Research Article

Central Obesity and Chronic Kidney Disease

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Abstract

Objective: to evaluate the role of central obesity in development of chronic kidney disease.

Methods: This study was performed on (٧٠) were divided into ٢ groups according to BMI, ٢٠ with BMI< ٢٥ kg/m٢ were included into the non-obese group while ٥٠ with BMI > ٣٠ kg/m٢ were included into the obese group. Complaining of chronic kidney disease attending to Minia University Hospital. **Results:** A/C Ratio was significantly higher in central obesity group than in non-obese group and ACR was positive correlated with BMI (p=٠.٠١٩), W.H.R. (p=٠.٠٠٤), WHtR(p=٠.٠٠٩) in the obese group this indicate that higher BMI is a risk factor for the development of microalbuminuria. **Conclusion,** our study showed a significant correlation between eGFR and BMI, serum leptin and serum Adiponectin There is significant correlation between A/C Ratio and B.M.I, W.H.R., WHtR and serum Leptin.

Key words: Central obesity, A/C Ratio, Waist to Hip Ratio, Chronic Kidney Disease

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Introduction

The worldwide prevalence of obesity has increased dramatically over the last several decades. In the United States alone, over ٦٠٪ of adults ٢٠ to ٧٤ years of age are now considered overweight or obese. (Hedley et al., ٢٠٠٤).

There is an increasing epidemic of obesity in the United States (USA) and worldwide. Obesity in the USA increased dramatically during the late ١٩٩٠s, with nearly one-third of all adults classified as obese (Body Mass Index (BMI)> ٣٠ kg/m^{٢}) at the end of the decade (Flegal et al., ٢٠٠٢).

The calculation of (BMI) has been used in the definition of obesity. The (BMI) equals a person's weight in kilograms (kg) divided by their height in meters squared (m^{٢}) (Al-Lawati & Jousilahti., ٢٠٠٨).

BMI varies between males and females and according to age and level of maturity. Thus, while male and female BMIs tend to be similar in childhood, they are higher among females in adolescence. In respect of age, BMI increases from birth to around one year, then declines to around age and six, then increases through the remainder of childhood and adolescence. The point at which BMI reaches its lowest level and begins to increase is termed 'adiposity rebound', with earlier adiposity rebound being associated with increased risk of subsequent overweight (Eisenmann et al., ٢٠٠٤).

The incidence and prevalence of end-stage renal disease (ESRD) continues to grow steadily. Although much less common than obesity, ESRD is an important health problem because of the high cost of renal replacement therapy, the associated high mortality and the effect on patients' quality of life. (US Renal Data System ٢٠٠٨).

The first sign of renal injury is microalbuminuria or frank proteinuria.The prevalence of microalbuminuria was positively increased with the increasing waist-to-hip ratio. In non-hypertensive subjects (Leise et al., ٢٠٠١). Microalbuminuria is actually considered as an ideal target for early prevention of the progression of kidney and vascular damage (Czekalski, ٢٠٠٦).

Adiponectin plays a role in the suppression of the metabolic derangements that may result in diabetes, obesity, atherosclerosis (Díez & Iglesias., ٢٠٠٣) non-alcoholic fatty liver disease (NAFLD) and an independent risk factor for metabolic syndrome (Renaldi et al., ٢٠٠٩).

The renin angiotensin system (RAS) is a major regulator of sodium and water homeostasis. Among all of the components in RAS, angiotensin II (Ang II) is pre-eminent, by binding to ٢ major receptor subtypes, angiotensin II type-١ receptor (AT١R) and type-٢ receptor (AT٢R) (Luo et al., ٢٠١٥).

Protein Energy Wasting (PEW) in uremic patients is defined by anorexia, increased energy expenditure, decreased protein stores with a low serum albumin, and loss of body weight and muscle mass. The pathophysiology of PEW in CKD is complex and many factors such as inflammation, metabolic acidosis, dysregulation of appetite controlling hormones and anorexia could play a key role. The major physiological role of leptin is to regulate hunger and satiety: as such, leptin suppresses food intake and increases energy expenditure (Zhang et al., ٢٠٠٥).

We aimed in the present study to evaluate the role of central obesity in development of chronic kidney disease.

Patient and Methods

This study included ٧٠ subject was carried out at (outpatient clinic of internal medicine department of El-Minia University Hospital and Ministry of Health and population EL-Minia General Hospital) According to their body mass index (BMI), they were divided into two groups: -

- **Group (I): (Obese group)**

Included fifty persons with BMI≥ ٣٠ kg/m²,

- **Group (II): (Control group)**

Included twenty lean persons with BMI from ١٨.٥ to ٢٤.٩ kg/m².

Inclusion criteria (obese group):

- ✓ Chronic kidney disease based on "function" determined by (GFR) and "damage" assessed by the presence of increased urine excretion of protein or albumin (National Kidney., ٢٠٠٢)
- ✓ Central obesity. Waist circumference ≥ ١٠٢ cm in men and ≥ ٨٨ cm in women (Lean et al., ١٩٩٥).
- ✓ waist-hip ratio (W.H.R) (>٠.٩ for men and >٠.٨٥ for women) (Molarius, ١٩٩٩).
- ✓ Patients ٣٠ years old or more.
- ✓ Non Pregnant women.

Exclusion criteria in obese group :

- ☒ waist circumference less than ١٠٢ cm in men and ٨٨ cm in women
- ☒ waist-hip ratio (W.H.R) (< ٠.٩ for men and < ٠.٨٥ for women)
- ☒ Pregnant women.
- ☒ Patients less than ٣٠ years old.
- ☒ Normal kidney function.
- ☒ Acute renal failure.
- ☒ Patients refused to be included in the study.

All patients have given a consent regarding the participation in the present study and having the right to be withdrawn from it, according to "ethics committee", faculty of medicine, Minia University.

Clinical Assessment:

Thorough clinical history and examination with special emphasis on those related to chronic kidney disease.

