Türk Kardiyol Dern Arş - Arch Turk Soc Cardiol 2012;40(8):671-680 doi: 10.5543/tkda.2012.92170

Kalsiyum skorunun koroner arterlerdeki segmenter dağılımı

Segmental distribution of calcium scores in the coronary arteries

Dr. Demet Erciyes, Dr. Murat Şener,# Dr. Cihan Duran,†

Dr. Mustafa Şirvancı,* Dr. Cemşit Demiroğlu, Dr. Murat Gülbaran#

ÖZET

Amaç: Koroner arterlerde kalsiyum birikimi aterosklerozun göstergesidir. Çalışmamızın amacı, kalsiyum birikiminin hangi koroner segmentinde daha fazla olduğunu ve demografik verilerle segmenter kalsiyum skorları arasındaki ilişkiyi göstermektir.

Çalışma planı: Çalışmaya 299 hasta alındı (192 erkek, 107 kadın; ort. yaş 59.08±10.7; dağılım 19-84 yıl). Tüm hastalarda 16 kesitli çok kesitli bilgisayarlı tomografi kullanılarak kalsiyum skoru ölçümleri yapıldı. Toplam kalsiyum skoru 1 Hounsfield ünitesi ve üzerindeki hastalarda koroner arterler 14 ayrı segmente ayrılarak her segmentin kalsiyum skoru ve lezyon sayısı hesaplandı.

Bulgular: Koroner arterlerin kalsiyum birikimleri incelendiğinde en fazla birikimin sol ön inen koroner arterin (LAD)proksimal segmentinde olduğu görüldü. Toplam kolesterolü ve LDL kolesterolü yüksek hastalarda toplam kalsiyum skorları, olmayanlara göre anlamlı olarak yüksek bulundu. Toplam kalsiyum skoru, hipertansiyonu olan hastalarda olmayanlara göre yüksekti. Sigara

ABSTRACT

Objectives: Calcium accumulation in the coronary arteries is a known indicator of atherosclerosis. The purpose of this study was to demonstrate both the correlation between patients' demographic characteristics and segmental calcium scores and also dominant topographic accumulation of calcium in the coronary arteries.

Study design: Two-hundred ninety-nine patients were included in the study (192 male, 107 female; mean age 59.08±10.7; range 19 - 84 years). All patients with total calcium scores of 1 Hounsfield unit or more underwent 16-slice multi-detector computed tomography with calcium scoring evaluation. Their coronary trees were divided into 14 different segments, then the number of lesions, and calcium score of each segment were calculated separately.

Results: When the coronary arteries were examined as for segmental calcium accumulation, the proximal segment of the LAD (left anterior descending coronary artery) had the highest calcium accumulation. Total calcium scores were higher in the patients with high total and LDL kullanan ve kullanmayan hastalar arasında kalsiyum skorları açısından anlamlı fark bulunmadı. Toplam kalsiyum skorları diyabeti olan hastalarda olmayanlara göre anlamlı olarak yüksekti. Koroner arter hastalığı aile hikâyesi olan hastaların toplam kalsiyum skorları ile olmayanların arasında fark saptanmadı. Yaş gruplarına göre incelendiğinde ileri yaş grubundaki hastalarda kalsiyum skorlarının anlamlı derecede yüksek olduğu görüldü.

Sonuç: Koroner arterlerdeki kalsiyum birikiminin en fazla LAD proksimal bölümü olmak üzere daha çok proksimal segmentlerde olduğunu saptadık. Koroner arter kalsifikasyonunun, hipertansiyon, yaş ve hiperlipidemi gibi koroner arter hastalığı risk faktörleri ile arttığı da görüldü.

cholesterol values than in normolipidemic patients. Total calcium scores were higher in patients with hypertension relative to the patients without hypertension. Calcium scores of smokers and non-smokers were not significantly different. Diabetic patients had higher calcium scores than patients without diabetes. No significant difference was identified between patients with and without a positive family history of coronary artery disease. Total calcium scores were significantly higher in the advanced age group, and in patients with hyperlipidemia.

Conclusion: Segmental analysis of calcium scoring demonstrates that calcium accumulation is mostly seen in the proximal LAD. Coronary artery calcification is observedly increased in the presence of coronary artery risk factors as hypertension, advanced age, and hyperlipidemia.

Geliş tarihi: 22.12.2010 Kabul tarihi: 09.08.2012
Yazışma adresi: Dr. Demet Erciyes. Şişli Florence Nightingale Hastanesi, Kardiyoloji Bölümü, Abide-i Hürriyet Cad. No: 164, Şişli, İstanbul.
Tel: +90 212 - 224 49 50 e-mail: demeterciyes@yahoo.com

Submitted on: 12.22.2010 Accepted for publication on: 08.09. 2012
Address of correspondence: Dr. Demet Erciyes. Division of Cardiology, Şişli Florence Nightingale Hospital, , Abide-i Hürriyet Cad. No: 164, Şişli, İstanbul, Turkey
Phone: +90 212 - 224 49 50 e-mail: demeterciyes@yahoo.com

Abbreviations:

CAD Coronary artery disease
Cx Circumflex artery
DM Diabetes mellitus
HDL-C: high- density lipoprotein cholesterol
HL: Hypercholesterolemia
LAD: Left anterior descending artery
LDL-C: low- density lipoprotein cholesterol
LMCA: Left main coronary artery
MSCT: multi-slice computed tomography
RCA: Right coronary artery

Coronary artery disease (CAD) is one the foremost causes of mortality, and morbidity in the developed countries.[1] Nowadays, with the introduction of multi-slice computed tomography (MSCT) which can record multiple images simultaneously into clinical practice, CT imaging modality has become more prevalent. Accumulation of calcium in the coronary arteries is known to be an indicator of atherosclerosis for a long time. Amount of calcium accumulated in coronary artery plaques consists nearly 20 % of the plaque volume. However the presence of plaques more vulnerable to rupture but without any calcium accumulation is also acknowledged.[2] In untreated patients, it has been demonstrated that a 15-20 % annual increase in coronary calcium burden occurs, but lipid lowering treatment could halt or slow down the rate of accumulation. .[3] It has been also reported that the likelihood of stenosis due to soft coronary plaque(s) was very low in patients with a calcium score of 0 HU.[4]

A study investigating the risk of development of serious cardiac events as mortality, myocardial infarction, stroke, and revascularization in different groups of patients without any intergroup difference as for atherosclerotic risk factors, has demonstrated that these risks were 22 –fold higher in cases with a calcium score of >160 Hounsfield ünits relative to those with a calcium score of 0 HU.[5]

In our study, we aimed to identify, and demonstrate the coronary artery segment(s) with increased accumulation using MSCT, and whether or not calcium distribution is affected by coronary artery risk factors as advanced age, hypertension, and hyperlipidemia.

