 DOI: 10.18231/2394-6377.2017.0079

Prevalence of metabolic syndrome in obese medical students –a case control study

D. Sridevi[1,*], G. Lakshmi Chaitanya[2], Amrut A. Dambal[3], V. Sampath Kumar[4], Ch. Anila Sushma[5]

[1]Assistant Professor, [3]Associate Professor, [4]Professor & HOD, [5]Junior Resident, Dept. of Biochemistry, [2]Tutor, Dept. of Medical Biochemistry, Malla Reddy Institute of Medical Sciences, Hyderabad, Telangana

***Corresponding Author:**
Email: sridumedico81@gmail.com

Abstract

Introduction: The Metabolic Syndrome is a constellation of risk factors predictive of future Cardio Vascular Accidents (CVA) and Type 2 Diabetes Mellitus (T2DM) in adults. Recently several studies revealed that metabolic syndrome is present in younger populations, suggesting that the origin of chronic metabolic diseases manifest relatively early in life.

Objectives: To assess the risk of metabolic syndrome in young obese students by assessing physical and biochemical parameters.

Materials and Method: This study comprised of students studying MBBS at Guntur Medical College. 30 Males and 31 Females between 18 to 22 years, with increased BMI were considered as cases and age matched 30 Male and 34 Female students with normal BMI were considered controls. Physical Parameters like Weight, Height, Blood Pressure, Waist Circumference and Biochemical parameters like Fasting Plasma Glucose and Lipid profile were analyzed for risk assessment of metabolic syndrome.

Results: It was observed that 23.33% of male cases and 19.35% of female cases had Metabolic Syndrome.

Conclusion: In our study, young obese subjects were more prone to Metabolic Syndrome. The metabolic syndrome and its future consequences, including CVA and T2DM, continue to increase until we find ways to prevent obesity. This can be achieved through adopting the required healthy life style changes.

Keywords: Metabolic Syndrome (MetS), Body Mass Index(BMI), Triglycerides (TG), High Density Lipoprotein Cholesterol (HDLC), Fasting Plasma Glucose (FPG), Type 2 Diabetes Mellitus (T2DM),Cardio Vascular Accidents (CVA).

Received: 21st June, 2017 **Accepted:** 5th August, 2017

Introduction

The Metabolic Syndrome (MetS) is a constellation of risk factors predictive of future cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) in adults.[1,2] Recently several studies[3-5] found that the metabolic syndrome is prevalent in younger populations, suggesting the origin of chronic metabolic diseases manifesting relatively early in life. Although the underlying pathophysiology of Met S is unclear, insulin resistance is thought to be a central abnormality in the pathogenesis of the disorder.[6] It is observed that overweight youth with the metabolic syndrome are significantly more insulin resistant when compared with those not having Met S.[3] The increasing prevalence of obesity, metabolic syndrome and insulin resistance in the young population is a global health issue, especially given that these conditions in youth may be antecedents to adult disease.[7] Given the epidemic of pediatric obesity and emergence of T2DM among adolescents, it shouldn't be surprising that the Met S has been reported to exist in adolescents and youth. Estimated prevalence rates range from 5% to 39% in the general population of youth and overweight adolescents, respectively.[8,9] Current literature supports the notion that the presence of the Met S in youth may be an important predictor of future risk for T2DM and CVD.[10] So a study was taken up to identify the prevalence of Met S in young overweight students.

Materials and Method

The prospective study was carried out in Guntur Medical College. After obtaining approval from Institutional Ethical Committee, informed consent was taken from the subjects included in the study. This study was taken up from February 2011 to September 2011.

This study comprised of students studying MBBS at Guntur Medical College. 30 Males and 31 Females obese students between 18 to 22 years, were considered as cases and age matched 30 Male and 34 Female non obese students were considered controls. Physical Parameters like Blood Pressure, Waist Circumference and Chemical parameters like Fasting Plasma Glucose and Lipid profile were analyzed.

Inclusion Criteria

Cases: Students between 18 to 22 years with BMI ≥30

Controls: Students between 18 to 22 years with BMI between 18 to 25

Exclusion Criteria: Students with known endocrine disorders or receiving any medications that may alter lipid levels, and Students with abdominal mass or ascites were excluded.