A- History taking:

- **Personal history**: including name, age, sex, residence, occupation, marital status and special habits.
- **History of the present illness**: with special emphasis on symptoms suggestive of chronic kidney disease as fatigue, dyspepsia, fever, itching, change in the colour of sclera and skin, abdominal pain, abdominal distension, lower limbs swelling, bleeding tendencies, disturbed level of consciousness, and encephalopathy, etc. Symptoms related to the other systems were also recorded.
- **Past history**: particularly that related to the cause of CKD as DM, Hypertension, GN, and UTI.

B- Clinical examinations:

- **General examination:**

With special emphasis on signs of chronic kidney diseases, as disturbed level of consciousness (alert, confused, drowsy, stupor, and coma), scratch marks, pallor, earthy look, flapping tremors, and oedma of lower limbs, muscle wasting, etc.

- Measurement of blood pressure*:*

With a standard sphygmomanometer. Three measurements will be taken while the individual seated and recorded the lowest value. (Hypertension was defined as a history of

hypertension (blood pressure >١٤٠/٩٠ mmHg) that required the initiation of antihypertensive therapy by the primary physician)

- **Anthropometric measurements:**
- **Height and weight**: using full length stadiometer for height and the mass meter.
- **Body mass index (**Which equal weight(kg) /{height (cm)}^{٢}):
  * Below ١٨.٥ as underweight
  * ١٨.٥-٢٤.٩ normal
  * ٢٥–٢٩.٩ as 'overweight' (or 'pre-obese');
  * >٣٠ as 'obese

- **Waist to hip ratio (WHR):**

Waist circumference was measured using tape at mid-distance between bottom of rib cage and iliac crest. Hip circumference was measured opposite the gluteal region. WHR provide index of relative accumulation of abdominal fat (normal in men below ٠.٩ and below ٠.٨٥ in women).

- **Waist to height ratio (WHtR):**

WhtR were calculated by dividing the Waist circumference and the body height**.**

- Abdominal examination:

Inspection: for abdominal contour, divercation of recti, subcostal angle, hernias, hair distribution, scars, pigmentation and dilated veins.
Palpation: for the size, surface, consistency, edge, tenderness on the kidneys, liver and spleen.
Percussion: for ascites.
Auscultation: for bruit of renal angle.

III- Investigations:

A- Abdominal ultrasonography:

It was done using a real time equipment (Fukuda Denshi-٤٥٠٠) linear machine. A transducer with frequency of ٣.٥ MHZ was used. Aquasonic gel was spread as a film on the abdomen to prevent interposition of any air between the transducer and the skin. Examination was done in supine, left and right lateral position. The abdominal ultrasonography examination included detailed report about the following: kidneys for stones, back pressure changes or parenchymal echogenicity, liver size (average, hepatomegaly or shrunken), surface (smooth or irregular), echopattern (normal, bright, fine coarse, hepatic focal lesion), splenic size, and lastly presence or absence of ascites.

B- Laboratory investigations:

- The sample of renin in adult human is taking in the upright position
- The sample of Serum Adiponectin Overnight fasting is required.
- Diabetes mellitus was defined as a fasting glucose level of ١٤٠ mg/dL, non-fasting glucose level of ٢٠٠ mg/dL, or a history of treatment for diabetes

- **CBC**
- **Serum glucose level.**
- **Kidney function tests.**
- **Lipid profile**
- **Spot urine specimens were collected for complete urine Analysis, ACR**
- **Serum Insulin**
- **Serum Renin**
- **Serum Angiotensin**١
- **Serum adiponectin.**
- **Serum leptin**

C- Estimation of GFR:

CKD-EPI CREATININE EQUATION (٢٠٠٩)

eGFR = ١٤١ x min (S_{Cr}/κ, ١)$^{\alpha}$ x max (S_{Cr} /κ, ١)$^{-١.٢٠٩}$ x ٠.٩٩٣^{Age} x ١.٠١٨ [if female] x ١.١٥٩ [if Black] (Levey et al., ٢٠٠٩)

Abbreviations / Units
eGFR (estimated glomerular filtration rate) = mL/min/١.٧٣ m^{٢}
S_{Cr} (standardized serum creatinine)=mg/dL
κ = ٠.٧ (females) or ٠.٩ (males)
α = -٠.٣٢٩ (females) or -٠.٤١١ (males)
min = indicates the minimum of S_{Cr}/κ or ١
max = indicates the maximum of S_{Cr}/κ or ١
age = years

Statistical analysis

The data of all patients were fed into an IBM-compatible computer and statistical software packages namely (SPSS) for windows student version ٢٠.٠ was used to analyze these data. Data were expressed as mean ± SD for parametric variables and as numbers and percent for non-parametric variables.

Statistical analysis was done to evaluate the difference between groups under study as regard the various parameters using t-student test. The non-parametric variable version compared by chi-square test.

Correlation was tried in-between the essential studied parameters by Pearson s correlation tests. This was expressed as weak correlation ٠-٠.٢٤, fair, ٠.٢٥-٠.٤٩, and moderate ٠.٥٠-٠.٧٤, strong > ٠.٧٥. This was expressed as

probability of value (p value) the difference was considered significant if p value < ٠.٠٥.

Results

Demographic data of all patients:

The study included ٧٠ According to their body mass index (BMI), and Waist to Hip Ratio they were divided into two groups :

Group (I): Obese group.

This group include fifty persons with chronic kidney disease and BMI≥ ٣٠ kg/m², there were ٣٥males (٧٠٪), and ١٥ females (٣٠٪). their ages were ranged between ٣١and ٦٥ with a mean ٤٩.٩±٨.٢ years and their mean BMI is ٣٥.٢±٣.٣kg/m². The number of smoker was ٣٠, number of nonsmoker was ٢٠.

Group (II): Control group.

This group include twenty lean person with BMI from was ١٩ to ٢٤.٦ kg/m², their ages ranged from ٣١ to ٦٢ years with a mean ٤١.٨±٨.٦ years their mean BMI is ٢٢.١±١.٣ kg/m². There were ١٢males (٦٠٪), and ٨ females (٤٠٪). Normal kidney function the number of smoker was ٥ (٢٥٪), number of nonsmoker was ١٥(٧٥٪).

