Transaminases serum concentrations in obese children and adolescents.

Short title: Transaminases and obesity

[1]González-Gil, E.M.
[2]Bueno-Lozano, G.
[2]Bueno-Lozano, O.
[1,3]Moreno, L.A.
[2]Cuadrón-Andres, L
[2]Huerta-Blas, P
[2]Garagorri, J.M.
[2]Bueno, M.

[1]Growth, Exercise, Nutrition and Development (GENUD) research group, University of Zaragoza (Spain).
[2]University Clinical Hospital "Lozano Blesa", paediatric department.
[3]School of Health Science, University of Zaragoza (Spain).

Address for correspondence and reprint requests:

Dr. Luis A. Moreno
E.U. Ciencias de la Salud
Universidad de Zaragoza
C/ Domingo Miral s/n
50009 Zaragoza
Spain
Phone: + 34 976 761000
Fax: + 34 976 761752
E-mail: lmoreno@unizar.es

Abstract

The aim of the study was to assess transaminases concentrations and to investigate the relationship between liver enzymes and metabolic syndrome (MS) features in obese children and adolescents.

A total of 132 children and adolescents (73 males and 59 females) aged 8 – 16, participated in the study. All were studied at the department of Paediatrics, University Hospital of Zaragoza (Spain). Inclusion criteria were the existence of obesity as defined by body mass index (BMI) according to Cole cut-off values (when BMI was higher than the age and sex specific equivalent to 30 kg/m^2). For the definition of metabolic syndrome the International Diabetes Federation criteria was chosen. Weight (kg), height (cm), waist circumference (cm), blood pressure and BMI were measured. Laboratory determinations after overnight fasting included: transaminases (ALT, AST, GGT), fasting glucose, insulin, triglycerides and HDL-C.

The MS was found in 21.6% of the obese children and adolescents and the prevalence was higher in males (25.9%) than in females (15.9%). Transaminases (ALT, AST and GGT) mean serum concentrations were higher in males than in females, and decreased during pubertal development. The obese children and adolescents with the MS did not show higher transaminases concentrations when compared with those without the MS. Some MS manifestations (mainly waist circumference) showed a relationship with ALT, although all transaminases values were normal according to adult references**.** Transaminases, a surrogate marker of NAFLD, didn't showed an early and consistent manifestation of its abnormalities in the obese children and adolescents studied. In order to define the presence of the disease, it would be necessary to obtain aminotransferase reference standards for children and adolescents, considering pubertal stage and gender.

Las concentraciones séricas de transaminasas en niños obesos y adolescentes. ¿Son marcadores tempranos de la enfermedad de hígado graso no-alcohólico (NAFLD)?

El objetivo del estudio fue valorar las concentraciones de transaminasas en niños y adolescentes obesos e investigar la relación entre enzimas hepáticas y marcadores de síndrome metabólico (SM) en un grupo de niños y adolescentes obesos.

Un total de 132 niños y adolescentes (73 chicos y 59 chicas), en edades entre 8-16 años, participaron en el estudio. Todos fueron valorados en el departamento de Pediatría del Hospital Universitario de Zaragoza. El criterio de inclusión fue la existencia de obesidad definida mediante el índice de masa corporal (IMC) de acuerdo con los valores de Cole et. al (cuando el IMC era mayor que el equivalente a 30 kg/m^2 para una edad y sexo especifico). Para definir el síndrome metabólico, se eligieron los criterios de la Federación Internacional de Diabetes. Peso (Kg), altura (cm), perímetro de la cintura y tensión arterial fueron medidos. Las determinaciones de laboratorio tras ayuno fueron: transaminasas (ALT, AST, GGT), glucosa, insulina, triglicéridos y HDL-C.

Presentaron síndrome metabólico el 21,6% de los niños y adolescentes obesos y la prevalencia fue mayor en chicos (25,9%) que en chicas (15.9%). Los componentes más frecuentes del síndrome metabólico fueron: obesidad abdominal (exceso de circunferencia de cintura, 93%) y la tensión arterial elevada (34,3%). Los valores medios de las concentraciones séricas de transaminasas (ALT, AST, GGT) fueron mayores en chicos que en chicas, y disminuyeron según el desarrollo puberal.

Los niños y adolescentes obesos con síndrome metabólico no presentaron mayores concentraciones de transaminasas cuando se comparaban con los que no tenían síndrome metabolico. Algunas manifestaciones de SM (en particular el perímetro de la cintura) se asociaron con ALT, aunque los valores de transaminasas fueron normales según las referencias usadas para adultos. En los niños estudiados, las transaminasas, un marcador secundario de hígado graso no-alcohólico (NAFLD), no fueron una manifestación temprana y consistente de estas anormalidades. Para definir la presencia de la enfermedad, seria necesario obtener valores de referencia de transaminasas para niños y adolescentes, considerando el estadío puberal y el sexo.

