Original Article

Prevalence and Correlates of Metabolic Syndrome in the Adolescents of Rural Wardha

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ABSTRACT

Background and Objective: Metabolic syndrome is a major concern as a precursor of cardiometabolic diseases. The present study was designed to study the magnitude and correlates of metabolic syndrome among the adolescents of rural Wardha. **Materials and Methods:** A cross-sectional study was carried out among the adolescents (10-19 years) of Anji PHC. A sample of 405 was selected by random sampling from the sampling frame available with department of Community Medicine. We collected data about their sociodemographic variables and other cardiometabolic risk factors. Fasting blood sample was collected to measure lipid profile and blood glucose. Blood pressure and anthropometric measurements (height, weight, and waist circumference) were also taken. **Results:** Prevalence of metabolic syndrome using ATP-III criteria modified for adolescents was found to be 9.9% (95% CI: 7.3-13.1) in the study population and lower level of high-density lipoprotein (HDL) cholesterol was found with a prevalence of 58.3% (95% CI: 53.4-63.0). The prevalence of metabolic syndrome was found to be significantly ($P < 0.05$) associated with the presence of obesity and hypertension among family members. **Interpretation:** There was a moderately high prevalence of metabolic syndrome among rural adolescents. **Conclusion:** The early identification of cardiometabolic risk factors such as hypertension and obesity can help prevent metabolic syndrome, diabetes, and cardiovascular disease.

Keywords: Adolescent, metabolic syndrome, dyslipidemia

Introduction

Metabolic syndrome is a group of cardiometabolic abnormalities, which include abdominal obesity, hypertension, dyslipidemia, and hyperglycemia.[1] The metabolic syndrome is a risk factor for cardiovascular diseases and an increasing trend has been reported even among adolescents.[2,3] People with metabolic syndrome have a greater risk of coronary heart disease and risk of cardiovascular mortality.[4]

Increasing trend of obesity among children and adolescents has led to increased prevalence of insulin resistance, paving the way for cardiometabolic risk factors such as high cholesterol, triglyceride level, and raised blood pressure; high insulin and sugar level promote inflammation conditions in body.[5] The coronary lesions or plaques stick to the inside of the arteries that have become rough due to inflammation and cause hardening of arteries, which ultimately lead to heart attacks and strokes.[6]

Autopsy studies in youth have shown that early stages of coronary atherosclerosis are associated with the presence of cardiovascular risk factors. The extent of lesions is directly proportional to the number of cardiovascular risk factors. Therefore, the high prevalence of the metabolic syndrome among adolescents could lead to an increase in cardiovascular disease in adulthood. Early recognition of the problem can help formulate strategies to counter this adverse cascade. Hence, it becomes important to study metabolic syndrome in adolescents.[7]

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Because of lifestyle changes and urbanization, the prevalence of metabolic syndrome is becoming common in rural areas as well.[8,9] However, very little information is available in this regard from rural areas of India, especially among adolescents. Hence, we studied the prevalence of metabolic syndrome and its correlates among adolescents of rural Wardha.

Materials and Methods

Study design, study area, and study population
A cross-sectional study was carried out in a rural area of Primary Health Centre, Anji situated in Wardha District of Central India. All adolescents in the age group of 10-19 years of Primary Health Centre, Anji were included in the study. The study was carried out between June 2008 and May 2009.

Sample size and sampling technique
Considering prevalence of metabolic syndrome to be 4.2% among adolescents,[8] a sample size of 405 was required at α-error of 5%, allowable error of 2%, and non-response rate 5%. The subjects were selected by using simple random sampling using computer-generated random numbers. The sampling frame available with the Department of Community Medicine was used for generating the random numbers. The subjects who were not willing to remain fasting or were not willing to participate in the study were excluded. We obtained approval from the Institutional Human Ethics Committee of Mahatma Gandhi Institute of Medical Sciences, Sevagram. The subjects were selected after obtaining written informed consent from the parents and assent was obtained from the subjects. In case of children aged 18 years and above, consent was taken from them.

Data collection and measurements
House visits were made to collect the data using a pre-designed and pre-tested interview schedule. Information was collected on age, sex, caste, education, type of family, physical activity, family income, and family history of obesity, hypertension, and diabetes.