Table (١): Demographic data of all patients with chronic Kidney Disease.

Demographic data	Obese (n=٥٠) Mean±SD	Non obese (n=٢٠) Mean±SD
Age (years):	٤٩.٩±٨.٢	٤١.٨±٨.٦
Range	٣١-٦٥	٣١-٦٢
Sex:		
Male, N(%)	٣٥ (٧٠٪)	١٢(٦٠٪)
Female, N(%)	١٥ (٣٠٪)	٨(٤٠٪)
Smoking NO	٢٠	١٥
YES	٣٠	٥

Table (٢): DM, HTN and Metabolic syndrome in central obesity patients.

Diabetes	
Yes	١١ (٢٢٪)
NO	٣٩ (٧٨٪)
Hypertension	
Yes	١٣ (٢٦٪)
No	٣٧ (٧٤٪)
Metabolic Syndrome	
Yes	١٤(٢٨٪)
No	٣٦(٧٢٪)

Diabetes mellitus in central obesity patients were ١١(٢٢٪) patients and ٣٩(٧٨٪) patients were non Diabetics. Hypertension in central obesity patients were ١٣(٢٦٪) and ٣٧(٧٤٪) were not Hypertensive. Patients with criteria of Metabolic syndrome were ١٤(٢٨٪) and ٣٦ (٧٢٪) were not Metabolic Syndrome.

Table (٣): Stages of CKD in central obesity patients

CKD Stages	N (%)
Stage ١	٠ (٠٪)
Stage ٢	٠ (٠٪)
Stage ٣	٤٧ (٩٤٪)
Stage ٤	٣ (٦٪)
Stage ٥	٠ (٠٪)

Stages of chronic kidney disease in central obesity patients showing CKD Stage ١{٠(٠٪)}, CKD Stage ٢{٠(٠٪)}, CKD Stage ٣ {٤٧(٩٤٪)}, Stage ٤ {٣(٦٪)}and CKD Stage٥{٠(٠٪)}

Table (٤): A/C Ratio in all patients in the present study.

A/C Ratio	Central obese group	Control group
A١ (< ٣٠) (mg/g)	٨ (١٦٪)	٢٠ (١٠٠٪)
A٢ (٣٠-٢٩٩) (mg/g)	٢٤ (٤٨٪)	
A٣(> ٣٠٠) (mg/g)	١٨ (٣٦٪)	

A/C Ratio in all patients in the present study. in central obesity patients showing A١ (< ٣٠) (mg/g) ٨ (١٦٪), A٢ (٣٠-٢٩٩) (mg/g) ٢٤ (٤٨٪) and A٣(> ٣٠٠) (mg/g) ١٨ (٣٦%). In other hand A/C Ratio in control group A١ (< ٣٠) (mg/g) ٢٠ (١٠٠٪).

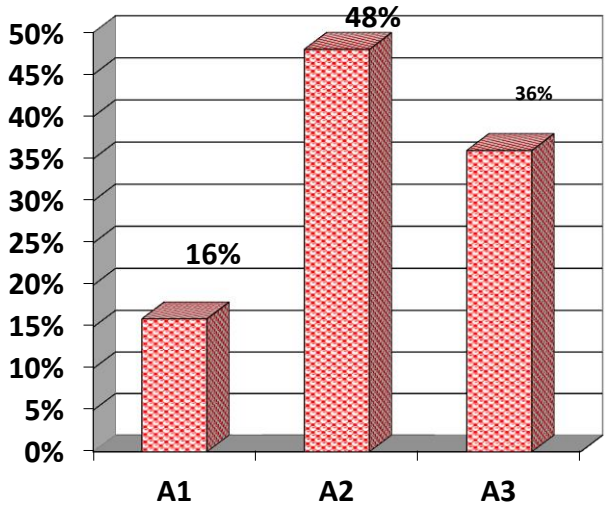

Fig. (١): A/C Ratio in obese in obese group

Table (٥): Abdominal ultrasonography in all patients in the present study.

Abd/us	Central obese group	Control group
Normal	٧ (١٤٪)	٢٠ (١٠٠٪)
Grade ١	٣٠ (٦٠٪)	
Grade ٢	١٣ (٢٦٪)	

Abdominal ultrasonography in all patients in the present study. in central obesity patients showing Normal Echogenicity was ٧ patients, Echogenicity Grade ١ was ٣٠ patients and Echogenicity Grade ٢ was ١٣ patients in other hand Abdominal ultrasonography in control group Normal Echogenicity was ٢٠ patients.

Table (٦): Comparison between central Obese and control according to the following parameters

