obesity and microalbuminuria. Microalbuminuria was highly prevalent among obese subjects compared to the controls (OR = 15.33, 95% CI: 5.83 to 40.32, P < 0.001). The study supports a significant link between obesity and the presence of microalbuminuria in adults.

Shastry et al., (2011)[90] conducted a study to compare the estimated GFR (eGFR) calculated by CG and MDRD formulae and to study the correlation between body mass index (BMI) and eGFR in healthy South Indian men. Mean (±SD) eGFR was 91.05 (± 15.04) and 86.43 (± 13.61) by CG and MDRD equations respectively; this difference though statistically significant (p=0.01), was clinically insignificant. BMI positively correlated with CG-GFR (r=0.471, p< 0.001), and negatively correlated with MDRD-GFR (r=-0.268, p< 0.001). The study concluded that normal eGFR seems to be lower in the South Indian population compared to the western standards. CG and MDRD formulae may need to be validated before these can be applied for staging of kidney function in a healthy Indian population.

LACUNAE OF LITERATURE

The prevalence of obesity-related glomerulopathy is increasing in parallel with the worldwide obesity epidemic and microalbuminuria is a risk factor for morbidity and mortality in obese individuals. There are very few studies done on microalbuminuria in obese people in the Indian context using BMI as index and the current study is an attempt to bridge this gap in literature.

MATERIALS & METHODS

MATERIALS & METHODS

Study population: All Individuals coming to RLJH and research centre in the department of General Medicine at Sri Devaraj Urs Academy Of Higher Education And Research were considered as study population.

Study design: The current study was a cross sectional study

Sample size: Sample size is estimated using microalbuminuria i.e, 11.66% according to the study conducted by Bhatt et al[17]

Formula

$$n = \frac{Z^2_{1-\alpha/2} p(1-p)}{d^2}$$

Where,

p : Expected proportion

d : Absolute precision

1- α/2 : Desired Confidence level

P= 0.116%

d= absolute precision at 6%

desired confidence interval (%) = 095

required sample size= 109

Sampling method: All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the study was done between January 2020 to May 2021 for a period of 1 year 5 Months.

Inclusion Criteria:

All patients who require Individuals >18 years of age, Individuals of BMI >25 kg/m2

Exclusion criteria:

Patients having diabetes mellitus(fasting blood sugars >126mg/dl or hbA1c >6.5%), Patients having hypertension, hyperlipidemia, Patients having any cardiovascular disorders, ischaemic heart disease, congestive heart failure, Patients having chronic kidney disease, urinary tract infections, acute febrile illness, History of drug intake like steroids, Having any other endocrine disorders like hypothyroidism, cushings syndrome.

Ethical considerations: Study was approved by institutional human ethics committee. All study participants gave their informed written consent and only those participants willing to sign the informed consent were incorporated in the study. The risks and benefits involved in the study and voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

Methodology:

- Patients were selected as per the inclusion and exclusion criteria.
- Patient who were obese (BMI >25kg/m^2) were selected
- They were explained about the procedure and their consent was taken and they were subjected to blood investigations
- Yes, our study requires investigations such as following :
    1) complete blood count
    2) Renal function test
    3) Liver function tests
    4) Urine routine including albumin, sugar
    5) Urine ACR.

6) Fasting lipid profile
7) FBS
8) PPBS
9) TSH
10) Serum uric acid

- Clinical, laboratory and sociodemographic data was elicited and recorded in pre defined proforma.

STATISTICAL METHODS:

- Urinary albumin excretion, eGFR, etc., were considered as the primary outcome of interest. BMI was considered as an explanatory variable.
- Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency, and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagrams and pie diagrams.
- Quantitative parameters were cross verified by the visual representation as histograms and Q-Q plots and appropriate statistical tests like the Shapiro-Wilk test.
- The association between continuous explanatory and outcome parameters was reported using Pearson's correlation and its significant value.
- A P-value of <0.05 was noted as statistically significant. Data were analyzed by using SPSS software, V.22.[91]

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OBSERVATIONS AND RESULTS

RESULTS:

A total of 109 cases included into the final study.

Table 3: Descriptive analysis of age in study population (N=109)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Age	37.78 ± 15.5	36.00	19.00	80.00	34.84	40.72

The mean age was 37.78 ± 15.5 years, ranged between 19 to 80 years in the study population. (Table 3)

Table 4: Descriptive analysis of age groups in the study population (N=109)

Age Groups (years)	Frequency	Percentages
<=25	31	28.44%
26-40	38	34.86%
41-55	22	20.18%
>55	18	16.51%

Among the study population, 31 (28.44%) participants were aged up to 25 years, 38 (34.86%) participants were aged between 26 to 40 years, 22 (20.18%) participants were aged between 41 to 55 years, and 18 (16.51%) participants were aged >55 years. (Table 6 & Figure 3)

Figure 3: Bar chart of age groups in the study population (N=109)

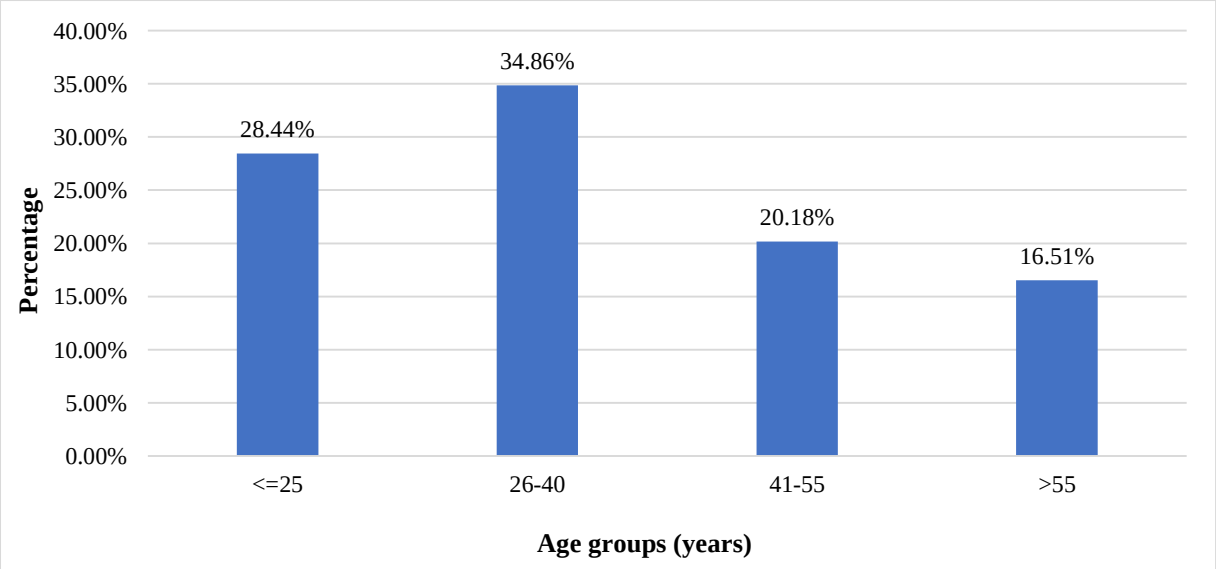


Table 5: Descriptive analysis of gender in the study population (N=109)

