TACHYCARDIA - SYMPTOMS, CAUSES, DIAGNOSIS, TREATMENT, COMPLICATIONS.

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Abstract. *Tachycardia is not a disease, but a symptom, as it can be observed as a manifestation of many diseases. Its most common causes are disorders of the autonomic nervous system, diseases of the endocrine system, hemodynamic disorders, and various forms of arrhythmia.*

Pathological tachycardia can cause negative consequences:

First, when the heart beats fast, its efficiency decreases, because the ventricles do not have time to fill with blood, as a result, blood pressure decreases and the quality of its delivery to the organs deteriorates.

Secondly, the heart's own blood supply is also impaired, because it does more work per unit of time and, accordingly, requires more oxygen, its poor blood supply increases the risk of coronary heart disease and subsequent heart attacks.

Key words: *Classification, Etiology, Diagnosis, Tachycardia treatment*

ТАХИКАРДИЯ - СИМПТОМЫ, ПРИЧИНЫ, ДИАГНОСТИКА, ЛЕЧЕНИЕ, ОСЛОЖНЕНИЯ.

Аннотация. *Тахикардия является не болезнью, а симптомом, так как может наблюдаться как проявление многих заболеваний. Наиболее частыми причинами его являются нарушения вегетативной нервной системы, заболевания эндокринной системы, нарушения гемодинамики, различные формы аритмии.*

Патологическая тахикардия может вызвать негативные последствия:

Во-первых, при быстром сокращении сердца снижается его работоспособность, так как желудочки не успевают наполниться кровью, в результате снижается артериальное давление и ухудшается качество его доставки к органам.

Во-вторых, нарушается и собственное кровоснабжение сердца, поскольку оно выполняет больше работы в единицу времени и, соответственно, требует большего количества кислорода, его плохое кровоснабжение увеличивает риск ишемической болезни сердца и последующих инфарктов.

Ключевые слова: *классификация, этиология, диагностика, лечение тахикардии.*

INTRODUCTION

CLASSIFIED SINUS TACHYCARDIA

This type of tachycardia is caused by the sinus nodes, which control the heart rhythm, generating an impulse or by disrupting the passage of impulses through them. Arrhythmias are detected by electrocardiogram.

PAROXYSMAL TACHYCARDIA

This is a heart attack that begins with a tostat and ends in the same way, and the number of contractions can reach 150-300 times per minute. There are 3 forms of it:

Subjunctive;

Knotted;

Ventricular.

The etiology of rapid heartbeat is the same as extrasystole, but supraventricular paroxysmal tachycardia is often associated with activation of the sympathetic nervous system, and ventricular forms - with serious dystrophic changes in the myocardium.

In almost all cases, ventricular tachycardia is observed in persons with cardiac pathology.

In 85% of cases, this is due to ischemic heart disease.

It is observed 2 times more often in men than in women.

In only 2% of cases, attacks are detected in patients without any reliable clinical and instrumental signs of heart damage.

Paroxysmal ventricular tachycardia usually causes hemodynamic disturbances (arterial hypotension, loss of consciousness), myocardial ischemia.

METHOD AND METHODOLOGY

VENTRICULAR FIBRILLATION

Ventricular fibrillation (VF) is characterized by chaotic contraction of myocardial fibers with a frequency of 250-480 per minute, without coordinated contraction of the ventricles. In this case, the heart stops performing its pumping functions and the blood supply to the whole body stops (not to be confused with a cardiac arrest). QF is often a complication of extensive transmural myocardial infarction.

It is conventionally accepted to distinguish primary, secondary and late fibrillation of the ventricles.

Primary QF develops in the first 24-48 hours of myocardial infarction and reflects electrical instability of the myocardium due to acute ischemia. This condition is the main cause of sudden death of patients with myocardial infarction.

Secondary QF develops against the background of left ventricular circulatory failure and cardiogenic shock.

Late QF occurs 48 hours after the onset of a heart attack, usually by 2-6 weeks of the disease. It often develops in patients with myocardial infarction. The mortality rate in late QF is 40-60%.

Ventricular fibrillation always starts suddenly and goes like this:

3-5 seconds after the start of fibrillation, dizziness and weakness are felt, after 15-20 seconds the patient loses consciousness.

Characteristic convulsions (tonic contraction of muscles) appear after 40 seconds. At this time, involuntary urination and defecation are usually observed.

After 40-45 seconds, the pupil expands and reaches its maximum size in 1.5 minutes. The maximum expansion of the pupil indicates that half of the time that brain cells can regenerate has passed.

Noisy (wheezing), frequent breathing gradually decreases and stops completely within 2 minutes (clinical death occurs).

The diagnosis of clinical death is made on the basis of:

Loss of consciousness;

Shortness of breath or agony-type breathlessness;

Absence of a pulse in the sleep area;

Enlargement of the pupils, paleness of the face.

