Original Article

A study of anthropometeric markers in patients of type 2 diabetes mellitus with metabolic syndrome

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Abstract

Introduction: Diabetes Mellitus is the commonest metabolic abnormality in the world. Type 2 diabetes the commonest form of diabetes constitutes nearly 90% of diabetic population in any country. Type 2 diabetes mellitus (DM) is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production, and abnormal fat metabolism. Metabolic Syndrome (MS) is a multifactorial complex trait that is influenced by both environmental and genetic factors. The major features of metabolic syndrome include central obesity, hypertriglyceridemia, low HDL cholesterol, hyperglycemia, and hypertension. MS is an independent clinical indicator of macrovascular and microvascular complication in DM. Obesity, particularly visceral or central, as evidenced by the waist-hip ratio (WHR) is very common in type 2 diabetes mellitus and the rising prevalence of obesity in India is associated with increasing co-morbidities of obesity like cardiovascular disease. Obesity is associated with increase in plasma triglycerides, reduced HDL cholesterol, and increased numbers of small, dense LDL particles. **Materials and Methods-**100 patients of newly diagnosed type 2 diabetes mellitus in the age group of 25-60 years were included in the study. Patients with Gestational diabetes mellitus were excluded from the study. **Results:** Of the 100 patients studied, 58 patients has metabolic syndrome according to the International Diabetes Federation (IDF) criteria. Out of the total subjects with metabolic syndrome in our study, 66% were overweight (Body Mass Index: 25.0-29.9kg/m^2) and 34% were obese (Body Mass Index: >30kg/m^2).Among the studied subjects 33 males had increased WHR>0.90(Normal <0.90)**,**whereas all the females had WHR>0.85(Normal <0.85).Mean waist circumference in males with MS was 96.26±1.01cm as compared to those without MS (85.65±0.57) and mean waist circumference in females with MS was 90.34±0.68 as compared to those without MS (75.63±0.25).The mean HbA1c level was 1.09 times higher in patients with metabolic syndrome as compared to without metabolic syndrome. This difference in the mean level was found to be statistically significant (p<0.01).Waist circumference had significant positive correlation with TG (p<0.01).The mean TG and VLDL levels were markedly raised in patients of metabolic syndrome as compared to without metabolic syndrome. This difference was found to be significant (p<0.009), (p<0.007) respectively. **Conclusion:** Identifying patients with metabolic syndrome in type 2 DM and advising simple lifestyle modifications like weight loss can improve deranged lipid profile parameters and prevent cardiovascular complications. Therefore physicians treating type 2 diabetics should consider metabolic syndrome in their patients with greater emphasis.

Keywords: Diabetes Mellitus, Anthropometric markers, Metabolic Syndrome.

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INTRODUCTION

The global epidemic of diabetes will challenge our generation to develop novel strategies to prevent and treat this lifelong condition. Every 10 seconds, two people develop diabetes and one person dies from diabetes-related causes.[1]In 2007, 246 million people worldwide had diabetes. That number is expected to climb to 380 million by 2030 (International Diabetes Federation, 2007).[2] In most developed countries, diabetes is the fourth or fifth leading cause of death and there is concern that it will become an epidemic in many developing and newly

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industrialized nations. The Metabolic Syndrome (syndrome X, insulin resistance syndrome) consists of a constellation of metabolic abnormalities that confer increased risk of cardiovascular disease and diabetes mellitus. The major features of the metabolic syndrome include central obesity, hypertriglyceridemia, low HDL cholesterol, hyperglycemia, and hypertension. According to the new International Diabetes Federation (IDF) 2006 definition, for a person to be defined as having the metabolic syndrome they must have:

- Central Obesity (defined as waist circumference with ethnicity specific values).
- Plus any two of the following four factor:
- Raised Triglycerides > 150 mg/dl (1.7 mmol/L) or specific treatment for this lipid abnormality.
- Reduced HDL Cholesterol < 40 mg/dl (1.03 mmol/L) in males, < 50 mg/dl (1.29 mmol/L) in females or specific treatment for this lipid abnormality.
- Raised Blood pressure- Systolic Blood pressure >130 mmHg or Diastolic Blood pressure > 85 mmHg or treatment of previously diagnosed Hypertension.
- Raised Fasting Plasma Glucose >100 mg/dl (5.6 mmol/L) or previously diagnosed type 2 diabetes. If above 5.6 mmol/L or 100 mg/dl, oral glucose tolerance test (OGTT) is strongly recommended but is not necessary to define presence of the syndrome.

