Evaluation of Epicardial Fat Tissue Thickness and Serum Omentin Levels In Cardiac Syndrome X Patients

Kardiyak Sendrom X'li Hastalarda Epikardiyal Yağ Dokusu Kalınlığının ve Serum Omentin Seviyelerinin Değerlendirilmesi

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ABSTRACT

Introduction: The aim of this study was to evaluate the correlation between epicardial fat tissue thickness (EFTT) and serum omentin levels in patients with cardiac syndrome X (CSX).

Materials and Method: 51 patients admitted to our clinic with CSX were included in our patient group and 46 healthy objects were included in our control group. Demographic informations, routine laboratory tests and hsCRP levels of all patients were registered. EFTT was measured with transthoracic echocardiyography (TTE). Serum omentin levels were measured with enzyme - linked immunosorbent assay (ELISA).

Results: EFTT was measured significantly higher in CSX group ($P < 0.001$). Serum omentin levels were significantly lower in CSX patients in comparison to the control group ($P < 0.001$). Median age was significantly higher in CSX group ($P < 0.001$). WBC and hsCRP levels showed no significant difference between CSX and control group (P : 0.46, p : 0.49, respectively).

Conclusion: In our study we found CSX patients have incresed EFT thickness and decreased serum omentin levels, similarly with the current literatture. According to these findings, increased EFT thickness may play a role in the pathophysiology of CSX by causing a decrease in serum omentin level.

Keywords: epicardial fat tissue; Omentin; Cardiac syndrome X

ÖZET

Giriş: Bu çalışmanın amacı kardiyak sendrom x'li hastalarda epikardiyal yağ dokusu ile serum omentin seviyeleri arasındanki ilişkiyi değerlendirmektir.

Hastalar ve Metod: Kliniğimizde tanısı konan 51 kardiyak sendrom x hastası, hasta grubuna ve 46 sağlıklı olgu ise kontrol grubuna dahil eidlmiştir. Demografik veriler, rutin labarotuar testleri hsCRP seviyeleri çalışıldı. Epikardiyal yağ dokusu kalınlığı transtorasik ekokardiyografi ile serum omentin seviyeleri ise ELISA yontemi ile ölçüldü.

Bulgular: Epikardiyal yağ dokusu kalınlığı hasta grubunda anlamlı olarak daha kalındı ($P < 0.001$). Kontrol grubu ile karşılaştırıldığında, serum omentin seviyeleri hasta grubunda anlamlı olarak daha düşüktü ($P < 0.001$). Kardiyak sendrom xli hastalarda medyan yaş anlamlı olarak daha yüksekti ($P < 0.001$). Lökosit sayımı ve hsCRP seviyleri iki grup arasından anlamlı farklılık göstermedi (sırasıyla; P : 0.46, P : 0.49)

Sonuç: Güncel literatüre benzer şekilde, yapmış olduğumuz çalışmamızda, kardiyak sendrom xli hastalarda epikardiyal yağ dokusu kalınlılığının artmış ve serum omentin seviyelerinin düşük olduğunu gösterdik. Bu bulgular ışığında artmış epikardiyal yağ dokusu , serum omentin seviylerindeki düşüşe neden olarak koroner sendrom x hastalığının patolojisinde rol oynayabilir.

Anahtar Kelimeler: Epikardiyal yağ dokusu; Omentin; Kardiyak sendrom X

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1. Introduction

Coronary arteries are observed to be normal in approximately 30% of patients having typical chest pain and positive cardiac stress test results [1]. This condition in which normal epicardial coronary arteries are observed in spite of the existence of myocardial ischemia evidence was defined as Cardiac Syndrome X (CSX) in 1973 by Kemp et al [2]. Although it is assumed that multiple factors are effective in CSX pathophysiology, microvascular ischemia caused by endothelial dysfunction is the most acknowledged abnormality [3]. In 20 - 30% of CSX patients, symptoms worsen and the quality of life is deteriorated [4]. Furthermore; the mortality rate from myocardial infarctus is 1.2% and a recurrant unstable anginal attack rate is 8.4% in non - st elevation acute coronary syndrome patients with normal coronary arteries [5].

