Diabetes & Metabolic Syndrome: Clinical Research & Reviews 10S (2016) S86–S89

Contents lists available at ScienceDirect

Diabetes & Metabolic Syndrome: Clinical Research & Reviews

journal homepage: www.elsevier.com/locate/dsx

Original article

The association between Metabolic Syndrome and serum levels of lipid peroxidation and interleukin-6 in Gorgan

Hamide Mojaz Sarbijani [a], Masoud Khoshnia [b], Abdoljalal Marjani [c,*]

[a] Department of Biochemistry and Biophysics, Metabolic Disorders Research Center, Gorgan Faculty of Medicine, Golestan University of Medical Sciences, Gorgan, Golestan province, Iran
[b] Golestan Research Center of Gastroenterology and Hepatology, Gorgan Faculty of Medicine, Golestan University of Medical Sciences, Gorgan, Golestan province, Iran
[c] Metabolic Disorders Research Center, Department of Biochemistry and Biophysics, Gorgan Faculty of Medicine, Golestan University of Medical Sciences, Gorgan, Golestan province, Iran

ARTICLE INFO

Keywords:
Metabolic syndrome
Lipid peroxidation
Interleukin-6
Gorgan

ABSTRACT

Background: There are limited studies on the relationship between inflammatory marker such as IL-6 and lipid peroxidation and metabolic syndrome.
Objective: The aim of present study was to assess IL-6 and lipid peroxidation in subjects with and without the metabolic syndrome and their association with metabolic syndrome components.
Methods: Age and gender matched 40 subjects with metabolic syndrome and 40 control groups took part in this study.
Results: The mean malondialdehyde level was significantly higher in overweight and obese subjects with metabolic syndrome than control groups ($P < 0.05$). The mean level of IL-6 in men and the mean level of malondialdehyde in women with metabolic syndrome was significantly higher than control groups ($p < 0.05$). There were significant positive correlation between malondialdehyde and fasting blood glucose, triglyceride and systolic blood pressure ($p < 0.05$).
Conclusions: Our results suggest that higher levels of IL-6 and malondialdehyde may cause insulin resistance and metabolic disorders in all subjects with metabolic syndrome. Malondialdehyde level shows strong association with some metabolic syndrome components. This means the greater risk of metabolic syndrome.

1. Introduction

Metabolic syndrome (MS) is known as an important risk factor for cardiovascular disease and type II diabetes [1]. It is reported recently that the prevalence of the metabolic syndrome in U.S. adults above20 years old was almost 23.7% [2,3]. Marjani et al. showed that metabolic syndrome and lipid peroxidation alter in different ethnic and age groups, postmenopausal women and different diseases [4–15]. Oxidative stress occurs when there is instability between tissue, free radicals, and reactive oxygen species and antioxidants system. This instability causes oxidative damage [16]. Oxidative stress shows an important role in the pathogenesis of different diseases [17]. Studies have indicated that oxidative stress damage glucose uptake by muscle and fat cells [18,19]. It also reduces insulin secretion from pancreatic β cells [20]. Oxidative stress may be elevated by components of metabolic syndrome such as insulin resistance, type II diabetes, hypertension, dyslipidemia, visceral obesity [21–23], impaired vascular function, inflammation, thrombosis, and atherosclerosis and vascular disease [24]. These diseases may associate with metabolic syndrome. Studies have revealed that elevated oxidative stress and inflammatory stress play a significant role in beginning and development of atherosclerotic vascular disease [25,26]. It has reported that low density lipoprotein oxidation as a marker of oxidative stress increased in patients with coronary heart disease [25] and subclinical atherosclerosis [27]. It has shown that the metabolic syndrome reveals an association with elevated oxidative stress and inflammatory burden [28,29]. The risk of coronary heart disease was more seen in obese subjects with metabolic syndrome when compared to subjects without it [30]. IL-6 is a proinflammatory cytokine. Macrophages and smooth muscle cells secret it in

* Corresponding author at: Metabolic Disorders Research Center, Department of Biochemistry and Biophysics, Gorgan Faculty of Medicine, Golestan University of Medical Sciences, Gorgan, Golestan province, Iran. Tel.: +98(171)4421651; fax: +98(171)4440225.
E-mail address: abdoljalal@yahoo.com (A. Marjani).