Gender	Frequency	Percentage
Male	46	42.20%
Female	63	57.80%

Among the study population, 46 (42.20%) were male participants, and 63 (57.80%) were female. (Table 5 & Figure 4)

Figure 4: Pie chart for distribution of gender (N=109)

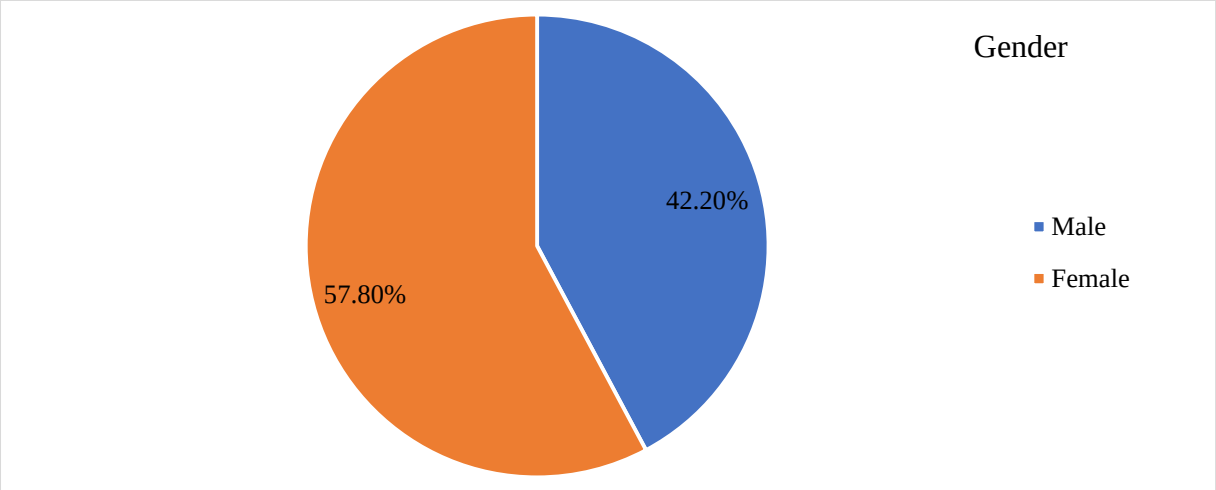


Table 6: Descriptive analysis of anthropometric and blood pressure parameters in the study population (N=109)

Parameter	Mean ± S. D	Median	Minimum	Maximum	95% CI	
					Lower CI	Upper CI
SBP (mmHg)	115.17±6.93	116.00	100.00	132.00	113.87	116.48
DBP (mmHg)	73.87±5.12	70.00	60.00	84.00	72.91	74.83
Weight (in kg)	73.50±10.78	68.00	57.00	93.00	71.47	75.52
Height (in cm)	163.49±10.65	158.00	150.00	184.00	161.49	165.48
BMI (kgm2)	27.38±2.04	27.05	25.10	36.50	27.00	27.77

The mean systolic and diastolic blood pressure was 115.17±6.93 and 73.87±5.12. The mean weight was 73.50±10.78 kg, mean height was 163.49±10.65 cm, and mean BMI was 27.38±2.04 kgm2). (Table 6)

Table 7: Descriptive analysis of BMI categories in the study population (N=109)

BMI categories	Frequency	Percentage
Obese	109	100.00%

Overall study, all of them 109 (100%) were obese. (Table 7)

Table 8: Descriptive analysis of CBC and RFT in the study population (N=109)

Parameter	Frequency	Percentage
Complete blood count: Normal	109	100.00%
RFT: Normal	109	100.00%

Among the study population, all of them 100% were reported normal CBC and RFT. (Table 8)

Table 9: Descriptive analysis of urine albumin in the study population (N=109)

Urine Albumin	Frequency	Percentage
1+	8	7.34%
Nil	101	92.66%

Out of 109 participants, 8 (7.34%) participants had urine albumin 1+. (Table 9 & Figure 5)

Figure 5: Bar chart for urine albumin in the study population (N=109)

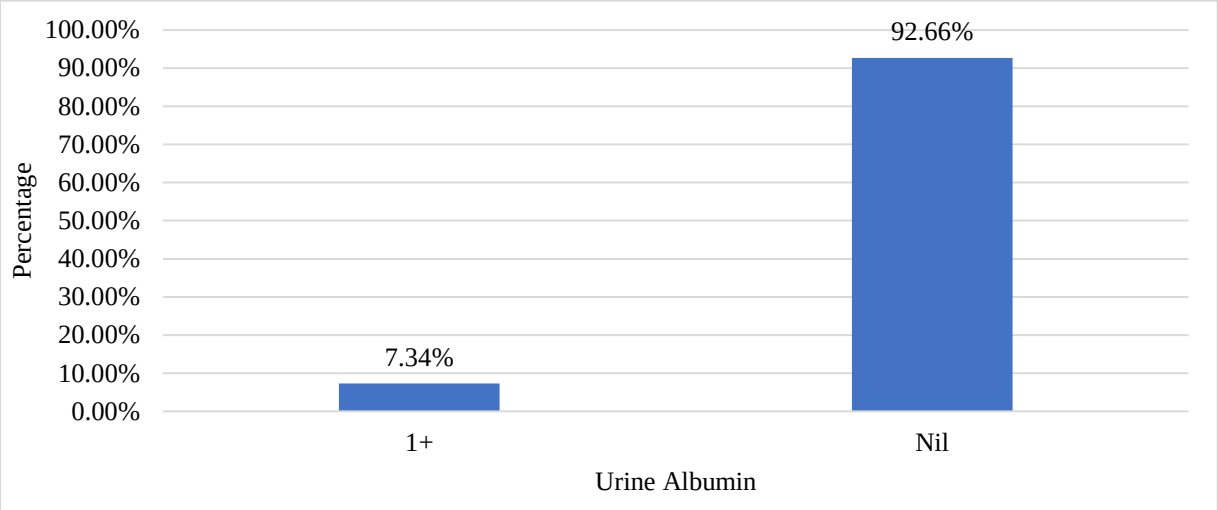


Table 10: Descriptive analysis of urine proteins in the study population (N=109)

Urine Proteins	Frequency	Percentage
1+	3	2.75%
Nil	106	97.25%

Among the study population, 3 (2.75%) participants had urine proteins 1+. (Table 10 & Figure 6)

Figure 6: Bar chart for urine proteins in the study population (N=109)