	Obese (n=٥٠) Mean±SD	Range	Non obese (n=٢٠) Mean±SD	Range	P-value	
Weight (Kg)	١٠٢.٦± ١٢.١	٧٩-١٢٦	٦٥.٨±٦.٣	٥٣-٧٩	٠.٠٠٠١	HS
Height (Cm)	١٧٠.١±٥.٢	١٥٩-١٨١	١٧٠±٦.١	١٦٤-١٨١	٠.٤٨٤	NS
B.M.I (Kg/m٢)	٣٥.٢±٣.٣	٣١-٤٣	٢٢.١±١.٣	١٩-٢٤.٦	٠.٠٠٠١	HS
W.H.R.	١.٢±٠.٢	١.٠-١.٤	٠.٨٣±٠.٠٩	٠.٧-١	٠.٠٠٠١	HS
WHtR	٠.٥٩±٠.٠٤٥	٠.٥١-٠.٦٨	٠.٤١±.٠٢٢	٠.٣٧-٠.٤٥	٠.٠٠٠١	HS
SBP (mmHg)	١٣٠.٧±١٩	١٠٠-١٨٠	١١٤.٧±٧.٣	١٠٠-١٣٠	٠.٠٠٢	HS
DBP (mmHg)	٨٢.٦±١٢	٦٠-١١٠	٧٣.٥±٦.٧	٦٠-٨٠	٠.٠١٠	S
FBS(mg/dl)	١٠٠.٨±٢١.٩	٦٥-١٤٣	٨٢.٢±٨.٦	٦٩-٩٦	٠.٠٠١	HS
PPBS(mg/dl)	١٧٤.٢± ٦١.٦	١٢١-٣٥٠	١١٣.٢±١٠.٩	٩٠-١٢٧	٠.٠٠١	HS
F.Insulin (mU/L)	١٢.٤±٥.٣	٦-٢٧	٩.٣±١.٦	٧-١٢	٠.٠٠٧	HS
PP.Insulin (mU/L)	٦٩.٥±٤١.٦	١٩-١٧١	٣١.٨±١.٩	٢٩-٣٥	٠..٠٠٣	HS
Urea (mg/dl)	٨٠.٧±١٠.٧	٥٤-١١٢	٢٥.٣±٧.١	١٥-٣٩	٠.٠٠٠١	HS
Creatinine (mg/dl)	١.٩±٠.٣	١.٦-٢.٧	٠.٩٦±٠.٢	٠.٦-١.٣	٠.٠٠٠١	HS
eGFR ml/m	٤٣.٤±٨.٣	٢٧.٣±٥٨.١	١٢٧.٢±٩.١	١١٢-١٤٢	٠.٠٠٠١	HS
A/C Ratio(mg/g)	٣٠٧.١±٣٣٧.٢	١٤-١٥٤٠	١٤.٦±٤.٠	٨-٢١	٠.٠٠١	HS
Cholesterol(mg/dl)	٢١٠.٤±٢٦.٩	١٦٨-٢٥٩	١٦٨.٦±٢٣.٢	١٢٧-٢٣٩	٠.٠٠٠١	HS
LDL (mg/dl)	١٢٤.٥±٣٧.٤	٧٢-١٨٤	٨٤.٩±٧.٤	٧٣-٩٩	٠.٠٠٠١	HS
HDL (mg/dl)	٤١.٤ ± ٤.٤	٣٦-٥٧	٤٨.٣±٦.١	٤٠-٥٩	٠.٠٠٠١	HS
Triglycerides(mg/dl)	١٨٠.٩±٦٤.٢	٧٨-٣٢٧	٨٠.٧±٥.٦	٧١-٩٠	٠.٠٠٠١	HS
Renin (pg/ml)	٥٦.٤±١٢.٠	٣٨-٨٩	٦.٩±١.٩	٣-١٠	٠.٠٠٠١	HS
Angiotensin١(ng/ml)	٢٠.٥±٣.١	١٦-٢٧	٢.٥±١.٠	٠.٩ -٤.١	٠.٠٠٢	HS
Adiponectin (ng/ml)	٦.٢±٢.٢	٣-١١	٨.٤±٢.١	٥-١٢	٠.٠٠٢	HS
Leptin (ng/ml)	٥٠.١±١٦.٨	٢٧-٧٧	١١.٧±٤.١	٦.٨-١٨.٢	٠.٠٠٠١	HS

Group (I): Obese group.
The mean Weight (Kg) was ١٠٢.٦± ١٢.١, mean Height (Cm) was ١٧٠.١± ٥.٢, mean BMI ٣٥.٢±٣.٣ mean W.H.R. was ١.٢±٠.٢, mean WHtR٠.٥٩±٠.٠٤٥. The mean SBP (mmHg)was ١٣٠.٧±١٩, mean DBP (mmHg) was ٨٢.٦±١٢, mean FBS(mg/dl) ١٠٠.٨±٢١.٩ mean PPBS (mg/dl) was ١٧٤.٢± ٦١.٦, mean F.Insulin (mU/L) ١٢.٤±٥.٣ and mean PP.Insulin (mU/L) ٦٩.٥±٤١.٦. the mean Urea (mg/dl) was ٨٠.٧± ١٠.٧, mean Creatinine (mg/dl) was ١.٩±٠.٣, mean eGFR ml/m ٤٣.٤±٨.٣ and mean A/C Ratio was ٣٠٧.١±٣٣٧.٢, the mean Cholesterol (mg/dl) was ٢١٠.٤±٢٦.٩, mean LDL (mg/dl) was ١٢٤.٥±٣٧.٤, mean HDL (mg/dl) ٤١.٤ ± ٤.٤ and mean Triglycerides (mg/dl) was١٨٠.٩±٦٤.٢, the mean Renin (pg/mL) was ٥٦.٤±١٢.٠, mean Angiotensin١ (ng/ml) was ٢٠.٥±٣.١, mean Adiponectin (ng/ml) ٦.٢±٢.٢ and mean Leptin (ng/ml) was ٥٠.١±١٦.٨.

Group (II): Control group.
The mean Weight (Kg) was ٦٥.٨±٦.٣, mean Height (Cm) was ١٧٠±٦.١, mean BMI ٢٢.١±١.٣, mean W.H.R. was ٠.٨٣±٠.٠٩, mean WHtR ٠.٤١±.٠٢٢, the mean SBP (mmHg) was ١١٤.٧±٧.٣, mean DBP (mmHg) was ٧٣.٥±٦.٧, mean FBS(mg/dl) ٨٢.٢±٨.٦ mean PPBS (mg/dl) was ١١٣.٢±١٠.٩, mean F.Insulin (mU/L) ٩.٣±١.٦ and mean PP.Insulin (mU/L) ٣١.٨±١.٩, the mean Urea (mg/dl) was ٢٥.٣±٧.١, mean Creatinine (mg/dl) was ٠.٩٦±٠.٢and mean eGFR ml/m ١٢٧.٢±٩.١ mean A/C Ratio was١٤.٦±٤.٠, the mean cholesterol(mg/dl) was ١٦٨.٦±٢٣.٢, mean LDL (mg/dl) was ٨٤.٩±٧.٤, mean HDL (mg/dl) ٤٨.٣±٦.١ and mean Triglycerides (mg/dl) was٨٠.٧±٥.٦, the mean Renin (pg/mL) was ٦.٩±١.٩, mean Angiotensin١ (ng/ml) was ٢.٥±١.٠, mean Adiponectin (ng/ml) ٨.٤±٢.١ and mean Leptin (ng/ml) was ١١.٧±٤.١.