http://dx.doi.org/10.1016/j.dsx.2015.09.024
1871-4021/

atherosclerotic lesions [31,32]. Plasma IL-6 is released from white blood cells (T cells and macrophages) and adipocytes [33–36]. It has associated with obesity and some components of the metabolic syndrome such as insulin resistance and dyslipidemia [37–39]. Studies have indicated that there is an association between elevated risk of coronary heart disease [40–43] and type 2 diabetes mellitus with IL-6. It has shown that IL-6 has principal role in the relationship among inflammation, obesity and cardiovascular disease [44]. Some other findings have shown that increased interleukin (IL)-6 used as a marker of systemic inflammation and diagnostic marker of future atherosclerotic events [45,46]. Clinical studies showed that the level of interleukin-6 (IL-6) is used as inflammatory marker for diagnosis of early stages of coronary artery disease [47]. It is recently reported that low-grade inflammation shows an important effect on the pathobiology of metabolic syndrome [47,48]. There are limited studies on the relationship between inflammatory marker such as IL-6 and lipid peroxidation and metabolic syndrome. The aim of present study was to assess IL-6 and lipid peroxidation in subjects with and without the metabolic syndrome and their association with metabolic syndrome components.

2. Methods

Age and gender matched 40 subjects with metabolic syndrome and 40 control groups took part in this study. Blood samples were collected after overnight fasting from all subjects who were referred to the Jelleyin health center in Golestan University of Medical Sciences, 2014. Commercial kit (With the use of photometer techniques, Model CLINIC II-Photometer) were used for determination of serum fasting glucose, triglycerides, LDL-cholesterol and HDL-cholesterol levels in the Metabolic Disorders Research Center, Gorgan Faculty of Medicine. Weight measurement of all subjects was done, while subjects were minimally clothed without shoes, using digital scales. Height measurement of all subjects was carried out in standing position using tape meter while the shoulder was in a normal position. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Subjects with BMI = 25.0–29.9 kg/m^2 and BMI $\geq$30 kg/m^2 were indicated as overweight and obese subjects, respectively [38]. Waist circumferences were determined at the point halfway between the lower border of ribs and the iliac crest in a horizontal plane [49]. Systolic and diastolic blood pressure was measured in sitting position from the right hand. Subjects with 3 or more of the below mentioned criteria were considered as a metabolic syndrome subjects according to Adult Treatment Panel III definition [50]:

A. Waist circumference >102 cm in men and >88 cm in women.
B. Serum triglycerides level $\geq$150 mg/dl.
C. Low HDL-cholesterol: <40 mg/dl in men and <50 mg/dl in women.
D. Systolic blood pressure (SBP) $\geq$130 mmHg and/or diastolic blood pressure (DBP) $\geq$85 mmHg or on treatment for hypertension.
E. Serum glucose level $\geq$110 mg/dl or on treatment for diabetes.

Serum IL-6 (Picogram/ml) and malondialdehyde (Nano mol/l) were determined by Immunoassay (LOT: IL6041453, REF: EIL06001, Germany) and Kei Satoh (using spectrophotometer technique, JENWAY6305) [51] methods, respectively ((the level of lipid peroxidation expressed as Malondialdehyde (MDA)). Collected data was analyzed using SPSS–16 version software. Statistical analysis of data expressed as percentage and means and standard deviations. Chi squared, independent sample t and Pearson's correlation tests were used to evaluate data. Statistical differences were considered significant if $p < 0.05$.

Table 1
Characteristic of subjects with metabolic syndrome and control groups.

Parameter	Subject with metabolic syndrome	Control groups	*P*-value
Age (years)	**48.50 ± 7.80**	**46.20 ± 9.70**	**0.227**
BMI (kg/m^2)	**29.95 ± 3.82**	**26.35 ± 4.14**	**0.0001**
Overweight			
IL-6 (pg/ml)	**3.74 ± 0.24**	**3.50 ± 0.01**	**0.342**
Malondialdehyde (nmol/l)	**3.40 ± 0.20**	**1.90 ± 0.19**	**0.02**
Obese			
IL-6 (pg/ml)	**6.85 ± 2.07**	**4.40 ± 0.93**	**0.063**
Malondialdehyde (nmol/l)	**3.58 ± 050**	**1.89 ± 0.15**	**0.01**

The *p*-value of tables is in bold.