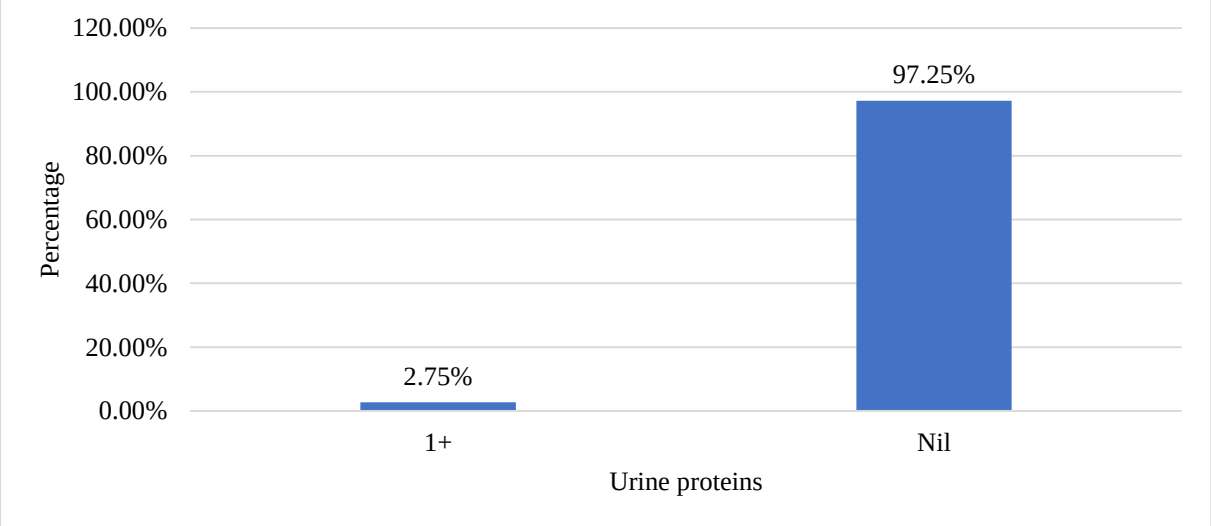


Table 11: Descriptive analysis of lab parameters in the study population (N=109)

Parameter	Mean± S. D	Median	Minimum	Maximum	95% CI	
					Lower	Upper
Albumin to creatinine ratio	27.20±22.63	22.00	0.39	132.00	22.95	31.45
EGFR mL/min/1.73 m2	127.24±39.81	121.00	59.00	221.00	119.77	134.71
FBS (mg/dl)	86.89±8.71	88.00	70.00	112.00	85.25	88.53
PPBS (mg/dl)	118.97±9.22	120.00	102.00	144.00	117.24	120.70
TSH Levels (mlu/l)	2.48±1.03	2.78	0.46	4.20	2.29	2.68

The mean Albumin to creatinine ratio was 27.20±22.63, mean EGFR mL/min/1.73 m2 was 127.24±39.81, mean fasting blood sugar was 86.89±8.71 (mg/dl), mean PPBS was 118.97±9.22 (mg/dl), and the mean TSH levels were 2.48±1.03 (mlu/l) in the study population. (Table 11)

Table 12: Descriptive analysis of EGFR in the study population (N=109)

EGFR	Frequency	Percentages
G3a (45-59)	1	0.92%
G2 (60-89)	21	19.27%
G1 (>=90)	87	79.82%

Majority of 87 (79.82%) participants had G1 EGFR (>=90) and 21 (19.27%) had G2 EGFR (60 to 89) followed by 1 case (0.92%) with G3a EGFR. (Table 12 & Figure 7)

Figure 7: Pie chart of EGFR in the study population (N=109)

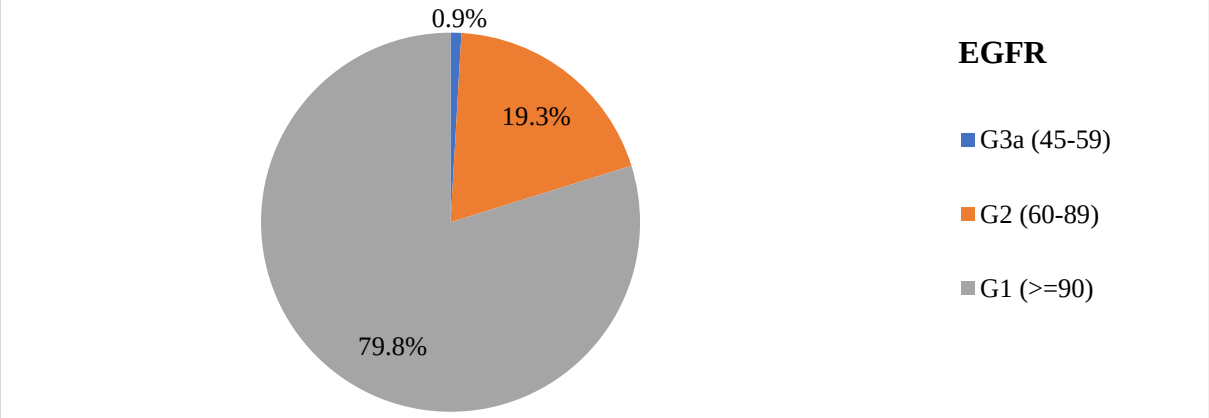
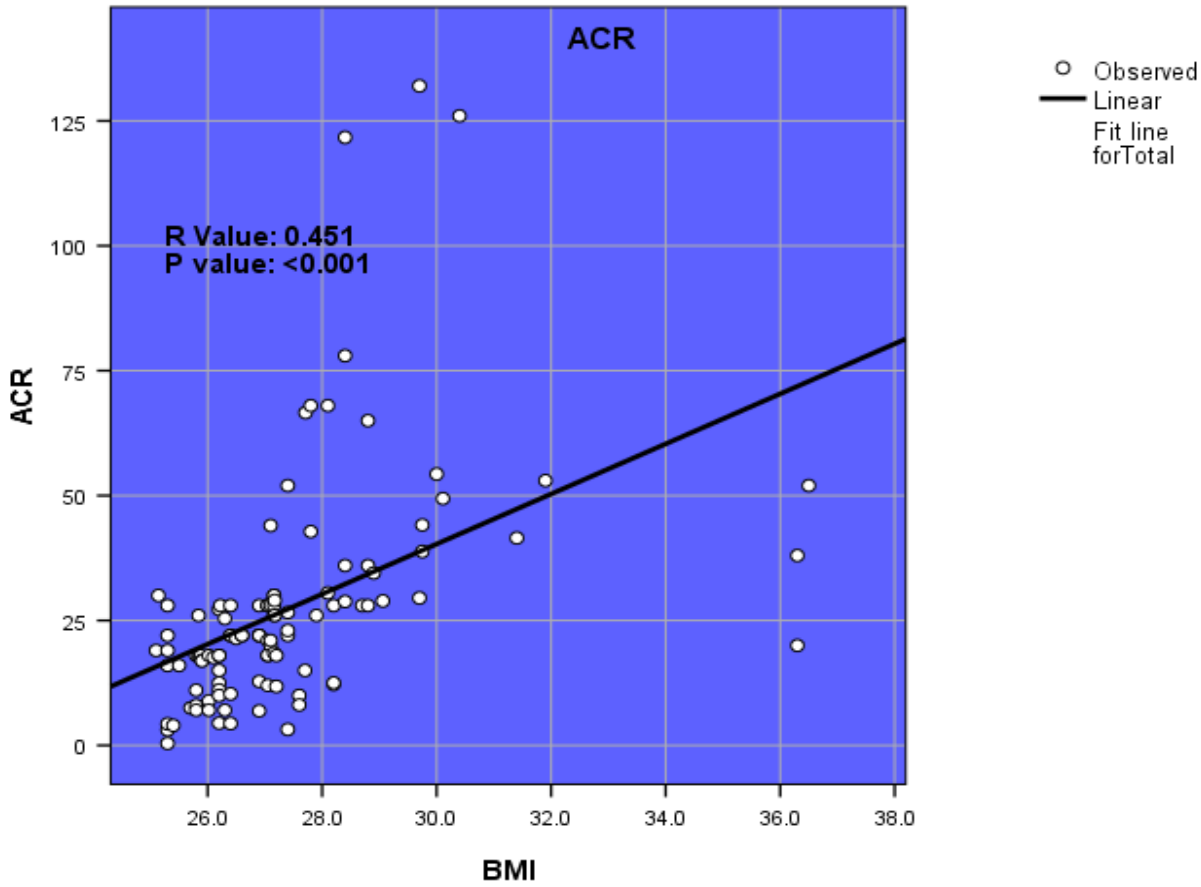