Physical activity was measured by using the Physical Activity Scoring System following instructions by Ramachandran *et al.*[9] Ramachandran used this system for adults. We modified the system for use in adolescents by modifying the categories of occupation/work.

We collected 10 ml blood through venepuncture in the morning after overnight fasting for fasting blood glucose level, lipid profile comprising total cholesterol, triglyceride, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL).

We measured height and weight of the subjects with a non-stretchable measuring tape and weighing machine to the nearest 0.1 cm and 0.5 kg, respectively. Waist circumference was measured in a horizontal plane at the midpoint between the bottom of the rib cage and above the top of the iliac crest with person breathing silently. Body mass index (BMI) was calculated by dividing weight (kilograms) by height squared (meter^2). Blood pressure was recorded three times with a mercury sphygmomanometer (Diamond Co., Industrial Electronics and Allied Products, Electronics Cooperative Estate, Pune, Maharashtra) in the right arm in sitting position in subjects after a 10-minute rest between each recording. The systolic and diastolic high blood pressure is defined by blood pressure value >90th percentile for age, sex, and height.[10,11]

The presence of obesity in the family was assessed by measuring BMI of parents.[12] At least one parent being obese was considered as family history of obesity. Similarly, for family history of hypertension, blood pressure measurements of parents were taken. At least one parent being hypertensive by JNC-VII criteria was considered as family history of hypertension. For family history of diabetes, parents were asked whether they are known diabetics.

Biochemical analysis
Fasting plasma glucose was measured by the glucose oxidase peroxidase method.[12] Total cholesterol was measured by CHOD-PAP method, Triglyceride by GPO-Trinder method, and HDL cholesterol (HDLc) by phospotungstic acid method using XL-300 autoanalyser, and LDL cholesterol value was calculated.[13-16] Coefficient of variation (CV) of glucose by the aforesaid method in our lab lies within 4%, and that of total cholesterol, triglyceride, and HDLc is within 3.5%, 6.5%, and 7%, respectively, indicating a good precision of the methods in use.

Definition of metabolic syndrome
The National Cholesterol Education Program (ATP III) definition modified for age has been used to define abnormal level of cardiometabolic risk factors in adolescents.[17] The criteria for abnormal level of cardiometabolic risk factors were:
1. Waist circumference at or above the 90th percentile value for age and sex from sample population classified as was having abdominal obesity using CDC reference population,
2. Triglyceride level ≥110 mg/dl,
3. HDL cholesterol level ≤40 mg/dl,
4. Systolic and diastolic high blood pressure is defined by blood pressure value ≥90th percentile for age sex and height,

5. Fasting blood glucose levels ≥100 mg/dl. Presence of any three of these five risk factors above constituted metabolic syndrome.[18]

Statistical analysis

Statistical analysis was conducted by using SPSS 12.0 software. The chi-square test was applied to test the significance of difference between two groups, and *P* value <0.05 was considered as significant. The odds ratio was calculated to evaluate the risk factors along with their 95% confidence interval. Multivariate logistic regression using the backward LR method was carried out to derive a final model of correlates of metabolic syndrome. All nine risk factors were pushed into the model.

RESULTS

Study subjects

Out of the 405 adolescents studied, 39.2% were in early adolescence (age <15 years), 44.9% were male, 46.2% were from other backward caste (OBC), 90.4% were from nuclear family, and 71.9% were involved in light physical activity. About 5.4% of the subjects were overweight (95th percentile ≥ BMI ≥85th percentile). None of them was obese. About 10.6% subjects had a family history of obesity, and 5.9% and 3.2% subjects had a family history of hypertension and diabetes, respectively.

Cardiovascular abnormalities

The mean systolic and diastolic blood pressure was found to be 112.0 mmHg and 73.0 mmHg, respectively. The mean values were 61.5 cm for waist circumference, 17.1 Kg/m² for body mass index, 38.9 mg/dl for HDLc, 92.8 mg/dl for TG, and 127.2 for total cholesterol. Mean fasting glucose was found to be 87.3 mg/dl [Table 1].