(If BMI is > 30 kg/m^2, central obesity can be assumed and waist circumference does not need to be measured.) Obesity is a chronic health problem affecting increasing number of people worldwide and is now recognized as a global epidemic. In India, obesity is emerging as an important health problem particularly in urban areas, paradoxically co-existing with under nutrition. Almost 30-65% of adult urban Indians are either overweight or obese or have abdominal obesity.[3]The rising prevalence of obesity in India has a direct correlation with the increasing prevalence of obesity-related co-morbidities; hypertension, the metabolic syndrome, dyslipidemia, type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD).[4] Simple measures of obesity are widely used in clinical practice like Body Mass Index (BMI), and waist-to-hip circumference ratio (WHR). The most widely used method to define thinness and fatness is BMI, a ratio of weight in kilograms divided by height in meters squared (kg/m^2). It has been correlated to morbidity and mortality risk in various populations.[5] Abdominal obesity is defined by easy-to-use parameters such as WHR. Though BMI, WHR correlate well with each other, it is also believed that combined use of these parameters of generalized andabdominal obesity may be better in identifying people at risk of cardiovascular disease (CVD) than either of them alone.[6,7] The currently recommended cut-offs of BMI recommended by World Health Organization include 18.5-24.9 kg/m^2 for normal, 25.0-29.9 for overweight and >30 kg/m^2 for obesity.[8] The currently recommended cut-offs of waist circumference (>102 cm in men and >88 cm in women) are not applicable to all the populations due to heterogeneity in the average levels of measurements and different relationship with cardiovascular risk. [9] International Diabetes Federation (IDF) has adopted different cut offs for waist circumference in different ethnicities. For south Asians the cut off values are ≥ 90 cm for males and ≥ 80 cm for females (Alberti KG *et al;*2005). Dyslipidemia, the hallmark of the metabolic syndrome, is summarised as (a) increased flux of free fatty acids, (b) raised TG values, (c) low high density lipoprotein (HDL) cholesterol values, (d) increased small, dense low density lipoprotein (LDL) values, and (e) raised apolipoprotein (Apo) B values. Dyslipidemia is widely established as an independent risk factor for cardiovascular disease. Low HDL cholesterol and hypertriglyceridemia have been found to be independently and significantly related to myocardial infarction/stroke in patients with metabolic syndrome. Additionally, a combination of high fasting glucose and low HDL cholesterol were shown to have primary predictive ability for coronary heart disease.[10, 11] Considering the important role of waist circumference, WHR and BMI in predicting risk of dyslipidemias and the fact that metabolic syndrome has been found to be an independent clinical indicator of macrovascular and microvascular complication in diabetes mellitus,[12,13]the objectives of the present study were to evaluate the anthropometric markers in patients of type 2 diabetes mellitus with metabolic syndrome and to study their correlation with parameters of lipid profile.

MATERIALS AND METHODS

The present study was carried out in the Department of Biochemistry, in association with the Department of Medicine, Government Medical College, Haldwani. 100 patients of newly diagnosed type 2 diabetes mellitus attending the Medicine OPD of GMC, Haldwani, were in the age group of 25-60 were included in the study**.** Patients with Gestational diabetes mellitus were excluded from the study. All patients were subjected to detailed history and thorough physical examination.

Statistical Analysis

The data were compiled and entered in MS Excel sheet and the analysis was carried out using the Statistical Package for the Social Sciences (SPSS 19.0.2) program for windows. Unpaired "t" test was used to analyze all the data for statistical significance. Correlation and regression

coefficient were also calculated among relevant parameters.

RESULTS

Of the 100 patients studied, 58 patients has metabolic syndrome according to the International Diabetes Federation (IDF) criteria.

Table 1: Age and Sex distribution of patients with metabolic syndrome

Age group (years)	Male		Female		Total	
	No	%	No	%	No	%
25-35	0	0	6	10	6	10
36-45	12	21	13	22	25	43
46-55	8	14	14	24	22	38
56-60	3	5	2	4	5	9
Total	**23**	**40**	**35**	**60**	**58**	**100**

Table 1 shows the age and sex distribution of patients with metabolic syndrome. 43 % of the patients with metabolic syndrome were in the age group 36-45 years followed by 38% within 46-55 years, 10% within 25-35 years and 9% within 56-60 years.