Figure 8: Correlation of BMI with albumin creatinine ratio in study population (N=109)



There was a weak positive correlation between BMI and Albumin creatinine ratio (r value=0.451, P value=<0.001). (Figure 8)

DISCUSSION

DISCUSSION:

One of the most powerful risk factors for the new-onset CKD is a high BMI. A compensatory hyperfiltration develops in obese people to match the higher metabolic demands of their greater body weight.[17] Microalbuminuria (increased urine albumin excretion) is a sign of endothelial dysfunction and indicates renal and systemic endovascular damage. Microalbuminuria, which was once used to predict the onset of overt diabetic nephropathy in diabetic patients, is now thought to be an early indicator of renal injury in non-diabetic people.[92] Obesity has been connected to microalbuminuria, though the occurence of microalbuminuria in overweight groups is not well documented. We conducted a cross-sectional study on individuals with BMI >25 kg/m2 with no known co-morbid conditions to estimate the urinary albumin excretion, calculate the estimated-glomerular filtration rate(eGFR) using MDRD formula and to correlate microalbuminuria with severity of obesity based on BMI. A total of 109 individuals attending R. L Jalappa Hospital from January 2020 to May 2021 meeting the inclusion and exclusion criteria are included in the study. Urinary albumin excretion, eGFR are considered as primary outcome of interest. BMI is considered as explanatory variable.

The mean age of the study population is 37.78 ± 15.5 years with 42.20% males and 57.80% females. The mean BMI is 27.38±2.04 kgm^2. Hemayati et al. had a slightly younger population in their cross-sectional study with the mean age of men 33.8 ± 10.8 years and of women 34 ± 9.3 years with a preponderance of female subjects at 76.5%. 68% of them were obese with BMI above 30 kg/m2 and 32% were overweight with 29.9> BMI ≥25 kg/m2.[11] Minoo et al. studied much younger group with a mean age of 30.4 ± 10.7 years with a higher mean BMI at 32.8 ± 2.6, among which 15.5% suffered from severe obesity (BMI > 35 kg/m2).[7] Kittiskulnam et al. studied much older group with a mean age of 52.4±15.2 years with 40% men. Their study also included 35.1% diabetes. The mean BMI was

31.5±5.8kg/m2.[79] The mean age was 42.1 ± 11.3 years and the mean BMI was 43.9 ± 8.1 in Rosenstock et al.'s study which included 25% diabetics and 47% hypertensive patients.[85]

Higher BMI has been linked to decreased eGFR, eGFR loss over time, and the development of end-stage renal disease (ESRD). Obesity is thought to cause or aggravate CKD, but the exact process is unknown. Comorbid illnesses such as hypertension and diabetes mediate some of the negative effects of fat. Obesity's effects on adipokines, inflammation, oxidative stress, stimulation of the renin-angiotensin-aldosterone system, sympathetic activation, insulin resistance, and aberrant lipid metabolism may all have an effect on the kidneys.[93] The prevalence of albuminuria is 7.34% and proteinuria is 2.75% in our study. Ray et al. reported a prevalence of 3.0% proteinuria in their study.[81] Mean eGFR (mL/min/1.73 m2) is 127.24±39.81 in our study according to MDRD equation. Mean eGFR in overweight patients was 75.03±10.82 and obese patients was 68.3±10.14 in Ray et al.'s study.[81] In a case-control study, Bhatt et al. found that the cases had a significantly higher mean eGFR of 123.29 ± 20.49 mL/min/kg as compared with controls who had a mean eGFR of 106.59 ± 10.15 mL/min/kg.[17] The prevalence of proteinuria and albuminuria was 21% (95% CI: 15.8–27.1%) and 19.7% (95% CI: 14.2–26.2%) respectively in Rosenstock et al.'s study.[85]

The mean ACR is 27.20±22.63 in our study. The mean ACR was 21.20 ± 26.82 mg/g in Bhatt et al.'s study.[17] Median ACR was 3.9 mg/g with an interquartile range (IQR) of 2.5 to 6.0 mg/g. At baseline, only 2 of 146 subjects had ACR ≥30 mg/g in Rothberg et al.'s study.[80] ACR was significantly higher in obese than overweight and normal body weight participants and rose progressively with increasing BMI. Mean ACR was 35.5 in those with BMI > 40 and 2.5 in those with BMI 25 to 30.[87]

In our study, majority of the participants, at 79.82% had eGFR >=90 i.e. stage I CKD, 19.27% had eGFR 60 to 89, stage 2 CKD and 0.92% had eGFR 45-59 implying stage 3a

