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Isolated Coronary Ectasia is Associated with Impairment of Left Ventricular Myocardial Performance and Aortic Elastic Properties

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Abstract

Aim: To investigate ventricular functions and tissue Doppler echocardiography-derived myocardial performance index and the elastic characteristics of the aorta in patients with coronary artery ectasia.

Material and methods: 42 patients with coronary artery ectasia ; mean age 56.9 ± 9.1 years and 26 subjects with angiographically normal coronaries; mean age 54.1 ± 6.9 years, were included in the study. All the subjects underwent echocardiography and tissue Doppler imaging to determine left ventricular diastolic functions, myocardial performance index, and elastic characteristics of the aorta

Results: The LV diastolic functions and myocardial permanence were impaired in the ectasia group as compared with patients with normal coronary arteries. The values obtained for aortic elasticity, as aortic strain, β index and aortic distensibility were lower in the ectasia group when compared with the values of the normal group

Conclusion: Left ventricular myocardial performance index, and elasticity of the aorta were deteriorated in patients with isolated coronary ectasia.

Keywords: Myocardial performance index: Coronary artery ectasia: Aortic elasticity

Introduction

Isolated coronary artery ectasia (CAE) is localized or diffuse dilation of epicardial coronary arteries, 1.5 times the diameter of the adjacent normal coronary segment without any specific symptoms [1-3]. It is either congenital or acquired and its incidence is reported to be between 0.3 - 10% in different studies [1,4,5]. Atherosclerosis, congenital causes, inflammatory or connective tissue disorders are among the probable etiologic factors; however, exact pathophysiologic mechanism remains unclear despite some molecular, cellular and vascular mechanisms defined in various studies [5,6]. In some studies, CAE was shown to be a generalized disease affecting other vascular beds [2]. The essential histopathological finding in the diagnosis of CE is the replacement of coronary artery media layer smooth muscle cells with hyalinized collagen, as a consequence of the increased degeneration of the media layer [7]. As a result, the loss of musculo-elastic components is observed in the media [8]. Thus, progressive artery dilatation occurs. Moreover, along with this disease, the presence of extracardiac artery dilatation has been reported in the previous studies [9,10]. Arterial stiffness, which is defined as the arterial rigidity caused by the loss of elastic tissue in the artery wall, decreases the widening capacity of the artery. Arterial rigidity develops and arterial widening capacity is deteriorated. It has been established that as the stiffness of the large arteries such as aorta increases, cardiovascular mortality and morbidity also increase [11]. Consequently, aortic stiffness has recently been regarded as a risk factor that needs to be treated [12]. It has also been established that aortic stiffness is increased in individuals with coronary artery disease (CAD) and in those with atherosclerosis [13]. Although myocardial ischemia and left ventricular dysfunction have been found in patients with CAE who have no narrowness or obstruction in the coronary arteries, the ventricular function of these patients has not been well studied [14,15]. The left ventricular functions and diastolic parameters of a small set of patients with CAE and no obstructive coronary artery disease have been evaluated by conventional and tissue Doppler echocardiography (TDE) and compared to controls [16]. Equally important, right ventricular function, which is a prognostic indicator for most heart diseases [17,18], has not been studied in patients with CAE.

Aim: The aim of the study to determine both ventricular functions and TDE-derived MPI in patients with CAE, and to investigate the elastic properties of the aorta and the relationship between these parameters and LV diastolic functions.

Patients and Methods

Patients with isolated coronary ectasia, detected during coronary angiograms in El Minia university hospital between January 2011 and June 2015, were included in the study. 42 patients with isolated CAE and 26 control subjects with normal coronary arteries (NCA) were evaluated. The indication for coronary angiography was either the presence of typical angina or positive or equivocal results of stress test. All of the patients were questioned for their cardiovascular risk factors and the drugs used. Routine biochemical and hematologic laboratory tests were done.

