ISSN 2520-5110

IAM
INTERNATIONAL ANNALS
of Medicine

JIFACTOR 3.12

November, 2017 2017; Vol1; Issue10 http://iamresearcher.online

Microalbuminuria in Obese Children and Adolescents and the Metabolic Syndrome

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ABSTRACT

Insulin resistance is a common feature of childhood obesity and is considered to be an important link between adiposity and development of type 2 diabetes mellitus and cardiovascular disease. It is also a major contributing factor to renal injury. Microalbuminuria (albumin excretion 20-200 mg⁄min or 30-300 mg⁄gram creatinine) is now considered an early marker of renal damage in non-diabetic patients.

Objectives: to evaluate the association of obesity and microalbuminuria among obese subjects and its relation to metabolic syndrome components. **Methods:** This cross-sectional study was conducted on sixty-two obese children and adolescents randomly recruited from the Obesity Clinic, Pediatric Hospital, Ain-Shams University. Anthropometric data were collected, fasting serum insulin, glucose and serum lipid profile were measured. The homeostasis model assessment of insulin resistance (HOMA-IR) was used to calculate in vivo insulin resistance. Oral glucose tolerance test and urinary albumin concentrations were done. **Results:** Microalbuminuria was detected in 18 cases (29%) , metabolic syndrome in 4 cases (6.4%), impaired OGTT in 9.6%. Impaired fasting insulin and high serum insulin after 2 hours in OGTT in 3.2% of cases. Abnormal lipid profile was significantly associated with microalbuminuria. **Conclusion:** Microalbuminuria is strongly associated with impaired fasting insulin, and abnormal lipid profile.

Keywords: Obesity, Insulin resistance, Metabolic syndrome, Renal endothelial damage, Children.

1. INTRODUCTION

The prevalence and severity of obesity in children and adolescents is dramatically increasing worldwide and has reached approximately 10% in a large number of developing countries[(1)]. Childhood obesity is associated with several metabolic and cardiovascular complications[(2)] and it increases the risk of kidney diseases[(1)], as well as its progression[(3)]. Obesity is strongly associated with the two most common causes of end-stage renal disease (ESRD), namely diabetes and hypertension[(4)]. Insulin resistance is a common feature of childhood obesity and is considered to be an important link between adiposity and the associated risk of type 2 diabetes mellitus and cardiovascular disease. It is also a key component of the metabolic syndrome. Reduced insulin sensitivity and hyperinsulinemia are among the most important factors of metabolic syndrome contributing to renal injury[(5)]. Microalbuminuria (albumin excretion 20-200 mg⁄min or 30-300 mg⁄gram creatinine) is now considered an early marker of renal damage in non-diabetic patients[(6)].

The objective of the study was to evaluate the association of obesity and microalbuminuria among obese children and adolescents and its relation to metabolic syndrome

To Cite This Article: Heba H El Sedfy, Nadin N Toaima, Mayada A Ibrahim, Lerine B El Din. Microalbuminuria in Obese Children and Adolescents and the Metabolic Syndrome. *International Annals of Medicine. 2017;1(10).* https://doi.org/10.24087/IAM.2017.1.10.301

2. METHODS

This was a cross-sectional study comprising 62 children and adolescents with simple obesity. Their age ranged from 4-14 years with mean age 10.9 ± 3.11 years randomly recruited during the period from April 2015 to March 2016.

Patients were included in the study if the body mass index (BMI) was ≥ 95th centile for age and sex according to the U.S. Centers for Disease Control and Prevention (CDC). Patients with fever, infections, renal diseases, systemic lupus erythematosus, genetic obesity syndromes, secondary obesity as endocrine causes of obesity and drug-induced obesity or diabetes and patients with albuminuria associated with urinary tract infections were excluded from the study.

All parents signed an informed consent prior to recruitment and so did the patients if this was deemed appropriate after full explanation of the study. The study protocol was approved by the local Ethical Committee of Pediatric Hospital, Ain-Shams University. Demographic and clinical data of these children were recorded.

