DIAGNOSTIC CRITERIA AND ATTENTIVE REVIEWS IN THE TREATMENT OF CARDIAC X SYNDROME ANICIZED PATIENTS

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Abstract: To assess coronary hemodynamic status in patients with cardiological x-syndrome. Those with cardiological x-syndrome had a much lower coronary reserve. Less myocardial vascularization, more severe diffuse cardiosclerosis that reduced the capillaries' dilatory reserve, and injury localization at the prearteriole, arteriole, and capillary levels (i.e., in the region of the coronary vascular bed most responsible for dilator reserve) were the factors that led to low coronary flow reserve values.

Key words: microcirculatory vascular bed, coronary reserve, cardiological x-syndrome.

Introduction. It has been shown that certain angina patients do not show any changes in their coronary arteries since coronary ventriculography was employed in clinical settings. According to the results of a 1973 study by Arbogast R. and Bouras-sa M.G., those with angina syndrome and normal coronary arteries, as well as those with angina pectoris and coronary artery stenosis, reacted differently to preoperative stimulation. In patients with normal coronary arteries, atrial stimulation resulted in ST segment ischemia depression, but it had no effect on myocardial contractile performance. In the editorial discussion of this publication, H.G. Kemp first used the term "X-syndrome" to refer to this patient population. There are two main theories on the mechanisms underlying cardiological X syndrome (CSX). The first claims that the condition is caused by ischemia resulting from metabolic disorders that cause the myocardium to deviate from its energy substrate consumption or from structural or functional abnormalities of the coronary microcirculation. One sign of the ischemic nature of CSX is the presence of characteristic ST segment depression episodes during activity testing and daily ECG monitoring that are indistinguishable from those observed in patients with coronary artery disease. It should be noted that in these patients, angina pectoris, temporary myocardial ischemia, and BT segment depression during activity testing may persist for years.

Studies that have demonstrated that myocardial scintigraphy in these individuals shows regions of hypoperfusion of the heart muscle during physical activity and testing with dipyridamole, as well as in some patients at rest, further corroborate the ischemic etiology of CSX. Myocardial ischemia is known to be characterized by an increase in lactate generation along with a reduction in its consumption. It has been demonstrated that during the atrial stimulation test, individuals with CSX exhibit an increase in the amount of lactate in their coronary sinus blood. Evidence of myocardial ischemia in CSX patients also indicates that the heart's regional and global contractility is compromised during exercise

Microvascular dysfunctions The primary challenge in evaluating coronary microcirculation is the inability to directly study the anatomy and functioning of tiny coronary arteries. Therefore, efforts to demonstrate the presence of coronary microvessel pathology in CSF relied on two kinds of studies: those that evaluated the coronary blood flow's response to vasoactive stimuli or the coronary vessels' resistance to them, depending

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on the microvessels' integrity; and those that sought to directly demonstrate the existence of myocardial ischemia.

Myocardial ischemia assessment According to the traditional definition of CCC, ST segment depression during activity testing is the primary indicator of myocardial ischemia. Furthermore, as was previously noted, ST segment depression in patients with CCC can be identified by 48-hour Holter monitoring and may be brought on by atrial stimulation or medications that produce ischemia. About 50–90% of individuals with CSH have reversible perfusion abnormalities that can be identified during loading myocardial scintigraphy with 201Tl.

Microvascular dysfunction mechanisms There are a number of known factors that may lead to abnormalities in coronary microcirculation in CSH patients. As previously mentioned, coronary microvascular dysfunction is likely caused by conventional risk factors for cardiovascular illnesses, such as diabetes mellitus and hypertension, specifically by impairing endothelium-dependent vasodilation. Patients with CSH have also been shown to have insulin resistance, estrogen insufficiency (in women), and chronic inflammation, among other conditions that might cause endothelial dysfunction.