Exclusion criteria

Previous history of myocardial infarction, percutaneous coronary intervention, cardiomyopathies, congenital heart diseases, cardiac valve diseases, ventricular hypertrophy, branch blocks, chronic obstructive lung disease and/or cor pulmonale, active infection, renal failure, neoplastic disease, antioxidant drug usage and alcohol abuse.

Coronary angiography

Coronary angiography was performed to all the patients by the General Electric (GE) Innova (USA), with the standard Judkins technique. Two cardiologists evaluated the coronary angiographies without knowing the clinical or routine bio-

Chemistry results of the patients. When there was no identifiable adjacent normal segment, the mean diameter of the corresponding coronary segment in the control group served as the normal value.

Echocardiography

Two-dimensional pulsed-wave Doppler and TDE were performed for all patients using a 2.5 -MHz transducer (GE vivid 3, USA) in the left decubitus position during normal respiration according to the recommendations of American Society of Echocardiograpgy [19]. The diameters of the left ventricular and the thicknesses of diastolic walls were measured from the parasternal window with two-dimensional M-mode echocardiography. Left ventricular ejection fraction was calculated using the modified Simpson's method [20].

From the apical four-chamber view, Doppler recordings were obtained with the pulsed sample volume placed at the tip of the mitral leaflets. The peak early (E) and late (A) velocities, E-wave deceleration time (DT) and isovolumetric relaxation time (IVRT) were measured.

Pulsed-wave TDE parameters were measured by an echocardiographic device with active TDE functions. A 3.5 mm sample volume was used. The TDE cursor was placed from the apical 4-chamber view on the mitral annulus opposite the septal and lateral walls. Peak systolic velocity (Sm), peak early (Em) and late (Am) diastolic velocities for each segment were measured and the Em/Am ratio was calculated. The isovolumetric relaxation time (IRT) was measured from the end of Sm to the beginning of Em, the isovolumetric contraction time (ICT) was measured from the end of Am to the beginning of Sm and the time period of Sm was measured as the ejection time (ET). The MPI was calculated using the equation (ICT+IRT)/ET. The average of the Sm, Em and Am time intervals obtained from the mitral annulus-lateral and interventricular septum was used to calculate left ventricular mean MPI M-mode echocardiography. The aortic diameter was recorded at 3 cm above the aortic valve [21]. Internal aortic diameters were measured by means of a caliper in systole and diastole as the distance between the trailing edge of the anterior aortic wall and the leading edge of the posterior aortic wall. Aortic systolic (AoS) diameter was measured at the time of full opening of the aortic valve and diastolic (AoD) diameter was measured at the peak of QRS. Ten consecutive beats were measured routinely and averaged. The AoS and AoD indexes for each participant were calculated by dividing the AoS and AoD by the body surface area. The percentage change of the aortic root was calculated as:

$$\%Ao = 100 \times (AoS - AoD)/AoD$$

Aortic elasticity was assessed using the following indexes [22]:

$$\text{Aortic strain } (\%) = 100 \times (AoS - AoD)/AoD$$

$$\text{Aortic dispensability index (cm/ dyn 10)} = 2 \times \text{aortic strain} \times (SBP-DBP)$$

$$\text{Aortic stiffens index beta} = \text{In } (SBP/DBP)/\text{aortic strain.}$$

Statistical analysis

SPSS version 21 was used for the statistical analysis. All the data were expressed as mean ± standard deviation. Categorical variables were compared via chi-square test. Normally distributed variables were compared across groups by means of Student's t test, whereas variables that were not normally distributed were compared by Mann-Whitney U test. Pearson's correlation analysis was used to evaluate relations between the variables. A P value of < 0.05 was considered significant.

Results

The clinical characteristics of patients with CAE and normal coronary angiography individuals are presented in (Table 1). Age, sex, body mass index, diabetes mellitus, dyslipidemia, family history, and smoking status did not differ between the CAE patients and the control group. Also, there were no significant differences as regards laboratory data (Table 1).