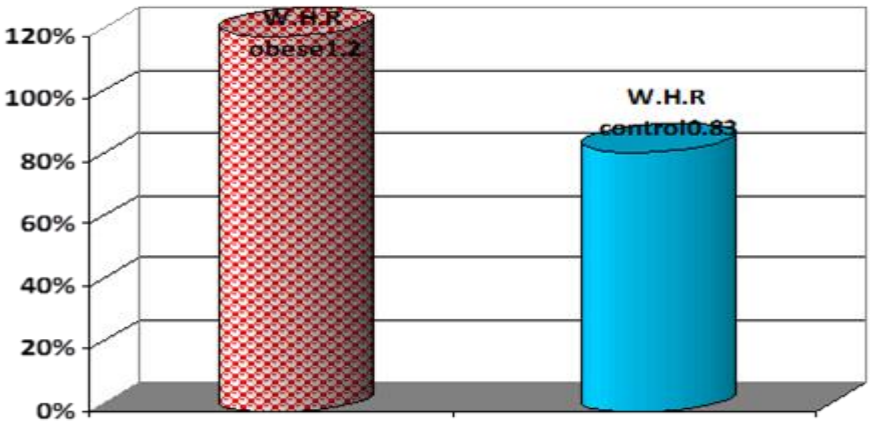

Fig. (٢): Mean of W.H.R in obese and non-obese groups

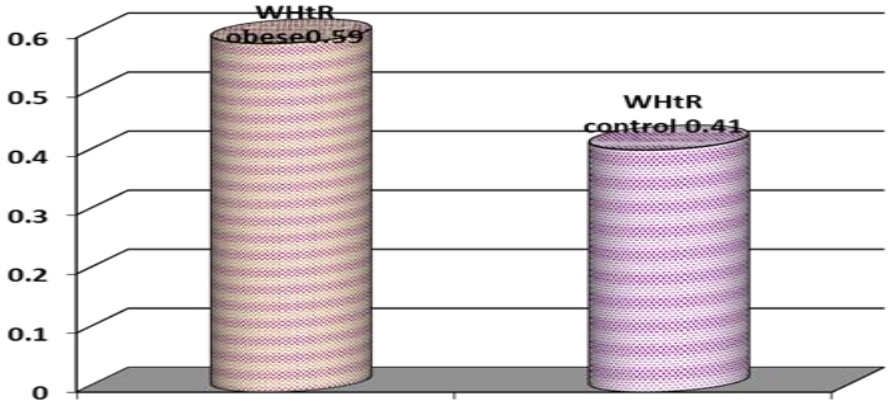

Fig. (٣): Mean of WHtR in obese and non-obese groups

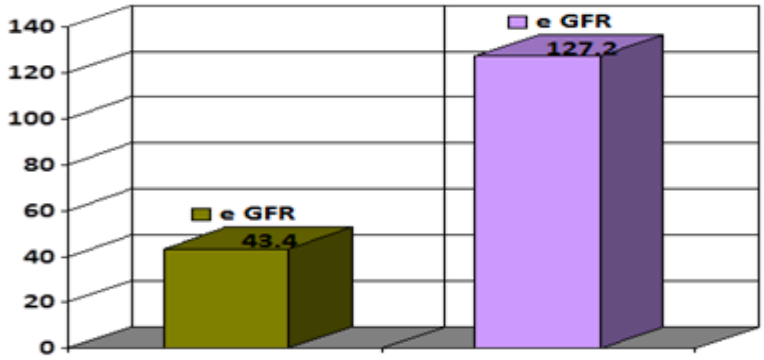

Fig. (٤): Mean of eGFR in central obese and control groups

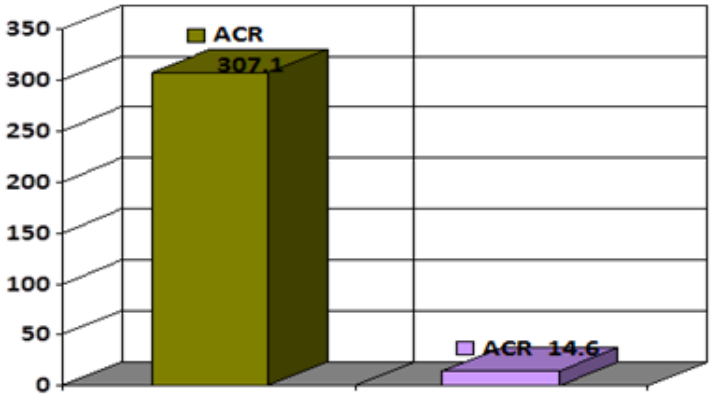

Fig. (٥): Mean of A/C Ratio in central obese and control groups

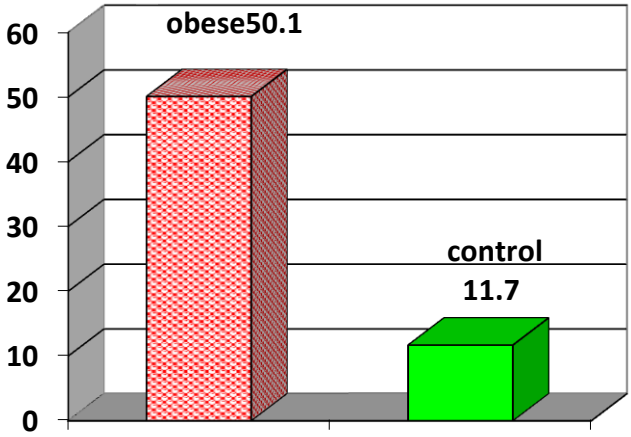

Fig. (٦): Mean of leptin in Obese and Non obese groups.