3. Results

The clinical and biochemical data of the subjects with metabolic syndrome and control group were shown in Table 1. The mean malondialdehyde level was significantly higher in overweight and obese subjects with metabolic syndrome than control groups ($P < 0.05$). Table 2 shows characteristic of the men and women with metabolic syndrome and control groups. The mean level of IL-6 in men and the mean level of malondialdehyde in women with metabolic syndrome was significantly higher than control groups ($p < 0.05$). Correlation between metabolic syndrome components and IL-6 and malondialdehyde are shown in Table 3. There were significant positive correlation between malondialdehyde and fasting blood glucose, triglyceride and systolic blood pressure ($p < 0.05$). There were no significant correlation between IL-6 and metabolic syndrome components. There were no significant correlation between IL-6 and malondialdehyde ($P = 0.917$, not shown in result section).

4. Discussion

In present study, we observed that the levels of IL-6 were significantly higher in subjects with the metabolic syndrome (Table 1). Studies have shown that there is an association between the participation of IL-6 and systemic inflammatory responses that causes to metabolic syndrome [52,53]. Higher levels of IL-6 in men than in women show that abdominal obesity, which is more prevalent in men, causes more proatherogenic cytokines production. This may show that men are more in higher risk for

Table 2
Characteristic of subjects with metabolic syndrome and control groups in men and women.

Parameter	Subject with metabolic syndrome	Control groups	*P*-value
Men			
Number of subjects (%)	18 (100%)	18 (100%)	
Age (years)	49.11 ± 8.74	45.72 ± 11.40	0.324
BMI (kg/m^2)	30.88 ± 3.53	25.98 ± 2.34	**0.0001**
IL-6 (pg/ml)	7.14 ± 0.24	3.50 ± 0.01	**0.0001**
Malondialdehyde (nmol/l)	3.36 ± 0.23	2.01 ± 0.11	0.262
Women			
Number of subjects (%)	22 (55%)	22 (55%)	
Age (years)	48 ± 7.11	46.59 ± 6.86	0.507
BMI (kg/m^2)	29.18 ± 3.96	26.65 ± 5.01	0.071
IL-6 (pg/ml)	3.77 ± 0.21	3.92 ± 0.42	0.351
Malondialdehyde (nmol/l)	3.65 ± 0.45	1.75 ± 0.14	**0.001**

The *p*-value of tables is in bold.

Table 3
Correlation between metabolic syndrome components and IL-6 and Malondialdehyde.

Parameters	Malondialdehyde (nmol/l)		IL-6 (pg/l)	
	R	*P*-value	*r*	*P*-value
Glucose (mg/dl)	0.474	**0.0001**	−0.007	0.948
Triglyceride (mg/dl)	0.224	**0.003**	0.065	0.565
HDL-C (mg/dl)	0.064	0.573	−0.131	0.247
Waist circumference (cm)	0.388	**0.0001**	0.193	0.256
Systolic blood pressure	0.232	**0.038**	0.126	0.256
Diastolic blood pressure	0.151	0.183	0.054	0.637

The *p*-value of tables is in bold.

cardiovascular diseases than women [39,54]. Men had higher IL-6 levels than women which is not in agreement with some other studies [52,55]. IL-6, as a pro-inflammatory cytokine was higher in obese subjects than overweight subjects with metabolic syndrome. This may shows a relationship between obesity and IL-6 levels [56]. Some studies have indicated that serum IL-6 were not associated with metabolic syndrome [57,58]. It revealed that IL-6 has an important role in development of insulin resistance [44,59] while some other studies reported IL-6 prevents insulin resistance [60]. Our results showed that there were no significant correlation between IL-6 and metabolic syndrome components. Thus, in contrast to some other studies, it may suggest that IL6 could not be used as a biomarker for the diagnosis of metabolic syndrome [57]. Oxidative stress may play a significant role in the development of metabolic syndrome [23]. In the present study malondialdehyde level was statistically significant in subjects with metabolic syndrome which was not in agreement with some studies [61,62] while it was in agreement with some other findings [16]. The results of this study show that the metabolic syndrome intensifies oxidative stress in subjects with metabolic syndrome. The present study also shows that oxidative stress was significantly higher in overweight and obese subjects with metabolic syndrome in comparison with subjects without metabolic syndrome which is in agreement with the findings of other studies that they have shown independent association of obesity and metabolic syndrome with elevated oxidative stress and inflammatory burden [28,29,45,46]. In the present study, we also determined the correlations between oxidative stress and metabolic syndrome components. Our findings showed that the oxidative stress was significantly positive correlated with triglyceride, glucose, systolic blood pressure and waist circumference in our study subjects (Table 3). It can be suggest that subjects with metabolic syndrome may have a higher level of oxidative stress. In general, abdominal obesity has seen in subjects with metabolic syndrome. Obesity makes happen oxidative stress that may cause to the decrease of antioxidant enzymes activities [63]. This may influence on inflammation which plays a pathogenic role in the development and progression of metabolic syndrome [64].