Variables	Patients	Control	P
Age (years)	56. 9 ± 9.1	54.1 ± 6.9	0.38
Sex (male/female)	25/20	8-Dec	0.33
Body mass index (kg/m)	26.1 ± 1.5	25.5 ± 1.5	0.13
Family history	5	3	0.45
Smoking	15	7	0.36
Diabetes Mellitus	14	6	0.26
Dislipidemia	19	7	0.35
Systolic blood pressure	136.2 ± 7	136.8 ± 10	0.8
Diastolic blood pressure	78 ± 9	83 ± 7	0.17
Glucose (mg/dl)	91.5 ± 7	89 ± 6	0.34
Serum creatinine (mg/dl)	0.99 ± 0.08	0.99 ± 0.1	0.88
Hemoglobin (g/dl)	13.1 ± 0.5	12.9 ± 0.4	0.31
Total cholesterol (mg/dl)	175.4 ± 25.6	172.4 ± 24,3	0.63
Triglycerides (mg/dl)	164.8 ± 24	173.6 ± 19	0.12
High density lipoprotein (mg/dl)	31.4 ± 5.6	30 ± 2.3	0.55
Low density lipoprotein (mg/dl)	148 ± 36	140 ± 27	0.33

Table1: Demographic and clinical data of both groups.

As regards conventional echocardiography parameters, there was no significant difference of LA, LVEDD, LVESD, PW, IVS, EF, and A velocity between the two groups. But the mitral E velocity was higher in the control group than the patients (88.6 ± 4.9, 71.2 ± 6.3) ($p = 0.01$), and E/A ratio was also higher in the control than the patients (1.2 ± 0.2, 1 ± 0.11) ($p = 0.01$).

Aortic strain, aortic distensibility and index values, which are the elasticity parameters of the aorta, were found to be lower in the CE (7.1 ± 2, 3.1 ± 0.1, 1.4 ± 0.1 respectively), as compared with the values of the control group (12.8 ± 1.2, 6.05 ± 0.6, 2.01 ± 0.03 respectively), (p = 0.01) (Table 2).

Variables	Patients	Controls	P
LA diameter (mm)	33.6 ± 1.7	33.6 ± 1.9	0.87
LVEDD (mm)	47.5 ± 8.6	45 ± 4.9	0.17
LVESD (mm)	29.8 ± 2.1	31.3 ± 2.2	0.7
IVS (mm)	9.1 ± 0.4	9.2 ± 0.5	0.77
PW (mm)	8.6 ± 0.7	8.6 ± 0.4	0.75
EF (%)	58.5 ± 7.8	61.6 ± 5.2	0.44
E, cm/s	71.2 ± 6.3	88.6 ± 4.9	0.01
A, cm/s	68.9 ± 5.7	68.5 ± 4.7	0.78
E/A	1 ± 0.11	1.2 ± 0.2	0.01
Aortic systolic diameter	3.06 ± 0.5	2.85 ± 0.8	0.01
Aorta diastolic diameter	2.8 ± 0.09	2.6 ± 0.5	0.01
Aortic Strain	7.1 ± .2	12.8 ± 1.2	0.01
B Index	1.4 ± .1	2.01 ± .03	0.01
Aortic distensibility	3.1 ± 0.1	6.05 ± 0.6	0.01

LA: Left Atrium LVEDD: Left Ventricle End Diastolic Diameter, LVESD: Left Ventricle End Systolic Diameter, IVS: Interventriculer Septum, EF: Ejection Fraction, E: Mitral E velocity, A: Mitral A velocity.

Table 2: Echocardiographic parameters of both groups.

The TDE parameters obtained from the left ventricle were given in (Table 3). There was no significant difference between the two groups as regards Sm, Am, while there was significant difference between the two groups as regards Em, Em/Am, IRT, ICT, and ET. The patients (7.3 ± 1.6, 0.87 ± 0.1, 99.6 ± 10.4, 79 ± 21, 264 ± 25 respectively) the controls (13.1 ± 2.2, 1 ± 0.1, 88 ± 10, 65 ± 15, 285 ± 24 respectively), and Left ventricular MPI was significantly higher in the CAE group as compared with the control (0.64 ± 0.05, Vs 0.50 ± 0.05) (p = 0,01).