Introduction

The prevalence of overweight and obesity in children and adolescents is increasing worldwide (12). In Europe, on the year 2003, the combined prevalence of paediatric overweight and obesity was around 20% in some countries and around 30% in countries such Spain or Italy (19). Southern European countries reported the highest levels of childhood overweight. In Spain, currently, there is a continuously rising trend (23).

The epidemic of obesity has been accompanied by the appearance of many health-related complications. Co morbidities of obesity are rising and this is because of earlier onset and longer obesity duration (26). Non-alcoholic fatty liver disease (NAFLD) is the most common cause of unexplained abnormal liver function tests in the paediatric population and it is mainly associated with obesity (17). According to the American Association for the Study of Liver Diseases, NAFLD is defined as fat accumulation in the liver exceeding 5 % to 10% as determined from the percentage of fat-laden hepatocytes by light microscopy (25). In addition, findings from Fishbein et al. suggest that visceral adiposity has a role in the pathogenesis of fatty liver in children (9).

Cardiovascular disease risk factors tend to cluster in obese children (1) and this association is often recognized as the metabolic syndrome. Currently, the most widely accepted definition of the metabolic syndrome (MS) is the International Diabetes Federation (IDF) one. It includes increased waist circumference and at least 2 of the following: Increased fasting serum glucose, triglycerides, high blood pressure or increased HDL-C (37). It was observed that obese adolescents with NAFLD had a higher prevalence of the metabolic syndrome (4) and liver fat was linked with all the components of the MS independent of obesity**.** The increased fat accumulation in the liver was considered a marker of hepatic insulin resistance which is related with the MS. However, the mechanisms that support the excess fat deposition in the liver are still poorly understood (14).

The aim of this study was to investigate the links between liver enzymes (ALT, AGT, GGT) and the metabolic syndrome features, considering also the effect of gender and pubertal stage. In addition, the prevalence of the metabolic syndrome and the frequency of individual components of the MS among obese paediatric patients were also assessed.

Patients and methods

Subjects

The study included a total of 132 children and adolescents (73 males and 59 females) aged 8 – 16 y. Inclusion criteria were the existence of obesity according to Cole et al. cut off values (7). Pubertal stage was determined in each patient using Tanner's criteria. (20) With the exception of obesity, the children had no hepatic disease or any other apparent disease.

Anthropometric measurements

Height and weight were obtained for all individuals. Anthropometric measurements were taken by the same person. In order to define abdominal obesity, waist circumference's over percentile 90 was chosen according to Moreno et al. (22) Waist circumference was measured with an inelastic tape, the subject being in a standing position; the tape was applied horizontally midway between the lowest rib margin and the iliac crest (22). BMI was calculated as weight in kilograms by the height in square meters. Obesity was defined by body mass index (BMI) according to Cole et al. cut-off values when BMI was higher than the age and sex specific equivalent to 30 kg/m^2 (6).

Laboratory determinations

After overnight fasting, blood was obtained by vein puncture between 08.00 and 09.30. All the assays were performed at the Department of Biochemistry, University Hospital of Zaragoza. Lipid profile [(triglycerides (TG), total cholesterol (C), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), glucose, and transaminases (ALT, AST, GGT) were determined in the fasting blood samples. Insulin was determined by inmunometric assay with an Immulite analyser. The HOMA (Homeostatic Model Assessment) index was calculated as the product of the fasting plasma insulin level (mU/mL) and the fasting plasma glucose level (mmol/L), divided by 22.5 (21).

Blood pressure

Blood pressure was measured three times by the same examiner, using a mercury sphygmomanometer and we considered the mean of the three measurements. The first, fourth and fifth Korotkoff phases were recorded each time.

Metabolic syndrome criteria

There are a lot of criteria for defying the metabolic syndrome. Recently the IDF published a worldwide definition of the condition in children and adolescents. The new IDF definition considers the following age groups: 6 to <10, 10 to <16 and 16 yr. In children 10 to16 IDF defines paediatric MS when waist circumference is ≥90^{th} percentile plus any two of the following abnormalities: Hypertriglyceridemia ≥ 1.7mmol/L (150 mg/dL), HDL-C <1,03 mmol/L, blood pressure ≥ 130/85 mm/Hg and fasting glucose ≥ 5.6 mmol/L (100mg/dL) (37).

Statistical analysis

All statistical analyses were performed with the SPSS for Windows (Language System Corp, Chicago). Descriptive values are given as means, standard deviations and percentages. Comparison between means in both sexes were carried out by using Student t tests and Mann – Whitney U tests, depending on whether the variables were normally distributed or not, respectively. Also we used the correlation coefficients of Spearman and Pearson, depending on the distribution of every variable.

Results

115 out of 132 obese children were aged 10 to 16 years. The prevalence of the MS in this obese population was 21.6 %, **(Figure 1)**. There were differences in prevalence by gender (males 25.9 %, females 15.9%). In both sexes the most frequent features of the MS were abdominal obesity (excess waist circumference, 93%), and blood pressure (34.3%); and the conditions with the lowest frequency in our patients were dyslipidemia (9.9%) and fasting hyperglicemia (17.5%). **(Figure 2).**

In relation with the serum concentrations of liver enzymes, they were within normal ranges in these patients, according to the current adult reference standards. Mean values of transaminases serum concentrations were higher in males than in females and ALT and AST were related with gender ($p<0.05$) **(Figure 3).** Furthermore, they decreased according to pubertal development ($p<0.05$); specially, AST was strongly related with pubertal development ($p<0.01$)**(Figure 4).** There were not association between transaminases serum concentrations and BMI.