PATIENTS AND METHOD

Selection of patients

Between January 2005, and December 2007, among 1094 patients who were referred with suspect diagnosis of coronary artery disease and undergone calcium scoring using MSCT, 299 cases (192 male, 107 female; mean age 59.08±10.7 yrs; range. 19-84 yrs) with calcium scores of > 0 HU were included in the study .(Table 1).

Exclusion criteria

Patients with a total calcium score of 0 HU were excluded from the study. Patients with damaged intimal, and medial layers of the coronary artery wall secondary to surgical or percutaneous revascularization or patients who had undergone revascularization because of increase in calcification were also excluded from the study. One of the reasons for the exclusion of the cases who had undergone stent revascularization using percutaneous coronary intervention was the blooming effect of the coronary artery stent which precluded healthy evaluation of the regional calcification. Cases referred from other centers whose demographic data could not be obtained were also excluded from the study.

CAD risk factors

American Diabetes Association criteria were used to make a diagnosis of diabetes mellitus (DM).[6] Diagnosis of DM was established in cases with a fasting blood glucose (FBG) of ≥ 126 mg/dl and/or those using oral antidiabetics and/or insulin.

In accordance with "Adult Treatment Panel III'' (ATP III) guidelines, diagnoses were based on the following criteria: hypercholesterolemia: serum total cholesterol >200 mg/dl or LDL cholesterol >100 mg/dl, use of lipid- lowering drugs, and hypertriglyceridemia (HTg): serum triglyceride > 150 mg/dL or use of triglyceride-lowering drugs .[7]

In compliance with "Joint National Committee VII" (JNC VII) guideline, hypertension was defined as systolic blood pressure of >140 mmHg or diastolic blood pressure of > 90 mm Hg, and use of antihypertensive drugs.[8]

Positive family history was defined as the presence of CAD in the first-degree relatives (males < 55, and females < 65 years of age)[7]

Current or past smokers were assessed as having a positive smoking history.

Table 1. Demographic data of the patients

	n	%
Male	192	64
Female	107	36
Family history	**88**	29
Hypertension	104	35
Diabetes mellitus	47	16
Smokers	80	27
Total cholesterol (>200 mg/dl)	73	24
LDL-C (>100 mg/dl)	95	32
HDL-C (<40 mg/dl)	72	24
Triglyceride (>150 mg/dl)	52	17

LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol

MSCT imaging

Single breath-hold non-contrasted multi-slice images were recorded within nearly 5 seconds. With the aid of electrocardiographic gating, 3-mm thick sections were obtained beginning from aortic root to the apex. MSCT data were retrieved using "Sensation 16 slice scanner" (Siemens Medical Solutions, USA)

Calculation of calcium scores

Semi-automatic programs were used for these calculations, and the results were expressed as Agatston scores. Agatston scoring system is the most frequently used measurement method for the determination of the amount of calcium in coronary arteries. In this method the calcium score is the product of area of the calcific plaque multiplied by the highest HU density. In the Agatston scoring lesions with > 130 HU density in two contiguous >1 mm^2 sections with 2-3 pixels were interpreted as calcifications. [9]

Coronary arteries were virtually divided into 14 different segments as proximal, mid- , and distal segments of the right (RCA), left deccending (LAD) , the circumflex (Cx) coronary arteries; the left main coronary artery (LMCA); posterolateral (RPL), and posterior descending (RPD) branches of RCA, and Cx. Number of the lesions, and scores of calcium accumulation in each segment were calculated individually (Figure 1).

Statistical Analysis

The same segments of all patients were evaluated as a single data, and the difference between calcium accumulations in these and other segments were investigated. The relationship between distribution of calcium accumulation, and the basic characteristics of the patients was investigated.. Calcium accumulation in coronary arteries was assessed as total values for each major coronary artery separately, and correlations among them were evaluated.