Variables	Patients	Controls	P
Sm, cm/s	7.9 ± 0.43	7.9 ± 0.44	0.93
Em, cm/s	7.3 ± 1.6	13.1 ± 2.2	0.01
Am, cm/s	10.8 ± 0.85	10.90.39	0.61
Em/Am	0.87 ± 0.1	1 ± 0.1	0.01
IRT, ms	99.6 ± 10.4	88 ± 10	0.01
ICT, ms	79 ± 21	65 ± 15	0.01
ET, ms	264 ± 25	285 ± 24	0.01
MPI	0.64 ± 0.05	0.50 ± 0.05	0.01

E: Mitral E velocity, A : Mitral A velocity, IRT: isovolumetric relaxation time; , ICT: isovolumetric contraction time, ET: ejection time, MPI: myocardial performance index

Table 3: Tissue Doppler parameters of the Left ventricle of both groups.

Table 4 showed the properties of the ectatic vessels of the CE patients. One vessel, 2 vessel and 3 vessel ectasia were found to be present in 7 (16%), 13 (31%) and 22 (52%) patients respectively. The stepwise linear multivariable analyses showed that aortic elasticity parameters had the strongest diagnostic power for detection of the abnormalities in the LV diastolic function. Among these parameters, the aortic strain had been found to possess the strongest diagnostic power for E/A rate, and IVRT (r=0.54; p=0.01, and r=0.42; p=0.01, respectively).

Average diameter of ectasia (mm)	5.44 ± 0.66
Number of ectasia segments	3.5 ± 1.2
Distribution of ectasic segments	
1 segment	5/42 (11.9%)
2 segment	9/42 (21.4%)
3 segment	12/42 (28.5%)
4 segment	7/42 (16.6%)
5 segment	6/42 (14.2%)
6 segment	3/42 (7%)
Distribution of coronary artery ectasia	
1 vessele ectasia	7/42 (16.6%)
2 vessele ectasia	13/42 (30.9%)

3 vessele ectasia	22/42 (52.3%)

Table 4: Ectatic properties of the patients.

Discussion

Coronary artery ectasia is the dilatation of epicardial coronary arteries more than 1.5 times and its basic pathophysiologic mechanisms are destruction of elastic layers of arterial tunica media and deposition of collagen and elastin which resulting in thinning of the arterial wall [23]. Coronary atherosclerosis is detected in more than 50% of the patients; however, connective tissue disorders and vasculitis can also be seen during pathologic examination [24]. Yetkin et al. [25] reported that carotid intima media thickness was thinner in CAE patients with stenotic CAD when compared to patients with CAD only and stated that ectasia was not an atherosclerotic process limited to coronary arteries. In some previous studies, peripheral artery disease, aortic aneurism, varicose dilatations of lower extremity veins, basilar artery aneurisms and varicoceles were shown to be increased in isolated CAE patients [26-28]. Papadakis et al. [29] found the coronary flow velocity measured using the TIMI frame count method to be lower in patients with CAE than both the obstructive CAD and control groups. The TIMI frame count method is a simple technique used to evaluate the quantitative index of coronary blood flow [30]. MPI of the CAE patients was significantly higher than the control group. This result consistent with the study of Gulec et al. [31]. Diastolic disorders in ischemic heart diseases are seen earlier than systolic dysfunctions [32]. In coronary artery disease a prolonged MPI has been shown to be an important precursor of the disease before the development of systolic dysfunction [33]. MPI is a simple, reproducible, and noninvasive method to assess systolic and diastolic functions [34,35]. Also, MPI is related to left ventricular dysfunction and clinical severity of heart failure and is a powerful parameter for the prognostic assessment of these patients [36]. Ozdemir et al. found that increased Doppler-derived MPI was related to mortality in a variety of cardiac diseases [37]. And also his study had shown that while Doppler-derived MPI was affected by preload and heart rate, TDE-derived MPI was not affected by either [37]. In this study, left ventricular MPI was significantly higher in CAE patients than the control group.

