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Research Progress on Pathological Mechanism and Treatment of Sudden Cardiac Death

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Abstract

With the development of society, people's pace of life is quickening, and the pressure of life is increasing. The incidence rate of sudden cardiac death is increasing year by year. Sudden cardiac death has the characteristics of sudden onset, rapid progress of disease course and difficult rescue. This paper summarizes the research progress on the pathological factors and treatment of sudden cardiac death in recent years.

Keywords

Sudden cardiac death; Diagnosis; Etiology; Research progress.

1. INTRODUCTION

Sudden cardiac death refers to a malignant condition described from an etiological source as sudden death or death within a short period of time caused by a cardiac disease, also known as sudden cardiac death. The most recent definition of SCD was developed by the European Society of Cardiology, the American Heart Association, and the American College of Cardiology, and it refers to death occurring within 1 h of the onset of acute symptoms attributable to a cardiac disease, whether occurring on the way to hospital, outside the hospital, or in the emergency department. Patients with SCD are usually detected with loss of consciousness as the main symptom, and their characteristics of sudden onset and rapid progression result in patients who cannot be easily rescued and successfully. Between 17 and 450000 cases have been estimated in recent years due to cardiac deaths in the United States, which outnumber all major cancers (lung, breast, colorectal) [1]. It is therefore important for the study and summary of SCD.

2. CLASSIFICATION AND PATHOPHYSIOLOGY OF SUDDEN CARDIAC DEATH

2.1. Ischemic Cardiomyopathy

The common forms of ischemic cardiomyopathy that can cause SCD include four categories: coronary heart disease with myocardial infarction or angina, coronary embolism, non iatrogenic CAD (arteritis, dissection and congenital anomalies of the coronary arteries), and coronary spasm. The first two categories may be related to the formation of blood clots or atherosclerosis (as), the main triggers of which include hemodynamic abnormalities, plasma total cholesterol values (TC) or triglyceride (TG) abnormalities, hypertension, smoking, other diseases causing secondary hyperlipidemia and non negligible genetic factors. Studies have shown that around 25% of coronary heart disease (CHD) is caused by SCD as the first clinical sign, while 80% of patients who have had an episode of SCD have suffered from CHD and its complications, among which also a non negligible 75% of patients have a history of myocardial infarction (MI) [2].

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2.2. Nonischaemic Cardiomyopathy

Nonischemic cardiomyopathies that clinically cause SCD mainly include hypertrophic cardiomyopathy, dilated cardiomyopathy, valvular heart disease, congenital heart disease, arrhythmogenic right ventricular cardiomyopathy, myocarditis, cardiac sarcoidosis, cardiac tamponade, neuromuscular diseases (e.g., Duchenne, Becker, and Emery Dreifuss muscular dystrophy), acute myocardial rupture, and aortic dissection, among others. Such cases are dominated by primary and secondary structural abnormalities. Structural abnormalities are exemplified by valvular heart disease: when the heart exhibits early mild mitral stenosis with elevated left atrial internal pressure, the left atrial wall gradually develops compensatory hypertrophy with hyperplasia, at which point it affects the volume of the left ventricular filling phase and gradually decreases ventricular pumping volume. As the stenosis of the mitral valve worsens, the left atrium gradually decompensates in a highly dilated state, which leads to pulmonary congestion, presenting symptoms of left heart failure. If mitral stenosis is accompanied by damage at this time, further progression can occur to global heart failure or the resulting SCD.

2.3. Structurally Normal Sub Cardiac SCD or Present with Primary Electrophysiological Abnormalities

This category mainly includes idiopathic ventricular fibrillation or J-wave syndrome, Brugada syndrome, long QT syndrome, short QT syndrome, catecholaminergic polymorphic myocardial infarction, pre-excitation syndrome, high degree atrioventricular block with torsades de pointes, and familial SCD, among others. Conduction system disorders such as league disease, Lev disease, and Brugada syndrome are, for example, caused by primary electrocardiographic abnormalities or ion channel dysfunction [3]. When a reentry loop or other trigger occurs in the myocardium, it is easy to trigger ventricular fibrillation, strenuous exercise, because of sudden anxiety, tension, fear, and other great mood swings, coronary spasm, platelet thrombus and so on may also show malignant arrhythmias. In addition, attention should also be paid to malignant arrhythmias caused by electrolyte imbalance, such as hypokalemia, which may cause torsade de points.