5. Conclusion

Our results suggest that higher levels of IL-6 and malondialdehyde may cause insulin resistance and metabolic disorders in all subjects with metabolic syndrome. Malondialdehyde level shows strong association with some metabolic syndrome components. This means the greater risk of metabolic syndrome.

Conflict of interest

None.

References

[1] Ferrannini E, Haffner SM, Mitchell BD, et al. Stern MP. Hyperinsulinaemia: the key feature of a cardiovascular and metabolic syndrome. Diabetologia 1991;34:34. 416-422.
[2] Shrestha S, Das BKI, Baral N, et al. Association of metabolic syndrome and its components with thyroid dysfunction in females. Int J Diab Dev Ctreis 2007;27:24–6.
[3] Pandey S, Baral N, Majhi S, et al. Prevalence of the metabolic syndrome in acute myocardial infarction and its impact on hospital outcomes. Int J Diab Dev Ctreis 2009;29:52–5.
[4] Marjani A, Hezarkhani S, Shahini N. Prevalence of metabolic syndrome among Fars ethnic women in north east of Iran World. J Med Sci 2012;7:17–22.
[5] Shahini N, Shahini I, Marjani A. Prevalence of metabolic syndrome in Turkmen ethnic groups in Gorgan. J Clin Diagnost Res 2013;7:1849–51.
[6] Marjani A, Shahini N, Agh Atabay O, Ghiyas Tabari R. Prevalence of metabolic syndrome among sistanee ethnic women advanced studies in biology. Adv Stud Biol 2012;4:363–72.
[7] Marjani A, Shahini N. Age related metabolic syndrome among Fars ethnic women in Gorgan. Iran J Pharm Biomed Sci 2013;30:929–35.
[8] Marjani A, Moghasemi S. The metabolic syndrome among postmenopausal women in Gorgan. Int J Endocrinol 2012;6.
[9] Marjani A, Veghari G, Badeleh MT. Serum lipid peroxidation and leptin levels in male and female type 2 diabetic patients in Gorgan (South East of Caspian Sea). Iran J Chin Clin Med 2010;5:26–35.
[10] Marjani A, Mansourian AR, Ghaemi EO, et al. Lipid peroxidation in the serum of hypothyroid patients (In Gorgan-South East of Caspian Sea). Asian J Cell Biol 2008;3:47–50.
[11] Marjani A, Mansourian AR, Veghari GR, et al. Age-related alterations of plasma lipid peroxidation and erythrocyte superoxide dismutase activity in different ethnic groups of Gorgan. J Appl Sci 2007;7:1795–9.
[12] Marjani A, Moradi A, Saeedi M. Plasma lipid peroxidation zinc and erythrocyte cu-zn superoxide dismutase enzyme activity in patients with type 2 diabetes mellitus in Gorgan City (South East of the Caspian Sea). J Med Sci 2007;7:585–90.
[13] Marjani A. Alterations in plasma lipid peroxidation and total antioxidant status during storage of blood Pakistan. J Biol Sci 2006;9:2520–3.
[14] Marjani A. Effect of haemodialysis on plasma lipid peroxidation and endogenous non-enzymic antioxidants in Gorgan (South East of Caspian Sea). J Med Sci 2006;6:681–5.
[15] Marjani A. Age-related alterations of plasma lipid peroxidation and erythrocyte superoxide dismutase activity in different age groups of Gorgan City. Iran Saudi Med J 2005;26:1647–8.
[16] Higdon JV, Frei B. Obesity and oxidative stress; a direct link to CVD. Arterioscler Thromb Vasc Biol 2003;23:365–7.
[17] Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature 2001;414:813–20.
[18] Maddux BA, et al. Protection against oxidative stress-induced insulin resistance in rat L6 muscle cells by micromolar concentrations of lipoic acid. Diabetes 2001;50:404–10.
[19] Rudich A, et al. Prolonged oxidative stress impairs insulin-induced GLUT4 translocation in 3T3-L1 adipocytes. Diabetes 1998;47:1562–9.
[20] Matsuoka T, et al. Glycation-dependent, reactive oxygen species-mediated suppression of the insulin gene promoter activity in HIT cells. J Clin Invest 1997;99:144–50.
[21] Ceriello A, Quatraro A, Giugliano D. Diabetes mellitus and hypertension: the possible role of hyperglycaemia through oxidative stress. Diabetologia 1993;36:265–6.
[22] Giugliano D, Ceriello A, Paolisso G. Diabetes mellitus, hypertension, and cardiovascular disease: which role for oxidative stress. Metabolism 1995;44:363–8.
[23] West IC. Radicals and oxidative stress in diabetes. Diabetic Med 2000;17:171–80.
[24] Giugliano D. Dietary antioxidants for cardiovascular disease prevention. Nutr Metab Cardiovasc Dis 2000;10:38–44.
[25] Toshima S, Hasegawa A, Kurabayashi M, et al. Circulating oxidized low density lipoprotein levels: a biochemical risk marker for coronary heart disease. Arterioscler Thromb Vasc Biol 2000;20:2243–7.
[26] Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation 2002;105:1135–43.
[27] Wallenfeldt K, Fagerberg B, Wikstrand J, et al. Oxidized low-density lipoprotein in plasma is a prognostic marker of subclinical atherosclerosis development in clinically healthy men. J Intern Med 2004;256:413–20.
[28] Hansel B, Giral P, Nobecourt E, et al. Metabolic syndrome is associated with elevated oxidative stress and dysfunctional dense high-density lipoprotein particles displaying impaired antioxidative activity. J Clin Endocrinol Metab 2004;89:4963–71.
[29] Festa A, D'Agostino RJR, Howard G, et al. Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). Circulation 2000;102:42–7.
[30] Lakka HM, Laaksonen DE, Lakka TA, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. JAMA 2002;288:2709–16.
[31] Dandona P, Aljada A, Chaudhuri A, et al. Metabolic syndrome: a comprehensive perspective based on interactions between obesity, diabetes, and inflammation. Circulation 2005;111:1448–54.
[32] Matsuzawa Y, Funahashi T, Kihara SS, et al. Adiponectin and metabolic syndrome. Arterioscler Thromb Vasc Biol 2004;24:29–33.