Arterial stiffness is known as the arterial rigidity that develops because of the loss of elastic tissue in the arterial wall, resulting in the loss of widening capacity of the artery. It has been reported that, cardiovascular mortality and morbidity rates increase with the increase of the stiffness in the large arteries. Therefore, aortic stiffness has recently been regarded as a risk factor which needs to be treated [38]. Some structural changes occur in the myocardium when the end-systolic stress increases. Thus, systolic and [38] diastolic stiffness develops in the myocardium. However the systolic function is preserved, while the diastolic function is impaired at the first stage of these compensatory [39] changes. Systolic function is impaired in the later stages. Besides the ventricular geometry, aortic functions are also [40], presumed to be responsible for the end systolic stress. Increased stiffness can be a potential factor for wall stress. When afterload is increased, elevated intraventricular pressure has to be generated first to open the aortic valve, and then during the ejection phase these increases in afterload and intraventricular pressure lead to an increase in myocardial wall stress. In animal models, loss of aortic distensibility directly affects the mechanical performance of the left ventricle, with increases noted in LV systolic pressure and [41] wall tension.

Conclusion

Aortic stiffness increased in patients with CE. The increase in aortic stiffness might be responsible from LV diastolic dysfunction. These results explain that, CE is a generalized vascular disorder rather than a microvascular disease; more studies are needed to explain these changes.

References

1. Swaye PS, Fisher LD, Litwin P, Vignola PA, Judkins MP, et al. (1983) Aneurysmal coronary artery disease. See comment in PubMed Commons below Circulation 67: 134-138.
2. Yetkin E, Waltenberger J (2007) Novel insights into an old controversy: is coronary artery ectasia a variant of coronary atherosclerosis? See comment in PubMed Commons below Clin Res Cardiol 96: 331-339.
3. Markis JE, Joffe CD, Cohn PF, Feen DJ, Herman MV, et al. (1976) Clinical significance of coronary arterial ectasia. See comment in PubMed Commons below Am J Cardiol 37: 217-222.
4. Hartnell GG Parnell BM, Pridie RB (1985) Coronary artery ectasia. Its prevalence and clinical significance in 4993 patients. See comment in PubMed Commons below Br Heart J 54: 392-395.
5. Befeler B, Aranda MJ, Embi A, Mullin FL, El-Sherif N, et al. (1977) Coronary artery aneurysms: study of the etiology, clinical course and effect on left ventricular function and prognosis. See comment in PubMed Commons below Am J Med 62: 597-607.
6. Chrissoheris MP, Donohue TJ, Young RS, Ghantous A (2008) Coronary artery aneurysms. See comment in PubMed Commons below Cardiol Rev 16: 116-123.
7. Rath S, Har-Zahav Y, Battler A, Agranat O, Rotstein Z, et al. (1985) Fate of nonobstructive aneurysmatic coronary artery disease: angiographic and clinical follow-up report. See comment in PubMed Commons below Am Heart J 109: 785-791.
8. Befeler B, Aranda MJ, Embi A, Mullin FL, El-Sherif N, et al. (1977) Coronary artery aneurysms: study of the etiology, clinical course and effect on left ventricular function and prognosis. See comment in PubMed Commons below Am J Med 62: 597-607.
9. Daoud AS, Pankin D, Tulgan h, Florentin RA (1963) Aneurysms of the coronary artery. Report of ten cases and review of literature. See comment in PubMed Commons below Am J Cardiol 11: 228-237.
10. Stajduhar KC, Laird JR, Rogan KM, Wortham DC (1993) Coronary artery ectasia, increased prevelance in patients with abdominal aneurysm as compared to occlusive atherosclorotic peripheral vascular disease. Am Heart J 125: 86-92.
11. Shokawa T, Imazu M, Yamamoto H, Toyofuku M, Tasaki N, et al. (2005) Pulse wave velocity predicts cardiovascular mortality: findings from the Hawaii-Los Angeles-Hiroshima study. See comment in PubMed Commons below Circ J 69: 259-264.
12. Hodes RJ, Lakatta EG, McNeil CT (1995) Another modifiable risk factor for cardiovascular disease? Some evidence points to arterial stiffness. See comment in PubMed Commons below J Am Geriatr Soc 43: 581-582.
13. Park SM, Seo HS, Lim HE, Shin SH, Park CG, et al. (2005) Assessment of arterial stiffness index as a clinical parameter for atherosclerotic coronary artery disease. See comment in PubMed Commons below Circ J 69: 1218-1222.
14. al-Harthi SS, Nouh MS, Arafa M, al-Nozha M (1991) Aneurysmal dilatation of the coronary arteries: diagnostic patterns and clinical significance. See comment in PubMed Commons below Int J Cardiol 30: 191-194.
15. Krüger D, Stierle U, Herrmann G, Simon R, Sheikhzadeh A (1999) Exercise-induced myocardial ischemia in isolated coronary artery

