

POTOMORPHOGENESIS OF SUDDEN CARDIAC DEATH IN PATIENTS WHO HAVE SUFFERED MYOCARDIAL INFARCTION

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Abstract:Sudden cardiac death (SCD) is one of the leading causes of death in adults worldwide. The presence of cardiovascular pathology (myocardial infarction (MI), heart failure, decreased ejection fraction, cardiac arrest, etc.) significantly increases the risk of developing SCD, although it is often the first, but lethal, manifestation of cardiovascular disease.

Keywords:Sudden cardiac death, treatment, method, myocardial infarction.

INTRODUCTION:Sudden cardiac arrest is one of the leading causes of death in adults worldwide. Most SCD occurs in patients with coronary artery disease (CAD), with the risk of developing SCD in patients who have had a myocardial infarction (MI) increasing 4 to 6 times more often than in the general population. The overall mortality rate after MI ranges from 24 to 40% of total mortality [1]. More than 15 million people suffer from ischemic heart disease in the United States, and about 720 thousand new or recurrent myocardial infarctions are registered here annually. SCD is the most common cause of death in this country and accounts for about 300-400 thousand deaths per year that could potentially be prevented, especially in the early period after MI [2].

MATERIALS AND METHODS:In patients with acute coronary syndrome, the risk of SCD is 5% per year, with a left ventricular (LV) ejection fraction (EF) < 35% and/or heart failure (HF) - 20%, after cardiac arrest or life-threatening events rhythm with successful resuscitation - 25%, in patients from the high-risk group after MI > 30% per year [1].

Therefore, there is an urgent need to evaluate effective risk stratification methods to select patients in the early period after MI who should maximize the optimization of treatment strategy and preventive measures [1].

In a significant number of patients, SCD occurs after rupture of an atherosclerotic plaque and acute MI. Many large observational and randomized studies have assessed SCD after MI.

RESULTS AND DISCUSSION:The most significant study to evaluate the risk of SCD depending on timing, the Valsartan Acute Myocardial Infarction Trial (VALIANT), included 14,609 patients with a history of myocardial infarction, either impaired LV function or HF.

The risk of SCD in the first 30 days after MI is 10 times higher and sharply decreases within 6 months, decreasing after 2 years.

When studying SCD risk data, three periods are distinguished after MI: early (48 hours - 40 days, subacute 41 days - 6 months) and late (6 months).

The pathogenesis and risk stratification of SCD in the early period after MI are extremely important for determining further treatment tactics and carrying out primary prevention measures.

The causes and mechanisms of development of SCD are numerous and not fully understood. SCD can develop in the form of asystole, pulseless electrical activity, bradyarrhythmia, ventricular tachycardia, or ventricular fibrillation.

Currently, the main risk factors for the development of SCD are: dyslipidemia, acute coronary syndrome, especially MI, HF, cardiomyopathies, myocarditis, arterial hypertension, increased heart rate (HR), obesity, diabetes mellitus, renal dysfunction, smoking, alcohol abuse, etc. [2].

In the acute phase of myocardial infarction, SCD develops as a result of ischemia, which can provoke lethal ventricular arrhythmias [1].

However, several autopsy studies have questioned the predominance of arrhythmias in the pathogenesis of SCD in the early period after MI. When analyzing autopsy records in VALIANT, it

was found that among 105 deaths that were attributed to SCD, the autopsy confirmed that 12.4% were due to cardiac rupture, 3.8% due to heart valve insufficiency, 26.6% due to recurrent cardiac arrest. MI. This study also suggested that death within 1 month after MI is more likely to have a mechanical etiology, and the risk of arrhythmic death increases after a few weeks.

The Optimal Trial in Myocardial Infarction with the Angiotensin II Antagonist Losartan (OPTIMAAL) study noted that recurrent myocardial infarction was confirmed in 55% of deaths clinically classified as SCD. However, in both VALIANT and OPTIMAAL, only a small percentage of patients (15% and 19%, respectively) underwent autopsy. In addition, data were collected retrospectively, without quality control of the autopsy.

At the same time, SCD can primarily manifest itself as a result of tachyarrhythmia and can be prevented with the help of a cardioverter-defibrillator (CD). The pathological prerequisite for the development of ventricular reentry arrhythmias can be various factors. Damage after MI contributes to the development of basic changes, which are based on disturbances in cardiac conduction, repolarization and autonomic regulation, which play a key role in the development of arrhythmia. As a result, occlusion of the epicardial coronary artery leads to ischemia and MI, impaired myocardial perfusion. As a consequence, remodeling occurs in the heart - a complex pathophysiological process leading mainly to thinning and dilatation of the ventricular wall.

This may contribute to a significant deterioration in left ventricular function in patients after MI and the development of heart failure. Activation of neurohumoral mechanisms, as well as ventricular dilatation, contribute to the progression of vasculopathy, left ventricular dysfunction and fibrosis, which causes electrical instability with impaired repolarization and reentry-type arrhythmias [1].

CONCLUSION: Thus, identifying post-MI patients at high risk of developing SCD is a big problem all over the world. Being one of the main causes of mortality, this multifaceted pathological process, given its high prevalence, requires a thorough and careful study for early and timely detection, prevention and treatment. To prevent SCD of arrhythmic etiology, several types of CD have been developed: implantable, subcutaneous, portable, each of which has its own advantages and disadvantages.

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