All subjects (controls and obese children) were subjected to a full medical history that included clinical examination, and routine laboratory investigations. Family history for obesity, hypertension type 2 diabetes mellitus, dyslipidemia, polycystic ovarian syndrome and metabolic syndrome were obtained by questionnaires filled in by the patients. Fasting blood glucose, fasting plasma insulin levels, fasting lipid profile were determined. Oral glucose tolerance test (OGTT) was done. Urinary albumin concentrations were measured with a solid-phase fluorescent immunoassay. The urinary albumin/creatinine ratio was expressed as milligrams of albumin per gram of creatinine after exclusion of urinary tract infections.

Clinical evaluation

Standing height was measured without shoes, to the nearest 0.1 cm, using Harpenden stadiometer (Holtain ltd, Croswell, Crymych, UK) and weight was measured using a digital scale, to the nearest 0·1 kg, wearing light clothing and without shoes. BMI was calculated using the formula kg/m2. Weight to height ratio was calculated by dividing weight by height. Standard deviation scores for weight, height, weight to height ratio[7], and BMI were calculated[8]. Waist and hip circumferences were measured using a flexible tape to the nearest 0.1 cm. Waist circumference (WC) was measured at the end of expiration midway between the lower rib margin and the iliac crest, and hip circumference (HC) was measured at the level of greater trochanter[9] Waist hip ratio (WHR) was calculated by dividing WC by HC, and standard deviation scores for WC and WHR were estimated[10]. All measurements were taken twice. Blood pressure was measured by a standard mercury sphygmomanometer, after the subject had rested for 5 min in the sitting position, using the appropriate cuff size and the 5th Korotkoff sound was taken for diastolic blood pressure categorization.

Biochemical evaluation: including fasting serum glucose level, fasting serum insulin level, oral glucose tolerance test (OGTT), and lipid profile. Urinary albumin concentrations were measured with a solid-phase fluorescent immunoassay (Beckman Instruments, Palo Alto, CA). The urinary albumin/creatinine ratio was expressed as milligrams of albumin per gram of creatinine after exclusion of urinary tract infections.

- A urine sample was taken in the morning; instructions were given to all subjects and parents for the former to avoid exercise and hyperactivity 24h prior to sampling. Urine was stored at −70°C for measurement of albuminuria.
- Fasting plasma glucose levels were measured with the modified hexokinase enzymatic method (NMLC code). Fasting insulin levels were measured using a commercially available radioimmunoassay kit Biosource Europe (Nivelles, Belgium).
- The Homeostasis Model Assessment for Insulin Resistance (HOMA-IR) was used to calculate in vivo insulin sensitivity according to the formula: fasting blood glucose (mmol/L) × fasting insulin (uU/L)/22.5. Insulin resistance was defined as a HOMA-IR score of > 4.34 without a history of DM or diabetes medication usage.
- After a 12-h overnight fast Oral glucose tolerance test (OGTT) was done for all subjects by giving them 1.75 grams of glucose/kg body weight, to a maximum dose of 75 grams of glucose solution to drink within a 5 minute time frame. Blood samples were withdrawn every 30 minutes after drinking the solution, for measurement of glucose and insulin levels.
- Serum glucose level after 2 hours was measured where plasma glucose level between 140 and 200 mg/dl indicate ‘impaired glucose tolerance’, and levels above 200mg/dl at 2 hours confirms a diagnosis of ‘diabetes mellitus’.

- Serum insulin level after 2 hours was measured where 60-100uU/ml is 'borderline diabetes mellitus', and more than 100uU/ml is 'diabetic'[11].
- Fasting serum triglycerides levels was measured enzymatically, LDL-cholesterol, HDL-cholesterol, total cholesterol levels were measured using heparin-manganese precipitation method.