Table 2: Distribution of study subjects according to their Body Mass Index (According to WHO criteria)

Weight (based on BMI) kg/m^2	Male		Female		Total %
	No	%	No	%	
Under weight (≤18)	1	1	2	2	3
Normal (18-24)	17	17	24	24	41
Over weight (25-29)	16	16	21	21	37
Obese (>30)	9	9	10	10	19
Total	**43**	**43**	**57**	**57**	**100**

Table 3: Distribution of patients with metabolic syndrome according to Body Mass Index

Weight (based on BMI) kg/m^2	Male with MS	%	Female with MS	%	Total no.	%
Overweight(25-29)	16	29	21	37	37	66
Obese (>30)	9	16	10	18	19	34
Total	**25**	**45**	**31**	**55**	**56**	**100**

Table 3 shows the distribution of patients with metabolic syndrome according to body mass index. Out of total subjects with metabolic syndrome 66% were overweight and 34% were obese.

Table 4: Distribution of patients according to Waist Hip Ratio

WHR	Male (n=43)		Female(n=57)		Total No
	<0.90	>0.90	<0.85	>0.85	
	10	33	0	57	100

Table 4 shows the distribution of WHR in the studied subjects. The normal WHR ratio in males was<0.90 and females was<0.85. Among the studied subjects 33 males had increased WHR, whereas all the females had WHR > 0.85.

Table 5: Waist Circumference in studied subjects (mean±SD)

Variables	Male with MS	Female with MS	Male without MS	Female without MS	Range (cm)
Waist circumference (cm)	96.26±1.01	90.34±0.68	85.65±0.57	75.63±0.25	Male < 90 Female <80

Table 5 shows mean waist circumference in diabetic patients with and without metabolic syndrome. Mean waist circumference in males with metabolic syndrome was 96.26±1.01 and mean waist circumference in females with metabolic syndrome was 90.34±0.68.

Table 6: HbA1c levels in studied subjects (mean ± SD)

Groups	No. of cases	HbA1c (%)	Range (%)
With MS	58	8.39±0.20	4.8-5.9
Without MS	42	7.30±0.25	

Significance level as compared to without metabolic syndrome: $p<0.01$. Estimation of HbA1c was done in all patients with metabolic syndrome (n=58) and without metabolic syndrome (n=42). The final result was expressed as percent (%). Table 6 shows the mean plasma HbA1c levels in patients with metabolic syndrome (8.39 ± 0.20) and without metabolic

syndrome (7.30±0.25). The mean level was 1.09 times higher in patients with metabolic syndrome as compared to without metabolic syndrome. This difference in the mean level was found to be statistically significant (p<0.01).

Table 7: Serum HDLc, LDLc, VLDLc, TG and ApoB levels in studied subjects

Parameters	With MS (n=58)	Without MS (n=42)	*P* value
HDLc (mg/dl)	40.03±1.12	42.51±1.51	NS
LDLc (mg/dl)	114.60±5.01	108.10±6.75	NS
VLDLc (mg/dl)	40.45±2.34	31.10±2.15	*P*<0.007
TG (mg/dl)	256.52±24.92	166.74±13.59	*P*<0.009
Apo B (mg/dl)	76.89±3.92	75.28±3.67	NS

Estimation of serum HDLc, LDLc, VLDLc, TG and ApoB was done in all patients with metabolic syndrome (n=58) and without metabolic syndrome (N=42). The final result was expressed as mg/dl. Table 7 shows the mean serum HDLc, LDLc, VLDLc, TG and ApoB levels in patients with metabolic syndrome (40.03±1.12),(114.60±5.01),(40.45±2.34),(256.52±24.92),(76.89±3.92) and without metabolic syndrome (42.51±1.51),(108.10±6.75),(31.10±2.15),(166.74±13.59),(76.89±3.920) respectively. No significant difference was found in HDLc, LDLc and ApoB levels in patients with metabolic syndrome as compared to without metabolic syndrome. The mean VLDLc and TG level was markedly raised in patients of metabolic syndrome as compared to without metabolic syndrome. This difference was found to be significant (p <0.007), (p<0.009) respectively.