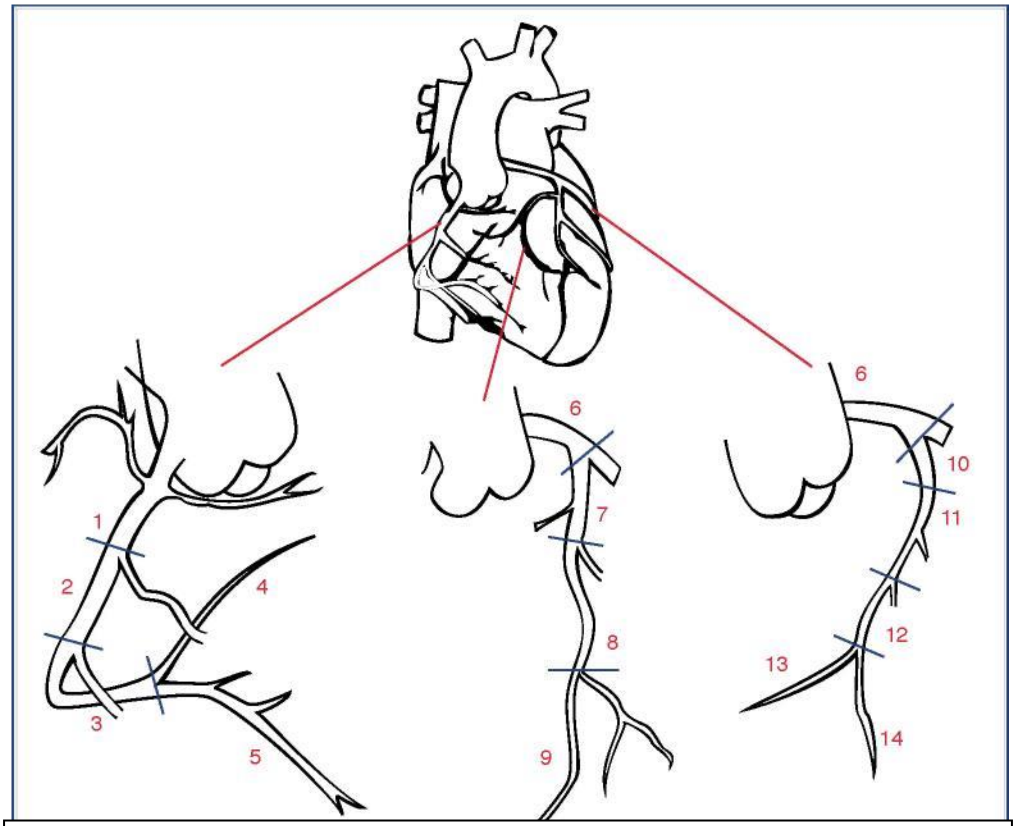

Figure 1. Segmental classification of coronary arteries modified from ACC/AHA criteria

For statistical evaluation SPSS (Statistical Package for the Social Sciences version 11.0, SSPS Inc, Chicago, III, USA) software program was used.

In intersegmental comparisons ANOVA (analysis of variance) test or its nonparametric equivalent Kruskal-Wallis or Mann-Whitney U-test was used. Homogeneity was evaluated using Levene, and Lillefor tests. Bonferroni test was used in post-hoc analysis. The impact of demographic data on segments, and calcium scores was evaluated with Student t-test or its nonprametric equivalent Mann-Whitney U-test. Correlation of parametric values were assessed with Pearson and Spearman correlation test, and bivariant linear regression analysis. For the comparison of nonparametric values *chi*-square or Fisher's exact test was used. The values were expressed as mean ± standard deviation (SD). $p<0.05$ was accepted as statistically significant.

RESULTS

A significant difference was not found between total calcium scores of male, and female patients. However when each segment was evaluated individually, calcium score of the LAD midsegment in men was found to be higher than that of the women (p=0.029).

The patients were divided into 3 age groups as those aged < 50 (Group 1: n=52), 50-65 (Group 2: n=167), and ≥ 65 years (Group 3: n=80),

Also a significant difference was found in calcium scores of Groups 1, and 2 as for LMCA (p=0.034), RCA midsegment (p=0.022), LAD proximal (p=0.045) and Cx proximal (p=0.021) segments. Still a significant difference was detected in calcium scores in patients aged < 50, and > 65 years as for LMCA (p<0.001), and proximal segments of LAD (p=0.003), Cx (p=0.007) , and RCA (p=0.045) (Figure 2).

Calcium scores of the proximal, mid, and distal segments were compared in patients aged <50, and 50-65 years with a significant difference in the proximal segment group (p=0.032). Still when the patients aged < 50, and ≥ 65 years were compared a significant difference was noted between proximal (p=0.001), and distal (p=0.048) segment groups.

A significant difference was revealed between patients aged 50-65, and ≥ 65 years regarding calcium scores of LMCA (p=0.014) , and LAD (p=0.010), while in comparisons between cases aged <50, and ≥ 65 years, calcium scores differed significantly as for LAD (p=0.003), RCA (p=0,005), Cx (p=0.008), and LMCA (p<0.001) (Figure 3).