According to the National Cholesterol Education Program (NCEP)[11] metabolic syndrome (MS) was diagnosed in the presence of three or more of the following five criteria: Increased waist circumference ≥ 95th or 90th centile (abdominal obesity)[11], elevated triglycerides > 135 mg/dl, decreased HDL < 35mg/dl, hypertension ≥ 95th or 90th centile for age and sex, and fasting serum glucose > 5.6 mmol/L (>100mg/dl) or active treatment for hyperglycemia[11].
Normal urinary excretion was defined as an albumin/creatinine ratio of <30mg/g. Microalbuminuria was defined as an albumin/creatinine ratio of ≥30mg/g and <300mg/g.

Statistical analysis:
SPSS for Windows version 10 was used for the analysis. All values are given as the mean ± standard deviation (SD). The Student t- test and chi-square test were used to compare data. Logistic regression models were developed to analyze associations with microalbuminuria using clinical and laboratory data as ± independent variables and adjusting for age, gender, BMI, metabolic syndrome (MS) and all of its different constituents. Correlation analysis was used to detect the relation between microalbuminuria and other variables. Number and percentage for categorical variables, –chi squared test or T test were used (P-value <0.05 was significant, p-value < 0.01 was considered as highly significant, p-value > 0.05 was considered insignificant).

3. RESULTS

In total, 62 cases were enrolled in the study. They were 30 males (48%) and 32 females (52 %). The mean birth weight was 3.18 ±1.06 kg. The mean age of weaning was 5.8 ± 2.6 months The mean age of onset of obesity was 5.9 ± 3.9 years. The progression of obesity was gradual in 30 cases (96.7%) and was rapid only in one case (3.2%). There were 4 cases (6.4%) of hypertension, 18 cases (29 %) of microalbuminuria, 14 cases (22.5%) of high triglycerides, 0 cases of high LDL, 12 cases (19%) of low HDL, 8 (25 %) of impaired fasting glucose levels, 18 cases (29%) of insulin resistance, 4 cases (6.4%) of metabolic syndrome (Table 1).

Table 1. Clinical and laboratory parameters of obese subjects in the study

Clinical and laboratory parameters	Patient cohort (n= 62)
Age (years)	10.9±3.11years
Male/Female (n)	30/32
Height SDS	-0.92 ± 1.25
Weight SDS	14.02 ± 4.34
BMI (kg/m2) (mean± SD)	31.4±3.9
BMI z-score (mean± SD)	2.7±0.4
Mean waist circumference (cm)	90.18 ± 16.9
Mean hip circumference (cm)	98.81± 17.04
Waist/hip ratio	0.92 ± 0.14
Abdominal obesity n(%)	46 cases (74%)
Hypertension n(%)	4 cases (6.4%)
Acanthosis negricans n(%)	4 cases (6.4%)
Microalbuminuria n(%)	18 cases (29 %)
High Triglycerides(>125mg/dl)	14 cases (22.5%)
High LDL(>100mg/dl)	0
Low HDL(<35mg/dl)	12 cases (19%)
Impaired fasting glucose level(mg/dl)	8 (12.9 %)
Insulin resistance (HOMA-IR)	18 cases (29%)
Metabolic syndrome n(%)	4 cases (6.4%)
Systolic blood pressure(mean± SD)	116.25±14.2
Diastolic blood pressure(mean± SD)	76.56±8.5

BMI= Body mass index, SD=Standard Deviation, LDL= LDL= Low density lipoprotein, HDL= High density lipoprotein, HOMA-IR= The Homeostasis Model Assessment for Insulin Resistance