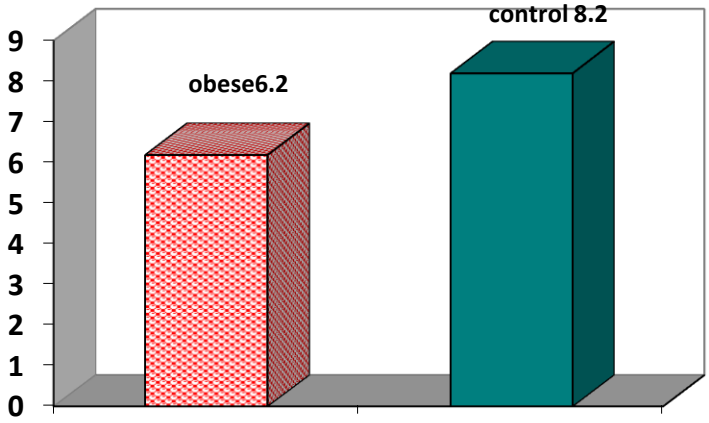

Fig. (٧): Mean of adiponectin in Obese and Non obese groups.

As shown in table (٧), the mean differences between Male and Female according to GFR, WHR, BMI, Adiponectin, renin and angiotensin in central Obese patients in present study. The significant differences were found with eGFR (p=٠.٠٠٠٢), leptin (p=٠. .٠٣٧) and ACR (p=٠. .٠٣٧) while there were no significant differences between (B.M.I, W.H.R., renin, Angiotensin١, and Adiponectin.

Table (٧): Comparison between male and female according to eGFR, WHR, BMI, Adiponectin, leptin, renin, angiotensin and A/C Ratio in obese subject.

	Male Mean ± SD	Female Mean±SD	P-value	Sig.
eGFR	٤٦.١±٧.٤	٣٧.١±٦.٨	٠.٠٠٠٢	HS
WHR	١.٢±.٠٨	١.٢±.٠٩	٠.٣٠٢	NS
WHtR	٠.٥٦±٠.٠٤٥	٠.٥٨±٠.٠٥	٠.٣٣٨	NS
BMI	٣٥.٢±٣.٤	٣٥.٢±٣.٠	٠.٩٧١	NS
Adiponectin	٦.٠±٢.٣	٦.٤±٢.٠	٠.٥٦٥	NS
Leptin	٤٨.٩±١٧.٤	٥٣.٠±١٥.٣	٠. ٤٣٧	NS
Renin	٥٥.٤±١٢.٢	٥٨.٧±١١.٥	٠.٣٩٧	NS
Angiotensin١	٢٠.٢±٢.٩	٢١.٢±٣.٤	٠.٣٠٩	NS
ACR	٢٤٢.٥±٢٨٨.٠	٤٥٧.٩±٤٠٢.٣	٠..٠٣٧	S

Table ٨: Correlation between A/C Ratio and the following parameter in obese subject

	r	P-value	Sig.
Age (Year)	-٠.١٢٧-	٠.٣٧٩	NS
Sex	٠.٣٧٩	٠.٠٣٧	S
Weight (Kg)	-٠.٣٨١-	٠.٠٠٦	HS
Height (Cm)	-٠.٢٣٤-	٠.١٠١	NS
B.M.I (Kg/m٢)	-٠.٣٣٠-	٠.٠١٩	HS
W.H.R.	-٠.٣٩٥-	٠.٠٠٤	HS
WHtR	٠.٣٦٦	٠.٠٠٩	HS
FBS(mg/dl)	-٠.٠١٩-	٠.٨٩٥	NS
PPBS(mg/dl)	٠.٠٧٩	٠.٥٨٥	NS
SBP (mmHg)	-٠.٣٣٠-	٠.٠١٩	HS
DBP (mmHg)	-٠.٢٩٦-	٠.٠٣٧	S
Urea (Mg/dl)	٠.٣٤٢	٠.٠١٥	S
Creatinine (Mg/dl)	-٠.٠٢٢-	٠.٨٧٨	NS
Cholesterol(mg/dl)	٠.٢٢١	٠.١٢٢	NS
LDL(mg/dl)	٠.١٤٥	٠.٣١٥	NS
HDL(mg/dl)	٠.١٤١	٠.٣٣٠	NS
Triglycerides(mg/dl)	٠.٢٤٢	٠.٠٩٠	NS
F.Insulin (mU/L)	-٠.١٤٦-	٠.٣١١	NS
PP.Insulin (mU/L)	-٠.١١٤-	٠.٤٣٢	NS
Renin (pg/mL)	-٠.٢٨٠-	٠.٠٥١	NS
Angiotensin١ (ng/ml)	-٠.٢٣٠-	٠.١٠٨	NS
Adiponectin (ng/ml)	٠.١٥١	٠.٢٩٧	NS
Leptin (ng/ml)	-٠.٣٩٧-	٠.٠٠٤	HS
Abd u/s	٠.٢٥٥	٠.٠٧٣	NS
eGFR	--٠.١١٢-	٠.٤٣٧	NS

As shown in table (٨) Significant correlation was found with sex (r= ٠.٣٧٩; p=٠.٠٣٧), Weight (kg) (r=-٠.٣٨١-; p= ٠.٠٠٦). BMI (r= -٠.٣٣٠-; p=٠.٠١٩), W.H.R. (r=-٠.٣٩٥-; p= ٠.٠٠٤) and WHtR (r=٠.٣٦٦; p= ٠.٠٠٩), systolic blood pressure (mmHg) (r= -٠.٣٣٠-; p=٠.٠١٩), diastolic blood pressure (mmHg) (r=-٠.٢٩٦- ; p= ٠.٠٣٧), Urea (mg/dl) (r= ٠.٣٤٢;٠.٠١٥), Leptin (ng/ml) (r=-٠.٣٩٧-; p= ٠.٠٠٤). While there was no significant correlation between A/C Ratio and other parameters.