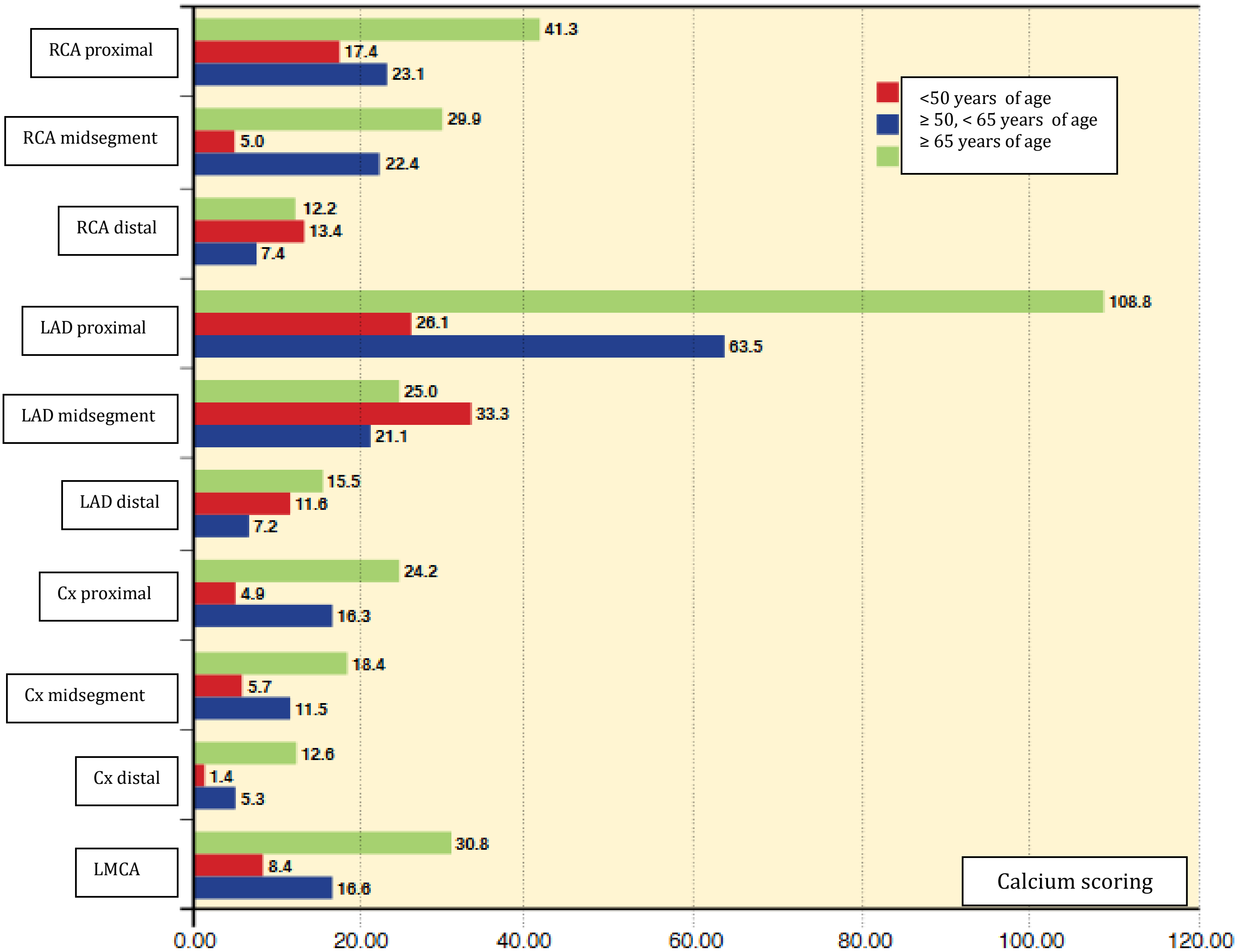

Figure 2. Comparison of calcium scores of coronary artery segments according to age groups
RCA: Right coronary artery; LAD: Left anterior descending artery; Cx: Circumflex artery; LMCA: Left main coronary artery

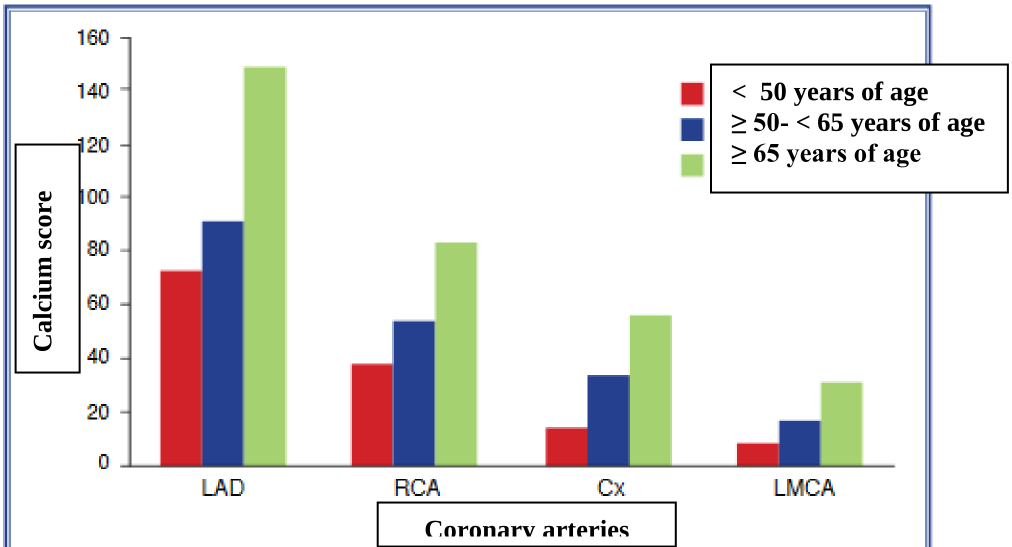

Figure 3. Comparison of coronary arteries as for calcium scores in various age groups RCA right coronary artery; LAD: Left anterior descending artery; Cx: Circumflex artery; LMCA: Left main coronary artery

When calcium accumulations in coronary arteries were analyzed on the base of segmental distribution irrespective of age groups, calcium accumulation was mostly observed in the proximal LAD, and then in the proximal RCA , midsegment of the LAD, proximal segments of LMCA, and Cx, in the decreasing order of frequency. (Figure 4).

When each major coronary artery was analyzed as one integral unit, and compared as for total calcium scores, the highest calcium score was estimated for LAD, and then RCA, Cx, and LMCA in decreasing order of frequency (Figure 5).

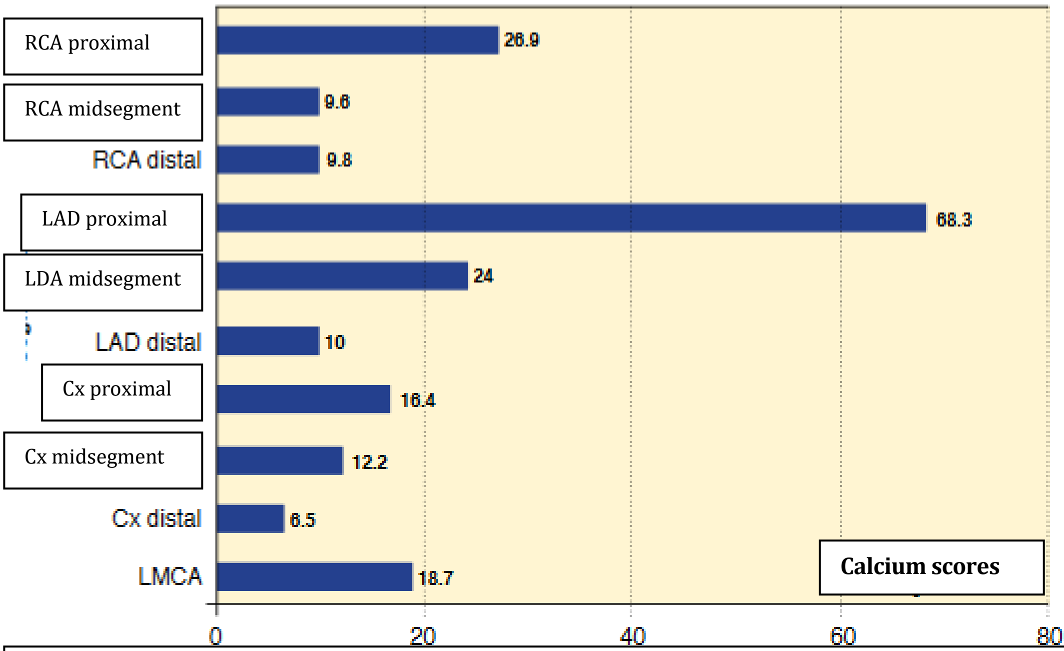

Figure 4. Segmental distribution of calcium scores RCA: right coronary artery: LAD: Left anterior descending artery; Cx: Circumflex artery; LMCA: Left main coronary artery