[33] Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med 2005;352:1685–95.
[34] Papanicolaou DA, Wilder RL, Manolagas SC, et al. The pathophysiologic roles of interleukin-6 in human disease. Ann Intern Med 1998;128:127–37.
[35] Heinrich PC, Castell JV, Andus T. Interleukin 6 and the acute phase response. Biochem J 1990;265:621–36.
[36] Fernandez-Real JM, Ricart W. Insulin resistance and chronic cardiovascular inflammatory syndrome. Endocr Rev 2003;24:278–301.
[37] Zuliani G, Volpato S, Ble A, et al. High interleukin-6 plasma levels are associated with lowHDL-C levels in community-dwelling older adults: the InChianti study. Atherosclerosis 2007;192:384–90.
[38] Choi KM, Ryu OH, Lee KW, et al. Serum adiponectin, interleukin-10 levels and inflammatory markers in the metabolic syndrome. Diabetes Res Clin Pract 2007;75:235–40.
[39] Hung J, McQuillan BM, Chapman CML, et al. Elevated interleukin-18 levels are associated with the metabolic syndrome independent of obesity and insulin resistance. Arterioscler Thromb Vasc Biol 2005;25:1268–73.
[40] Ridker PM, Rifai N, Stampfer MJ, et al. Plasma concentration of interleukin-6 and the risk of future myocardial infarction among apparently healthy men. Circulation 2000;101:1767–72.
[41] Jenny NS, Tracy RP, OggMS MS, et al. In the elderly, interleukin-6 plasma levels and the -174 G > C polymorphism are associated with the development of cardiovascular disease. Arterioscler Thromb Vasc Biol 2002;22: 2066–71.
[42] Lowe GDO, Rumley A, MacMahon AD, et al. West of Scotland Coronary Prevention Study Group. Interleukin-6, fibrin D-dimer, and coagulation factors VII and XIIa in prediction of coronary heart disease. Arterioscler Thromb Vasc Biol 2004;24:1529–34.
[43] Tuomisto K, Jousilahti P, Sundvall J, et al. C-reactive protein, interleukin-6 and tumor necrosis factor alpha as predictors of incident coronary and cardiovascular events and total mortality. A population-based, prospective study. Thromb Haemost 2006;95:511–8.
[44] Yudkin JS, Kumari M, Humphries SE, et al. Inflammation, obesity, stress and coronary heart disease: is interleukin-6 the link. Atherosclerosis 2000;148: 209–14.
[45] Keaney JF, Larson MG, Vasan RS, et al. Obesity and systemic oxidative stress: clinical correlates of oxidative stress in the Framingham Study. Arterioscler Thromb Vasc Biol 2003;23:434–9.
[46] Festa A, D'Agostino Jr R, Williams K, et al. The relation of body fat mass and distribution to markers of chronic inflammation. Int J Obes Relat Metab Disord 2001;25:1407–15.
[47] Luc G, Bard JM, Juhan-Vague I, et al. C-reactive protein, interleukin-6, and fibrinogen as predictors of coronary heart disease: the PRIME Study. Arterioscler Thromb Vasc Biol 2003;23:1255–61.
[48] Das UN. Metabolic syndrome X: an inflammatory condition. Curr Hypertens Rep 2004;6:66–73.