IDF 2005 guidelines to confirm Met S: Central adiposity (Waist circumference ≥ 90cm in males and ≥ 80 cm in females) plus two or more of the following four factors:[11]

1. Serum Triglycerides: ≥150mg/dl
2. Serum HDL cholesterol: <40 mg/dl in men and <50 mg/dl in women

3. Blood pressure: Systolic Blood Pressure ≥ 130 mmHg or
Diastolic Blood Pressure ≥85 mmHg and
4. Fasting plasma glucose concentration ≥100 mg/dl.

Sample Collection: Fasting venous blood samples were collected in respective vacutainers. The blood was allowed to clot and the specimens were centrifuged to separate the serum from the cells

Methods of Estimation: BMI was calculated by measuring the weight in kgs and height in meters and using the formula BMI= weight /height^2

Waist circumference was measured at the level of the umbilicus using a plastic anthropometric tape.

Blood Pressure was measured with a mercury sphygmomanometer by auscultatory method following the American Heart Association protocol.[12]

Fasting Plasma Glucose: Glucose Oxidase – Peroxidase method

Serum Triglycerides: Glycerol -3 – Phosphate Oxidase method

Serum HDL Cholesterol: Phosphotungstic Acid Method

Statistical Analysis: Data was analyzed as mean and standard deviation (mean ± SD). Mean and standard deviations were compared between control and case groups by unpaired student 't' test. Descriptive statistical analysis was carried out in the present study by using SPSS package version 14.

Results

Body Mass Index (BMI) in male controls was 22.67 ± 1.12 compared to male cases with 33.72 ± 2.01 displaying highly Significant statistical difference ($p < 0.001$). Body Mass Index (BMI) in female controls was 21.02 ± 1.86 compared to female cases with 32.19 ± 1.07 displaying highly Significant statistical difference ($p < 0.001$). (Table 1 & 2, Fig. 1 & 3)

Waist Circumference (WC) in male controls was 80.17±5.46 compared to male cases with 97 ±6.86 displaying highly Significant statistical difference ($p < 0.001$). Waist Circumference (WC) in female controls was 72.71 ±5.01 compared to female cases with 86.32 ±6.16 displaying highly Significant statistical difference ($p < 0.001$). (Table 1 & 2, Fig. 1 & 3)

Systolic Blood Pressure (SBP) in male controls was 117.53 ±6.00 compared to male cases with 123.43 ±7.27 displaying highly Significant statistical difference ($p<0.005$). Diastolic Blood Pressure (DBP) in male controls was 76.93 ±4.66 compared to male cases with 81.33 ±7.83 displaying Significant statistical difference ($p < 0.01$). Systolic Blood Pressure (SBP) in female controls was 107.65 ±9.43 compared to female cases with 115.1 ±11.35 displaying Significant statistical difference ($p < 0.01$). Diastolic Blood Pressure (DBP) in female controls was 67 ±6.39 compared to female cases with 73.94 ±8.21 displaying highly Significant statistical difference ($p < 0.001$) (Table 1 & 2, Fig 1 & 3).

Fasting Plasma Glucose (FPG) in male controls was 75.73±5.2 compared to male cases with 80.2 ±6.86 displaying Significant statistical difference ($p < 0.01$). FPG in female controls was 75.21 ±5.20 compared to female cases with 77.16 ±5.78. The increase in FPG in females is not Significant ($p < 0.1$). (Table 1 & 2, Fig. 2 & 4)

Serum Triglycerides (TG) in male controls was 86.3 ±32.85 compared to male cases with 105.6 ±31.79 displaying Significant statistical difference ($p <0.05$). TG in female controls was 81.12 ±27.09 compared to female cases with 81.84 ±31.54. The increase in TG in female is not Significant ($p >0.1$). (Table 1 & 2, Fig. 2 & 4)

Serum High Density Lipoprotein Cholesterol (HDLC) in male controls was 49.5 ±10.19 compared to male cases with 38.9 ±8.91 displaying highly Significant statistical difference ($p < 0.001$). HDLC in female controls was 52.53 ±8.88 compared to female cases with 41.84 ±7.46 displaying highly Significant statistical difference ($p < 0.001$). (Table 1 & 2, Fig. 2 & 4)

The overall risk analysis for Metabolic Syndrome in the 30 (Thirty) male cases, 7 (Seven) were with 3 risk factors which represents 23.33% of male cases having Metabolic Syndrome. But risk analysis for Metabolic Syndrome in the 30 (Thirty) Control male group, No single subject with 3 risk factors was detected.