Covering more than three fourths of the heart, epicardial fat tissue (EFT) is a specific type of visceral adipose tissue located between myocardium and visceral pericardium [4,6]. That EFT is an active tissue secreting several mediators such as adipokinin was demonstrated in previous studies [6,7]. It is assumed to be a risk factor for cardiovascular diseases as it secrets several pro - inflammatory cytokines under the effect of paracrine [8,9]. Though EFT can be measured with transthoracic echocardiography (TTE), computerized tomography (CT) and magnetic resonance imaging (MRI); TTE is conceived to be inexpensive, easier - to - apply and a more reliable method among the others [6].

Nowadays, adipose tissue has been declared as not only a storage of triglycerides and source for free fatty acids but also as an endocrine organ which takes part in many metabolic procedures as it secrets several mediators by means of the mature adipocytes it embodies [10]. Omentin, also called intelectin, is a new adipokinin discovered in 2005 [11]. Taking charge in insulin mediated glucose transportation in human adipocytes, omentin is secreted more from the visseral fat tissue in comparison to the subkutan fat tissue [12]. It has been demonstrated that; omentin mRNA is predominantly expressed in human epicardial and omental adipose tissue rather than subcutaneous and internal mammary artery periadventitial adipose tissue reserves. Omentin like other periadventitial epicardial adipokines, may have an important role in CVD pathogenesis. [11] It is demonstarated that omentin increases vasodilatation through nitric oxide (NO) yet; it decreases migration, angiogenesis and vascular inflammation by reducing the release of tumor necrosis factor - alfa (TNF - α) from endothelial cells

[11, 13]. Omentin - 1 levels are decreased in obese individuals and there is an inverse ratio between body mass index (BMI) and omentin level [12]. Serum omentin levels are also found lower in obesity - related conditions such as insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus and policystic ovarian syndrome [13].

In accordance with the information provided above, current study aims to investigate the relationship between the thickness of EFT and serum omentin levels of the CSX patients with microvascular dysfunction as the most common pathophysiology, as well as comparing their results with the normal population.

2. Methods

2.1. Study Design

Approved by the Ethic Committee of İstanbul Bakırköy Dr. Sadi Konuk Training and Research Hospital, current study was designed as an observational cross - sectional study. All the participants were informed of objectives of the study in advance and signed a letter of consent in accordance with the Helsinki Declaration Standards.

2.2. Study Population

51 consecutive patients (16 men, 35 women) diagnosed with CSX in our clinic between dates of January and July 2014 were included in our study. CSX refers to the patients with typcial chest pain, ischemia evidence in myocardial perfusion scintigraphy and normal epicardial coronary lumenogram in coronary angiography. The preliminary diagnosis of vasospastic angina was ruled out in CSX patients, by performing the hyperventilation test following the coronary angiography. The control group is composed of 46 volunteers (17 men, 29 women) admitted to the clinic with atypical chest pain and with negative treadmill test results between the same dates as the patient group. Those with diabetes mellitus, renal dysfunction (creatinine level > 1.5 mg/dl or GFR < 60 ml/min), acute coronary syndrome, stable coronary artery disease, heart failure, inflammatory or autoimmune disease, chronic obstructive lung disease or any other respiratory problems, malignancy and the patients refusing to participate in the study were excluded.

Demographic information of both the patient and the control group were registered. Hypertension was defined as blood pressure ≥ 140/90 mmHg, or the use of anti - hypertensive treatment (14) while hyperlipidemia was defined as the use of a hypolipidemic medication or low - density lipoprotein cholesterol (LDL) ≥ 160 mg/dl or total cholesterol > 220 mg/dl or triglycerides (TG) ≥ 150 mg/dl (15). Finally, BMI was calculated as body weight divided by height squared.

2.3. Biochemical Analysis

Venous blood samples were collected from the antecubital vein followed by an over night fasting. The blood sampled were centrifuged at 2000 g (10 min) tore to move the serum. A liquots of serum samples were stored at - 80°C until analysis. Serum glucose, urea, creatinine, LDL, TG and other biochemical parameters were measured by Beckman Coulter AU 5800 system with commercial kits (Beckman CoulterInc, USA). Serum hsCRP levels were measured by nephelometry using BN II nephelometer (Siemens Healthcare Diagnostics, USA). Complete blood count was determined in a Coulter LH 750 auto analyzer (BeckmanCoulter, CA, USA)

Serum Human Intelectin - 1 (TLN1, Omentin) levels were determined by using Human Intelectin - 1 (TLN1, Omentin) Assay ELISA Kit, according to the manufacturer's instructions YH Biosearch Laboratory Systems (China, cat. no: YHB166 Hu). Intra - assay and inter - assay coefficients of variation were < 10% and 12% respectively. Omentin level was expressed as ng / L.