Resistance to insulin. The connection between metabolic syndrome and cardiac syndrome X One significant cause of reduced blood flow in tiny coronary arteries is endothelial dysfunction. In individuals with CSH, the occurrence of comparable problems in different organs and systems confirms that it causes broad diseases of smooth muscle tissues. Insulin resistance, which is a pathological disease that disrupts all forms of metabolism by violating the biological impact of insulin at the receptor and post-receptor levels with compensatory hyperinsulinemia, is directly linked to endothelial dysfunction. Hyperinsulinemia reduces the synthesis of prostacyclin and nitric oxide, which have vasodilating effects, and increases the endothelium's production of vasoconstrictor chemicals (endothelin-1 and thrombocyan A2).

Care for individuals with cardiac syndrome X CSX is treated conservatively. The mainstay of treatment for people with CSH is lifestyle modifications because cardiac risk factors are crucial to the progression of the illness. Many individuals who restrict their physical activity may benefit from cardiac rehabilitation to reduce the disease's symptoms. It has been shown to be useful in reducing illness symptoms and increasing tolerance to exercise.

Concerning cardiac X syndrome, or microvascular stenocardia
The symptom of chest discomfort known as microvascular stenocardia is brought on by the heart's tiny (microvascular) blood vessels malfunctioning. In this instance, even when the epicardial (large) coronary arteries are clean, the heart muscle experiences ischemia due to microcirculation problems.

The pathophysiology and causes
1. Endothelial dysfunction: microarteries' capacity to enlarge will be diminished.
2. Microvascular spasms: blood flow is disrupted by excessive constriction of tiny arteries.

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3. The cardiac vessels' inflammatory processes are impacted by oxidative stress and inflammation.

4. Dysfunction of the autonomic nervous system: Microcirculation is also impacted by excessive sympathetic nervous system activity.

Clinical indicators Similar to classical stenocardia, chest discomfort is frequently persistent (lasting more than ten minutes).It may worsen during physical exertion, but it can also happen during periods of stress or even while you're sleeping. Is there a sluggish reaction to nitroglycerin? There may be ST-segment depression or unexplained ECG abnormalities. The results of coronary angiography are normal. Diagnostics: 1. Stress tests Veloergometry or the Tredmil test evaluates how the heart reacts to loading. Stress-MRI or stress-ecography is used to examine the cardiac muscle's blood supply.

2. Angiography of the heart: A vasoreactivity test using acetylcholine or adenosine is utilized to identify microvascular dysfunction even in cases when the major coronary arteries are OK.

3. Holter surveillance: keeping an eye on heart activity throughout the day, particularly to identify ischemia alterations linked to symptoms.

4. Tests for biochemistry: Lipid profile: to determine cardiovascular disease risk. Catecholamines are occasionally used to investigate disorders of the autonomic nervous system.

Therapy: 1. A shift in lifestyle: Avoid being undernourished, anxious, and overweight. Frequent exercise, such as mild aerobics. .

2. Drugs: Metoprolol and bisoprolol are beta blockers that lower cardiac loading. Microvascular spasms are avoided with calcium channel blockers, such as diltiazem and amlodipine. Some patients benefit with nitrators (nitroglycerin, isosorbide dinitrate), but not all do. To treat endothelial dysfunction, statins (atorvastatin, rosuvastatin) are used. Ramipril and perindopril, two ACE inhibitors, aid in the enlargement of the heart's blood arteries.

3. Complementary and experimental therapies: Heart metabolism is enhanced by ranolazine.

Antioxidant treatment or L-arginine can help endothelial function.

Conclusion: Microvascular stenocardia is a disease that is difficult to diagnose and takes a long time to cure. It usually happens more often after menopause and in women. A correct diagnosis requires coronary angiography, stress testing, and microvascular functional testing. It may contribute to the development of myocardial ischemia by obstructing the delivery of oxygen substrates and oxidation to cardiomyocytes. Therefore, based on the analysis of data from the literature and our own research findings, we may conclude that the pathogenetic basis of CCC is damage to the coronary artery at the microcirculatory level.

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