Editorial Comment 621

Fragmented QRS frequency in patients with cardiac syndrome X

The term "cardiac syndrome X" (CSX) was introduced more than 40 year ago by Kemp (1) who described a group of patients with angina-like chest pains and normal coronary artery angiograms. Since then, the mosaic of knowledge has gradually built up; however, the letter "X" in this term characterizes the fact that evidence and knowledge on this syndrome are still limited and controversial (2).

CSX is seen more frequently in women and is defined as a combination of the following (3, 4):

- angina-like chest pains,
- ST segment depression during angina/exercise test,
- · normal coronary arteries at angiography,
- absence of known comorbidities associated with microvascular dysfunction.

Initially, this syndrome was assumed to have a benign prognosis. Gradually, the evidence on structural and functional alterations of myocardium and vascular involvement has been accumulated (5–7). It has been also shown that CSX is associated with worst prognosis and higher prevalence of adverse cardiovascular events (8, 9).

The pathophysiology of CSX is still open. Presently, two main theories are accepted (for review see example 4):

- microvascular dysfunction, i.e., myocardial ischemia due to impaired microvascular function and
- (2) abnormal cardiac pain sensitivity, i.e., exaggerated pain perception.

In the paper "Fragmented QRS frequency in patients with cardiac syndrome X" by Daman et al. (10) in this issue, the higher frequency of fragmented QRS (fQRS) complex in patients with CSX is documented in comparison with that in control group. The fQRS complex has been documented in a number of cardiac pathology cases (11–14). It was also shown that it is associated with structural changes of myocardium (11, 13, 15) and represents an adverse diagnostic and prognostic sign. In this context, the finding of fQRS complex in SCX patients is very interesting. It adds a new piece of evidence that perception of pain in these patients has structural and functional background.

Each piece of knowledge to the mosaic of this clinically significant syndrome is of a great value.

Thinking in terms of future meta-analysis and review papers, it would be of great value if papers with original investigation will provide more details on the clinical status, ECG findings at rest and during exercise, and coronarography.

Liuba Bacharova^{1,2}

¹International Laser Center; Bratislava-*Slovak Republic* ²Institute of Pathophysiology, Medical Faculty, Comenius University; Bratislava-*Slovak Republic*

References

- Kemp HG Jr. Left ventricular function in patients with the anginal syndrome and normal coronary arteriograms. Am J Cardiol 1973; 32: 375-6.
- Parsyan A, Pilote L: Cardiac syndrome X: mystery continues. Can J Cardiol 2012; 28 (Suppl): S3-6.
- Arthur HM, Campbell P, Harvey PJ, McGillion M, Oh P, Woodburn E, et al. Women, cardiac syndrome X, and microvascular heart disease. Can J Cardiol 2012; 28(2 Suppl): S42-9.
- Agrawal S, Mehta PK, Bairey Merz CN. Cardiac Syndrome X: update 2014. Cardiol Clin 2014; 32: 463-78.
- Hurst T, Olson TH, Olson LE, Appleton CP. Cardiac syndrome X and endothelial dysfunction: new concepts in prognosis and treatment. Am J Med 2006; 119: 560-6.
- Melikian N, De Bruyne B, Fearon WF, MacCarthy PA. The pathophysiology and clinical course of the normal coronary angina syndrome (cardiac syndrome X). Prog Cardiovasc Dis 2008; 50: 294-310.
- Jones E, Eteiba W, Merz NB. Cardiac syndrome X and microvascular coronary dysfunction. Trends Cardiovasc Med 2012; 22: 161-8.
- Jespersen L, Hvelplund A, Abildstrøm SZ, Pedersen F, Galatius S, Madsen JK, et al. Stable angina pectoris with no obstructive coronary artery disease is associated with increased risks of major adverse cardiovascular events. Eur Heart J 2012; 33: 734-44.
- Sedlak TL, Lee M, Izadnegahdar M, Merz CN, Gao M, Humphries KH. Sex differences in clinical outcomes in patients with stable angina and no obstructive coronary artery disease. Am Heart J 2013; 166: 38-44.
- Damar I H, Altunkaş F, Çelik A, Koç F, Karayakah M, Karaman K, et al. Fragmented QRS frequency in patients with cardiac syndrome X. Anatol J Cardiol 2016; 16: 616-20.
- 11. Das MK, Khan B, Jacob S, Kumar A, Mahenthiran J: Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation 2006; 113: 2495-501.
- Homsi M, Alsayed L, Safadi B, Mahenthiran J, Das MK. Fragmented QRS complexes on 12-lead ECG: a marker of cardiac sarcoidosis as detected by gadolinium cardiac magnetic resonance imaging. Ann Noninvasive Electrocardiol 2009; 14: 319-26.
- Başaran Y, Tigen K, Karaahmet T, Işıklar I, Çevik C, Gürel E, et al. Fragmented QRS complexes are associated with cardiac fibrosis and significant intraventricular systolic dyssynchrony in nonischemic dilated cardiomyopathy patients with a narrow QRS interval. Echocardiography 2011; 28: 62-8.
- Park SJ, On YK, Kim JS, Park SW, Yang JH, Jun TG, et al. Relation of fragmented QRS complex to right ventricular fibrosis detected by late gadolinium enhancement cardiac magnetic resonance in adults with repaired tetralogy of fallot. Am J Cardiol 2012; 109: 110-5.
- Das MK, Suradi H, Maskoun W, Michael MA, Shen C, Peng J, et al. Fragmented wide QRS on a 12-lead ECG: a sign of myocardial scar and poor prognosis. Circ Arrhythm Electrophysiol 2008; 1: 258-68.