Figure 1 shows the magnitude of Cardiometabolic abnormalities in the study subjects. About 2.2% (95% CI: 1.1-4.0) adolescents had waist circumference equal to or more than 90th percentile for the age and sex. Blood pressure levels were higher in 22.4% (95% CI: 18.6-26.7) adolescents while triglyceride levels were higher than cutoff in 27.9% (95% CI: 23.7-32.4) adolescents. About 58.3% (95% CI: 53.4-63.0) adolescents had HDLc levels

lower than desired, while fasting blood glucose was higher than cutoff among 13.8% (95% CI: 10.7-17.5) adolescents.

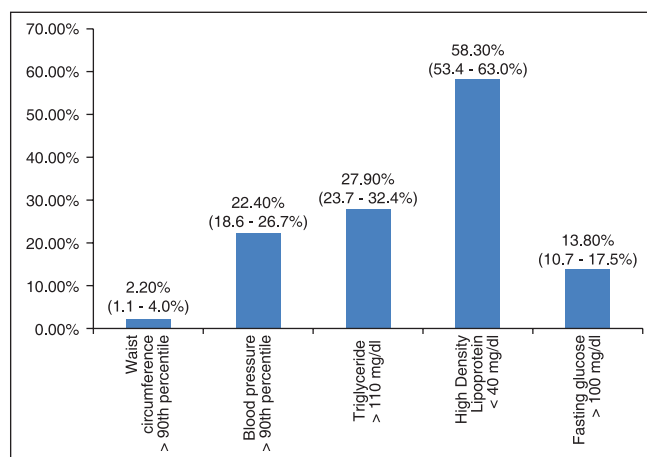### Prevalence of metabolic syndrome

The overall prevalence of metabolic syndrome was 9.9% (95% CI: 7.3-13.1). It was 10.7% (95% CI: 5.6-14.7) in early adolescence (<15 years) and 9.3% (95% CI: 6.2-13.4) in late adolescence (>15 years). Among male adolescents, it was observed to be 7.7% (95% CI: 4.5-12.2) while among female adolescents it was 11.7% (95% CI: 8.0-16.3). The difference in prevalence by age group and sex was not statistically significant [Table 2].

Risk factors

The risk of metabolic syndrome did not differ significantly in age group, sex, caste, type of family, education, physical activity, family income, and family history of diabetes. However, it differed significantly with a family history of obesity and by family history of hypertension. Adolescents having a family history of obesity had 2.90 times (95% CI: 1.51-5.60) higher odds of metabolic syndrome as compared to those who did not have a family history of obesity. Similarly, those who had a family history of hypertension were at 3.41 times (95% CI: 1.83-8.83) at higher odds of metabolic syndrome as compared to those who did not have a family history of hypertension. Nagekerke's R² for the final model was 0.054 [Tables 2 and 3].

DISCUSSION

In the present study, we found an overall prevalence of metabolic syndrome to be 9.9%, which is quite high as compared to another Indian study that reported 4.2% in adolescents aged 12-17 years.[8] National Health and Nutrition Examination Survey also reported 4.2% of the prevalence of metabolic syndrome in adolescents 12-19 years of age group, and it increased to 6.4% later study during 1999-2000.[19] A comparatively higher

Table 1: Mean values of cardiovascular abnormalities (*N* = 405)

Cardiovascular abnormality	Mean	Standard error
Systolic blood pressure (mmHg)	112.0	0.38
Diastolic blood pressure (mmHg)	73.0	0.32
Waist circumference (cm)	61.5	0.30
Body Mass Index (kg/m²)	17.1	0.13
High density lipoprotein (mg/dl)	38.9	0.48
Triglycerides (mg/dl)	92.8	1.57
Total cholesterol (mg/dl)	127.2	1.59
Impaired fasting glucose (mg/dl)	87.3	0.63
Low density lipoprotein (mg/dl)	68.4	0.85

Figure 1: Prevalence (95% CI) of cardiovascular abnormalities in rural adolescents of Wardha