CKD. Research undertaken on 12,672 hypertensive patients with eGFR at least 60 ml/min per 1.73 m at the Southern Medical University in China proved that patients with BMI ≥28.0 kg/m had a higher chance of developing CKD (OR = 1.82; 95% CI 1.15-2.90) and rapid renal function decline (OR = 1.26; 95% CI 0.95-1.67).[82] Du et al. in a cross-sectional study involving 2889 participants indicated that about 43.9% of the risk of elevated ACR in participants with both obesity as well as central obesity was attributed to the interaction between obesity and central obesity (the attributable proportion because of the interaction: 0.439; 95% CI: 0.110–0.768).[69] Ren et al. in a cross-sectional study in 8600 subjects aged 40 years or older reported that on Pearson's correlation analysis and multivariate linear regression analysis BMI, waist circumference and body fat content were significantly correlated with ACR (all $p<0.01$).[88] While all the above studies illustrated that obesity was a risk factor for elevated ACR, Seo et al. in a study of 3979 people in non-diabetic, non-hypertensive patients in Korea announced that the correlation only existed in low BMI (BMI<18.5 kg/m2).[8]

A descriptive, cross-sectional study of 41,085 with both central and peripheral obesity showed that compared with non-obese people, patients with both central and peripheral obesity had a risk of a higher ACR, while for those with only peripheral obesity or central obesity, there was no positive relationship.[66] Karthikeyan et al. noted in their study that obese participants were 11.29 times at higher chances of having microalbuminuria when compared to the non-obese patients and it was found to be statistically significant (p value <0.000). They also noted that waist circumference among obese males had a significant association with microalbuminuria whereas waist-to-hip ratio had not.[83] On analysis of 46,854 participants, Xiong et al. found that BMI combined with ACR was strongly associated with all-cause mortality in the general population. People with a low BMI and ACR ≥10 mg/g were among those at highest risk. For cumulative incidence of death, the BMI

≥30.0 kg/m2 with ACR ≥10 mg/g group was at the lowest risk, the BMI 18.5–24.9 kg/m2 with ACR ≥10 mg/g group was among those at the highest risk, and the BMI ≥30.0 kg/m2 with ACR <10 mg/g group was at the lowest risk.[94]

Bhatt et al. found no statistical correlation between anthropometric measurements and ACR, but observed a moderate statistical correlation between BMI and eGFR and between waist circumference and eGFR in study group, showing that as these anthropometric measurements increased, eGFR increased beyond normal range, suggesting hyperfiltration, in otherwise normal patients.[17] They reported that the prevalence of microalbuminuria in obese subjects was three times higher than non-obese subjects, but there is no statistically significant difference observed. [17] An observational study by Pavan et al. found a strong association between obesity and microalbuminuria. Microalbuminuria was highly prevalent among obese subjects compared to the controls (OR = 15.33, 95% CI: 5.83 to 40.32, P < 0.001).[16] In our study, we noted a weak positive correlation between BMI and ACR (r value=0.451, p value=<0.001). This can be attributed to the fact that our study consisted of a relatively younger age group with no co-morbidities. Minoo et al. also reported a similar result from their study with no significant correlation observed between BMI and ACR (r value 0.065, p value 0.376). They came to the conclusion that extreme obesity, as compared to lesser obesity, does not help predict increased urine albumin excretion and microalbuminuria.[7] Fouad et al. noted that the BMI did not differ significantly among those with and without proteinuria or albuminuria (p = 0.74 and p = 0.64, respectively). It was found in a Multivariable analysis that, of all the included factors (like diabetes mellitus, age, HTN, and ACE inhibitor/ARB use), DM was the only significant factor for proteinuria (p = 0.0001) and albuminuria (p = 0.0001).[87]

Osman et al.'s study showed no significant difference between the urine findings from obese subjects and the normal subjects regarding ACR and there was no correlation between

microalbuminuria and various indices of obesity in both normal and obese groups. This can be due to the fact that their study had a very young population with mean ages 21.8±1.6 and 21.9±1.8 in both normal BMI and obese groups. [84] Obese patients who had no other co-morbidities had much greater amounts of albuminuria and proteinuria than seen in the general United States population reflecting the adverse effect of obesity itself on renal physiology.[85]

SUMMARY

SUMMARY:

Obesity is becoming more widely recognized as a risk factor for renal illness, although the precise prevalence of microalbuminuria, especially when other risk factors are taken into consideration, remains unknown. Interventions to prevent or delay the onset or progression of early stage renal damage in obese people can help to delay or avoid ESRD and severe cardiovascular illnesses. Hence, the relationship between microalbuminuria and BMI in obese patients could be useful for clinical intervention. The current cross-sectional study is conducted on individuals attending R. L Jalappa Hospital from January 2020 to May 2021 meeting the inclusion and exclusion criteria to correlate microalbuminuria with severity of obesity based on BMI. Urinary albumin excretion, eGFR are considered as primary outcome of interest. BMI is considered as explanatory variable. A total of 109 individuals with a mean age of 37.78 ± 15.5 and mean BMI 27.38±2.04 kgm2 are included in the study. The mean ACR is 27.20±22.63 in our study. Our study noted a weak positive correlation between BMI and ACR (r value=0.451, p value=<0.001).

CONCLUSIONS

CONCLUSIONS:

- A total of 109 individuals attending R. L Jalappa Hospital from January 2020 to May 2021 are enrolled in the study.
- The mean age is identified as 37.78 ± 15.5 years with 42.20% males and 57.80% females.
- The mean weight was 73.50±10.78 kg, mean height was 163.49±10.65 cm.
- The mean BMI is 27.38±2.04 kgm2.
- The mean systolic and diastolic blood pressure was 115.17±6.93 and 73.87±5.12.
- The prevalence of albuminuria is 7.34% and proteinuria is 2.75% in our study.
- The mean ACR is 27.20±22.63 in our study.
- In our study, majority of the participants, at 79.82% had eGFR >=90 i.e. stage I CKD, 19.27% had eGFR 60 to 89, stage 2 CKD and 0.92% had eGFR 45-59 implying stage 3a CKD.
- Our study noted a weak positive correlation between BMI and ACR (r value=0.451, p value=<0.001).

LIMITATIONS AND RECOMMENDATIONS:

Small sample size and cross-sectional method are the limitations as being a cross-sectional study, the causal relationship between obesity and the ACR cannot be established. To assess obesity, other anthropometric measures such as WC and WHR are recommended to be used in conjunction with BMI.

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ANNEXURES

Study of microalbuminuria in obese individuals as an early indicator of nephropathy

PROFORMA

Name: UHID

Age / Sex:

Residential Address:

Mobile No:

Case History:

Other known Illness:

BP: Pulse rate:

Temperature: Respiratory rate

Pallor- icterus- clubbing-

Cyanosis- lymohadenopathy- edema-

HEIGHT:

WEIGHT:

WAIST CIRCUMFERENCE:

BMI:

CVS-

RS-

P/A-

CNS-

Outcome Measures:

Urine ACR	
Total count	
Serum creatinine	
Blood urea	
Urine routine • Proteins • Pus cells • Albumin • Any other	
Fasting lipid profile	
FBS	
PPBS	
TSH	
eGFR	

Signature

PATIENT INFORMATION SHEET

Study Title: Study of microalbuminuria in obese individuals as an early indicator of nephropathy

Study site: R.L Jalappa hospital, Tamaka, Kolar.