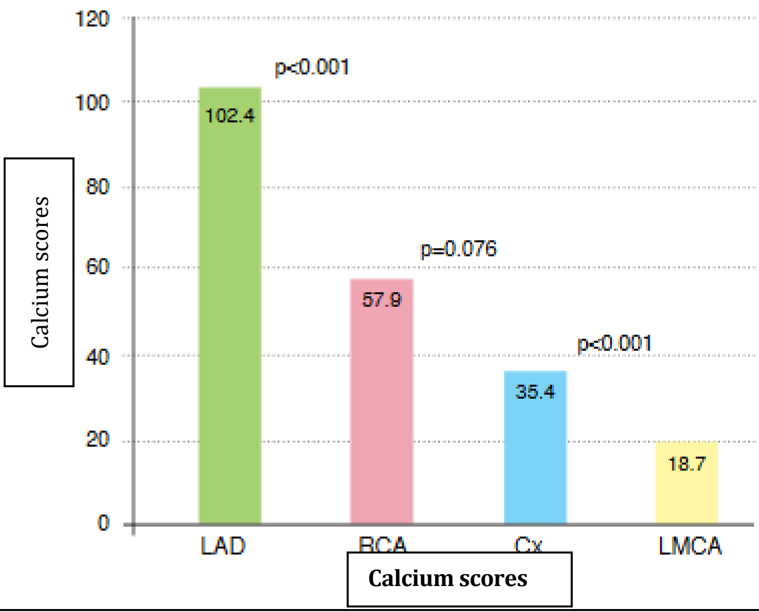

Figure 5. Calcium scores of coronary arteries aligned in order of decreasing frequency. RCA: right coronary artery:LAD: Left anterior descending artery; Cx: Circumflex artery; LMCA: Left main coronary artery

When segments were grouped based on their distance from coronary ostium as proximal (segments 1, 7, and 10), midsegments (segments 2, 8, 11), and distal (segments 3, 4, 5, 9, 12, 13, and 14) segments, the highest calcium score was detected in the proximal group, and then midsegments, and distal segments in order of decreasing frequency (p<0.001).

Total calcium score was relatively higher in hypertensives when compared with normotensive patients (p=0.008). While LAD (p=0.001) and Cx (p=0.03) arteries differed in this respect, RCA and LMCA was not significantly different. Besides, calcium scores of both proximal (p=0.012), and distal (p=0.01) segments were higher in hypertensives when compared with those of the normotensive patients. In addition calcium scores calculated for RCA distal (p=0.03), LAD proximal (p=0.003), and Cx mid- (p=0.024), and distal (p=0.028) segments were relatively higher.

A significant difference as for calcium scores of all segments could not be found between smokers, and non-smokers.

Total calcium scores of the diabetics were significantly higher than those of the non-diabetics (p<0.001).

Calcium scores of coronary artery segments in diabetics were generally higher than those of the non-diabetics. Only calcium scores of LMCA, and proximal segments of Cx were not different (Figure 6).

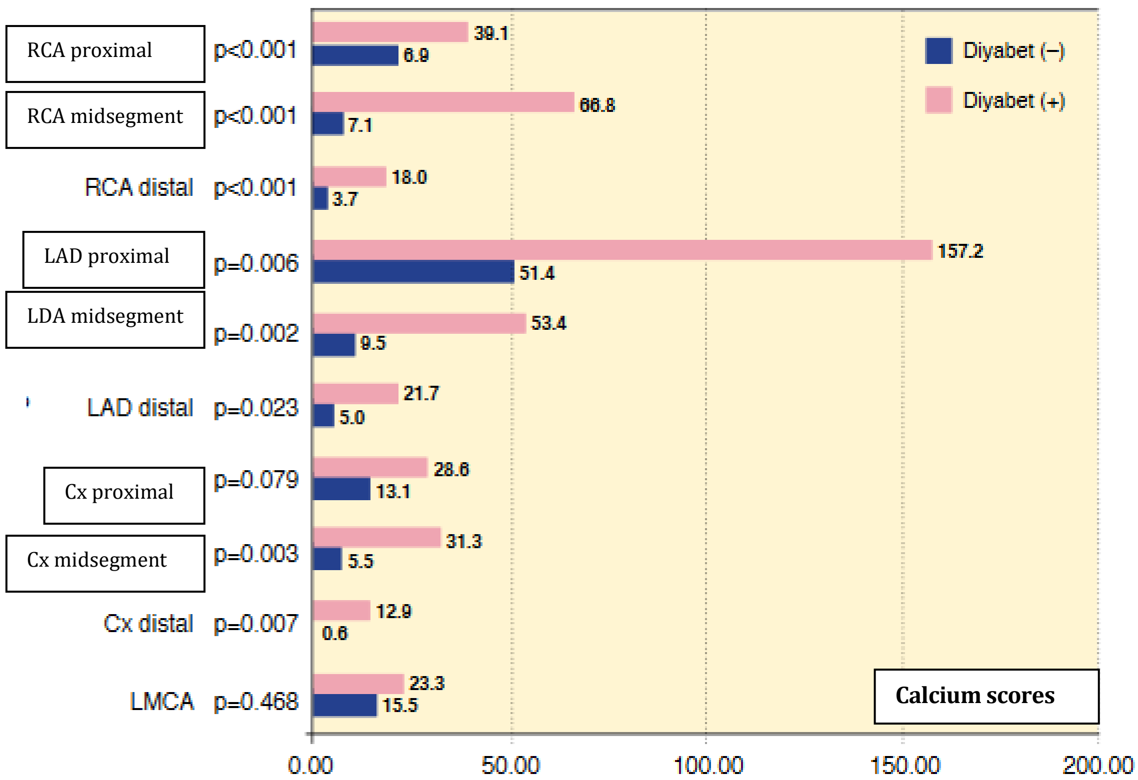

Figure 6. Comparison of segmental calcium scores between patients with and without diabetes. RCA: right coronary arteryLAD: Left anterior descending artery; Cx: Circumflex artery; LMCA: Left main coronary artery