Table 2: Prevalence of metabolic syndrome and its correlates

Variables	Examined number (%)	Metabolic syndrome numbers (%; 95% CI)	Crude OR (95% CI)
Overall	405 (100)	40 (9.9; 7.3-13.1)	—
Age (years)			
<15	159 (39.2)	17 (10.7; 6.6-16.2)	1
≥15	246 (60.8)	23 (9.3; 6.2-13.4)	0.94 (0.47-1.87)
Sex			
Male	182 (44.9)	14 (7.7; 4.5-12.2)	1
Female	223 (55.1)	26 (11.7; 8.0-16.3)	1.58 (0.80-3.21)
Caste			
General	47 (11.6)	4 (8.5; 2.8-19.2)	1.23 (0.33-3.89)
OBC	187 (46.2)	24 (12.8; 8.5-18.2)	1.95 (0.95-4.16)
ST/SC	171 (42.2)	12 (7.0; 3.9-11.6)	1
Type of family			
Joint	39 (9.6)	7 (17.9; 8.2-32.3)	2.20(0.84-5.24)
Nuclear	366 (90.4)	33 (9.0; 6.3-12.2)	1
Physical activity			
Light	291 (71.9)	25 (8.6; 5.7-12.2)	0.62 (0.32-1.25)
Moderate	114 (28.1)	15 (13.2; 7.8-20.3)	1
Family income			
1st Quartile (INR < 6000)	117 (28.9)	9 (7.6; 3.8-13.6)	1
2nd Quartile (INR 6001-8000)	115 (28.4)	10 (8.6; 4.5-14.9)	1.14 (0.44-3.62)
3rd Quartile (INR 8001-10000)	89 (21.9)	11 (12.3; 6.6-20.4)	1.69 (0.66-4.42)
4th Quartile (INR > 10000)	84 (20.8)	10 (11.9; 6.2-20.1)	1.62 (0.61-4.31)
Family history of obesity			
Yes	43 (10.6)	8 (18.6; 9.0-32.2)	2.35 (1.01-5.51)
No	362 (89.4)	32 (8.8; 6.2-12.1)	1
Family history of hypertension			
Yes	24 (5.9)	6 (25.0; 10.8-44.9)	3.39 (1.16-8.91)
No	381 (94.1)	34 (8.9; 6.7-12.7)	1
Family history of diabetes			
Yes	13 (3.2)	2 (15.4; 2.6-42.2)	1.69 (0.25-7.13)
No	392 (96.8)	38 (9.7; 7.1-12.9)	1

Table 3: Correlates of metabolic syndrome: Final model by multivariate logistic regression

Variables	Odds ratio	95% Confidence interval
Presence of family history of obesity	2.90	1.51-5.60
Presence of family history of hypertension	3.41	1.83-8.83

Nagekerke's R-square = 0.054

prevalence of metabolic syndrome reported in our study can be attributed to differences in study setting and population. It can also be due to the difference in the definition used for metabolic syndrome. Since the criteria for adolescents are not well defined, modifications were made, which may yield different results due to various ethnicities. Singh *et al.* demonstrated how lowering of cut-off level of fasting plasma glucose increased prevalence of metabolic syndrome.[8] Similarly, Ferranti *et al.* discussed how different definitions of metabolic syndrome are used and how that has led to different estimates of prevalence of the disease.[20] Further, it is worth mentioning that Cook *et al.*[21] modified the criteria for fasting blood glucose to be 110 mg/dl (from ADA criteria) whereas in our study, it

has been fixed to a lower value of 100 mg/dl. As reported by Singh *et al.*,[8] in India, the prevalence was found to increase as the fasting blood glucose level had been lowered to 5.5 mM (100 mg/dl) level. Thus, the observed higher prevalence may in part be attributed to the changed criterion. A study by Esmaillzadeh *et al.* conducted in the capital city of Iran reported a very high prevalence of 10.1% for the metabolic syndrome in adolescents aged 10-19 years (10.3% in boys and 9.9% in girls).[22] Thus, the emerging global picture is getting murkier in the recent years, appearing to develop serious epidemiological menace in cardiovascular disorder.

Advent of rapid urbanization and wide cultural intermingling ushered a revolution in the lifestyle over the last one or two decades. The adolescent of today might be considered as the mirror of this changed lifestyle. Hence, it is worthwhile to look for the sociobiological factors that may contribute to this condition in this age group.