Whereas the overall risk analysis for Metabolic Syndrome in the 31 (Thirty One) female cases, 6 (Six) were with 3 risk factors which represents 19.35% of female cases having Metabolic Syndrome. But risk analysis for Metabolic Syndrome in the 34 (Thirty Four) Control female group, No single subject with 3 risk factors was detected.

Table 1: Comparison of Physical and Biochemical Parameters in Male Cases and Controls

Parameter	Controls	Cases	p Value	Significance
	Mean ± SD	Mean ±SD		
BMI	22.67 ± 1.12	33.72 ± 2.01	<0.0001	Highly Significant
WC	80.17 ± 5.46	97 ± 6.86	<0.0001	Highly Significant
SBP	117.53 ± 6	123.43 ± 7.27	0.0011	Very Significant
DBP	76.93 ± 4.66	81.33 ± 7.83	0.0105	Significant
FPG	75.73 ± 5.2	80.2 ± 6.86	0.0062	Significant
TG	86.3 ± 32.85	105.6 ± 31.79	0.0243	Significant

HDL-C	49.5 ± 10.19	38.9 ± 8.91	<0.0001	Highly Significant

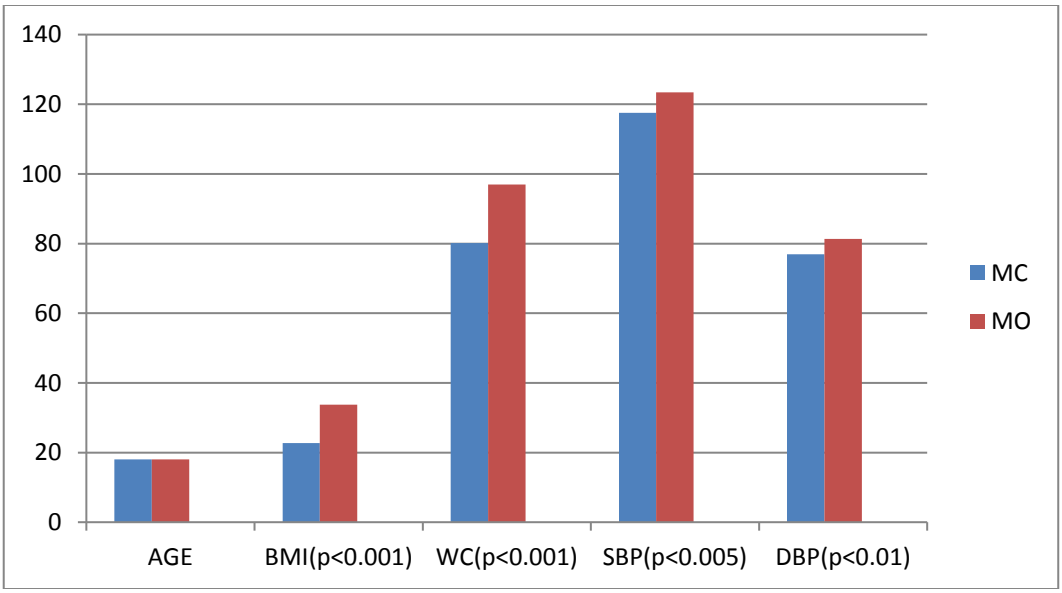

Fig. 1: Comparison of Physical Parameters in Male Cases and Controls

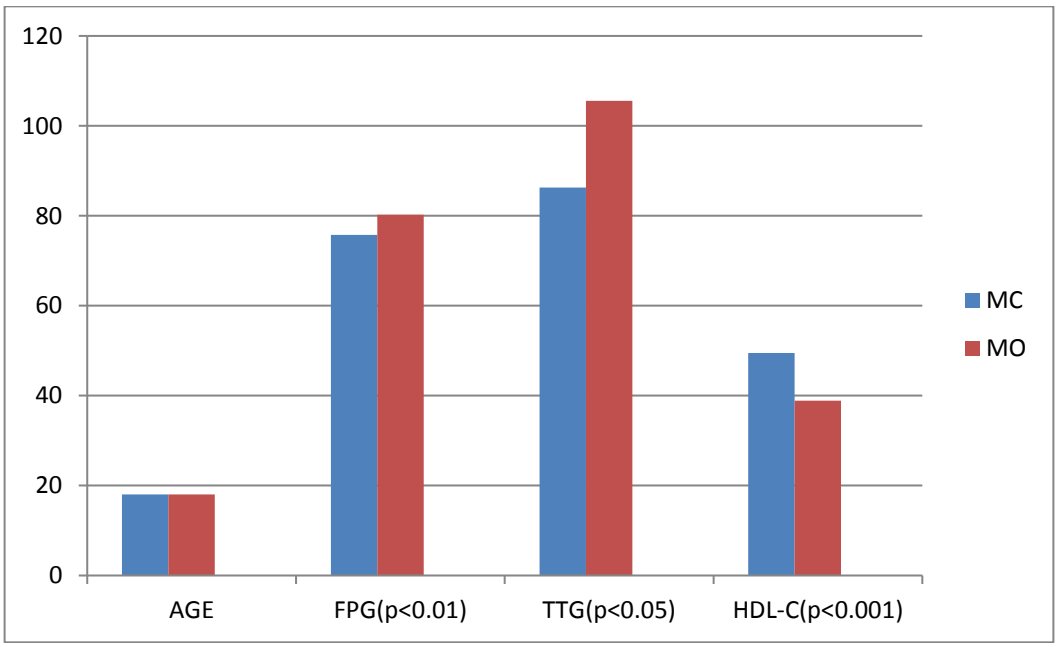

Fig. 2: Comparison of Biochemical Parameters in Male Cases and Controls

Table 2: Comparison of Physical and Biochemical Parameters in Female Cases and Controls

Parameter	Controls	Cases	p Value	Significance
	Mean ± SD	Mean ± SD		
BMI	21.02 ± 1.86	32.19 ± 1.07	<0.0001	Highly Significant
WC	72.71 ± 5.01	86.32 ± 6.16	<0.0001	Highly Significant
SBP	107.65 ± 9.43	115.1 ± 11.34	0.0053	Very Significant
DBP	67 ± 6.39	73.94 ± 8.21	0.0003	Highly Significant
FPG	75.21 ± 5.2	77.16 ± 5.78	0.1561	Not Significant
TG	81.12 ± 27.09	81.84 ± 31.54	0.9214	Not Significant
HDL-C	52.53 ± 8.88	41.84 ± 7.46	<0.0001	Highly Significant

