

Preliminary Results of Assessment of Systolic and Diastolic Function in Patients with Cardiac Syndrome X Using SPECT CT

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Summary: The Cardiac syndrome X includes patients, mainly women, with the triad of angina pectoris, a positive exercise electrocardiogram for myocardial ischemia and angiographically smooth coronary arteries. The most common manifested clinical symptom in those patients is chest pain. In most cases, the syndrome is associated with debilitating symptomatology, increased psychological morbidity and a poor quality of life. All the heterogeneity of the syndrome makes the treatment approaches to those patients unclear and uncertain.

Keywords: Cardiac syndrome X, SPECT CT, Systolic function, Diastolic function

1. INTRODUCTION

The concept of cardiac syndrome X includes patients, mostly women, before or in their period of menopause fulfilling the following three criteria: chest pain, positive exercise-strain test, normal coronary angiogram [1, 2, 10]. The chest pain is a leading symptom of these patients, usually stronger and more prolonged than the typical angina pectoris, which is also more difficult to control by the standard antiischemic therapy [3, 7]. As a result, patients are repeatedly re-hospitalised, new unnecessary angiograms are being done and quality of life of these patients worsens [4].

Some clinical studies related to prognosis of these patients show that their life-expectancy does not differ considerably from the rest of the population, excluding those with rhythm and conductance disorders such as left-bundle branch block who develop dilatated cardiomyopathy more frequently (NLHBI WISE study with mean duration of follow-up- 5.2 years) [5].

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In May 2009 [5] the results from St. James Women Take Heart (WTH) Project were announced. The project gives two main results. The first one is that "...women with symptoms and signs suggestive of ischemia, but without obstructive CAD are at elevated risk for cardiovascular events compared with asymptomatic community-based women". The second one defines that based on the finding from this and other studies, linking endothelial dysfunction and future cardiovascular events, it is recommendable that all women with symptoms suggestive of ischemia undergo initial evaluation for obstructive CAD, and if there is no evidence of obstructive CAD, such women need further assessment for endothelial dysfunction.

All these give enough reasons for further investigation, despite that all cardiac syndrome X keep being diagnostically and therapeutically obscure. Many authors work on finding the **pathogenesis of this syndrome**:

1. myocardial ischemia,
2. endothelial dysfunction,
3. metabolic and hormonal factors,
4. change in pain perception.

Heterogeneous pathogenesis of cardiac syndrome X makes therapeutic approach undefined [2].

Statistical data show that not less than 33% of women exhibiting typical angina chest pain have clear coronary vessels, proved by selective coronary angiogram [5].

The aim of the study is to investigate the changes in the LV kinetics and the perfusion-reversible or not reversible changes in patients with cardiac syndrome X.

2. MATERIAL AND METHODS

A prospective randomized study includes 18 consecutive patients (women) at the mean age 55.29 ± 9.47 who have fulfilled the criteria for cardiac syndrome X, hospitalized in the Clinic of Cardiology, Department of Internal Medicine, Medical University of Sofia, and who are selected from the four cath-Labs in Sofia.

All randomized patients with Cardiac Syndrome X in the study underwent **Myocardial perfusion SPECT CT**. Before performing the study, all patients were carefully made familiar with the protocol of the study.

The protocol of the study includes rest and stress SPECT and after application of nitrates with depot effect.

Radiopharmaceutical: ^{99m}Tc Tetrofosmin. Single day protocol. Activity applied: 250 MBq at exertion and 750 MBq at rest with total activity- 1000 MBq. Records are made at exertion on the 30 min. after the application of the radiopharmaceutical and on the 1 and 2 h at the rest. Just after the first record at rest, patients are given per os 10 mg Isodinit. All records are made in γ -camera SPECT-CT with ECG synchronization. Framing - eight frames for a cardiac cycle.

Stress test: treadmill. Step by step exertion until the patient complains from chest pain or ECG changes in the repolarization. If there are no such complains, the test continues until reaching 90% from the maximum calculated heart rate. The radiopharmaceutical is infused at the maximal exertion.