According with the presence of different components of the MS and transaminases only ALT was related with MS ($p=0.031$). The obese children and adolescents with MS did not show higher transaminases concentrations when compared with those without the MS.

Correlations of MS components with transaminases serum concentrations are shown in table I. In males (Table I) there was only a positive significant correlation between ALT and waist circumference and a negative correlation with glucose, insulin and HOMA. In females (Table I), all the three transaminases were significantly correlated with total and LDL-cholesterol. ALT also correlated with systolic and diastolic blood pressure, and GGT correlated with fasting insulin and the HOMA index.

Discussion

In order to asses transaminases concentrations and to investigate the relationship between liver enzymes and MS, a clinical sample of obese children and adolescents was studied. Many of the metabolic and cardiovascular complications of obesity are already present during childhood and are closely related to the presence of insulin resistance/ hyperinsulinemia, the most common abnormality seen in obesity (3).

Childhood obesity is the most powerful predictor for developing the metabolic syndrome. Furthermore, many studies have shown that the metabolic syndrome prevalence in children and adolescents has increased and the prevalence was higher when the subjects were obese (3,10). There is no consensus on the diagnostic criteria; therefore, the results are very different. The prevalence of MS is reported to be 30 – 50 % among obese children and adolescents (7,35). The prevalence seems to be linked with the severity of obesity and reached 50 per cent on severely obese youngsters (35). The different MS prevalence observed in the paediatric literature depends on the criteria for the definition. The most recently recommended criteria of MS for children was proposed by the International Diabetes Federation (IDF) Task force on Epidemiology and Prevention on Diabetes (37).

There are only a few studies using the IDF criteria, and those diagnosed by using its definition have lower rates of MS than when using the NCEP (National Cholesterol Education Program) one. The most frequently mentioned components of childhood metabolic syndrome are: visceral obesity, hypertension, hyperinsulinemia / insulin resistance / type 2 diabetes mellitus and dyslipemia (hypertrigliceridemia, low HDL cholesterol). The most frequent component of MS in our paediatric patients was waist circumference (93 %) and the second blood pressure (34.3%); the less frequent were triglycerides (9,9 %) and glucose (17.5 %). Waist circumference is one of the most frequent clinical features (2) and that's why seems to be the best predictor of children with the metabolic syndrome in the paediatric population (24). Fatty liver is the most common liver abnormality in children age 2 to 19 (30). In addition, obesity is by far the strongest risk factor for developing Non – alcoholic fatty liver disease (NAFLD) (11). Liver fat content is increased in subjects with MS (16). Therefore we can consider visceral adiposity as a risk factor for paediatric NAFLD (9).

In our study, like in others, it has been observed that the best correlate of MS components with fatty liver was waist circumference (14). Risk factors, like waist

circumference, should be taken into consideration for the screening of NAFLD in paediatric population (31). Increased lipid content in the liver may also independently be linked with hipoadiponectemia, and increased visceral adiposity with peripheral and hepatic insulin resistance (16).

The best way to diagnose NAFLD is liver biopsy but is invasive and expensive and not feasible in our population for ethic reasons (36). The development of non invasive surrogate markers for the screening of NAFLD are needed on large populations at risk, and specially in obese children and adolescents (26).

In the studied population, there were different transaminases concentrations (ALT and AST) according to gender. As in previous studies, transaminases serum concentrations were higher in males than in females. (4, 33). Furthermore, transaminases were linked to pubertal stage, specially AST.

Although there is a strong correlation between the degree of liver fatty infiltration and the liver enzymes increase, the exact pathogenesis of raised ALT in NAFLD remains unclear (28). ALT increases are more correlated with the metabolic syndrome than the other liver enzymes (29, 13). In males, we observed correlation between ALT and waist circumference and a negative correlation with glucose, insulin and HOMA and in female sample all the three transaminases were significantly correlated with total and LDL-cholesterol. ALT also correlated with systolic and diastolic blood pressure, and GGT correlated with fasting insulin and the HOMA index. Deterioration in glucose and lipid metabolism seems to be associated with modest ALT elevations (4). Furthermore, in some studies aminotransferase elevations are strongly associated with features of the metabolic syndrome (5). In our study we observed some relations between MS manifestations and ALT but no differences between those with and without MS. Intervention studies have shown that liver fat can be decreased by weight loss, and insulin therapy (15). In general, modifications through diet and exercise should be performed (26).