ectasias and aneurysms ('dilated coronopthy'). J Am Coll Cardiol 34: 1461-1470.

16. Saglam M, Barutcu I, Karakaya O, Esen AM, Akgun T, et al. (2008) Assessment of left ventricular functions in patients with isolated coronary artery ectasia by conventional and tissue Doppler imaging. See comment in PubMed Commons below Angiology 59: 306-311.
17. Polak JF, Holman BL, Wynne J, Colucci WS (1983) Right ventricular ejection fraction: an indicator of increased mortality in patients with congestive heart failure associated with coronary artery disease. J Am Coll Cardiol 2: 217-224.
18. Di Salvo TG, Mathier M, Semigran MJ, Dec GW (1995) Preserved right ventricular ejection fraction predicts exercise capacity and survival in advanced heart failure. See comment in PubMed Commons below J Am Coll Cardiol 25: 1143-1153.
19. Pearlman AS, Gardin JM, Martin RP, Parisi AF, Popp RL, et al. (1987) Guidelines for optimal physician training in echocardiography. Recommendations of the American Society of Echocardiography Committee for Physician Training in Echocardiography. See comment in PubMed Commons below Am J Cardiol 60: 158-163.
20. Schiller NB, Shah PM, Crawford M, De Maria A, Devereux R, et al. (1989) Recommendations for quantitation of the left ventricle by two-dimensional echocadiography. American Society of Echocardiography Committee on Standards, Sub-committee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 2: 358-367.
21. Stefanadis C, Stratos C, Boudoulas H, Kourouklis C, Toutouzas P (1990) Distensibility of the ascending aorta: comparison of invasive and non-invasive techniques in healthy men and in men with coronary artery disease. See comment in PubMed Commons below Eur Heart J 11: 990-996.
22. Lacombe F, Dart A, Dewar E, Jennings G, Cameron J, et al. (1992) Arterial elastic properties in man: a comparison of echo-Doppler indices of aortic stiffness. See comment in PubMed Commons below Eur Heart J 13: 1040-1045.
23. Isner JM, Donaldson RF, Fortin AH, Tischler A, Clarke RH (1986) Attenuation of the media of coronary arteries in advanced atherosclerosis. See comment in PubMed Commons below Am J Cardiol 58: 937-939.
24. Demopoulos VP, Olympios CD, Fakiolas CN, Pissimissis EG, Economides NM, et al. (1997) The natural history of aneurysmal coronary artery disease. See comment in PubMed Commons below Heart 78: 136-141.
25. Yetkin E, Acikgoz N, Aksoy Y, Bariskaner E, Sivri N, et al. (2005) Decreased, carotid intima-media thickness in patients with coronary artery ectasia compared with patients with coronary artery disease. Coron Artery Dis 16: 495-498.
26. Yetkin E, Kilic S, Acikgoz N, Ergin H, Aksoy Y, et al. (2005) Increased prevalence of varicocele in patients with coronary artery ectasia. See comment in PubMed Commons below Coron Artery Dis 16: 261-264.
27. Kahraman H, Ozaydin M, Varol E, Aslan SM, Dogan A, et al. (2006) The diameters of the aorta and its major branches in patients with isolated coronary artery ectasia. See comment in PubMed Commons below Tex Heart Inst J 33: 463-468.
28. Papadakis MC, Leontiadis E, Manginas A, Voudris V, Pavlides G, et al. (2004) Frequency of coronary artery ectasia in patients undergoing surgery for ascending aortic aneurysms. See comment in PubMed Commons below Am J Cardiol 94: 1433-1435.