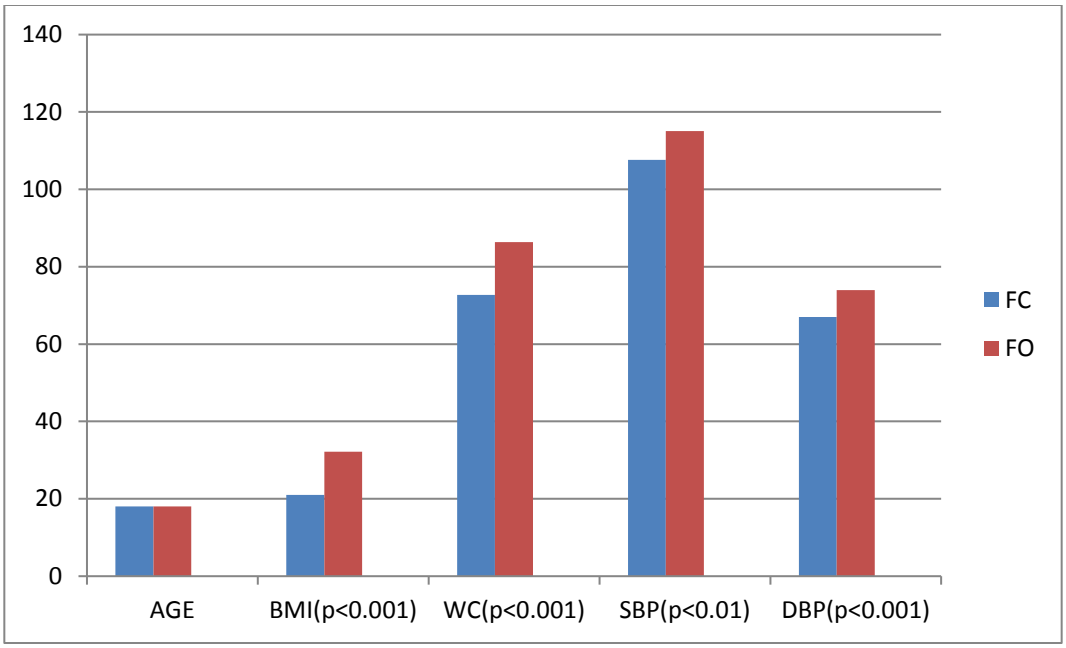

Fig. 3: Comparison of Physical in Female Cases and Controls

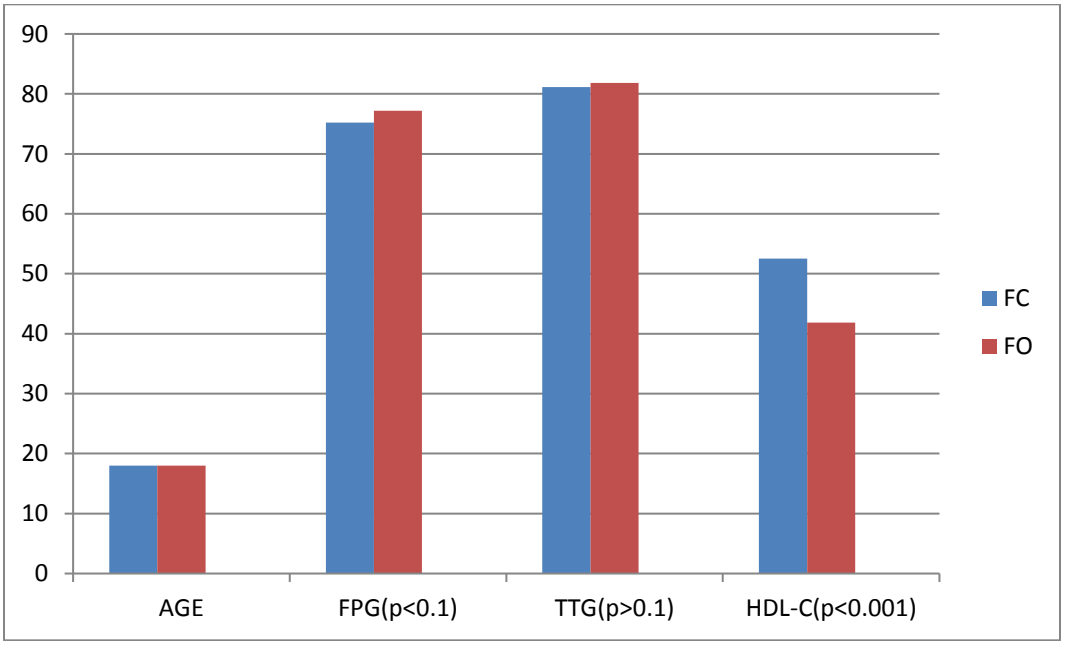

Fig. 4: Comparison of Biochemical Parameters in Female Cases and Controls

Discussion

Multiple set of risk factors that commonly appear together, form as the 'Metabolic Syndrome'. This 'cluster' of abnormal metabolic activities that occur in an individual appear to confer a substantial additional risk for CVA and T2DM.[13,14] There is an overwhelming moral, medical and economic imperative for early identification of individuals with metabolic syndrome because it is associated with double the risk of the cardiovascular accidents and five times increased risk for Type 2 Diabetes mellitus (T2DM).[15]

So in this study we intended to investigate and identify the components of metabolic syndrome among young obese subjects.