Calcium scores of patients with higher total cholesterol levels were found to be higher than those normocholesterolemic patients ($p<0.001$).

Even though a significant difference could not be found as for total calcium scores of the patients with and without higher HDL or LDL-cholesterol levels, significantly increased calcium scores were detected in cases with higher LDL cholesterol values relative to those with normal levels ($p=0.001$).

Calcium scores of coronary artery segments in patients with and without total cholesterol levels were analyzed, and significant intergroup differences in these levels were noted as for proximal, mid-, and distal segments of RCA, proximal, and midsegments of LAD, and midsegments of Cx. ($p<0.05$) (Figure 7).

A significant difference did not exist regarding calcium scores of coronary artery segments in patients with and without HDL cholesterol levels below and above 40 mg/dl.

Calcium scores of proximal segments of LAD ($p=0.001$), and RCA ($p=0.020$), and distal segments of RCA ($p=0.029$) differed significantly between patients with LDL –cholesterol levels above and lower 100 mg/dL.

Calcium scores of the coronary artery segments of the patients with triglyceride levels below, and above 150 mg/dl were analyzed, and a significant intergroup difference was observed only between calcium scores of the LAD midsegments ($p=0.013$).

Still total calcium scores calculated for all segments were not different between patients with and without positive familial history for CAD.

When calcium scores were compared as for dominancy of coronary artery segments, only calcium scores of the patients with left RCA dominancy were lower with those of right coronary artery dominancy ($p=0.004$) and cases with balanced coronary artery laterality ($p=0.001$).

In our study, a significant correlation was observed among total calcium scores of RCA, and proximal segments of RCA, and calcium scores of LMCA.

DISCUSSION

Coronary artery disease is among the most prevalent diseases of our time, and continues to be one of the predominant causes of death worldwide. CAD does not only affect survival, and quality of lives of the individuals, but also has an adverse impact on the community with its increased financial burden. A consensus has been established that the likelihood of future cardiovascular events is quite low (annual incidence of 0.15 %) in patients with calcium scores of 0 HU .[10,11]

In our study, when calcium scores were compared between genders, calcium scores related to only midsegment of LAD were observedly higher in male patients. Total calcium scores of LAD were found to be higher in hypertensive patients when compared with normotensive cases. Besides calcium scores related to proximal, and distal segment groups were significantly higher in the hypertensive patients. In the midsegment group, hypertensive patients had higher calcium scores relative to the normotensive cases without any statistically significant difference between them. If the number of patients were higher, we could detect statistically significant difference between the groups.

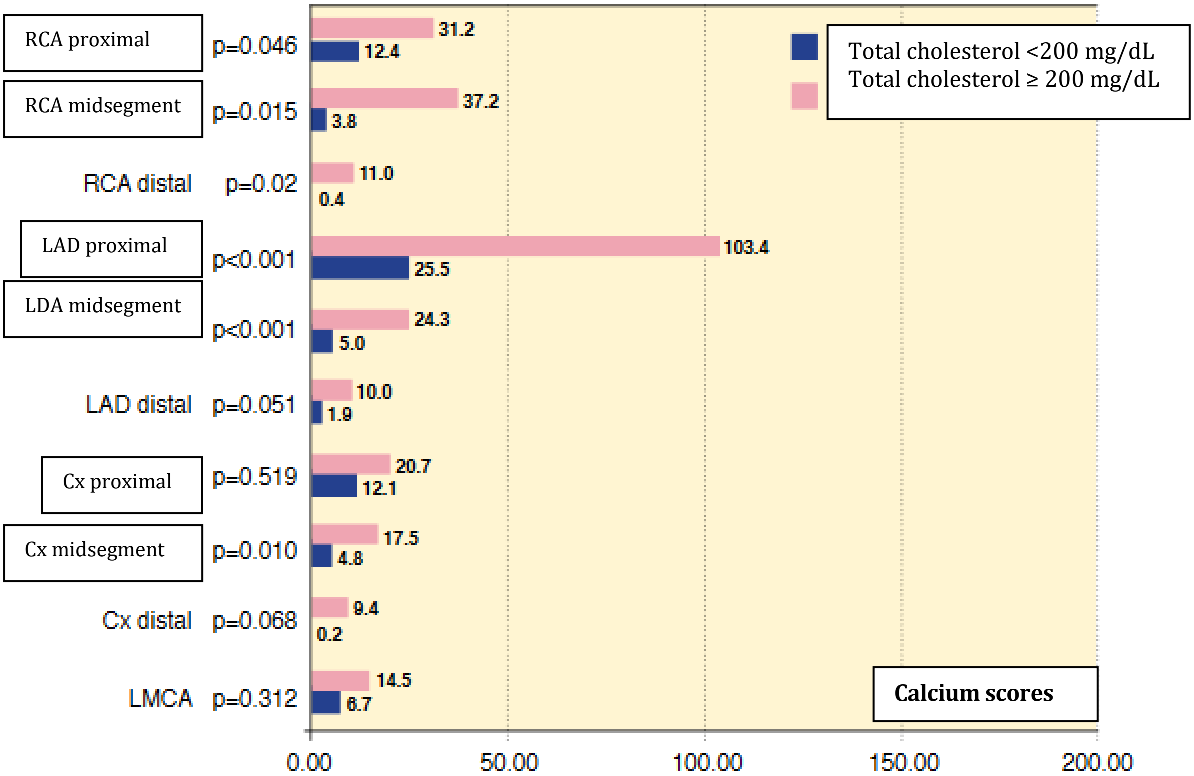

Figure 7. Calcium scores of coronary artery segments according to total cholesterol levels RCA: right coronary artery: LAD: Left anterior descending artery; Cx: Circumflex artery; LMCA: Left main coronary artery

The association between smoking, and atherosclerosis is not known. In our study, in none of the comparisons, calcium scores of smokers were different from those of the nonsmokers. This lack of difference might stem from scarce number of our study patients.