Aim:

1) To estimate the urinary albumin excretion in obese individuals.
2) To calculate the estimated glomerular filtration rate (eGFR) using MDRD formula in these patients
3) To correlate microalbuminuria with severity of obesity based on BMI.

Microalbuminuria is nowadays considered as the most important risk factor for the morbidity and mortality in obese individuals. There are many studies to prove that diabetes and hypertension are considered as risk factors for nephropathy but there are very few studies to prove that obesity itself can serve as a risk factor for nephropathy. Since there is lack of information on microalbuminuria in healthy obese Indian adults and whether these obese adults deserve targeted identification and clinical intervention, this study would serve as a tool to help in the decision making for intervention in nephropathy.

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

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. And the cost of the investigations required for the study will be paid by chief investigator of the study. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. Javeria Afshan

Mobile no: 8105844528

Email: afsjaveria@gmail.com

CONSENT FORM

I ---------- participant, hereby give consent to participate in the study entitled **"Study of microalbuminuria in obese individuals as an early indicator of nephropathy"**

I have been explained that;

1. I would have to provide a blood sample for the study purpose.
2. I have to answer the questionnaires related to project.
3. The data generated from my clinical examination and laboratory tests and other reports will be used in the study (which may be subsequently published) without revealing my identity in any manner.

I affirm that I have been given full information about the purpose of the study and the procedures involved and have been given ample opportunity to clarify my doubts in my mother tongue. In giving my consent, I have not faced any coercion. I have been informed that, notwithstanding this consent given, I can withdraw from the study at any stage.

For any further clarification you can contact the study investigator:

Dr. Javeria Afshan

Mobile no: 8105844528

Email: afsjaveria@gmail.com

Signature of participant: Place:

Name of participant: Date:

ಒಪ್ಪಿಗೆ ಪತ್ರ

ನಾನು ---------- ಭಾಗವಹಿಸುವವರು, ‘‘ಮೈಕ್ರೋಅಲ್ಬುಮಿನೂರಿಯಾ ಅಧ್ಯಯನದಲ್ಲಿ’’ ಎಂಬ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಈ ಮೂಲಕ ಸಮ್ಮತಿ ನೀಡುತ್ತೇನೆ ಆರೋಗ್ಯಕರ ಸ್ಥೂಲಕಾಯದ ವ್ಯಕ್ತಿಗಳು ನೆಫ್ರೋಪತಿಯ ಆರಂಭಿಕ ಸೂಚಕವಾಗಿ ’’

ನನಗೆ ಅದನ್ನು ವಿವರಿಸಲಾಗಿದೆ;

1. ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ನಾನು ರಕ್ತದ ಮಾದರಿಯನ್ನು ಒದಗಿಸಬೇಕಾಗಿತ್ತು.

2. ಯೋಜನೆಗೆ ಸಂಬಂಧಿಸಿದ ಪ್ರಶ್ನಾವಳಿಗಳಿಗೆ ನಾನು ಉತ್ತರಿಸಬೇಕಾಗಿದೆ.

3. ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ಪ್ರಯೋಗಾಲಯ ಪರೀಕ್ಷೆಗಳು ಮತ್ತು ಇತರ ವರದಿಗಳಿಂದ ಉತ್ಪತ್ತಿಯಾದ ಡೇಟಾವನ್ನು ಯಾವುದೇ ರೀತಿಯಲ್ಲಿ ನನ್ನ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸದೆ ಅಧ್ಯಯನದಲ್ಲಿ ಬಳಸಲಾಗುತ್ತದೆ (ನಂತರ ಅದನ್ನು ಪ್ರಕಟಿಸಬಹುದು).

ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಒಳಗೊಂಡಿರುವ ಕಾರ್ಯವಿಧಾನಗಳ ಬಗ್ಗೆ ನನಗೆ ಸಂಪೂರ್ಣ ಮಾಹಿತಿ ನೀಡಲಾಗಿದೆ ಮತ್ತು ನನ್ನ ಮಾತೃಭಾಷೆಯಲ್ಲಿ ನನ್ನ ಅನುಮಾನಗಳನ್ನು ಸ್ಪಷ್ಟಪಡಿಸಲು ಸಾಕಷ್ಟು ಅವಕಾಶವನ್ನು ನೀಡಲಾಗಿದೆ ಎಂದು ನಾನು ದೃ irm ಪಡಿಸುತ್ತೇನೆ. ನನ್ನ ಒಪ್ಪಿಗೆ ನೀಡುವಲ್ಲಿ, ನಾನು ಯಾವುದೇ ಬಲಾತ್ಕಾರವನ್ನು ಎದುರಿಸಲಿಲ್ಲ. ಈ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಿದ್ದರೂ, ನಾನು ಯಾವುದೇ ಹಂತದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

Dr. Javeria Afshan

Mobile no: 8105844528

Email: afsjaveria@gmail.com

Signature of participant: Place:

Name of participant: Date:

ಮೈಕ್ರೋಅಲ್ಬ್ಯುಮಿನೂರಿಯಾವನ್ನು ಇತ್ತೀಚಿನ ದಿನಗಳಲ್ಲಿ ಸ್ಥೂಲಕಾಯದ ವ್ಯಕ್ತಿಗಳಲ್ಲಿನ ಕಾಯಿಲೆ ಮತ್ತು ಮರಣದ ಪ್ರಮುಖ ಅಪಾಯಕಾರಿ ಅಂಶವೆಂದು ಪರಿಗಣಿಸಲಾಗಿದೆ. ಮಧುಮೇಹ ಮತ್ತು ಅಧಿಕ ರಕ್ತದೊತ್ತಡವನ್ನು ನೆಫ್ರೋಪತಿಗೆ ಅಪಾಯಕಾರಿ ಅಂಶಗಳೆಂದು ಪರಿಗಣಿಸಲು ಅನೇಕ ಅಧ್ಯಯನಗಳಿವೆ ಆದರೆ ಸ್ಥೂಲಕಾಯತೆಯು ನೆಫ್ರೋಪತಿಗೆ ಅಪಾಯಕಾರಿ ಅಂಶವಾಗಿ ಕಾರ್ಯನಿರ್ವಹಿಸುತ್ತದೆ ಎಂಬುದನ್ನು ಸಾಬೀತುಪಡಿಸಲು ಬಹಳ ಕಡಿಮೆ ಅಧ್ಯಯನಗಳಿವೆ. ಆರೋಗ್ಯವಂತ ಸ್ಥೂಲಕಾಯದ ಭಾರತೀಯ ವಯಸ್ಕರಲ್ಲಿ ಮೈಕ್ರೊಅಲ್ಬ್ಯುಮಿನೂರಿಯಾ ಬಗ್ಗೆ ಮಾಹಿತಿಯ ಕೊರತೆ ಇರುವುದರಿಂದ ಮತ್ತು ಈ ಬೊಜ್ಜು ವಯಸ್ಕರು ಉದ್ದೇಶಿತ ಗುರುತಿಸುವಿಕೆ ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ಹಸ್ತಕ್ಷೇಪಕ್ಕೆ ಅರ್ಹರಾಗಿದ್ದಾರೆಯೇ, ಈ ಅಧ್ಯಯನವು ನೆಫ್ರೋಪತಿಯಲ್ಲಿ ಹಸ್ತಕ್ಷೇಪ ಮಾಡುವ ನಿರ್ಧಾರ ತೆಗೆದುಕೊಳ್ಳುವಲ್ಲಿ ಸಹಾಯ ಮಾಡುವ ಸಾಧನವಾಗಿ ಕಾರ್ಯನಿರ್ವಹಿಸುತ್ತದೆ.