We studied the serum concentrations of the liver enzymes in our patients and all the results were within normal ranges, using reference standards for adults. It would be necessary to obtain aminotransferase reference standards for children and adolescents because there are reports of obese children with radiographic evidence of fatty liver who have normal aminotransferase serum concentrations (8, 32). Reference ranges of serum aminotransferase levels has been called into question because reference ranges were determined by population samples perhaps including undiagnosed liver diseases like

NAFLD (27). These findings suggest that a critical revision of transaminases limits would require the definition of “healthy ranges” rather than a generic update of “normal ranges”, specially in the paediatric population.

Transaminases are surrogate markers of NAFLD instead of invasive techniches, but they should always be complemented with another diagnostic techniques. It would be necessary to obtain aminotransferase reference standards for children and adolescents, and for assessing their values we should take into account pubertal stage and gender.

According with our results it seems transaminases serum concentrations do not show strong association with the MS and its individual manifestations in obese children and adolescents. If we identify an obese children or adolescent with abnormal transaminases concentratrions, he/she could have already an advanced liver disease. Studies with bigger population samples and combining biochemical results with other diagnostic techniques are needed to corroborate these results.

References

1. Brambilla, P., Lissau, I., Flodmark, C. E., Moreno, L. A., Widhalm, K., Wabitsch, M., et al. (2007): Metabolic risk-factor clustering estimation in children: to draw a line across pediatric metabolic syndrome. Int J Obes (Lond*),* 31(4), 591-600.

2. Bueno, G., Moreno, L.A., Bueno, O., Morales, J., Perez-Roche, T., Garagorri, J.M. (2007): Metabolic risk-factor clustering estimation in obese children. J Physiol Biochem, 63(4):347-55.

3. Bueno, G., Bueno, O., Moreno, L.A., Garcia, R., Tresaco, B., Garagorri, J.M. (2006): Diversity of metabolic syndrome risk factors in obese children and adolescents. J Physiol Biochem, 62(2):125-33.

4. Burgert, T.S., Taksali, S.E., Dziura, J., Goodman, T.R., Yeckel, C.W., Papademetris, X. (2006): Alanine aminotransferase levels and fatty liver in childhood obesity: associations with insulin resistance, adiponectin, and visceral fat. J Clin Endocrinol Metab, 91(11):4287-94.

5. Clark, J.M., Brancati, F.L., Diehl, A.M. (2003): The prevalence and etiology of elevated aminotransferase levels in the United States. Am J Gastroenterol, 98(5): 960-7.

6. Cole, T.J., Bellizzi, M.C., Flegal, K.M., Dietz, W.H. (2000): Establishing a standard definition for child overweight and obesity worldwide: international survey. Bmj, 320(7244):1240-3.

7. Cook, S., Weitzman, M., Auinger, P., Nguyen, M., Dietz, W.H. (2003): Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc Med, 157(8):821-7.

8. Fishbein, M.H., Miner, M., Mogren, C., Chalekson, J. (2003): The spectrum of fatty liver in obese children and the relationship of serum aminotransferases to severity of steatosis. J Pediatr Gastroenterol Nutr, 36(1):54-61.

9. Fishbein, M.H., Mogren, C., Gleason, T., Stevens, W.R. (2006): Relationship of hepatic steatosis to adipose tissue distribution in pediatric nonalcoholic fatty liver disease. J Pediatr Gastroenterol Nutr, 42(1):83-8.

10. Ferranti, S.D., Gauvreau, K., Ludwig, D.S., Neufeld, E.J., Newburger, J.W., Rifai, N. (2004): Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutrition Examination Survey. Circulation, 110(16):2494-7.

11. Haynes, P., Liangpunsakul, S., Chalasani, N. (2004): Nonalcoholic fatty liver disease in individuals with severe obesity. Clin Liver Dis, 8(3):535-47, viii.

12. Hedley, A.A., Ogden, C.L., Johnson, C.L., Carroll, M.D., Curtin, L.R., Flegal, K.M. (2004): Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. Jama, 291(23):2847-50.

13. Ioannou, G.N., Weiss, N.S., Boyko, E.J., Kahn, S.E., Lee, S.P. (2005): Contribution of metabolic factors to alanine aminotransferase activity in persons with other causes of liver disease. Gastroenterology, 128(3):627-35.

14. Kotronen, A., Westerbacka, J., Bergholm, R., Pietilainen, K.H., Yki-Jarvinen, H. (2007): Liver fat in the metabolic syndrome. J Clin Endocrinol Metab, 92(9):3490-7.

15. Kotronen, A., Yki-Jarvinen, H. (2008): Fatty liver, a novel component of the metabolic syndrome. Arterioscler Thromb Vasc Biol, 28(1):27-38.

16. Koska, J., Stefan, N., Permana, P.A., Weyer, C., Sonoda, M., Bogardus, C. (2008): Increased fat accumulation in liver may link insulin resistance with subcutaneous abdominal adipocyte enlargement, visceral adiposity, and hypoadiponectinemia in obese individuals. Am J Clin Nutr, 87(2):295-302.

17. Lavine, J.E., Schwimmer, J.B.(2004): Nonalcoholic fatty liver disease in the pediatric population. Clin Liver Dis, 8(3):549-58, viii-ix.