Table ٩: Correlation between eGFR and the following parameter in obese patients

	r	P-value	Sig.
Age (Year)	-٠.١٠٤	٠.٤٧٢	NS
Weight (Kg)	-.٣٢٩	٠.٠٢٠	S
Height (Cm)	٠.٠٨٤	٠.٥٦٢	NS
B.M.I (Kg/m٢)	-٠.٤٥٨	٠.٠٠١	HS
W.H.R.	-٠.١٣٠-	٠.٣٦٧	NS
WHtR	-.٠١٣-	٠.٩٢٧	NS
FBS(mg/dl)	٠.٠٧٢	٠.٦١٨	NS
PPBS(mg/dl)	٠.٠٨٤	٠.٥٦٣	NS
SBP (mmHg)	-٠.٠١٥	٠.٩١٦	NS
DBP (mmHg)	-.٠٥٨	٠.٦٨٨	NS
Urea (Mg/dl)	-٠.٣٠٨	٠.٠٣٠	S
Creatinine (Mg/dl)	-٠.٧٣٣	٠.٠٠٠١	HS
Cholesterol (mg/dl)	-٠.١٦٥	٠.٢٥١	NS
LDL (mg/dl)	-٠.٠٧٧	٠.٥٩٤	NS
HDL (mg/dl)	-٠.٢٢٨-	٠.١١١	NS
Triglycerides (mg/dl)	-٠.٠٠١	٠.٩٩٥	NS
F.Insulin (mU/L)	٠.٠٠٩	٠.٩٥٣	NS
PP.Insulin (mU/L)	-٠.٠٨٥	٠.٥٥٦	NS
Renin (pg/mL)	-٠.٠٢٢	٠.٨٧٩	NS
Angiotensin١ (ng/ml)	٠.٠٣٤	٠.٨١٤	NS
Adiponectin (ng/ml)	٠.٢٨٥	٠.٠٤٥	S
Leptin (ng/ml)	-٠.٤٧٩	٠.٠٠٠١	HS
Abd u/s	-٠.٥١١	٠.٠٠٠١	HS
ACR	-٠.١١٢	٠.٤٣٧	NS

As shown in table (٩) Correlation between eGFR and other variables, Significant correlation was found with BMI (r= - ٠.٤٥٨; p=٠.٠٠١), Weight (kg) (r=-.٣٢٩; p= ٠.٠٢٠), Urea (Mg/dl) (r= - ٠.٣٠٨; p=٠.٠٣٠), Creatinine (Mg/dl) (r= -٠.٧٣٣; p=٠.٠٠٠١) Adiponectin (ng/ml) (r= ٠.٢٨٥; p= ٠.٠٤٥), Leptin (ng/ml) (r= -٠.٤٧٩; p= ٠.٠٠٠١) and Abdominal ultrasonography (r=-٠.٥١١ ; p=٠.٠٠٠١) ,while there was no significant correlation between eGFR and (Age ,Height, W.H.R., WHtR, FBS, PPBS, SBP, DBP, Triglycerides, LDL, HDL, cholesterol, F. Insulin, PP.Insulin, renin and Angiotensin١.

As shown in table (١٠) associations between indexes of central obesity and CKD, multivariate logistic regression model with adjustment for age and gender was used. there are significantly associated with Waist Height Ratio (WHtR) Cholesterol, post prandial insulin, serum creatinine, A/C Ratio.

Table (١٠): Multiple logistic regression of CKD and selected risk factors.

Dependent variables	B	S.E.	Sig.	Odds Ratio	٩٥.٠٪ C. I.for Odds Ratio	
					Lower	Upper
Weight	.٠٤١	.٠٢٨	.١٤٠	NS	.٩٨٧	١.١٠١
WHR	٤.٦٩٥	٣.٦٥٢	.١٩٩	NS	.٠٨٥	١٤٠٥٦٦.٥٥٠
WHtR	٥٨.٠٧٣	٢٩.٢٦٣	.٠٤٧	S	٢.٠٥٠	١.٣٤٧E+٠٥٠
BMI	.٠٨١	.١٠١	.٤٢٥	NS	.٨٨٩	١.٣٢١
leptin	-.٠٠٨-	.٠٣٦	.٨١٩	NS	.٩٢٤	١.٠٦٥
F.Insulin	-.٠٥٦-	.٠٥٩	.٣٤١	NS	.٨٤٢	١.٠٦١
PP.Insulin	-.٠١٦-	.٠٠٨	.٠٣٩	S	.٩٧٠	.٩٩٩
B.Urea	-.٠٤١-	.٠٣١	.١٨٨	NS	.٩٠٣	١.٠٢٠
S.Creatinine	-٣.٦٥٧-	١.٢٨٠	.٠٠٤	S	.٠٠٢	.٣١٧
FBS	-.٠٠٨-	.٠١٤	.٥٨١	NS	.٩٦٤	١.٠٢١
PPBS	-.٠٠٤-	.٠٠٥	.٤٢١	NS	.٩٨٧	١.٠٠٦
TRIGLYCIRED	-.٠٠٧-	.٠٠٥	.١٦٨	NS	.٩٨٣	١.٠٠٣
HDL	.١٤٠	.٠٩٨	.١٥٠	NS	.٩٥٠	١.٣٩٣
LDL	-.٠١١-	.٠٠٩	.٢١٠	NS	.٩٧٢	١.٠٠٦
CHOLESTEROL	-.٠٢٨-	.٠١٣	.٠٣٢	S	.٩٤٨	.٩٩٨
angiotensin١	-.٠٣٠-	.١٠٣	.٧٦٧	NS	.٧٩٤	١.١٨٦
Renin	.٠٠٤	.٠٢٧	.٨٧٧	NS	.٩٥٣	١.٠٥٨
Adiponectin	-.٠٦٧-	.١٣٩	.٦٢٧	NS	.٧١٢	١.٢٢٧
SBP	.٠٠٩	.٠١٧	.٥٩٤	NS	.٩٧٦	١.٠٤٤
DBP	.٠٠٥	.٠٢٧	.٨٦٥	NS	.٩٥٣	١.٠٥٨
ACR	.٠٠٣	.٠٠١	.٠٣٠	S	١.٠٠٠	١.٠٠٦
Smoker	-١.١٧٠	١.٢٦١	.٣٥٣	NS	.٠٢٦	٣.٦٧٤
Metabolic Syndrome	.٢٦٨	١.٢٦٧	.٨٣٢	NS	.١٠٩	١٥.٦٧٩

Discussion

Some of the major questions related to treatment of chronic kidney disorders are whether weight loss interventions by diet and lifestyle changes, pharmacological therapy, or surgical interventions, such as bariatric surgery, are effective in preventing and/or slowing development and progression of CKD. Also, the most effective therapies for hypertension and diabetes in obese subjects have not been fully elucidated with appropriate clinical trials (Hall et al., ٢٠١٤).