ಮೇಲಿನ ಸಂಬಂಧಿತ ತನಿಖೆಗಾಗಿ ರಕ್ತದ ಮಾದರಿಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಈ ಮಾಹಿತಿಯು ನಿಮಗೆ ಅಧ್ಯಯನದ ಸಾಮಾನ್ಯ ಹಿನ್ನೆಲೆಯನ್ನು ನೀಡಲು ಉದ್ದೇಶಿಸಿದೆ. ದಯವಿಟ್ಟು ಈ ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮ್ಮಿಂದ ಅಥವಾ ಇಬ್ಬರಿಗೂ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾದ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತು ಬಹಿರಂಗಗೊಳ್ಳುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆಗೆ ಸಹಿ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

MASTER SHEET

S.no	AGE	GENDER	HEIGHT (in cm)	WEIGHT (in kg)	BMI	SBP (mmHg)	DBP (mmHg)	CBC (ABNORMALITIES EG. INCRAESED LEUCOCYTES ETC	RFT (U- C-)	URINE PROTEINS	FBS	PPBS	ACR	TSH Levels	EGFR
1	56	Female	152	65	28.1	110	70	Normal	Normal	nil	90	111	30.5	2.22	69
2	38	Female	155	63	26.2	100	60	Normal	Normal	1+	77	109	15	0.9	147
3	26	Female	155	65	27.05	110	70	Normal	Normal	nil	76	136	21	1.9	128
4	35	Female	159	69	27.2	130	70	Normal	Normal	nil	89	114	18	2.1	121
5	30	Male	170	78	26.9	120	84	Normal	Normal	nil	78	109	28	0.8	93
6	33	Male	172	81	27.2	130	80	Normal	Normal	nil	88	122	11.8	1.6	151
7	63	Female	168	85	30.11	124	70	Normal	Normal	nil	99	119	49.4	2.8	77
8	38	Female	155	88	36.3	130	74	Normal	Normal	nil	78	128	20	1.9	59
9	49	Female	157	90	36.5	122	70	Normal	Normal	nil	76	136	52	1.55	71
10	54	Male	167	89	31.9	110	76	Normal	Normal	1+	88	119	53	2.78	93
11	47	Male	172	93	31.4	120	80	Normal	Normal	nil	78	123	41.5	1.14	96
12	44	Male	180	88	27.16	116	80	Normal	Normal	nil	102	112	18.5	3.18	97
13	47	Female	155	63	26.22	114	70	Normal	Normal	nil	99	126	28	1.98	189
14	34	Female	161	67	25.84	122	78	Normal	Normal	nil	78	121	18	0.88	150
15	33	Female	157	68	28.8	132	70	Normal	Normal	nil	88	108	65	2.16	77
16	22	Female	162	66	25.14	126	80	Normal	Normal	nil	83	129	30	3.78	164
17	31	Female	161	67	25.84	110	70	Normal	Normal	nil	78	131	26	2.1	153
18	26	Male	178	90	28.4	118	80	Normal	Normal	nil	89	124	78	4.1	214
19	41	Male	180	88	27.17	122	80	Normal	Normal	nil	78	109	26	1.08	99
20	19	Male	181	83	25.3	110	78	Normal	Normal	nil	78	133	22	0.46	116
21	31	Male	184	87	26.2	120	70	Normal	Normal	nil	77	123	4.45	2.54	105
22	28	Female	161	68	26.2	100	68	Normal	Normal	nil	79	133	12.5	2.18	163
23	29	Female	158	65	26.02	104	70	Normal	Normal	nil	103	128	8.9	1.98	126
24	45	Female	155	63	26.2	112	80	Normal	Normal	nil	78	132	27.2	0.9	145
25	30	Male	180	88	27.16	120	80	Normal	Normal	nil	104	112	30	1.16	93
26	23	Male	175	83	27.1	110	80	Normal	Normal	nil	99	114	20	2.1	169
27	19	Female	155	63	26.2	110	70	Normal	Normal	nil	92	120	18	1.4	98
28	22	Female	155	65	27.05	110	70	Normal	Normal	nil	99	124	28	3.8	95
29	37	Male	178	90	28.4	110	70	Normal	Normal	nil	92	126	36	4.1	161
30	65	Female	155	88	36.3	100	70	Normal	Normal	nil	100	132	38	2	107
31	63	Male	180	88	27.16	110	70	Normal	Normal	nil	112	132	30	3.2	145
32	20	Female	155	64	26.6	114	70	Normal	Normal	nil	102	128	22	2.7	167
33	62	Male	178	86	27.1	116	70	Normal	Normal	nil	98	144	44	2.9	179
34	57	Male	181	83	25.3	114	70	Normal	Normal	nil	98	114	28	1.9	148
35	22	Female	154	65	27.4	116	70	Normal	Normal	nil	96	122	22	3.1	133
36	29	Male	180	88	27.16	120	82	Normal	Normal	nil	82	120	26	1.7	169
37	56	Female	157	68	28.8	110	70	Normal	Normal	nil	88	122	36	2.8	110
38	53	Male	182	90	27.17	116	70	Normal	Normal	nil	92	132	29	1.1	150
39	75	Female	154	66	27.8	132	70	Normal	Normal	nil	96	128	68	3.2	74
40	20	Female	154	67	28.2	120	80	Normal	Normal	nil	88	132	28	3.8	167
41	21	Female	155	65	27.05	120	70	Normal	Normal	nil	78	122	12.01	2.2	166
42	19	Female	158	65	26.02	110	80	Normal	Normal	nil	81	131	18	1.9	169
43	25	Male	181	83	25.3	122	80	Normal	Normal	nil	90	122	16	3.4	129
44	20	Female	154	64	26.9	118	80	Normal	Normal	nil	88	120	22	0.9	169
45	51	Male	176	80	25.8	120	70	Normal	Normal	nil	90	122	18	2.9	112
46	59	Male	174	90	29.7	118	70	Normal	Normal	nil	92	132	132	3.1	85
47	25	Male	175	83	27.1	112	80	Normal	Normal	nil	88	111	21	4.2	129