ETIOLOGY

Causes of a fast heart rate include:

Organic injury of the myocardium;

Chronic forms of CKD;

Myocarditis;

The presence of additional anomalous conduction of WPW (Wolf-Parkinson-White) pathways;

Significant autonomic and humoral disorders in patients with neurocirculatory dystonia.

In 90-95% of cases, ventricular tachycardia is observed in patients with organic damage to the heart.

The largest share of them corresponds to chronic ischemic heart disease. In 1-2% of cases, this is in patients who have experienced a heart attack. In the last case, it lasts for a few seconds or minutes and returns to normal by itself.

Poisoning with cardiac glycosides can also cause ventricular tachycardia (in about 20% of cases).

Other causes include rheumatic and congenital heart defects, myocarditis, cardiomyopathy, mitral valve prolapse syndrome, congenital prolongation of the Q-T interval syndrome, mechanical damage to the heart, pheochromocytoma, strong negative emotions (fear), quinidine, isadrine, Complications of therapy with adrenaline, some anesthetics, psychotropic drugs take place.

In very rare cases, rapid heartbeat is observed in almost healthy people.

DISORDER OF THE VEGETATIVE NERVOUS SYSTEM AND ENDOCRINE SYSTEM

Increased stimulation of the sympathetic nervous system can cause increased heart rate both by direct action on sympathetic nerve fibers in the heart and by acting on the adrenal gland, leading to an increase in adrenaline secretion. Tachycardia caused by the influence of the sympathetic nervous system is observed in completely healthy people as a result of anxiety and caffeine consumption.

Endocrine disorders associated with increased production of adrenaline also lead to tachycardia.

RESEARCH RESULTS

HYPODYNAMIC RESPONSE

As a result of feedback mechanisms that support blood pressure, heart rate increases when blood pressure decreases.

Therefore, tachycardia occurs in response to a decrease in blood volume (for example, as a result of blood loss or dehydration). In addition, a sudden change in body position can lead to a decrease in blood pressure and, accordingly, an acceleration of the heartbeat.

DIAGNOSIS

CLINIC

An attack develops suddenly, heart activity changes to another rhythm. The number of heart contractions in the ventricular form usually reaches 150-180 pulses per minute, in the supraventricular form - up to 180-240 pulses. Often, during an attack, the jugular veins pulsate.

On auscultation, a pendulum-like rhythm (embryocardia) is characteristic, no difference between I and II tone is noticeable. The duration of an attack can last from a few seconds to several days. Nodular and compartmental paroxysmal tachycardia does not significantly affect central hemodynamics.

However, heart failure may worsen and swelling may increase in patients with IHF. Supraventricular paroxysmal tachycardia increases myocardial oxygen demand and can lead to an attack of acute coronary insufficiency. Characteristically, the sinusoidal form does not start suddenly and, in the same way, gradually ends.

DISCUSSION

ELECTROCARDIOGRAPHIC (ECG) SIGNS

In the electrocardiogram:

QRS complexes do not change in the supraventricular form.

The QRS complex has changed in the shape of the ventricles.

In supragrantricular form, P-tooth merges with T.

In conditions of altered QRS, P-waves are not detected, only sometimes P-waves are seen before the deformed QRS complex.

In contrast to the supraventricular form, ventricular paroxysmal tachycardia always leads to heart failure, gives the appearance of collapse and can end in the death of the patient.

TREATMENT OF TACHYCARDIA

Treatment of ventricular tachycardia requires the use of antiarrhythmic drugs, treatment of the primary disease, and measures to eliminate factors that cause arrhythmia (glycoside intoxication, electrolyte disturbances, hypoxemia).

CONCLUSION

The main antiarrhythmic agent used in the treatment is lidocaine, which is administered intravenously in a dose of 1 mg per 1 kg of the patient's weight (average 70-100 mg) in a few minutes. If there is no effect, after 10-15 minutes, the drug is injected again in the same dose. In recurrent tachycardia, lidocaine is administered intravenously at a dose of 1-2 mg per minute for 24-48 hours.

If ventricular tachycardia is accompanied by a drop in blood pressure, blood pressure should be increased to 100-110 mmHg by intravenous noradrenaline or other pressor amines. Failure to observe the effect serves as an indication for the use of electroimpulse therapy.

In cases where lidocaine fails to stop ventricular tachycardia, novocainamide, aymalin, β-blockers are used.

In the treatment of patients with ventricular tachycardia caused by cardiac glycoside intoxication, potassium chloride and lidocaine are administered intravenously or by slow-flow oral administration.

The prognosis of patients with ventricular tachycardia is negative, because it is a manifestation of severe myocardial damage in most of them. Among patients suffering from acute myocardial infarction, heart failure, and hypotension, the death rate is especially high.

Tachycardia is also treated with minimally invasive surgery - under local anesthesia, without leaving stitches. It can be radiofrequency catheter ablation, artificial pacemaker installation, etc.

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