Table 8: Correlation between waist circumference with TG, HDLc, Apo B, and HbA1c levels in patients of metabolic syndrome (n= 58)

	WC (cm)	TG (mg/dl)	HDLc (mg/dl)	Apo-B (mg/dl)	HbA1c (%)
MEAN±SD	92.11±0.72	256.52±24.92	40.03±1.12	76.89±3.92	8.39±0.20
Correlation coefficient (r)		0.956	0.392	0.245	0.454
S.E.OF 'r'		0.363	0.740	0.239	0.426
Pvalue		<0.01	<0.05	NS	<0.05
Regression Coefficient(b)		0.885	0.111	0.284	0.696
'a' value		25.39	26.97	27.38	26.80

Table 8 and Fig. 1, 2 and 3 shows the correlation of waist circumference with TG, HDLc, Apo B, and HbA1c in patients of metabolic syndrome. Waist circumference had significant positive correlation with TG (r=0.095; p<0.01) and HbA1c (r=0.45; p<0.05).HDLc (r=0.039; p<0.05) levels had negative correlation with waist circumference. Waist circumference had no significant correlation between Apo B. Linear relationships were observed between the parameters.

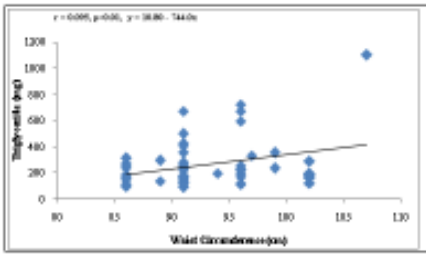

Figure 1

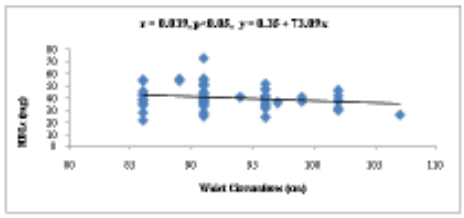

Figure 2

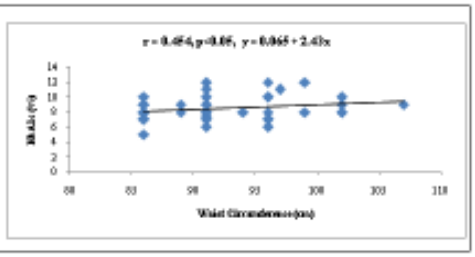

Figure 3

Legend

Figure 1: Correlation between waist circumference and triglyceride levels in patients with metabolic syndrome (n=58)

Figure 2: Correlation between waist circumference and HDLc levels in patients with metabolic syndrome (n = 58)

Figure 3: Correlation between waist circumference and HbA1c levels in patients with metabolic syndrome (n = 58)

DISCUSSION

Metabolic syndrome is a cluster of cardiovascular risk factors. The components of metabolic syndrome are obesity, hypertension, low glucose tolerance and dyslipidemia. Association of metabolic syndrome with diabetes mellitus increases the risk of cardiovascular complications significantly. Diabetes is the most feared disease because it leads to a variety of complications including end-stage vascular disease, cardiovascular damage and retinal abnormalities. As a consequence, a large burden is put on the National Health System of all countries around the world. In the present study 100 patients of newly diagnosed type 2 DM attending the Medicine OPD were included. According to the new International Diabetes Federation (IDF) 2006, out of these 100 patients studied, 58 patients met the criteria of