Ectopic deposition of lipids into nonadipose tissues, such as the kidney, often occurs in obesity. Excessive lipid deposition into nonadipose organs can lead to accumulation of toxic metabolites, such as diacylglycerols and ceramides, derived from metabolism of fats/fatty acids and sphingolipids (Unger & Scherer., ٢٠١٠).

Obesity is associated with glomerular hyper-filtration and hypertension. Obesity related glomerulopathy (ORG) is clinically characterized by moderate proteinuria, minimal edema, lower serum cholesterol and higher serum albumin (Srivastava., ٢٠٠٦).

ORG has been described as a secondary form of focal segmental glomerulosclerosis (FSGS) occurring in obese patients. The first research between obesity and renal injury was reported in ١٩٧٤ (Weisinger et al., ١٩٧٤). However, the improvement in proteinuria might not correlate with histological change. The pathology of ORG may be biased by the fact that most of the kidney samples were obtained in patients with proteinuria. It suggested that ORG could not be the histopathological feature in nonproteinuric obese individuals with renal dysfunction (Ding et al., ٢٠١٥).

Most of central obese patients in our study were ٣٥males (٧٠٪), and ١٥ females (٣٠٪). The mean age of them was ٤٩.٩±٨.٢ years (range: ٣١-٦٥ years). Also in non-obese person's males were ١٢(٦٠٪) and females ٨ (٤٠٪).

Our study the mean differences between Male and Female in central obesity patients according to eGFR, WHR, WHtR, BMI, Adiponectin, leptin, renin and angiotensin١ in present study. The significant differences were found with eGFR (p=٠.٠٠٠٢),), A/C Ratio (٠.٠٣٧) while there were no significant differences between (B.M.I, W.H.R., WHtR, renin, Angiotensin١ and Adiponectin.

The BMI–CRF risk relationship seemed to be somewhat stronger and evident in a lower BMI range in men than in women. However, no BMI gender interactions attained statistical significance. Therefore, the observed difference is likely to be a chance finding (Hsu et al., ٢٠٠٦).

In our study A/C Ratio was significantly higher in central obesity group than in non-obese group and ACR was positive correlated with BMI (p=٠.٠١٩), W.H.R. (p=٠.٠٠٤), WHtR (p=٠.٠٠٩) in the obese group this indicate that higher BMI is a risk factor for the development of microalbuminuria. Our findings are consistent with other reports that link higher BMI with albuminuria (Kramer et al., ٢٠٠٥).

In a cross sectional study, Bello et al., ٢٠١٠ found that the main determinants of microalbuminuria on the population level were increased age, diabetes, obesity and family history of hypertension and obesity had greater odds for microalbuminuria than diabetes and hypertension.

In our study Significant correlation was found with Urea (mg/dl) (p=٠.٠٣٠), Creatinine (mg/dl) (p= ٠.٠٠٠١) and Abd u/s (p= ٠.٠٠٠١).in agree with our result Siddappa, et al. found A statistically significant positive correlation between serum creatinine and cortical echogenicity grading (P=٠.٠٠٤). Renal echogenicity and its grading correlates better with serum creatinine in CKD than other sonographic parameters like longitudinal size (P = ٠.٠٨٥), parenchymal thickness (P = ٠.٠٤٦), and cortical thickness (P = ٠.٦٥٦). As serum creatinine is an indicator of kidney function, renal echogenicity is a better parameter to estimate renal function with the added advantage of irreversibility when compared to serum creatinine, which improves with kidney replacement therapy like hemodialysis, peritoneal dialysis, and renal transplantation in chronic kidney disease (Siddappa, et al., ٢٠١٣).

Most adipocytokines are positively correlated with obesity; however, adiponectin is negatively correlated with obesity and appears to be down-regulated in more obese patients (Hotta et al., ٢٠٠٠ and Ryan et al., ٢٠٠٣).

In our obese subjects, the mean s. adiponectin (٦.٢ng/ml) was significantly lower than in non-obese subjects (٨.٤٢ng/ml), this results is agree with (Tadokoro et al., ٢٠١٠) Study who found adiponectin level are paradoxically lower in obese than in lean humans, despite increased adipose tissue mass.

It is clear that excess weight gain, especially when accompanied by increased visceral fat, is associated with many features of the metabolic syndrome which increase the risk for the development of CKD. Ectopic fat accumulation in and around the kidney may also have adverse consequences on renal function. Markers of visceral adiposity such as waist circumference is easily obtained in the clinic setting and may provide valuable prognostic information. More detailed examinations of specific fat depots evaluated with magnetic resonance and computed tomography imaging may also provide useful information related to the risk for development of CKD.

In agree with our study the most recent Prospective cohort study. Chang et al., ٢٠١٦; Overweight and obesity are associated with an increased incidence of CKD in metabolically healthy young and middle-aged participants. These findings show that metabolically healthy obesity is not a harmless condition and that the obese phenotype, regardless of metabolic abnormalities, can adversely affect renal function.

In conclusion, our study showed a significant correlation between eGFR and BMI, serum leptin and serum Adiponectin There is significant correlation between A/C Ratio and B.M.I, W.H.R., WHtR and serum Leptin. There is significant correlation between BMI and serum Adiponectin and serum Leptin. an

association between WHR and serum Adiponectin and serum leptin. Our results suggested the possibility that adiponectin and leptin plays a role as an endogenous protective factor against obesity-related initial renal injury. This was a hypothesis-generating survey, however, and longitudinal and intervention studies will be needed to clarify our hypothesis.

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