2.4. Measurement of the Epicardial Fat Tissue

All EFTT were measured after coronary angiography (CAG), by the same cardiology speacialist who was blind to the patients, using a Philips iE33 Ultrasound Machine with a S5 - 1 transducer (1 - 5 MHz) according to Standard techniques in accordance with the recommendations of the American Society of Echocardiography [16]. Aortic annulus was accepted as a reference point for the measurement of EFTT from the parasternal short and long - axis view of the free wall of the right ventricle (Figure 1). The average measurements for both short and long axis views were calculated.

2.5. Coronary Angiography

Standart selective coronary angiography was perfomed on all patients having ischemia evidence in myocardial perfusion scintigraphy with Philips Allura Xper FD10 X - ray system. Coronary angiography

was applied to all patients using right femoral catheterization with Judkins technique and the results were evaluated by a cardiologist blind to study. The control group was not evaluated with CAG.

2.6. Statistical Analysis

For the comparison of numerical variables between CSX and control group, Student t test was used in case of normal distribution of the variables while Mann - Whitney U - test was employed in the existence of a non - normal distribution. Numerical variables were determined as median value ± standart deviation and categorical variables were determined as ratio (%). Categorical variables of the groups were compared by chi square test or in case of need by Fisher's exact test. Furthermore; in correlation analysis while it is the Pearson test to be used for the relation between numerical variables with normal distribution, Spearman Rho test was employed for the relation between numerical variables with non - normal distribution. As for the significant correlation results, the strength and the direction of correlation were defined by Rho value. The diagnostic power of EFTT and serum omentin levels showing significant difference between CSX patients and the control group were demonstrated by means of ROC analysis and ROC curve with the AUC (area under curve) values. Moreover, the conditions where p value was less than 0.05 were accepted as statistically significant. Finally, SPSS 16.0 software (SPSS Inc., Chicago, Illinois, USA) was used for all statistical analysis.

3. Results

As it aforementioned, the CSX group is composed of 51 patients (16 men, 35 women) while the control group consists of 46 volunteers (17 men, 29 women). Median age in CSX group (56 ± 8.7 years) was significantly higher in comparison to the control group (49.5 ± 9.3 years) ($P < 0.001$). Yet, hyperlipidemia was found to be significantly higher in control group (P : 0.029). No significant difference was observed in BMI between CSX patients and the control group (respectively 29.4 ± 4.8, 28.5 ± 6.1, P : 0.54). Other characteristics and comparison of CSX patients and the control group was illustrated in Table 1.

No significant difference was observed in renal function tests, white blood cells, hemoglobin and platelet levels between CSX patients and the control group (Table 2). Also, hsCRP levels were similar between CSX patients and the control group (2.1 ± 4.8, 1.6 ± 3, respectively, P : 0.49). However, TG

levels were significantly higher in the control group (184 ± 77.6 mg/dl) in comparison to CSX patients (132 ± 69.3 mg/dl) (P : 0.045).

EFTT in CSX group was significantly higher than the control group 4.1 ± 0.9, 2.8 ± 0.8 mm; respectively (P < 0.001) (Table 1, Figure 2). Still, serum omentin levels of CSX patients were significantly lower than those of the control group 361.9 ± 201.6, 372.9 ± 191.5 ng/L, respectively, P < 0.001) (Table 2, Figure 3).

The correlation analysis illustrated a weak but significant negative correlation between BMI and omentin; weak but significant positive correlation between BMI and EFTT and finally a moderately significant positive correlation between EFTT and age (Table 3).

Finally, the diagnostic power of EFTT for CSX was observed to be higher than omentin levels in ROC analysis (Figure 4).

4. Discussion

The omentin levels, a newly defined adipokine, were observed to be lower in CSX patients in our study. In accordance with the literature, we found out that EFTT is higher in CSX patients and there is a weak but significant negative correlation between BMI and serum omentin levels. That the diagnostic power of EFTT is higher than serum omentin levels may indicate that several mediators secreted from the epicardial fat tissue may take part in the development of pathophysiology of the disease.