The incidence of impaired OGTT in this study was 9.6% (6 cases), where the serum glucose level after 2hrs was between (140-200ng/dl). Impaired insulin level after 2hrs was found in 3.2% (2 cases) where the serum insulin level was 62.1 uU/ml and 8 cases (12.9%) were diabetic (their serum insulin levels were more than 100uU/ml).
Cases in the study were stratified according to presence or absence of microalbuminuria. Children with urine albumin levels < 20mg/24 hours = normoalbuminuria group (44 cases; 70.9 %) and the second group included children with urine albumin levels ≥ 20mg/24 hours = microalbuminuria (18 cases; 29 %). The mean urinary albumin in microalbuminuria group was 70.83± 21.81mg.
There was no significant difference between the two groups in terms of age, sex, BMI and BMI z score (p>0.05) (Table 2). There was significant decrease of

birth weight in normoalbuminuria group: 3.17±0.35 compared to microalbuminuria group: 3.47± 0.14 Kg ($p \leq 0.05$) (Table 2). Also Triglycerides and LDL were higher in microalbuminuria group but not statistically significant. HDL was lower in microalbuminuria group but non-significant. HOMA-IR was higher in microalbuminuria group, however non-significant.

Table 2. Clinical and laboratory parameters of obese subjects categorized according to the presence of microalbuminuria or normoalbuminuria

Variables (mean±SD)	Microalbuminuria (≥20mg/24hrs)(n=18 cases) (29%)	Normoalbuminuria (<20mg/24hrs)(n=44 cases) (70.9%)	T-test	P-value
Age (years)	10.33±3.05	10.9±3.2	0.92	0.79
Male/Female (n)	8/10	22/22		
Birth weight (kg)	3.47±0.14	3.17±0.35	Z= -1.938	0.050*
Weight SDS	15.50±4.77	13.03±3.99	-1.086	0.297
Height SDS	-1.30±1.28	-0.67±1.24	-1.065	0.287
BMI (kg/m2) (mean± SD)	30±3.3	31.6±4.1	0.7	0.52
BMI z-score (mean± SD)	0.7±0.4	0.7±0.3	0.7	0.52
Waist circumference(cm)	77.3±20.4	93.1±15.4	1.2	0.3
Waist/hip ratio	0.76±0.18	0.95±0.1	1.7	0.21
Systolic blood pressure(mmHg)	113.3±5.7	116.9±15.6	0.56	0.52
Diastolic blood pressure(mmHg)	73.3±5.7	77±9	0.95	0.38
Triglycerides(mg/dl)	107.8±2	97.85±37.4	0.052	0.95
LDL(mg/dl)	94±25.4	77.37±13	-0.141	0.890
HDL(mg/dl)	45.38±2.3	50.83±7.9	0.195	0.849
Total cholesterol(mg/dl)	168.00±13.48	161.00±28.35	-0.559	0.586
Insulin resistance(HOMA-IR)	3.3±1.3	1.2±0.96	2.5	0.1
Fasting glucose level(mg/dl)	84.66±10.7	87.15±10.8	0.36	0.743
Fasting insulin level(mU/L)	16.5±7.8	5.5±3.8	2.35	0.13
Time spent weekly in exercise(min/week)	175.00±96.49	92.78±50.32	-2.176	0.049*

BMI= body mass index, SD=Standard Deviation, LDL= LDL= Low density lipoprotein, HDL= High density lipoprotein, HOMA-IR= The Homeostasis Model Assessment for Insulin Resistance, p-value < 0.05 is significant.

Multivariate analysis (Logistic regression) was done to assess the association between different variables and the presence of microalbuminuria in obese children. No significant difference between obese children with microalbuminuria and those without regarding to abdominal obesity, hypertension, acanthosis negricans, and metabolic syndrome (Table 3).

Table 3. Association of microalbuminuria with different clinical parameters in obese children.

Parameter	Microalb. No.	Microalb. %	Normoalb. No.	Normoalb. %	TTotal	XP- value	Odds Ratio(CI)
HTN						00.246(0.62)	0.09(0.005-1.5)
+ve	2	50	2	50	4		
-ve	11	20	47	80	558		
Acanthosis negricans					2	0	
+ve	0	0	2	100	2	0.527(0.46)	
-ve	13	21	47	79	560		
Metabolic syndrome					2	0	
+ve	0	0	4	100	44	0.246(0.62)	
-ve	12	20	46	80	258		

CI= Confidence Interval.