metabolic syndrome (Table 1). Alshkri and Elmehdawi found that prevalence of metabolic syndrome among type 2 diabetes mellitus patients in Libya was 92% according to NCEP-ATP III criteria and 80.8% according to IDF criteria.[13] Lin *et al,* found it to be 70% in USA, according to NCEP-ATP III criteria.[14]Monami*et al*, found in Italy to be68.4% according to NCEP-ATP III criteria and 73.7% according to IDF criteria.[15] Lu *et al*, found that prevalence of metabolic syndrome among type 2 diabetes mellitus patients in UK was 61% according to NCEP-ATP III criteria and 54% according to IDF criteria.[16] Our study showed that 60% of the patients of metabolic syndrome were female. There was an overall female predominance in patients with metabolic syndrome. In the study of Alshkri and Elmehdawi, out of 99 patients, 61 were female and 38 were males.[13]the results are comparable with the present study. Ford *et al,* found that prevalence of metabolic syndrome were more common in male which is in contrast with this study.[17] Obesity has been known to be positively related to insulin resistance. Increased secretion of free fatty acids, inflammatory cytokines and decreased secretion of adiponectin are molecules mediating obesity and insulin resistance.[18] Visceral obesity is closely linked to insulin resistance, and is currently regarded as a principle component of the metabolic syndrome. It is well documented that insulin resistance is predictive of the risk of type 2 diabetes and cardiovascular disease.[19] Abdominal obesity characterized by high waist circumference is a stronger predictor than generalized obesity defined by elevated Body Mass Index (BMI) of subsequent development of major coronary event, vascular mortality, diabetes and metabolic syndrome. In our study the mean waist circumference in patients of metabolic syndrome was (96.26±1.01 cm) in males as compared to those without MS (85.65±0.57and (90.34±0.68 cm) in females as compared to those without metabolic syndrome (75.63±0.25). (Table 5). In a study by Oscar H *et al.*, 2009 waist circumference in normal subjects was (89.016±13.056cm) and in diabetics (102. 656± 11.52 cm), and incidence of metabolic syndrome was 40.6%.[20] BMI was calculated by the formula weight in kg/ (height in meter) ^{2}. Overweight is defined as a BMI of 25-29 and obese as a BMI >30.[21] It is known that Indians are prone to developing diabetes at a lower BMI in comparison to the western population.

Shekhar *et al.,* 2005 in their study in Mysore on urban patients found the BMI to be 23.9 in males and 25.3 in females.[22] In the study of Alshkri and Elmehdawi, 2008 out of 99 patients, 61 were females and 38 were males of all patients, 74.4% were obese and obesity was significantly more frequent among females ($p<0.001$). Mean BMI was 33.6 kg/m.^{2} In our study according to BMI, obesity was predominant in females as compared to males (Table 3), which is comparable with the above studies. The suggestion for the use of proxy anthropometric indicators arose from a 12-year follow-up of middle-aged men, which showed that abdominal obesity (measured as waist–hip ratio) was associated with an increased risk of myocardial infarction, stroke and premature death, whereas these diseases were not associated with measures of generalized obesity such as BMI.[23] In our study WHR was increased in all the females studied (>0.85) while 33% of males had increased WHR (> 0.90) (Table 4). Various studies have shown that WHR is an important cardiovascular risk factor and greater levels are associated with multiple risk factors. Gupta R *et al*., 2003 reported that WHR >0.9 in men and >0.8 in women is associated with a significant increase in multiple risk factors.[24] HbA1c is the most commonly measured parameter for long term monitoring of diabetes mellitus. The level of HbA1c has been widely accepted as an indicator of mean daily blood glucose concentration over the preceding 8-12 weeks. In our study HbA1c levels were significantly elevated ($p < 0.01$) in patients of metabolic syndrome as compared to without metabolic syndrome (Table 6). Osei *et al.,* 2003, reported that in 219 non-diabetic, obese, first-degree relatives of African-American patients with Type 2 diabetes, the upper tertile of HbA1c reflected some components of metabolic syndrome.[25] These results suggest that HbA1c may be a surrogate marker not only of future diabetes, but also of CVD. Although there are many studies which report the utility of HbA1c in predicting CVD and diabetes, there are few which investigate the usefulness of HbA1c as a predictor of metabolic syndrome. Jasmin S *et al.,* 2008, observed that metabolic syndrome group showed significantly higher glucose, HbA1c levels and waist circumference.[26] Dyslipidemia, the major constituent of the metabolic syndrome, is characterised as an increased free fatty acid, triglyceride, small dense LDLc and low HDLc levels. Dyslipidemia is a widely accepted risk factor for coronary heart disease. The hypertriglyceridemia seen with abdominal obesity and insulin resistance is related to the oversecretion of triglyceride-rich VLDL particles. An increased rate of hepatic FFA uptake stimulates the secretion of Apo B-100, leading to increased numbers of Apo B containing particles and possibly hypertriglyceridemia.[27] In our study serum VLDLc ($p<0.007$) and serum triglyceride ($p<0.009$) levels were significantly elevated in patients of metabolic syndrome as compared had without metabolic syndrome (Table 7), whereas no significant difference was found between HDLc and LDLc (Table 7). Apo B is one of the component found in chylomicron, VLDL, intermediate density lipoprotein and LDL. The association of Apo B with diabetes and metabolic