Interpretation: point of interest were the changes in the perfusion (reversible or not reversible), and the left ventricle (LV) kinetics. Using the quantitative gated SPECT (QGS), the total defect in perfusion was calculated in percentage of the myocardium of the LV. The following parameters were defined: total exertion, rest and differential scores: summed stress score (SSS), summed rest score (SRS), summed difference score (SDS). The following parameters of LV function were calculated at rest and exertion: ejection fraction of the left ventricle (EFLV), end diastolic volume (EDV), end systolic volume (ESV), max velocities of filling and ejection: peak emptying rate (PER), peak filling rate (PFR).

3. RESULTS

Changes in the LV kinetics were found in 3 patients (16.7%): 2 with perfusion defect (1 with no change at rest) and 1 without such defect (Fig. 1).

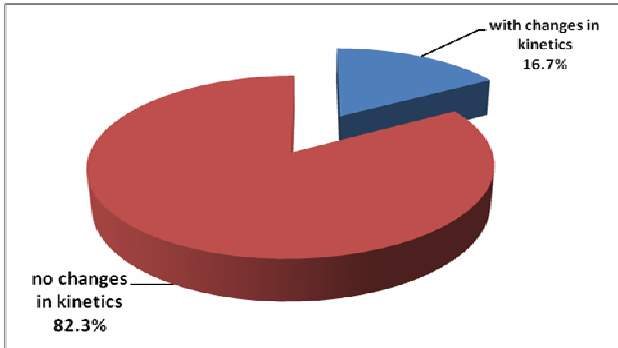


Fig. 1 Patients from the studied group with data from SPECT CT for changes in myocardial kinetics

In 10 (55.6%) patients from the studied group there were different degrees of perfusion abnormalities: SDS max 9, min 2, mean 3.6 ± 2.497 (Fig. 2). From patients with $SDS > 0.6$ (60%) recovered at rest and 2 (20%) normalized after receiving nitrates with depot effect.

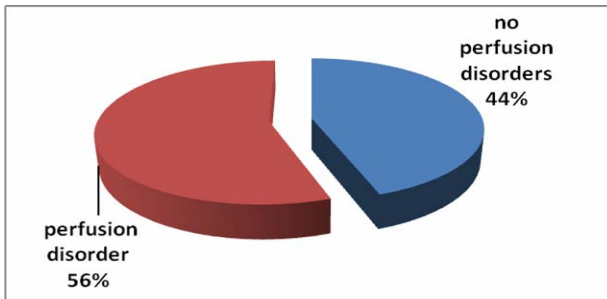


Fig. 2 Distribution of patients according to perfusion disorders

Comparing parameters of systolic function in patients with perfusion defects there was not significant increase of EFLV ($p = 0.235$) (Fig. 3) and no significant change of the ESV ($p = 0.498$) (Fig. 4). There is a preserved reaction of stress of the PER ($p = 0.05$) in those patients but no significant difference of the PFR ($p = 0.674$) as a parameter of diastolic function (Fig. 5).

In patients without perfusion defects, there is a preserved reaction to stress of the EFLV ($p = 0.08$) (Fig. 6) and the parameter of diastolic function PFR ($p = 0.068$) (Fig. 7).

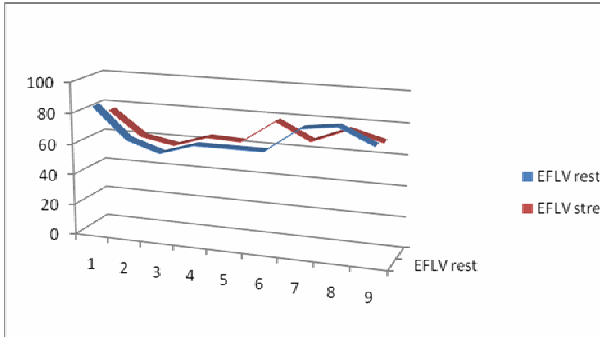


Fig. 3 EFLV at rest and stress in patients with perfusion disorder ($p = 0.235$)