There was no significant difference between the two groups regarding fasting plasma glucose level (p=0.743) or 2 hrs postprandial blood glucose (p=0.24). However, the mean of serum insulin level after 2hrs in OGTT was significantly higher in patients with microalbuminuria than those without (96.3±29.17 versus 29.89±32.8) (p= 0.03*) as shown in table (4).

Table 4. Serum glucose and serum insulin after 2 hrs and HOMA-IR in obese children categorized according to the presence of microalbuminuria or normoalbuminuria.

	Microalb.	Normoalb.
Serum glucose after 2hrs (Mean±SD) mg/dl	116±11.5	105±16.7
T test	1.34	
P- value	0.24	
Serum insulin level after 2hrs (Mean±SD) uU/ml	96.3±29.17	29.89±32.8
T test	3.47	
P- value	0.03*	
HOMA-IR (Mean±SD)	3.3±1.3	1.2±0.96
T test	2.5	
P- value	0.1	

SD= Standard Deviation

Our study also showed a significant positive correlation between microalbuminuria and time spent weekly in exercise (minute/week) (life style) (r= 0.544, p= 0.036*). There was a significant decrease in time spent during exercise or walking (minutes/week) in normoalbuminuria group (55± 62.4 minutes) compared to microalbuminuria group (80± 31.2 minutes).

Also according to the OGTT (oral glucose tolerance test); a significant positive correlation between microalbuminuria and postprandial blood glucose at 1 hr and 1.5 hrs in OGTT was found (r= 0.509, p= 0.035 and r=0.503, p=0.046 respectively). Other variables showed no significant correlation with microalbuminuria.

4. DISCUSSION

Renal dysfunction may start long before the appearance of hypertension or diabetes in adulthood[4]. The long-term cardiovascular and renal impact of obesity, although deferred to adult life, has its origin in childhood[6].

Assessment of early markers of cardiometabolic risk may be useful in stratifying the future risk of obese children. One of these markers is microalbuminuria, which denotes subtle abnormalities of endothelial permeability and demonstrated as a marker of risk to develop type 2 diabetes as well as cardiovascular and renal risk in adults[12].

The prevalence of microalbuminuria in this study was 29 % which was higher than that reported by Okpere et al.[13], in 2012, who found a prevalence of 3.5% . The present study also noted a higher prevalence of microalbuminuria compared to the 19% reported by Ibadin et al. in 2004[14] in Benin City, Nigeria and the 7.8% reported by Jones et al. in 2002 in the United States of America[15]. The lower prevalence by Ibadin et al., 2004 may be due to exclusion of subjects with obesity and hypertension from their study, while that by Jones et al., 2002 may be due to the larger sample size and the wide variation in the ages of the study population.

Excess weight is thought to increase intraglomerular capillary pressure, resulting in glomerular hyperfiltration, a permissive environment or condition for end-organ damage. In this setting, hypertension, impaired fasting glucose or DM may provide a second hit, causing endothelial dysfunction that leads to microalbuminuria[12].

In the present study, males represented 22 (50 %) of group 1 (microalbuminuria < 20mg/dl) and 8 (44.4 %) of group 2 (microalbuminuria ≥ 20mg/dl) with no significant difference between both groups as regard sex distribution. These results disagree with that reported by Ibadin et al., 2004 who reported a higher prevalence in males (20.8%) compared to females (16.7%)[14].

On the other hand, these results are different from those reported by Okpere et al. 2012[13] who reported significantly higher gender specific prevalence of microalbuminuria in females (45.3%) compared to males (20.4%); and their results are similar to the findings recorded by Jones et al. 2002 in the USA, who reported a gender specific prevalence of 9.7% in females and 6.1% in males[15]. The higher prevalence of microalbuminuria (MA) in girls than boys could be attributed to the accompanied bigger muscle mass and urinary creatinine excretion of the latter resulting in their smaller albumin creatinine ratio (ACR) values[16].