Even though the etiopathogenesis of CSX is not clear yet; endothelial dysfunction, inflammation and abnormal pain sensation are perceived to be the most effective factors [17]. The study by Egashira et al. in 1993 showed that endothelium dependent dilatation of coronary arteries was impaired in CSX patients [18]. Furthermore, Quyyumi et al. concluded that endothelial dysfunction can contribute to decreased coronary reserve during stres or pain [19]. Microvascular dysfunction (MVD), also known as microvascular angina, can be displayed in a significant proportion of CSX patients by using several objective methods [20,21]. A study by Murthy et al. (2014), illustrated that patients having MVD had higher rates of clinical endpoints such as cardiac death or hospitalization due to heart failure and non - fatal myocardial infarctus [22].

EFT is an intrathoracic component of the visceral adipose tissue, located especially in interventricular and atrioventricular sulcus, lateral wall of the right ventricle and around coronary arteries [23]. Even though EFT thickness can be measured by various methods (CT, MRI); echocardiography is usually the primary choice as it is inexpensive, easily accessible, has no radiation disadvantage and has correlated results with MRI, [23]. By secreting adipokines and cytokines, EFT creates paracrine and vasocrine effects on heart [24]. EFT can have anti - inflammatory and anti - atherogenic effects by secreting adiponectin, adrenomedullin; but can also increase the risk of cardiovascular disease with monocyte chemotactic protein (MCP) - 1, interleukin (IL) - 1β, IL - 6 ve TNF - α (24,9). Sade et al. (2009) found out significantly higher EFTT, C - reactive protein (CRP) and insulin resistance; and decreased adiponectin levels in CSX patients, i.e. women with MVD [25]. The study by Benedicte et al. on healthy volunteers showed that increased EFTT is associated with decreased coronary microvascular response and that EFT can affect endothelial function in the early period (26). Moreover; the studies by Gedikli et al. and Mohammd et al. showed increased EFTT in CSX patients [4,6].

Discovered in 2005, omentin is a new adipokine taking part in insülin - dependent glucose transport in human visceral adipocytes [11]. Serum omentin level is observed to be inversely proportional to obesity, insulin resistance and BMI but directly proportional to HDL and plasma adiponectin levels [12]. A study showed that after omentin treatm[ent to rat aorta, noradrenaline dependent vasoconstriction decreased. This result indicates that omentin contributes to vasodilatation through endothelium derived nitric oxide [11]. Moreno et al. demonstrated that serum omentin levels are associated with age, BMI, waist hip ratio, systolic and diastolic blood pressure, endothelium dependent vasodilatation, IL - 6 and CRP levels [27]. The same study showed that omentin can be used as an indicator of endothelial function [27]. Yamawakive et al. found out that omentin has anti - inflammatory effects since it regulates TNF - α - induced cyclooxygenase - 2 secretion in vascular endothelial cells [28]. Zhong et al. (2011) detected decreased serum omentin levels in patients with coronary artery disease as well as a negative correlation between serum omentin levels and BMI and IL - 6 levels [29]. Accordingly, it can be concluded that omentin reduces endothelial dysfunction, serves as an anti - inflammatory molecule and has protective effects in cardiovascular diseases [11].

The relation between visceral adipose tissue thickness and cardiometabolic diseases, especially the role of various molecules secreted from adipocytes, has been examined via clinical researches. In the

light of these studies; parallel to the increased visceral adipose tissue thickness, adipokine levels secreted from adipocytes protective against cardiovascular disease, decrease and precipitating molecule levels increase. In our study we also found that CSX patients have increased EFT thickness and decreased omentin levels. We haven't directly investigated the relation between EFT thickness and serum omentin levels but we may conclude that serum omentin levels may have a negative correlation with EFT thickness.

5. Study Limitations

The number of patients and the control group included in the study are the most important limiting factor. Another limiting factor was control group was not consisted of age and sex matched healthy volunteers, because of our study design we had to include patients with aypical chest pain and negative treadmill test. Also; the inability to show microvascular dysfunction in CSX patients objectively, the measurement of EFTT by transthoracic echocardiography, not studying any inflammatory marker other than hsCRP and not analysing insulin resistance are considered to be other restrictive factors. However; as is stated in the introduction and the discussion sections, EFTT measurement by echocardiography is not considered as a major limitation as it is inexpensive, easily accessible and has correlated results with MRI [6,23].