The prevalence of metabolic syndrome was found to be higher in females (11.7%) than in males (7.7%).

However, the higher prevalence among females was not statistically significant. Igleseder *et al.* suggested greater cardiogenic risk in females with metabolic syndrome,[6] although a considerable number of reports did not suggest any significant age- and sex-dependant difference in prevalence of metabolic syndrome. The cross-sectional survey in adolescents in Ho Chi Minh City showed that overall, the prevalence of metabolic syndrome was 4.6%, and there was no difference by sex but prevalence of metabolic syndrome was slightly higher in females (4.7%) than in males (4.6%).[23] Contrary to our finding, Cook *et al.* showed higher prevalence in males (6.1%) than in females (2.1%).[19] Difference in prevalence of metabolic syndrome between males and females is a rather complicated issue; differential impact of sex hormone on lipoprotein metabolism and HDL, in particular, may play significant role. Besides, this stress level and physical activity has possible interaction with the various components of metabolic syndrome, which may have varied sex predilection.[24]

The result of this study, however, did not reveal any socioeconomic aspect to be associated with metabolic syndrome. On the contrary, biological factors such as family history of obesity and hypertension were found to be significantly associated with this condition. In this context, it is worth mentioning that obesity is an anthropometric parameter that relies upon the waist circumference, which reflects only the subcutaneous fat. In recent years, the growing database made a paradigm shift in our understanding of the metabolic stress and gradually, we tend to appreciate the more definitive role of visceral adiposity.[25] The conventional anthropometric obesity indicators fail to measure this. The circulatory triglyceride level might be considered as a justifiable representative of such ectopic adiposity.[26] In our study, we found a sizeable fraction of the adolescent population (27%) to have high triglyceride level. The significance of triglyceride as a harbinger of impending cardiovascular disease, particularly in adolescence, is gradually being appreciated.[27] It would be interesting to look for the lipid profile of the family members of the patients for further correlation. A major percentage of populace under study showed low HDL level, which definitely indicates an altered lipoprotein metabolism with a skew favoring cardiometabolic stress. Such metabolic stress associated with visceral adiposity has been reported to be attributed through derangement of rennin–angiotensin axis leading to hypertension.[25] This might explain the greater statistical impact of the family history of hypertension in our study. Interestingly, we also recorded significantly higher systolic and/or diastolic pressure in 22% adolescent population under study. In this context, a relevant report confirmed the definitive contribution of high triglyceride and fasting insulin level in development of hypertension.[28] Of late, an Indian group also reported hypertension in the adolescent population to be attributed to obesity, higher serum lipids, and family history of hypertension.[29] The study undeniably highlighted the importance of familial traits in the cardiometabolic disorder.

The limitations of the study were that we did not study dietary intake, which is one of the important correlates of metabolic syndrome. The other limitations being family history of diabetes was taken as reported; similarly, family history of obesity was based on current BMI status. These might have led to some degree of misclassification of exposure status.

Finally, we conclude that there is a definite cause of concern regarding the moderately high prevalence of metabolic syndrome in rural communities of India. Identification of cardiometabolic risk factors, i.e., family history of diabetes and hypertension at the early age can be useful to prevent consequent diabetes and cardiovascular disorders.

References

1. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 1988;37:1595-607.

2. Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, *et al.* Obesity and the metabolic syndrome in children and adolescents. N Engl J Med 2004;350:2362-74.

3. Ramachandran A, Snehalata C, Yamuna A, Murugesan N, Narayan KM. Insulin resistance and clustering of cardiometabolic risk factor in urban teenagers in southern India. Diabetes Care 2007;30:1828-33.

4. Isomaa B, Almgren P, Tuomi T, Forsén B, Lahti K, Nissén M, *et al.* Cardiovascular morbidity and mortality associated with the Metabolic Syndrome. Diabetes Care 2001;24:683-9.

5. Dandona P, Aljada A, Bandyopadhyay A. Inflammation: The link between insulin resistance, obesity and diabetes. Trends Immunol 2004;25:4-7.

6. Iglseder B, Cip P, Malaimare L, Ladurner G, Paulweber B. The metabolic syndrome is a stronger risk factor for early carotid atherosclerosis in women than in men. Stroke 2005;36:1212-7.

7. Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med 1998;338:1650-6.

8. Singh R, Bhansali A, Sialy R, Aggarwal A. Prevalence of metabolic syndrome in adolescents from north Indian population. Diabet Med 2007;24:195-9.

9. Ramachandran A, Snehalatha C, Baskar AD, Mary S, Kumar CK, Selvam S, *et al.* Temporal changes in prevalence of diabetes and impaired glucose tolerance associated with life style transition occurring in rural population in India. Diabetologia 2004;47:860-5.

10. Luepker RV, Evans A, McKeigue P, Reddy KS. Cardiovascular survey methods. 3rd ed. Geneva: World Health Organization; 2004.

11. Update on the 1987 Task Force Report on High Blood Pressure in Children and Adolescents: A working group report from the National High Blood Pressure Education Program: National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. Pediatrics 1996;98:649-58.

12. WHO/IASO/IOTF. The Asia-Pacific perspective: Redefining obesity and its treatment. Health Communications Australia Pty Ltd; 2000.

13. Trinder P. Quantitative determination of glucose using GOP-PAP method. Clin Biochem 1969;6:24-7.

14. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. Clin Chem 1974;20:470-5.

15. Trinder P. Triglyceride estimation by GPO-PAP method. Ann Clin Biochem 1969;6:24-7.

16. Burstein M, Scholnic HR, Morfin K. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. J Lipid Res 1970;11:583-95.

17. Friedewald WT, Levy RI, Fredrickson DS. Estimation of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499-502.

18. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholesterol Education Program (ATP III) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001;285:2486-97.

19. National Institutes of Health. National Cholesterol Education Panel Report of the Expert Panel on blood Cholesterol Level in Children and Adolescents, Bethesda; Md. National Institute of Health; 1991. NIH publication No. 91-2732.

20. de Ferranti SD, Gauvreau K, Ludwig DS, Neufeld EJ, Newberger JW, Rifai N. Prevalence of the Metabolic Syndrome in American adolescents - Findings from the Third National Health and Nutrition Examination Survey. Circulation 2004;110:2494-7.

21. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescent: Findings from the Third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc Med 2003;157:821-7.

22. Esmaillzadeh A, Mirmiran P, Azadbekht L, Etemadi A, Azizi F. High prevalence of the metabolic syndrome in Iranian adolescents. Obesity (Silver Spring) 2006;14:377-82.

23. Nguyen TH, Hong KT, Kelly P, van der Ploeg HP, Dibley MJ. Association between physical activity and metabolic syndrome: A cross sectional survey in adolescents in Ho Chi Minh City, Vietna. BMC Public Health 2010;10:141.

24. Mayes PA, Botham KM. Lipid transport and storage. In: Harper's Illustrated Biochemistry; 26th ed. New York: McGraw-Hill Publishers; 2003.

25. Mathieu P, Poirier P, Pibarot P, Lemieux I, Després JP. Visceral Obesity: The link among inflammation, hypertension, and cardiovascular disease. Hypertension 2009;53:577-84.

26. Katsuki A, Sumida Y, Urakawa H, Gabazza EC, Murashima S, Maruyama N, *et al.* Increased visceral fat and serum levels of triglyceride are associated with insulin resistance in Japanese metabolically obese, normal weight subjects with normal glucose tolerance. Diabetes Care 2003;26:2341-4.

27. Miller M, Stone NJ, Ballantyne C, Bittner V, Criqui MH, Ginsberg HN, *et al*; American Heart Association Clinical Lipidology, Thrombosis, and Prevention Committee of the Council on Nutrition, Physical Activity, and Metabolism; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular Nursing; Council on the Kidney in Cardiovascular Disease Triglycerides and cardiovascular disease: A scientific statement from the American Heart Association. Circulation 2011;123:2292-333.

28. Haffner SM, Miettenen H, Gaskill SP, Stern MP. Metabolic predictors of hypertension: The San Antonio Heart Study. Arch Intern Med 1990;156:1994-2001.

29. Goel R, Misra A, Agarwal SK, Vikram N. Correlates of hypertension among urban Asian Indian adolescents. Arch Dis Child 2010;95:992-7.