In the present study, birth weight was significantly lower in group 1 normoalbuminuria in comparison to group 2 microalbuminuria (3.17±0.35 vs 3.47±0.14 respectively) (p=0.050). These results disagree with Keijzer-Veen et al. in 2005[17] who reported that, as far as birth weight is concerned; small for gestational age status has been linked to increased microalbuminuria (MA) in a cohort of Dutch young adults.

Our results revealed no significant difference between obese children with MA and those with normoalbuminuria regarding waist circumference, lipid profile or insulin levels. This is in contrast to results reported by Sanad and Gharib, 2011[18] in Zagazig University, who reported that, values for waist circumference were significantly higher in obese children with MA than in those with normoalbuminuria ($p < 0.05$). They also found that, Triglycerides and LDL were significantly higher in MA group ($p < 0.01$), while HDL was significantly lower in MA group ($p<0.01$). However, in our study, there was no significant difference between the two groups as regard the lipid profile (Triglycerides, LDL, HDL and Total cholesterol) where p-value was (0.95, 0.890, 0.849, 0.586 respectively). Sanad and Gharib, 2011 added, insulin resistance was significantly higher in obese children with MA than those with normoalbuminuria[18].

Also Burgert et al. found that the individuals with microalbuminuria had significantly higher glucose and insulin levels during an oral glucose tolerance test than individuals without microalbuminuria[19].

The present study showed no significant correlation between both waist/hip ratio and BMI versus microalbuminuria. These results disagree with many previous studies. There is strong evidence that obesity, in particular central body fat distribution, is an important risk factor for renal function abnormalities[19]. This difference may be attributed to the relatively young age of our cohort.

There was no significant difference in our study between the two groups regarding blood pressure. This disagrees with Sanad and Gharib who found that obese children presenting with microalbuminuria had significantly higher blood pressure[18] than those without MA. They also found, significantly higher prevalence of insulin resistance, impaired fasting glucose level and metabolic syndrome among their obese children with MA. Also MA in their study showed a significant positive correlation with BMI, waist circumference, systolic and diastolic blood pressure, TG level, LDL level, insulin resistance and fasting glucose level.

Insulin increases the effects of angiotensin II on mesangial cells, thus contributing to hypertension, raised intraglomerular pressure, exacerbation of proteinuria, induction of intrarenal inflammatory cytokines and growth factors and apoptosis[19]. Another explanation for this association between obesity and cardiovascular metabolic complications leading to renal injury may stem from the recently elucidated role of adipokines in influencing body weight and glucose via the effect of adiponectin on lipid metabolism in adipocytes[20]. This is mediated through adiponectin stimulation of the AMPK pathway, a key regulator of intracellular energy status with potent antiproliferative effects [20].

Our study had one limitation and that was the use of a single urine sample to estimate microalbuminuria, which might have led to overestimation, because albuminuria can be transient. However, we attempted to avoid orthostatic proteinuria by taking samples in the early morning and to decrease the overestimation of albuminuria induced by exercise via instructions to limit strenuous (hyperactivity) for 24 hours prior to sampling.

5. CONCLUSION

Obesity in childhood and adolescence is a risk factor for the development of microalbuminuria, which is significantly associated with metabolic syndrome and its different components. These findings raise the issue as to whether microalbuminuria could be proposed as an item in the diagnosis of metabolic syndrome in children and adolescents.