6. Conclusion

In our study, increased EFT thickness and decreased serum omentin levels are found to be associated with CSX disease. The higher diagnostic value of EFTT from serum omentin levels indicates that the visceral adipose tissue is a pool and affects the development of the disease by secreting various molecules. Our study needs to be supported by more extensive researches in this direction. Besides, in the extent of our knowledge, our study is the first to analyse both EFTT and serum omentin levels together in CSX patients.

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Table 1. Comparison of clinical features of cardiac syndrome X and control groups.

	Cardiac syndrome X group (%)	Control group (%)	P
Number of cases	51	46	
Age (median ± SD) (year)	56 ± 8.7	49.5 ± 9.3	< 0.001*
BMI (median ± SD)	29.4 ± 4.8	28.5 ± 6.1	0.54*
Gender			0.56
Male	16 (31.4)	17 (37)	
Female	35 (68.6)	29 (63)	
HT			0.24
(-)	26 (51)	18 (39.1)	
(+)	25 (49)	28 (60.9)	
Smoking			0.09
(-)	10 (19.6)	16 (34.8)	
(+)	41 (80.4)	30 (65.2)	
Family anamnesis			0.94
(-)	17 (33.3)	15 (32.6)	
(+)	34 (66.7)	31 (67.4)	
HPL			0.029
(-)	19 (37.3)	8 (17.4)	
(+)	32 (62.7)	38 (82.6)	
EFTT (mm) (median ± SD)	4.1 ± 0.9	2.8 ± 0.8	< 0.001*

Chi-square test, *Student's t test, SD: Standard deviation, HT: Hypertension,

HPL: Hyperlipidemia, BMI: Body mass index, EFTT: Epicardial fat tissue thicknes

Table2. Comparison of labaratory features of cardiac syndrome X and control groups

	Cardiac syndrome X group (median ± SD)	Control group (median ± SD)	P
hsCRP (mg/dl)	2.1 ± 4.8	1.6 ± 3.7	0.49*
Urea (mg/dl)	29 ± 7.2	26.5 ± 8.5	0.56
Creatinine (mg/dl)	0.7 ± 0.15	0.69 ± 0.15	0.46
LDL (mg/dl)	129 ± 32.9	127 ± 161	0.89*
Total cholesterol (mg/dl)	195 ± 43.4	209 ± 34.4	0.46
HDL (mg/dl)	48 ± 11	47.5 ± 9.8	0.83
Triglyceride (mg/dl)	132 ± 69.3	184 ± 77.6	0.045
HbA1C (%)	5.4 ± 0.37	5.4 ± 0.4	0.56
Omentin (ng/L)	361.9 ± 201.6	372.9 ± 191.5	0.042*
WBC	7000 ± 1380	7070 ± 1470	0.46
Hb (gr/dl)	13.1 ± 1.6	13.3 ± 1.4	0.97
PLT	261000 ± 55900	267000 ± 52800	0.71
Neutrophil count	3700 ± 950	3800 ± 1000	0.28
Lymphocyte count	2060 ± 580	2000 ± 640	0.86

Student's t test, *Mann - Whitney U test, SD: Standard deviation

Table 3. Summary of significant correlation results between serum omentin level, epicardial fat tissue thickness and other variables

Variables	Rho	P
Omentin - BMI	- 0.221	0.03
EFTT - Age	0.482	< 0.001*
EFTT - BMI	0.291	0.004*

Spearman's Rho test, *Pearson's test, BMI: Body mass index, EFTT: Epicardial fat tissue thickness

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Figure 1. Measurement of epicardial fat tissue (EFT, EAT) thickness by echocardiography. LV; left ventricle, LA; left atrium, RV; right ventricle, Ao; aorta.