syndrome has been shown, as it has potential role as a subclinical inflammatory agent.[28]Apo B is a good surrogate measure of increased LDL particle numbers in people with metabolic syndrome and insulin resistance and small LDL particle number was best correlated with Apo B (and triglycerides and HDL-cholesterol) in the Framingham Heart study.[29,30] The present study shows Serum Apo B levels was not significantly elevated in patients of metabolic syndrome (Table 7), whereas Lim *et al.*, 2011, found serum Apo B to be higher in diabetic patients with metabolic syndrome than those without metabolic syndrome, and this difference was significant even after correcting with LDLc.[31]Ryoo*et al.*, 2013, conducted a cohort study which followed up 25,193 healthy Korean males without metabolic syndrome for 5 years, and reported that Apo B was a predictive factor for metabolic syndrome.[32] During 5 years of follow up, 5,407 (21.5%) were diagnosed with metabolic syndrome, and there was a significant positive correlation between the occurrence of metabolic syndrome Apo B levels.

CONCLUSION

Defining metabolic syndrome in patients of type 2 Diabetes Mellitus can serve as a simple clinical approach to identify a person for intervention to reduce obesity and cardiovascular complications. This can lead to deceased morbidity and mortality from microvascular and macrovascular complication in patients of type 2 diabetes mellitus and significantly enhance their life style. Identifying patients with metabolic syndrome and proper intervention in type 2 diabetes mellitus will also significantly reduce the burden on state health services. Physicians in developing countries need to reiterate lifestyle modifications like weight loss, exercise of $\geq$ 30 min/day, intake of high dietary fibers which would reduce cardiovascular diseases arising from the epidemic of obesity and the metabolic syndrome. In addition, obese individuals should be targeted for intense lipid lowering therapy, when necessary.

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REFERENCES

1. IC Health. National Cardiovascular Disease Database. Available from: http: //www. Who India. org/LinkFiles/NMH_Resources_National_CVD_database -Final_Report. pdf, accessed on February 10, 2008.
2. International Diabetes Federation (IDF). (2007). Diabetes alas. Belgium: International Diabetes Federation.
3. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. J ClinEndocrinol Metabolism 2008; 93(11 Suppl1):S9-30.
4. Gupta R, Gupta VP, Sarna M, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch-2. Indian Heart J 2002; 54(1):59-66.
5. Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. N Engl J Med. 1999; 341(6):427-34.
6. Ardern CI, Katzmarzyk PT, Janssen I, Ross R. Discrimination of health risk by combined body mass index and waist circumference. Obes Res 2003; 11(1):135-42.
7. Meisinger C, Doring A, Thorand B, Heier M, Lowel H. Body fat distribution and risk of type 2 diabetes in the general population: are there differences between men and women? The MONICA/KORA Augsburg cohort study. Am J ClinNutr. 2006; 84(3):483-9. T13.
8. World Health Organization. Obesity: preventing and managing the global epidemic. Report of WHO consultation presented at: the World Health Organization; June 3-5, 1997; Geneva, Switzerland. Publication WHO/NUT/NCD/98. 1.
9. Genest JG Jr. Dyslipidemia and coronary artery disease. Can J Cardiol 2000; 16 (suppl A):3–4A.
10. Anderson JL, Horne BD, Jones HU, et al, for the intermountain heart collaborative (IHC) study. Which features of the metabolic syndrome predict the prevalence and clinical outcomes of angiographic coronary artery disease? Cardiology 2004; 101:185–93.
11. Su Q, Tsai J, Xu E, Qiu W, Bereczki E, Santha M, Adeli K (2009). "Apolipoprotein B100 acts as a molecular link between lipid-induced endoplasmic reticulum stress and hepatic insulin resistance". Hepatology 50 (1): 77–84. doi:10. 1002/hep. 22960. PMID 19434737.
12. Kahn R, Buse J, Ferrannini E, Stern Ml. The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care. 2005; 28:2289 –2304.
13. Alshkri MM, Elmehdawi RR. Metabolic Syndrome among Type-2 Diabetic Patients in Benghazi-Libya: A pilot study. Libyan J Med 2008; AOP: 080715: [Online], available from: www. ljm. org. ly. (accessed on 12 june 2008).
14. Lin CC, Liu CS, Li TC, et al: Microalbuminuria and the metabolic syndrome and its components in the Chinese population. Eur J Clin Invest 2007, 37:783–790.
15. Monami M, Marchionni N, Masotti G, Mannucci E. IDF and ATP-III definitions of metabolic syndrome in the