A significant prevalence of MetS of about 23.33% in males and 19.35% in female obese students was observed in the present study, the results in accordance with the studies of Du Bose et al and Goodman et al.[8,9] Our study is also in accordance with studies of Lara Nasreddine[16] and Singh R[17] who have recorded a MetS prevalence of 19.2% and 36.6% respectively among obese group.

In the present study, significant statistical difference was observed among male cases and controls for Systolic and Diastolic Blood Pressure, FPG, Serum TG and Serum HDL.

In this study, significant statistical difference was observed among female cases and controls for Systolic and Diastolic Blood Pressure and Serum HDL. No statistical difference was observed for FPG and Serum TG.

The results of this present study indicate that young adults with increased waist circumference are strongly associated with Metabolic Syndrome compared with age matched contemporary controls.

Accumulation of visceral fat as seen in obese subjects is characterized by a relatively high lipid turnover which may result in higher levels of free fatty acids in the portal circulation.[18] This, in turn, may contribute to the development of individual components of the MetS, for example, enhanced lipid synthesis, gluconeogenesis, and insulin resistance.[19,20] Furthermore, intra-abdominal fat correlates positively with activation of the sympathetic nervous system,[21] which may further enhance free fatty acid release into portal circulation.[22] Sympathetic activation may also contribute to the elevation of Blood Pressure through its effects on vasculature and renal handling of sodium and water.[23]

The worldwide increase in the prevalence of obesity in the recent decades is startling and is likely a cause of the rising incidence of insulin resistance and the MetS,[6,24] as well as CVD and T2DM.[24] The combination of obesity, physical inactivity, and consumption of an atherogenic diet is believed to lead to insulin resistance.[25]

Beck-Nielsen and Groop[26] put forth a three-stage hypothesis in the development of T2DM. **Stage 1**: includes normal or slightly increased blood glucose with fasting hyperinsulinemia. **Stage 2**: is characterized by prediabetic glucose intolerance with insulin resistance, and **Stage 3**: is development of T2DM. Unfortunately, many of the macro vascular variations in association with T2DM and CVA begin in stages 1 and stage 2, quite before diagnosis is done. Thus early detection of MetS, there is a hope that appropriate lifestyle changes can prevent the future development of prediabetes or T2DM in the younger population.

Conclusion

The prevalence of Metabolic Syndrome is increasing in obese young medical students both male and female students of Guntur Medical College, Guntur. As per International Diabetes Federation Criteria; among these medical students with increased waist circumference, 23.33% of male students and 19.35% of female students are with Metabolic Syndrome whereas none of the students are with Metabolic Syndrome having normal waist circumference.

This represents that there would be a serious threat to the current and future health of youth with central obesity. The metabolic syndrome and its many consequences, including CVD and T2DM, will continue to increase unless we can find ways to prevent obesity and thus MetS in young adults. This can be achieved through adopting the required healthy life style changes.