18. Lissau, I. (2004): Overweight and obesity epidemic among children. Answer from European countries. Int J Obes Relat Metab Disord, 28 Suppl 3:S10-5.

19. Lobstein, T., Frelut, M.L. (2003): Prevalence of overweight among children in Europe. Obes Rev, 4(4):195-200.

20. Tanner, J.M., Whitehouse, R.H. (1976): Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. Arch Dis Child, 51(3):170-179.

21. Matthews, D.R., Hosker, J.P., Rudenski, A.S., Naylor, B.A., Treacher, D.F., Turner, R.C. (1985): Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia, 28(7):412-9.

22. Moreno, L.A., Fleta, J., Mur, L., Rodriquez, G., Sarria, A., Bueno, M. (1999): Waist circumference values in Spanish children--gender related differences. Eur J Clin Nutr, 53(6):429-33.

23. Moreno, L.A., Mesana, M.I., Fleta, J., Ruiz, J.R., Gonzalez-Gross, M., Sarria, A.(2005): Overweight, obesity and body fat composition in spanish adolescents. The AVENA Study. Ann Nutr Metab, 49(2):71-6.

24. Moreno, L.A., Pineda, I., Rodriguez, G., Fleta, J., Sarria, A., Bueno, M. (2002): Waist circumference for the screening of the metabolic syndrome in children. Acta Paediatr, 91(12):1307-12.

25. Neuschwander-Tetri, B.A., Caldwell, S.H. (2003): Nonalcoholic steatohepatitis: summary of an AASLD Single Topic Conference. Hepatology, 37(5):1202-19.

26. Patton, H.M., Sirlin, C., Behling, C., Middleton, M., Schwimmer, J.B., Lavine, J.E. (2006): Pediatric nonalcoholic fatty liver disease: a critical appraisal of current data and implications for future research. J Pediatr Gastroenterol Nutr, 43(4):413-27.

27. Prati, D., Taioli, E., Zanella, A., Della Torre, E., Butelli, S., Del Vecchio, E. (2002): Updated definitions of healthy ranges for serum alanine aminotransferase levels. Ann Intern Med, 137(1):1-10.

28. Radetti, G., Kleon, W., Stuefer, J., Pittschieler, K. (2006): Non-alcoholic fatty liver disease in obese children evaluated by magnetic resonance imaging. Acta Paediatr, 95(7):833-7.

29. Sattar, N., Scherbakova, O., Ford, I., O'Reilly, D.S., Stanley, A., Forrest, E. (2004): Elevated alanine aminotransferase predicts new-onset type 2 diabetes independently of classical risk factors, metabolic syndrome, and C-reactive protein in the west of Scotland coronary prevention study. Diabetes, 53(11):2855-60.

30. Schwimmer, J.B., Deutsch, R., Kahen, T., Lavine, J.E., Stanley, C., Behling, C. (2006): Prevalence of fatty liver in children and adolescents. Pediatrics, 118(4):1388-93.

31. Schwimmer, J.B., Pardee, P.E., Lavine, J.E., Blumkin, A.K., Cook, S. (2008): Cardiovascular risk factors and the metabolic syndrome in pediatric nonalcoholic fatty liver disease. Circulation, 118(3):277-83.

32. Tazawa, Y., Noguchi, H., Nishinomiya, F., Takada, G. (1997): Serum alanine aminotransferase activity in obese children. Acta Paediatr, 86(3):238-41.

33. Wallace, T.M., Utzschneider, K.M., Tong, J., Carr, D.B., Zraika, S., Bankson, D.D. (2007): Relationship of liver enzymes to insulin sensitivity and intra-abdominal fat. Diabetes Care, 30(10):2673-8.

34. Wan, N.J., Mi, J., Wang, T.Y., Duan, J.L., Li, M., Gong, C.X. (2007): Metabolic syndrome in overweight and obese schoolchildren in Beijing. Zhonghua Er Ke Za Zhi; 45(6):417-21.

35. Weiss, R., Dziura, J., Burgert, T.S., Tamborlane, W.V., Taksali, S.E., Yeckel, C.W. (2004): Obesity and the metabolic syndrome in children and adolescents. N Engl J Med, 350(23):2362-74.

36. Wieckowska, A., Feldstein, A.E. (2008): Diagnosis of nonalcoholic fatty liver disease: invasive versus noninvasive. Semin Liver Dis, 28(4):386-95.

37. Zimmet, P., Alberti, G., Kaufman, F., Tajima,N., Silink, M., Arslanian, S. (2007): The metabolic syndrome in children and adolescents. Lancet, 369(9579):2059-61.