[49] Altekin E, Çoker C, Şişman AR, et al. The relationship between trace elements and cardiac markers in acute coronary syndromes. J Trace Elements Med Biol 2005;18:235–42.
[50] World Health Organization. Prevention and management of the global epidemic of obesity. Report of the WHO Consultation on Obesity. Geneva: WHO; 1998 [Technical Report Series, No. 894].
[51] 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 (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults Adult Treatment Panel III). JAMA 2001;285:2486–97. 2001.
[52] Satoh K. Serum lipid peroxide in cerebrovascular disorders determined by new colorimetric method. Clin Chim Acta 1978;90:37–43.
[53] Berg AH, Scherer PE. Adipose tissue, inflammation, and cardiovascular disease. Circ Res 2005;96:939–49.
[54] Drabkin DL, Austin JM. Spectrophotometric constants for commonhemoglobinderivativesinhuman, dogandrabbit blood. J Biol Chem 1932;98:719–33.
[55] Bae JH, Bassenge E, Kim KB, et al. Postprandialhypertriglyceridemiaimpairsendothelial function by enhanced oxidant stress. Atherosclerosis 2001;155: 517–23.
[56] Arita Y, Kihara S, Ouchi N, et al. Paradoxical decrease of an adipose-specific protein, adiponectin, in obesity. Biochem Biophys Res Commun 1999;257: 79–83.
[57] Bastard JP, Jardel C, Delattre J, et al. Evidence for a link between adipose tissue interleukin-6 content and serum C-reactive protein concentrations in obese subjects. Circulation 1999;99:2221–2.
[58] Salmenniemi U, Ruotsalainen E, Pihlajamäki J, et al. Multiple abnormalities in glucose and energy metabolism and coordinated changes in levels of adiponectin, cytokines, and adhesion molecules in subjects with metabolic syndrome. Circulation 2004;110:3842–8.
[59] Choi KM, Lee J, Lee KW, et al. Comparison of serum concentrations of C-reactive protein, TNF-alpha, and IL 6 between elderly Korean women with normal and impaired glucose tolerance. Diabetes Res Clin Pract 2004;64:99–106.
[60] Bermudez EA, Rifai N, Buring J, et al. Interrelationships among circulating IL-6, C-reactive protein, and traditional cardiovascular risk factors in women. Arterioscler Thromb Vasc Biol 2002;22:1668–73.
[61] Febbraio MA, Pedersen BK. Muscle-derived IL-6: mechanisms for activation and possible biological roles. FASEB J 2002;16:1335–47.
[62] Shrestha S, Chandra L, Aryal M, et al. Evaluation of lipid peroxidation and antioxidants' status in metabolic syndrome. Kathmandu University Med J 2010;8.
[63] Romero FG, Moran MR. Hypomagnesemia, oxidative stress, inflammation and metabolic syndrome. Diabetes Metab Res Rev 2006;22:471–6.
[64] Karaouzene N, Merzouk H, Aribi M, et al. Effects of the association of aging and obesity on lipids, lipoproteins and oxidative stress biomarkers: a comparison of older with young men. Nutr Metab Cardiovasc Dis 2011;21:792–9.