prediction of all-cause mortality in type 2 diabetic patients. Diabetes Obese Metab2007; 9(3): 350-353.
16. Lu B, Yang Y, Song X, Dong X. An evaluation of the International Diabetes Federation definition of metabolic syndrome in Chinese patients older than 30 years and diagnosed with type 2 diabetes mellitus. Metabolism 2006; 5(8): 1088-1096.
17. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: finding from the third National Health and Nutrition Examination Survey. JAMA 2002; 287: 356-359.
18. Matsuzawa Y, Funahashi T, Kimara S, Shimomura Adiponectin and metabolic syndrome. ArteriosclerThrombVascBiol 2004; 24: 29–33.
19. Abdul – Ghani MA, K. Williams, RA. DeFronzo, M. Stern (2007): What is the best predictor of future type 2 diabestes. Diabetes Care. ; 30:1544-1548.
20. Oscar H. M. Franco, Joseph. Massaro, Jacky Civil, R. Mark. Cobain (2009) : Form Unilever Corporate Research, Sharnbrook, UK (O. H. F., J. C., M. R. C., B. O.); University of Warwick, Warwick Medical School, Health Sciences Research Institute, Coventry, UK (O. H. F.); Department of Mathematics/Statistics and Biostatistics, Boston University, Boston, Mass (J. M. M.); and the National, Heart, Lung and Blood Institute's Framingham Heart Study, Framingham, Mass (R. B. D.). Circulation. ; 120:1943-1950.
21. World Health Organization. Obesity: preventing and managing the global epidemic. Report of WHO consultation presented at: the World Health Organization; June 3-5, 1997; Geneva, Switzerland. Publication WHO/NUT/NCD/98. 1.
22. MA Shekhar, HM Somashekhar, BS Vishwanath. Study of incidence of obesity in newly diagnosed type 2 diabetics using anthropometric measurements Int J DiabDev Countries 2005;25: 102-04
23. Larsson B, Svardsudd K, Welin L et al. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. British Medical Journal, 1984, 288(6428):1401-1404.
24. Gupta R, Rastogi S, Panwar RB, Soangra MR, Gupta VP, Gupta KD. Major coronary risk factors and coronary heart disease epidemic in India. South Asian J PrevCardiol2003; 7:11-40.
25. Osei K, Rhinesmith S, Gaillard T, Schuster D. Is glycosylated hemoglobin A1c a surrogate for metabolic syndrome in nondiabetic, first-degree relatives of African-American patients with type 2 diabetes? J ClinEndocrinolMetab2003; 88: 4596–4601.
26. Jasmin S, VenesaAbdie-Nekie et al, Study Of HbA1c As A Reliable Indicator For Metabolic Syndrome InNon Diabetic Patients 2008.
27. Marsh JB. Lipoprotein metabolism in obesity and diabetes: insights from stable isotope kinetic studies in humans. Nutr Rev. 2003; 61:363–375.
28. Faraj M, Messier L, Bastard JP, et al. Apolipoprotein B: a predictor of inflammatory status in postmenopausal overweight and obese women. Diabetologia 2006; 49:1637-46.
29. Williams K, Sniderman AD, Sattar N, D'Agostino R Jr, Wagenknecht LE, Haffner SM. Comparisons of the associations of apolipoprotein B and low-density lipoprotein cholesterol with other cardiovascular risk factors in the Insulin Resistance Atherosclerosis Study (IRAS). Circulation 2003; 108:2312-16.
30. Kathiresan S, Otvos JD, Sullivan LM, et al. Increased small low-density lipoprotein particle number: a prominent feature of the metabolic syndrome in the Framingham Heart Study. Circulation 2006; 113:20-29.
31. Lim JS, Lee DH, Park JY, Jin SH, Jacobs DR (2011). "Reliability of low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein B measurement". Journal of Clinical Lipidology 5 (4): 264–272. doi:10. 1016/j. jacl. 2011. 05. 004. PMID 17478563.
32. Ryoo JH, Park SK. Association of apolipoprotein B and incidence of metabolic syndrome in Korean men: a 5-years' follow-up study. Atherosclerosis 2013; 226:496-501.
33. Alberti KG, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group. The metabolic syndrome—a new worldwide definition. Lancet 2005; 366:1059–1062.

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