Table I.
Correlations between Metabolic Syndrome components and transaminases in males and females.
HDL-Cholesterol: High-density lipoprotein cholesterol.
LDL-Cholesterol: Low-density lipoprotein cholesterol.
HOMA: Homeostatic Model Assessment
SBP: Systolic blood pressure.
DBP: Diastolic blood pressure.
(**) $p < 0,001$ (*) $p < 0,05$

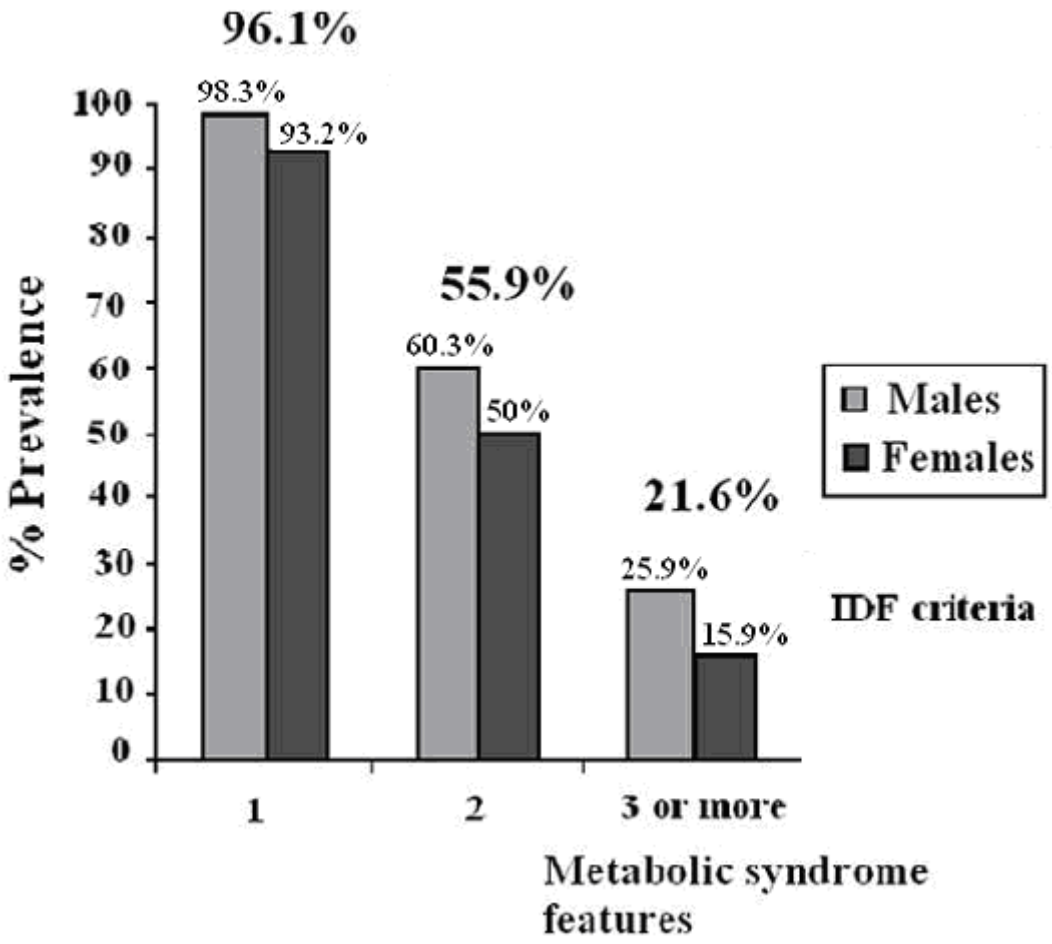
	Males			Females		
	AST	**ALT**	**GGT**	**AST**	**ALT**	**GGT**
Waist C. (cm)	- 0,047	0,295*	0,013	- 0,137	0,046	- 0,112
Triglyc.(mg/dL)	- 0,154	0,079	0,114	0,122	0,247	0,299*
Chol. (mg/dL)	0,057	0,023	0,162	0,390*	0,390*	0,447**
HDL-Chol.(mg/dL)	0,107	- 0,117	- 0,039	0,087	- 0,046	- 0,023
LDL-Chol.(mg/dL)	0,067	0,036	0,154	0,305*	0,429*	0,526**
Glycemia(mg/dL)	- 0,259*	- 0,054	- 0,099	- 0,169	0,196	0,104
Insulinemia(mg/dL)	- 0,246*	0,095	0,011	- 0,248	0,173	0,275*
HOMA	- 0,285*	0,069	- 0,047	- 0,232	0,187	0,269*
SBP (mmHg)	- 0,148	- 0,031	- 0,062	0,137	0,349*	- 0,020
DBP(mmHg)	- 0,270*	- 0,205	- 0,135	0,080	0,348*	0,102