REFERENCES

1. Wang Y, Chen X, Klag MJ, Caballero B. Epidemic of childhood obesity: implications for kidney disease. Adv Chronic Kidney Dis 2006; 13(4):336-351.
2. World Health Organization Obesity. Preventing and managing the global epidemic. Report of a WHO consultation. WHO technical Report Series 894; Geneva, Switzerland 2000.
3. Wang Y, Chen X, Song Y, Caballero B, and Cheskin LJ. Association between obesity and kidney disease: a systematic review and metaanalysis. Kidney Int 2008; 73(1): 19-33.
4. Wahba IM, Mak RH. Obesity and obesity-initiated metabolic syndrome: mechanistic links to chronic kidney disease. Clin J Am Soc Nephrol 2007; 2(3): 550-562.
5. Chiarelli F and Marcovecchio ML. Insulin resistance and obesity in childhood. Eur J Endocrinol 2009;159 Suppl 1:S67-74.

To Cite This Article: Heba H El Sedfy, Nadin N Toaima, Mayada A Ibrahim, Lerine B El Din. Microalbuminuria in Obese Children and Adolescents and the Metabolic Syndrome. *International Annals of Medicine. 2017;1(10).* https://doi.org/10.24087/IAM.2017.1.10.301

6. Verhave JC, Gansevoort RT, Hillege HL, Bakker SJ, De Zeeuw D, de Jong PE. An elevated urinary albumin excretion predicts de novo development of renal function impairment in the general population. Kidney Int Suppl 2004; 92: S18-S21
7. Tanner JM, Whitehouse RH: Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. Arch Dis Child 1976; 51:170- 9.
8. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 2000; 320:1240–3.
9. Schwandt P, Kelishadi R, Ribeiro RQ, Haas GM, Poursafa P. A three-country study on the components of the metabolic syndrome in youths: the BIG Study. Int J Pediatr Obes. 2010; 5(4): 334-41.
10. Fernandez JR, Redden DT, Pietrobelli A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. J Pediatr 2004, 145(4):439-444.
11. American Academy of Pediatrics. National cholesterol education program: report of the expert panel on blood cholesterol levels in children and adolescents. Pediatrics 89(3 Pt 2):525-84.
12. Srivastava T. Non-diabetic consequences of obesity on kidney. Pediatr nephrol 2006; 21(4):463-470.
13. Okpere AN, Anochie IC, Eke FU. Prevalence of Microalbuminuria among secondary school children. African Health Services 2012;12(2):140-147.
14. Ibadin MO, Onunu A, Unuigbe E et al. Microalbuminuria in adolescents/young adult offsprings of hypertensive Nigerian adults-a preliminary report. Nig J Clin Pract 2004;7:60-64.
15. Jones CA, Francis ME, Eberhardt MS. Microalbuminuria in the US population: Third National Health and Nutrition Examination Survey (NHANES III). Am J Kidney Dis 2002;39:445-459.
16. Tsioufis C, Mazaraki A, Dimitriadis K, Stefanidis CJ. Microalbuminuria in the pediatric age: current knowledge and emerging questions. Acta Paediatrica 2011; 100:1180-1184.
17. Keijzer-Veen M, Dekker F, Nauta J. Microalbuminuria and lower glomerular filtration rate at young adult age in subjects born very premature and after intrauterine growth retardation. J Am Soc Nephrol 2005; 16:2762-8.
18. Sanad M and Gharib A. Evaluation of microalbuminuria in obese children and its relation to metabolic syndrome. Pediatr Nephrol 2011; 26:2193-2199.Nguyen S, McCulloch C, Braeman P, Portale A, Hsu CY. Being overweight modifies the association between cardiovascular risk factors and microalbuminuria in adolescents. Pediatrics 2008;121(1):37-45.
19. Buergert TS, Dziura J, and Caprio S. Microalbuminuria in pediatric obesity: prevalence and relation to other cardiovascular risk factors. Int J Obes 2005;45:198-202.
20. Joachim H, Sharma K. Mechanisms linking obesity, chronic kidney disease, and fatty liver disease: the roles of fetuin-A, adiponectin, and AMPK. J Am Soc Nephrol 2010; 21(3):406-412.