48	38	Male	177	85	27.1	122	80	Normal	Normal	nil	91	132	28	1.3	92
49	40	Male	170	88	30.4	118	80	Normal	Normal	nil	92	130	126	3.8	74
50	65	Female	153	66	28.1	110	70	Normal	Normal	nil	98	114	68	0.8	67
51	40	Female	157	68	28.8	110	70	Normal	Normal	nil	88	122	28	2.8	84
52	46	Male	170	83	28.7	116	70	Normal	Normal	nil	82	120	28	1.1	82
53	19	Female	154	67	28.2	120	82	Normal	Normal	nil	82	120	12.2	1.7	169
54	23	Male	180	88	27.16	110	70	Normal	Normal	nil	88	122	28	2.8	110
55	20	Female	155	63	26.2	110	70	Normal	Normal	nil	92	120	18	1.4	169
56	21	Female	155	65	27.05	110	70	Normal	Normal	nil	99	124	28	3.8	166
57	30	Female	154	64	26.9	118	80	Normal	Normal	nil	70	120	22	0.9	154
58	57	Male	176	80	25.8	120	70	Normal	Normal	nil	92	122	11.05	2.9	124
59	28	Male	181	83	25.3	110	80	Normal	Normal	nil	90	120	19	1.3	210
60	19	Female	158	65	26.02	110	80	Normal	Normal	nil	81	111	7.05	1.9	169
61	21	Male	181	83	25.3	122	80	Normal	Normal	nil	76	102	3.09	3.4	181
62	55	Female	154	64	26.9	118	80	Normal	Normal	nil	88	108	22	0.9	110
63	19	Female	154	67	28.2	110	70	Normal	Normal	nil	88	102	12.5	3.8	169
64	20	Female	155	64	26.6	120	70	Normal	Normal	nil	78	122	22	2.2	169
65	20	Male	165	76	27.9	118	80	Normal	Normal	nil	99	121	26	3.1	177
66	21	Female	155	65	27.05	120	70	Normal	Normal	nil	78	122	18	0.9	166
67	22	Male	168	72	25.5	118	80	Normal	Normal	nil	92	130	16	3.8	221
68	23	Male	176	80	25.8	110	70	Normal	Normal	nil	98	114	8	0.8	219
69	21	Female	156	63	25.88	110	70	Normal	Normal	nil	92	120	18	1.4	166
70	22	Female	155	65	27.05	110	70	Normal	Normal	nil	99	124	28	3.8	164
71	22	Female	153	62	26.4	118	80	Normal	Normal	nil	70	120	22	0.9	164
72	22	Female	157	64	25.9	118	80	Normal	Normal	nil	92	130	17	3.8	169
73	47	Male	174	90	29.7	118	70	Normal	Normal	nil	92	132	29.5	3.1	96
74	80	Female	151	60	26.3	110	70	Normal	Normal	nil	88	122	25.4	2.8	102
75	42	Female	153	62	26.4	118	80	Normal	Normal	nil	70	120	28	0.8	117
76	19	Female	155	65	27.05	110	70	Normal	Normal	nil	99	124	18.05	3.8	169
77	39	Female	154	64	26.9	118	80	Normal	Normal	nil	70	120	12.8	0.9	153
78	38	Male	176	80	25.8	120	70	Normal	Normal	nil	92	122	7.05	1.8	198
79	26	Female	155	65	26.4	110	70	Normal	Normal	nil	78	112	4.37	3.1	78
80	28	Male	153	62	26.4	110	70	Normal	Normal	nil	84	107	10.3	3	107
81	40	Female	150	64	28.4	112	80	Normal	Normal	nil	90	107	28.8	3.9	74
82	27	Male	172	82	27.71	110	80	Normal	Normal	nil	79	112	66.6	3	95
83	42	Male	172	86	29.06	108	70	Normal	Normal	nil	89	112	28.9	3.1	78
84	50	Male	171	87	29.75	120	80	Normal	Normal	nil	87	108	44.1	3.2	84
85	65	Female	152	58	25.1	110	70	Normal	Normal	nil	77	112	19	2.9	67
86	35	Female	170	80	27.6	120	70	Normal	Normal	nil	88	110	10	2.2	117
87	45	Female	151	66	28.9	100	70	Normal	Normal	nil	81	108	34.5	4	82
88	67	Male	170	80	27.6	120	70	Normal	Normal	nil	90	117	8.1	2.9	143
89	35	Female	150	57	25.3	120	70	Normal	Normal	nil	90	111	4.3	3.2	121
90	42	Female	154	65	27.4	110	70	Normal	Normal	nil	88	110	23	4	98
91	29	Male	177	87	27.7	120	70	Normal	Normal	nil	76	136	15	2.2	142
92	43	Female	154	65	27.4	100	70	Normal	Normal	nil	70	112	52	2.9	79
93	45	Male	175	84	27.4	120	70	Normal	Normal	nil	90	102	3.19	3.2	96
94	76	Female	151	58	25.4	100	70	Normal	Normal	nil	76	112	4	4	127
95	23	Female	156	64	26.2	110	70	Normal	Normal	nil	78	102	11.1	3.2	110
96	39	Female	155	66	27.4	120	70	Normal	Normal	nil	92	112	26.6	2.2	100
97	67	Male	174	86	28.4	120	70	Normal	Normal	nil	88	122	121.7	3.1	79
98	55	Male	168	74	26.2	120	80	Normal	Normal	nil	88	112	9.98	4	149
99	36	Female	153	62	26.4	112	70	Normal	Normal	nil	90	109	22	2.8	120
100	40	Female	154	63	26.5	110	70	Normal	Normal	nil	92	107	21.4	4.02	99
101	62	Female	154	62	26.1	130	80	Normal	Normal	nil	78	117	17.6	2.1	108
102	50	Male	172	89	30	120	80	Normal	Normal	protein 1+	88	110	54.3	3.2	68
103	38	Female	154	66	27.8	110	80	Normal	Normal	nil	86	116	42.8	3.8	74
104	45	Female	150	58	25.7	110	70	Normal	Normal	nil	77	108	7.51	4.01	115
105	36	Female	152	60	25.9	110	70	Normal	Normal	nil	81	111	16.9	3.1	120

106	36	Male	172	75	25.3	120	80	Normal	Normal	nil	83	103	0.39	2.2	162
107	36	Female	154	64	26.9	120	80	Normal	Normal	nil	81	107	6.9	2.1	120
108	60	Male	170	86	29.75	112	80	Normal	Normal	nil	79	111	38.8	2.1	81
109	29	Male	172	78	26.3	110	70	Normal	Normal	nil	79	104	7.05	2.9	169