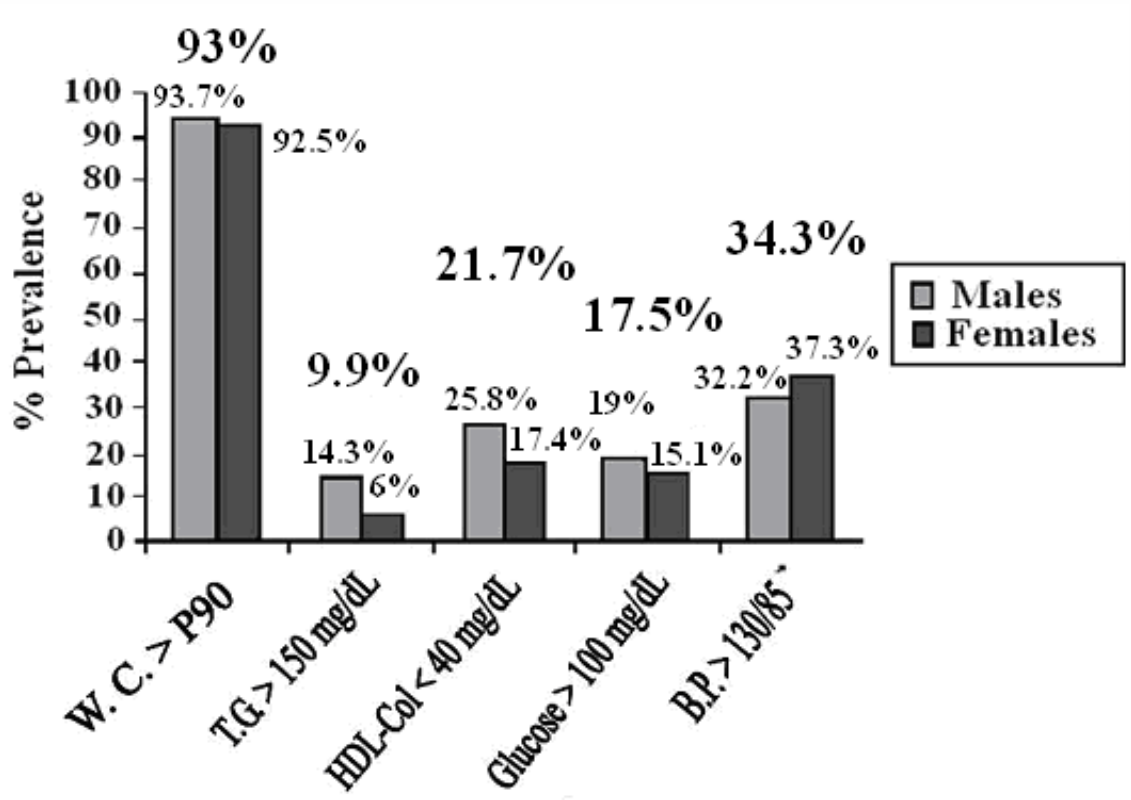
Figure 1. Prevalence of metabolic syndrome (MS) according to the number of components. International Diabetes Federation (IDF) criteria.

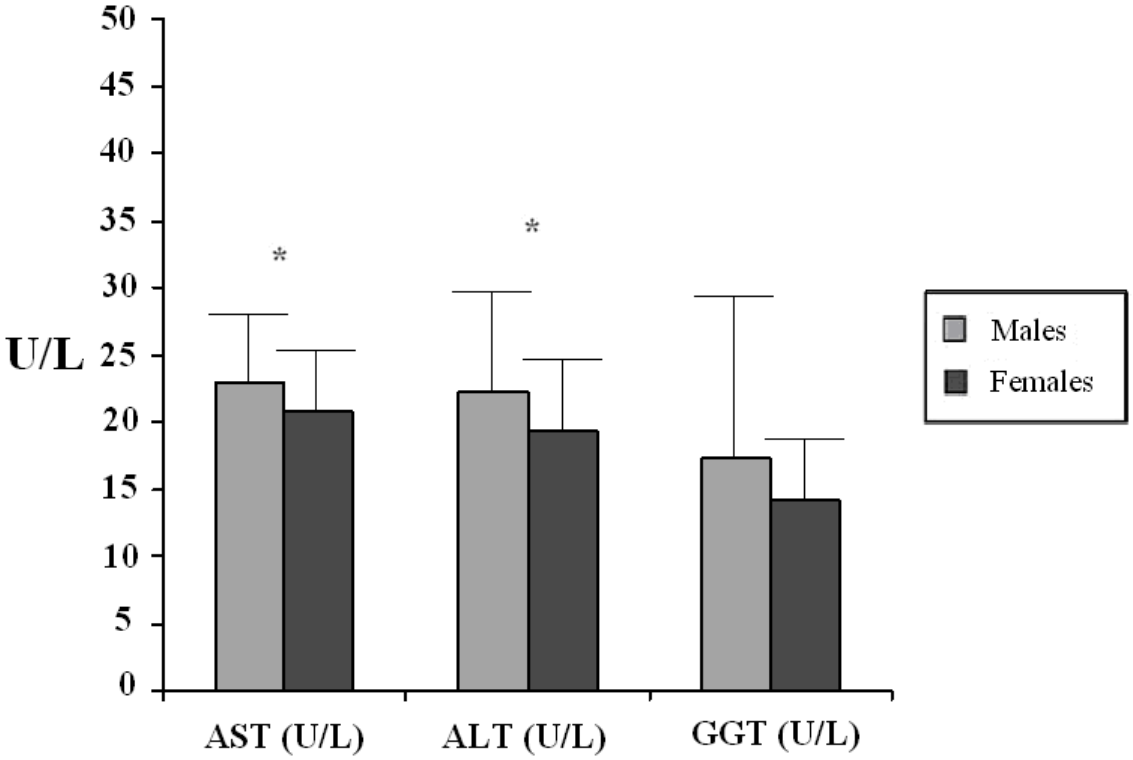
Figure 2. Prevalence of each component of metabolic syndrome.
W.C.: Waist circumference.
T.G.: Triglycerides.
B.P.: Blood pressure.