Calcium scores of diabetics were found to be significantly higher that those of non-diabetics.

Calcium scores did not differ between patients with and without positive family history, except for higher calcium scores detected for proximal segments of RCA in patients without any familial evidence of coronary artery calcification. This finding can be coincidental. Still, with higher patient population, the results might be different. When the patients with higher, and lower levels of total cholesterol, LDL-C, and HDL-C were stratified, calcium scores of most of the coronary artery segments in patients with higher total cholesterol, and LDL-C levels were found to be significantly increased relative to normocholesterolemic patients. This difference was not observed between patients with normal and increased HDL-C levels. In patients with higher triglyceride levels, only calcium score of the midsegment of LAD was found to be significantly higher than that of normotrigyceridemic patients.

Our study demonstrates unfavourable effects of calcium accumulation on lipid metabolism, emphasizes the importance of dyslipidemic metabolic states, and encourages conduction of further studies to that end.

In our study, a statistically significant increase was observed in the calcification of coronary arteries as the patient aged. This finding is in parallel with the results of previous studies. In a study performed by Mautner et al [12], calcium accumulation was detected in 93 % of the severely (> 75) , and 14 % of the mildly (< 25 %) stenotic luminal lesions. Demonstration of a significant (> 50%) coronary artery stenosis in coronary angiography is related to the presence of calcium accumulation as detected in EBT (electron beam tomography), however degree of luminal stenosis does not directly correlate with total calcium scores. In a study morphologically assessed decrease in the luminal diameter of 723 coronary artery segments was compared with the extent of calcium accumulation .[13] A weak correlation was detected between coronary artery calcification, and the severity of coronary artery narrowing, and the aıthors stated that the degree of calcification could not be used in the prediction or estimation of angiographic stenosis. This phenomenon was explained by positive remodeling which indicates an increase in coronary artery diameter so as to ensure luminal patency despite increased plaque load.[14,15] Noncalcified plaques usually lead to less than 50 % (typically < 20 %) decrease in luminal diameters.[13] In the light of these data, inability to detect coronary calcification indicates very low likelihood of occlusive coronary artery disease.

Calcium scoring takes calcium build-up in all coronary arteries into account. However it is known that each coronary artery has a unique calcium accumulation pattern. Some studies have suggested that individual assessment of each coronary artery plaque would increase the degree of sensitivity of calcium scoring.[16] We think that during the evaluation of total risk, individual assessment of calcium scoring of each coronary artery will decrease the impact of clinical bias during decision-making process based on these results. This logic has led us to consider coronary arteries as certain segments, and calculate calcium scores of each segment *per se*. Besides, we grouped segments as proximal, mid-, and distal segments, and compared total calcium scores of these groups among themselves. As a final step of this evaluation, segmental distribution of calcium accumulation in coronary arteries, and whether or not this distribution was affected by the basic clinical characteristics of the patients were investigated. Segmental evaluation of calcium distribution in coronary arteries demonstrated highest calcium build-up in proximal segments. Among proximal segments, calcium salts accumulated mostly in the proximal segment of LAD.

In our study current smokers, and quitters were evaluated as smokers. Daily cigarette consumption, and number of pack-years of cigarette smoking will have different effects on cardiovascular risk. However, assessment of smoking as the only risk factor *per se* requires these details, and as a prerequisite distribution of other risk factors in the studied group should be homogeneous. The results of the study performed by Schmermund et al, [17] which demonstrated distribution of calcium accumulation in coronary arteries using EBT generally resembles our study results. In both studies, among coronary artery segments, calcium accumulation was mostly observed in LAD, followed by RCA, and Cx. Schmermund et al. did not investigate the impact of demographic data on calcium scores, but only analyzed the distribution of calcium scores in coronary arteries. In some of the previous studies, atheromatous plaques were more frequently demonstrated in the proximal segments.[18] The results of our study also reinforce the findings of the cited studies.

Calcium more frequently accumulates in the proximal segments of coronary arteries. Calcification in distal segments is rarely seen without concomitant calcification of proximal segments.[19-21]

Up to date, many studies have defined CAD as luminal stenosis of > 50 %, and compared EMT with coronary angiography.[9,22] These studies have determined total sensitivity, and specificity of EBT in the detection of occlusive coronary artery disease as 95, and 43 %, respectively. Lower rates of specificity of EBT might be explained by its demonstration of coronary artery calcification, and the presence of atherosclerotic plaques which do not cause hemodynamically significant degree of stenosis.

Budoff et al.[23] observed higher rates of specificity with an increase in the number of calcified coronary arteries. Calcium scores higher than 100 HU have been suggested as highly sensitive, specific, and robust predictive factors for occlusive coronary artery disease .[24,25]

Conclusion

Based on the data obtained from our study, we can say that calcium accumulation in coronary arteries is more frequently seen in the proximal segments, mainly in the proximal segments of LAD. Our study has demonstrated that coronary artery calcification is adversely effected by the presence of risk factors as hypertension, hyperlipidemia, and advanced age.