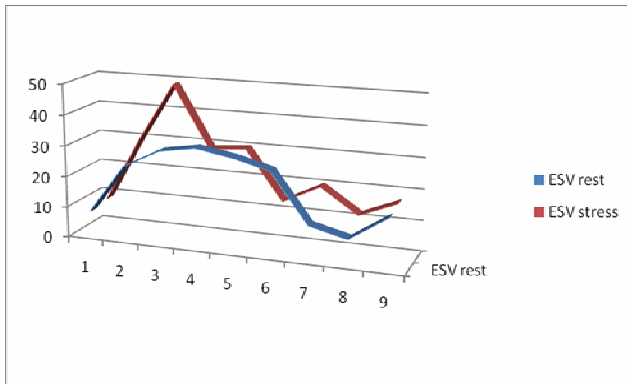


Fig. 4 ESV at rest and stress in patients with perfusion disorder ($p = 0.498$)

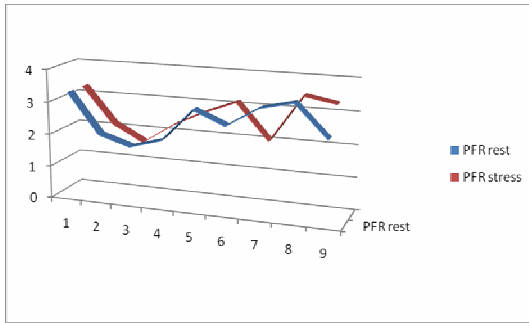


Fig. 5 PFR at rest and stress in patients with perfusion disorder ($p = 0.674$)

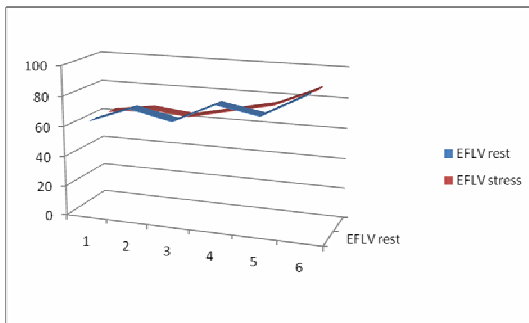


Fig. 6 EFLV at rest and stress in patients without perfusion disorder ($p = 0.08$)

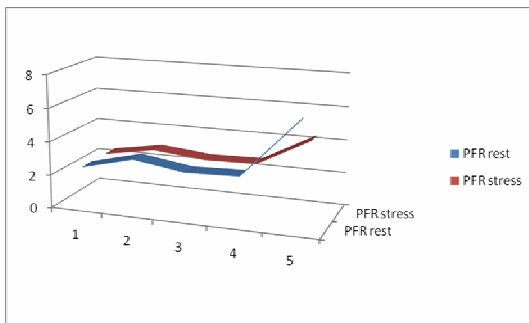


Fig. 7 PFR at rest and stress in patients without perfusion disorder ($p = 0.068$)

4. DISCUSSION

One of the most discussed ideas for the origin of cardiac syndrome X is myocardial ischemia. Publications suggest contradictory opinions about this idea. Cannon et al. proved that patients with disturbed coronary vasodilative reserve had decreased left-ventricle ejection fraction (EF) during states of increased strain compared to those with preserved coronary reserve². They concluded that these patients had signs of myocardial ischemia. Sax et al. investigated systemic vasodilative response by a method based on venous pletismography in order to compare patients to healthy controls¹¹. Their results showed a 21% decrease of vasodilative response in patients with cardiac X syndrome, appearing on the 3rd minute. The authors also found significantly increased vessel resistance in this group of patients. Supported by these results, Cannon concludes that X-syndrome is based on small coronary vessel dysfunction, i.e. “microvascular angina” [2, 3].

5. CONCLUSION

The results of the current study demonstrate that in patients with criteria for cardiac syndrome X there are more marked changes in the myocardial perfusion, which usually are not connected with changes in the kinetics. In those patients, there is no adequate increase of EFLV, which is probably related to the disturbed diastolic function assessed by PFR. These results correlates with previous studies that have demonstrated the presence of myocardial fibrosis in these patients.

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