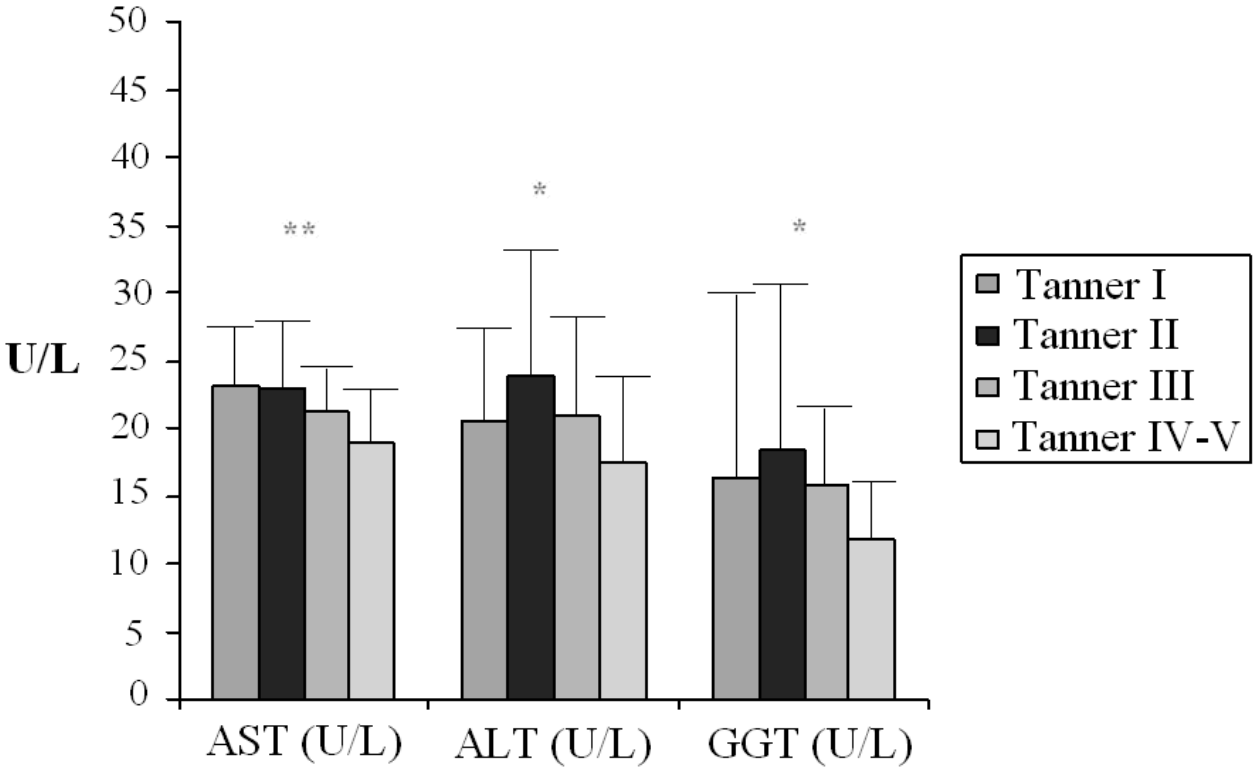
Figure 3. Serum concentration of transaminases according to gender (mean±SD).
Point lines are the upper limit concentration levels of transaminases.
** $p<0.05$*

Figure 4. Serum concentration level of transaminases according to pubertal stages.
**$p<0.05$*
***$p<0.01$*


96.1%
98.3%
93.2%
55.9%
60.3%
50%
21.6%
25.9%
15.9%
100
90
80
70
60
50
40
30
20
10
0
% Prevalence
1
2
3 or more
Metabolic syndrome features
Males
Females
IDF criteria


93%
21.7%
17.5%
34.3%
% Prevalence
100
90
80
70
60
50
40
30
20
10
0
93.7%
92.5%
9.9%
14.3%
6%
25.8%
17.4%
19%
15.1%
32.2%
37.3%
Males
Females
W. C. > P90
T.G > 150 mg/dL
HDL-Col < 40 mg/dL
Glucose > 100 mg/dL
B.P. > 130/85*


U/L
50
45
40
35
30
25
20
15
10
5
0
*
*
Males
Females
AST (U/L)
ALT (U/L)
GGT (U/L)


50
45
40
35
30
25
20
15
10
5
0
U/L
**
*
*
AST (U/L)
ALT (U/L)
GGT (U/L)
Tanner I
Tanner II
Tanner III
Tanner IV-V