29. Papadakis MC, Manginas A, Cotileas P, Demopoulos V, Voudris V, et al. (2001) Documentation of slow coronary flow by the TIMI frame count in patients with coronary ectasia. See comment in PubMed Commons below Am J Cardiol 88: 1030-1032.
30. Gibson CM, Cannon CP, Daley WL, Dodge JT Jr, Alexander B Jr, et al. (1996) TIMI frame count: a quantitative method of assessing coronary artery flow. See comment in PubMed Commons below Circulation 93: 879-888.
31. Gulec S, Atmaca Y, Kilickap M, Akyürek O, Aras O, et al. (2003) Angiographic assessment of myocardial perfusion in patients with isolated coronary artery ectasia. See comment in PubMed Commons below Am J Cardiol 91: 996-999.
32. García-Fernández MA, Azevedo J, Moreno M, Bermejo J, Pérez-Castellano N, et al. (1999) Regional diastolic function in ischaemic heart disease using pulsed wave Doppler tissue imaging. See comment in PubMed Commons below Eur Heart J 20: 496-505.
33. Kang SM, Ha JW, Rim SJ, Chung N (1998) Index of myocardial performance using Doppler-derived parameters in the evaluation of left ventricular function in patients with essential hypertension. See comment in PubMed Commons below Yonsei Med J 39: 446-452.
34. Tei C, Ling LH, Hodge DO, Bailey KR, Oh JK, et al. (1995) New index of combined systolic and diastolic myocardial performance: a simple and reproducible measure of cardiac function: a study in normal and dilated cardiomyopathy. J Cardiol 26: 357-366.
35. Tavil Y, Kanbay A, Sen N, Ciftçi TU, Abaci A, et al. (2007) Comparison of right ventricular functions by tissue Doppler imaging in patients with obstructive sleep apnea syndrome with or without hypertension. See comment in PubMed Commons below Int J Cardiovasc Imaging 23: 469-477.
36. Vizzardi E, Chiari E, Faggiano P, D'Aloia A, Bordonali T, et al. (2010) Measurement of the myocardial performance index in ambulatory patients with heart failure: correlation with other clinical and echocardiographic parameters and independent prognostic value. See comment in PubMed Commons below Echocardiography 27: 123-129.
37. Ozdemir K, Balci S, Duzenli MA, Can I, Yazici M, et al. (2007) Effect of preload and heart rate on the doppler and tissue doppler-derived myocardial performance index. See comment in PubMed Commons below Clin Cardiol 30: 342-348.
38. Tuzun N, Tanriverdi H, Evrengul H, Kuru DS, Ergene AO (2007) Aortic elastic properties in patients with coronary artery ectasia. See comment in PubMed Commons below Circ J 71: 506-510.
39. Weber KT (1989) Cardiac interstitium in health and disease: the fibrillar collagen network. See comment in PubMed Commons below J Am Coll Cardiol 13: 1637-1652.
40. Tanriverdi H, Evrengul H, Kaftan A, Kara CO, Kuru O, et al. (2006) Effect of obstructive sleep apnea on aortic elastic parameters: relationship to left ventricular mass and function. See comment in PubMed Commons below Circ J 70: 737-743.
41. Kelly RP, Tunin R, Kass DA (1992) Effect of reduced aortic compliance on cardiac efficiency and contractile function of in situ canine left ventricle. See comment in PubMed Commons below Circ Res 71: 490-502.