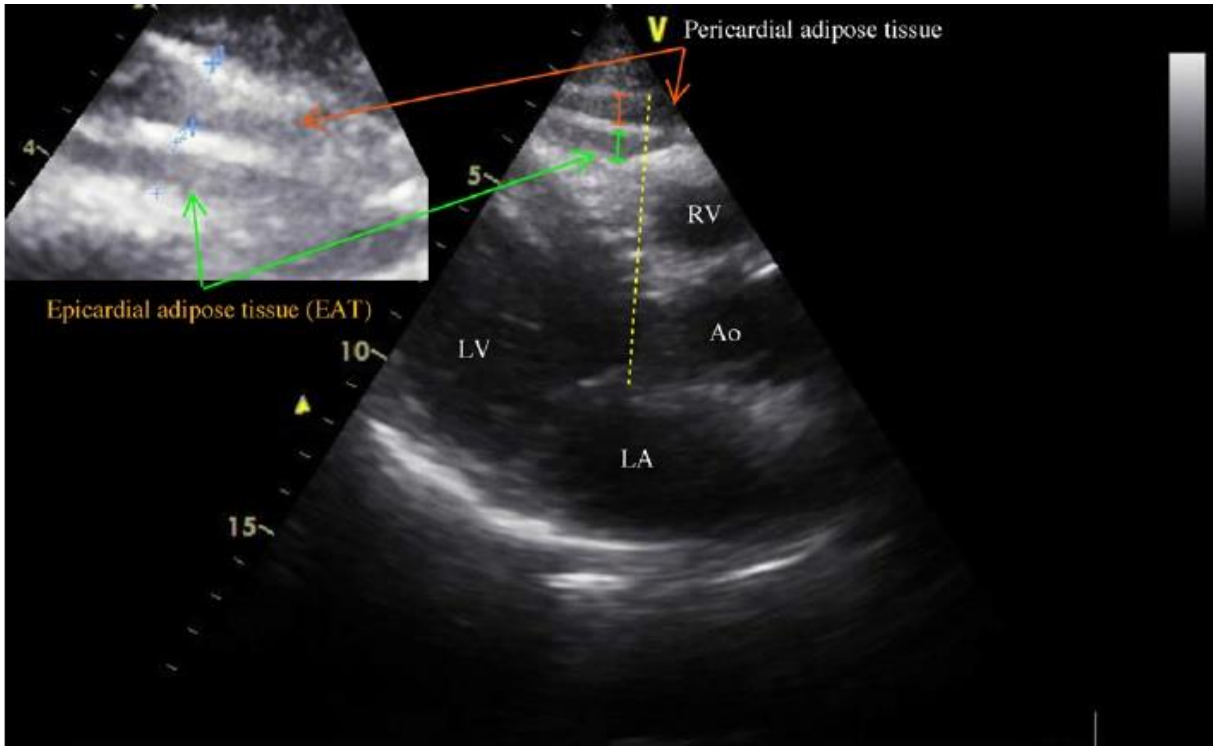

Figure 2. Distribution of epicardial fat tissue thickness in syndrome X and control groups (Box-plot)