References

1. Malik S, Wong ND, Franklin SS, Kamath TV, L' Italien GJ, Pio JR, Williams GR. Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. Circulation (2004); 110:1245–1250.
2. Laaksonen DE, Lakka HM, Niskanen LK, Kaplan GA, Salonen JT, Lakka TA. Metabolic syndrome and development of diabetes mellitus: application and validation of recently suggested definitions of the metabolic syndrome in a prospective cohort study. Am J Epidemiol (2002);156:1070–1077.
3. Cruz ML, Weigensberg MJ, Huang TT, Ball G, Shaibi GQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. J Clin Endocrinol Metab (2004); 89:108–113.
4. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988–1994. Arch Pediatr Adolesc Med (2003);157:821–827.
5. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, Allen K, Lopes M, Savoye M, Morrison J, Sherwin RS, Caprio S .Obesity and the metabolic syndrome in children and adolescents. N Engl J Med (2004); 350:2362–2374.
6. Reaven GM Banting lecture. Role of insulin resistance in human disease. Diabetes (1988); 37:1595–1607.
7. Morrison JA, Friedman LA, Wang P, Glueck CJ. Metabolic syndrome in childhood predicts adult metabolic syndrome and type 2 diabetes mellitus 25 to 30 years later. J Pediatr. (2008),152(2):201-206.
8. DuBose KD, Stewart EE, Charbonneau SR, Mayo MS, Donnelly JE. An evaluation of the prevalence of the metabolic syndrome in elementary school children. Acta Paediatr. (2006);95:1005-1011.
9. Goodman E, Daniels SR, Morrison JA, Huang B, Dolan LM. Contrasting prevalence of and demographic disparities in the World Health Organization and National Cholesterol Education Program Adult Treatment Panel III definitions of metabolic syndrome among adolescents. J Pediatr. (2004); 145:445–451.
10. Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S, Wong G, Bennett P, Shaw J, Caprio S. The metabolic syndrome in children and adolescents. Lancet, (2007);369:2059-2061.
11. International Diabetes Federation: The IDF consensus worldwide definition of the metabolic syndrome.
12. Perloff D, Grim C, Flack J, Frohlich ED, Hill M, McDonald M, Morgenstern BZ (2001): Human Blood Pressure Determination by Sphygmomanometry. Dallas, TX, American Heart Association.
13. Sattar N, Gaw A, Scherbakova O. Metabolic syndrome with and without C-reactive protein as a predictor of coronary heart disease and diabetes in the West of Scotland Coronary Prevention Study: Circulation (2003);108:414-9.

14. Golden SH, Folsom AR, Coresh J et al. Risk factor grouping related to insulin resistance and their synergistic effects on subclinical atherosclerosis: the atherosclerosis risk in communities study. Diabetes (2002);51:3069-76. 10 Hu G, Qiao Q, Tuomilehto J et al.
15. Cornier MA, Dabelea D, Hernandez TL, Lindstrom RC, Steig AJ, Stob NR, Van Pelt RE, Wang H, and Eckel RH. The Metabolic Syndrome. Endocrine Reviews (2008); 29(7):777–822.
16. Nasreddine L, Naja F, Tabet M, Habbal M-Z, Aida El-Aily A, Chrystel Haikal, Samira Sidani, Nada Adra & Nahla Hwalla. Obesity is associated with insulin resistance and components of the metabolic syndrome in Lebanese adolescents. Annals of Human Biology, (2012); 39(2): 122–128.
17. Singh R, Bhansali A, Sialy R, Aggarwal A. Prevalence of metabolic syndrome in adolescents from a north Indian population. Diabetic Medicine. (2007);(2):195–199.
18. Björntorp P. The regulation of adipose tissue distribution in humans. Int J Obes Relat Metab Disord. (1996);20(4):291-302.
19. Ritchie SA, Connell JM. The link between abdominal obesity, metabolic syndrome and cardiovascular disease. Nutr Metab Cardiovasc Dis. (2007);17(4):319-326.
20. Kanai H, Matsuzawa Y, Kotani K; et al. Close correlation of intra-abdominal fat accumulation to hypertension in obese women. Hypertension. (1990);16(5):484-490.
21. Alvarez GE, Beske SD, Ballard TP, Davy KP. Sympathetic neural activation in visceral obesity. Circulation. (2002);106(20):2533-2536.
22. Lönnqvist F, Thorne A, Large V, Arner P. Sex differences in visceral fat lipolysis and metabolic complications of obesity. Arterioscler Thromb Vasc Biol. (1997);17(7):1472-1480.
23. Hall JE. Pathophysiology of obesity hypertension. Curr Hypertens Rep. (2000);2(2):139-147.
24. Ginsberg HN Insulin resistance and cardiovascular disease. J Clin Invest (2000);106:453–458
25. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith Jr SC, Spertus JA, Costa F Diagnosis and management of the metabolic syndrome. An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Executive summary. Cardiol Rev (2005);13:322–327.
26. Beck-Nielsen H, Groop LC: Metabolic and genetic characterization of prediabetic states: sequence of events leading to non-insulin-dependent diabetes mellitus. J Clin Invest(1994); 94:1714 -1721.