Controversy still exists about higher vulnerability of calcified or soft plaques to rupture. *In vitro* studies have demonstrated that calcification of plaques weakens their durability.[11] Calcium load as detected by EBT is accepted as an indicator of the presence of coronary atheromatous disease, and the severity of atherosclerosis.[12]

Larger scale prospective studies are needed which would analyse segmental distribution of coronary artery calcification, and demographic factors which might effect this distribution

Conflict of interest: None declared

REFERENCES

1. Warburton RK, Tampas JP, Soule AB, Taylor HC 3rd. Coronary artery calcification: Its relationship to coronary artery stenosis and myocardial infarction. Radiology 1968;91:109-15.

2. Fayad ZA, Fuster V. Clinical imaging of the high-risk or vulnerable atherosclerotic plaque. Circ Res 2001;89:305-16.

3. Achenbach S, Ropers D, Pohle K, Leber A, Thilo C, Knez A, et al. Influence of lipid-lowering therapy on the progression of coronary artery calcification: a prospective evaluation. Circulation 2002;106:1077-82.

4. Rumberger JA, Simons DB, Fitzpatrick LA, Sheedy PF, Schwartz RS. Coronary artery calcium area by electron-beam computed tomography and coronary atherosclerotic plaque area. A histopathologic correlative study. Circulation 1995;92:2157-62.

5. Arad Y, Spadaro LA, Goodman K, Newstein D, Guerci AD. Prediction of coronary events with electron beam computed tomography. J Am Coll Cardiol 2000;36:1253-60.

6. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2008;31(Supp. 1):55-60.

7. Fedder DO, Koro CE, L'Italien GJ. New National Cholesterol Education Program III guidelines for primary prevention lipid-lowering drug therapy: projected impact on the size, sex, and age distribution of the treatment-eligible population. Circulation 2002;105:152-6.

8. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003;42:1206-52.

9. Agatston AS, Janowitz WR, Hildner FJ, Zusmer NR, Viamonte M Jr, Detrano R. Quantification of coronary artery calcium using ultrafast computed tomography. J Am Coll Cardiol 1990;15:827-32.

10. Detrano R, Guerci AD, Carr JJ, Bild DE, Burke G, Folsom AR, et al. coronary Coronary calcium as a predictor of events in four racial or ethnic groups. N Engl J Med 2008;358:1336-45.

11. Raggi P, Cooil B, Shaw LJ, Aboulhson J, Takasu J, Budoff M, et al. Progression of coronary calcium on serial electron beam tomographic scanning is greater in patients with future myocardial infarction. Am J Cardiol 2003;92:827-9.

12. Mautner GC, Mautner SL, Froehlich J, Feuerstein IM, Proschan MA, Roberts WC, et al. Coronary artery calcification: assessment with electron beam CT and histomorphometric correlation. Radiology 1994;192:619-23.

13. Sangiorgi G, Rumberger JA, Severson A, Edwards WD, Gregoire J, Fitzpatrick LA, et al. Arterial calcification and not lumen stenosis is highly correlated with atherosclerotic plaque burden in humans: a histologic study of 723 coronary artery segments using nondecalcifying methodology. J Am Coll Cardiol 1998;31:126-33.

14. Glagov S, Weisenberg E, Zarins CK, Stankunavicius R, Kolettis GJ. Compensatory enlargement of human atherosclerotic coronary arteries. N Engl J Med 1987;316:1371-5.

15. Clarkson TB, Prichard RW, Morgan TM, Petrick GS, Klein KP. Remodeling of coronary arteries in human and nonhuman primates. JAMA 1994;271:289-94.

16. Moselewski F, O'Donnell CJ, Achenbach S, Ferencik M, Massaro J, Nguyen A, et al. Calcium concentration of individual coronary calcified plaques as measured by multidetector row computed tomography. Circulation 2005;111:3236-41.

17. Schmermund A, Möhlenkamp S, Baumgart D, Kriener P, Pump H, Grönemeyer D, et al. Usefulness of topography of coronary calcium by electron-beam computed tomography in predicting the natural history of coronary atherosclerosis. Am J Cardiol 2000;86:127-32.

18. Gibson CM, Kirtane AJ, Murphy SA, Karha J, Cannon CP, Giugliano RP, et al. Distance from the coronary ostium to the culprit lesion in acute ST-elevation myocardial infarction and its implications regarding the potential prevention of proximal plaque rupture. J Thromb Thrombolysis 2003;15:189-96.

19. Beadenkopf WG, Daoud AS, Love BM. Calcification in the coronary arteries and its relationship to arteriosclerosis and myocardial infarction. Am J Roentgenol Radium Ther Nucl Med 1964;92:865-71.

20. Rifkin RD, Parisi AF, Folland E. Coronary calcification in the diagnosis of coronary artery disease. Am J Cardiol 1979;44:141-7.

21. Ambrose JA, Tannenbaum MA, Alexopoulos D, Hjemdahl-Monsen CE, Leavy J, Weiss M, et al. Angiographic progression of coronary artery disease and the development of myocardial infarction. J Am Coll Cardiol 1988;12:56-62.

22. Kennedy J, Shavelle R, Wang S, Budoff M, Detrano RC. Coronary calcium and standard risk factors in symptomatic patients referred for coronary angiography. Am Heart J 1998;135:696-702.

23. Budoff MJ, Georgiou D, Brody A, Agatston AS, Kennedy J, Wolfkiel C, et al. Ultrafast computed tomography as a diagnostic modality in the detection of coronary artery disease: a multicenter study. Circulation 1996;93:898-904.

24. Rumberger JA, Sheedy PF, Breen JF, Schwartz RS. Electron beam computed tomographic coronary calcium score cutpoints and severity of associated angiographic lumen stenosis. J Am Coll Cardiol 1997;29:1542-8.

25. Guerci AD, Spadaro LA, Goodman KJ, Lledo-Perez A, Newstein D, Lerner G, et al. Comparison of electron beam computed tomography scanning and conventional risk factor assessment for the prediction of angiographic coronary artery disease. J Am Coll Cardiol 1998;32:673-9.

Anahtar sözcükler: Ateroskleroz; kalsinoz; kalsiyum/analiz; koroner anjiyografi; koroner arter hastalığı.

Key words: Atherosclerosis; calcinosis; calcium/analysis; coronary angiography; coronary artery disease.