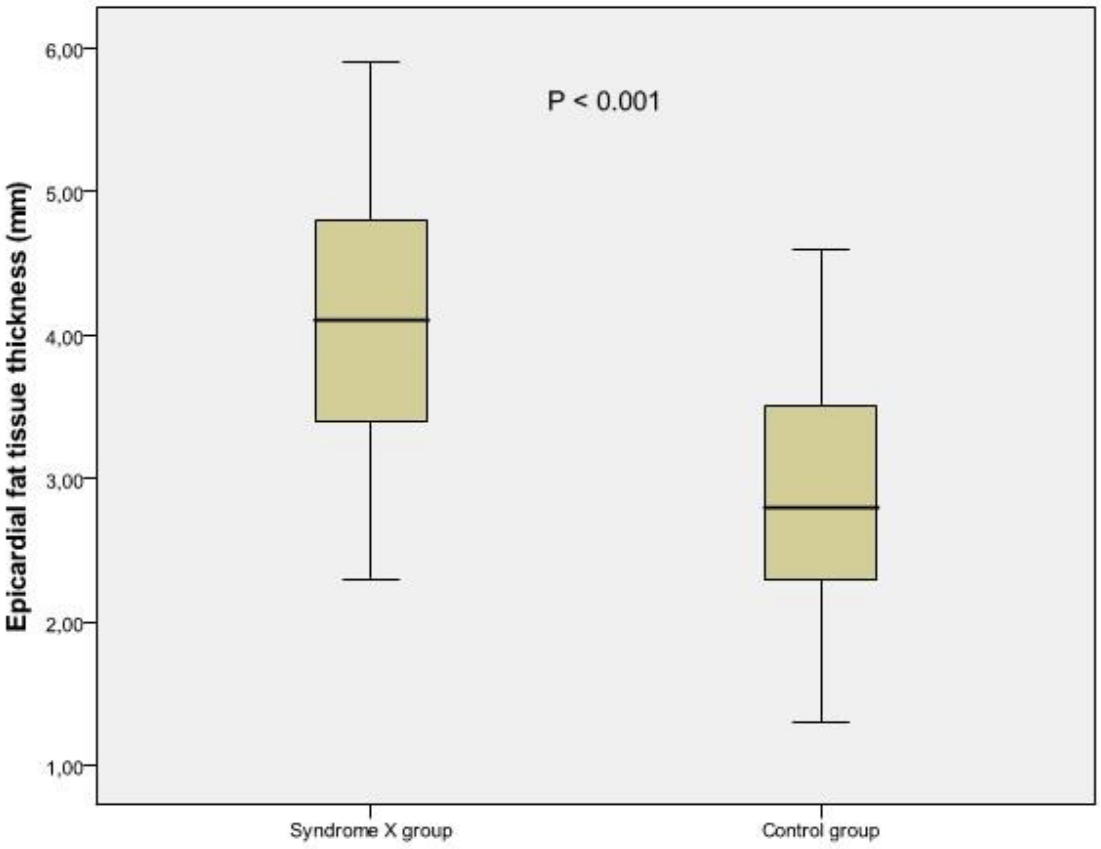

Figure 3. Distribution of serum omentin levels in syndrome X and control groups (Box-plot)

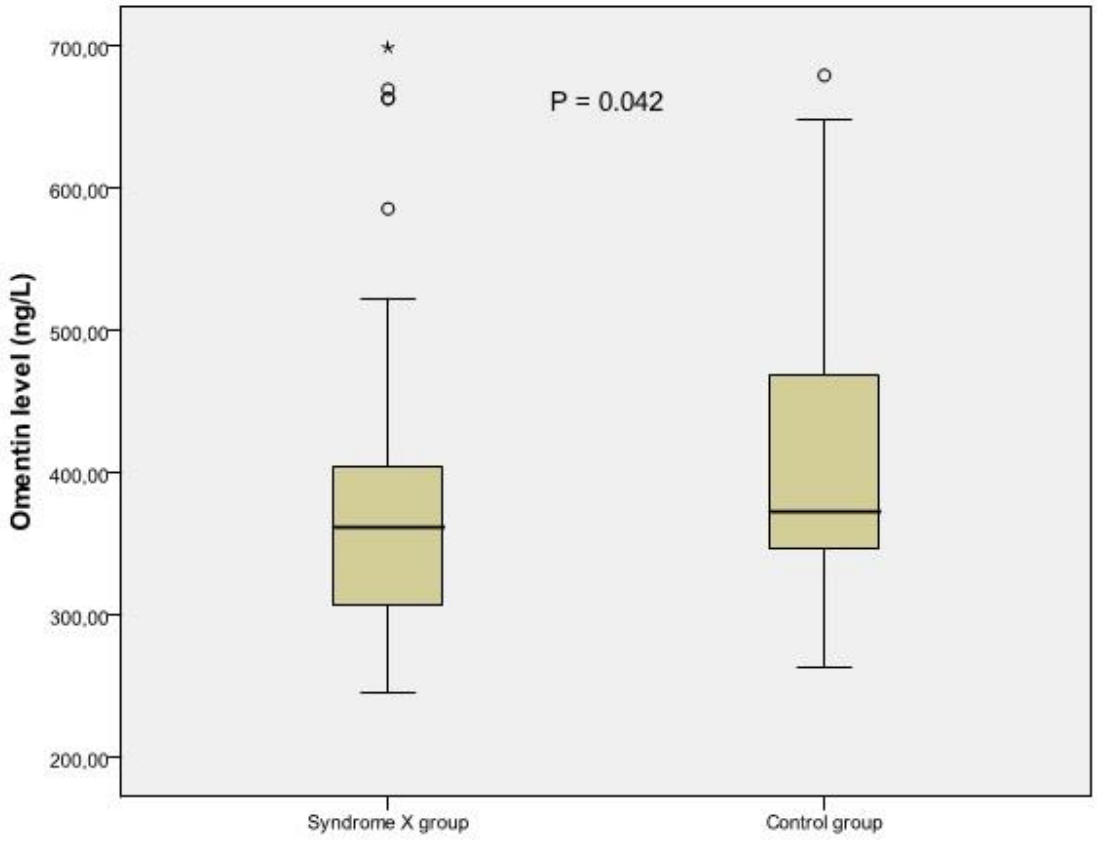

Figure 4. ROC curves of omentin levels and epicardial fat tissue thickness for prediction of syndrome X.