2.4. Non-cardiac SCD

Such include pulmonary embolism, intracranial hemorrhage, drowning, Pickwickian syndrome, drug overdose or toxic poisoning, central airway obstruction, sudden infant death syndrome, and others.

3. IDENTIFICATION OF SCD

SCD usually needs to be differentiated from NSCD, which refers to sudden death in a patient due to disease other than cardiac. There are many etiologies for NSCD, and abnormalities in each system may contribute to pathogenesis, and there is no clear definition of which of the respiratory or heart beats first stops, simply that stopping the other one at any time will subsequently stop, whereas SCD is clearly defined as the heart first stopping beating, so it can be clearly distinguished from SCD at this point. An additional point that should also be elucidated is the nature of SCD and differs from cardiac arrest (SCA), which is a hemodynamic abnormality caused by the disappearance of pumping function of the heart, in which out of hospital cardiac arrest (OHCA) can reestablish circulation through free cardiopulmonary resuscitation, and in which cardiac arrest in the emergency room or inpatient setting is usually given chest compressions Aggressive measures such as electrocution defibrillation and intravenous passage medication thus have a life-saving chance, and SCD is generally considered fatal. Failure to re-establish circulation in patients with SCA is referred to as SCD, and the mechanism of death is often thought of as the underlying arrhythmia [4,5].

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4. RESCUE OF SCD

Some key indicators such as heart rate, oxygen saturation, etc. should be strictly monitored in the rescue of SCD with out of hospital episodes. The rescue process should be divided into two parts: I) pre hospital first aid process and in-hospital first aid process: ① pre hospital first aid: after 120 scheduling center receives telephone call from patient or family member for rescue, the nearest hospital with the ability to rescue and go, the first aid car should be equipped with a defibrillator and the emergency medicine should be prepared, avoiding the accelerated deterioration of patient's condition because the equipment or drugs during the transfer process are missing. First active assessment of patient condition upon arrival of the emergency physician to return for rescue, necessary checks such as the first 12 lead ECG were performed according to the local emergency regime with attention to retention check results. Transport in route first responders should contact the hospital rescue room to be sent, anticipate the patient situation and get the rescue room side ready. ② In hospital first aid: after admission, take semi recumbent position or lying flat position according to personal condition, take the necessary basic disposal, including (establish venous access, continuous oxygen absorption, etc.) complete each examination in accordance with the importance and sequence of examination, should always monitor the patient's heart rate, blood oxygen saturation, blood pressure, biochemical indicators of hematuria. For patients with S-T abnormalities on ECG examination actively contact cardiology consultation, treatment with sublingual isosorbide dinitrate combined with intravenous 2-5 mg morphine is indicated for patients with myocardial infarction, and for patients who already have shock symptoms, aggressive rehydration according to the principle of rehydration, using vasoactive drugs against shock. For patients who have already had a respiratory arrest, immediate cardiopulmonary brain resuscitation with opening of the airway and removal of oral foreign bodies (including dentures, etc.) was administered along with intravenous epinephrine at a dose of one milligram each, five minutes apart, and shock defibrillation (generally biphasic wave defibrillator 200J) was administered immediately when ventricular fibrillation or other defibrillatory rhythms were present, with attention to the time interval between two defibrillations.

5. EARLY DIAGNOSIS OF SCD AND PREVENTION AGAINST TRIGGERS

Based on the perception of SCD, we can assess the risk of incident SCD at an early age, both life factors and genetic factors. We can give better advice on primary and secondary hierarchical assessment of potential patients with SCD, such as cases with SCD within families, individuals with different degrees and types of CVD, etc., so that more precise and effective interventions for SCD can be given. But this method also still has drawbacks because the onset of SCD mostly occurs when it is considered unprovoked by the patients themselves, so the spread of knowledge about SCD should be strengthened on the one hand, and public training on basic first aid skills, including the use of AEDs, implementation of CPR, etc. [6-11].

6. SUMMARY

For clinical work, SCD remains a significant problem with huge public health implications. Although a gradual and perfect rescue system has been established so far, there are still many unknown and imperfect factors in the rescue and treatment of SCD, such as the risk stratification of SCD is still not ideal, especially for patients with non-ischemic cardiomyopathy. More complete rescue for SCD therefore also hinges on further research.

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