Pro Index | Journal of Science in Medicine and Life

Volume: 2 Issue: 5 Year: 2024

http://journals.proindex.uz

MODERN VIEWS ON PREVENTION AND TREATMENT OF VENTRICULAR TACHYCARDIA, PATHOGENESIS

Burhonova Nigora

Shodiyev Xumoyun

Raxmonov Azamat

Komilova Moxinur

Samarkand State Medical University, Department of Therapy, Cardiology and Functional Diagnostics, 1st year ordinators

Abstract: Ventricular tachycardia 3 consecutive ventricular complexes with a frequency of 120 beats per minute. Depending on the duration of symptoms, they can range from no symptoms to palpitations, hemodynamic collapse, and death. Diagnosed by electrocardiography. Prolonged episodes are treated with cardioversion or antiarrhythmics depending on the symptoms. If necessary, long-term treatment is carried out using an implantable cardioverter defibrillator.

Some experts use a rate ≥ 100 beats per minute to test for ventricular tachycardia (VT). Ventricular rhythms that repeat at a lower rate are called accelerated idioventricular rhythms, or slow VT; as a rule, they are comfortable and do not require treatment if patients do not have clinical symptoms.

Most patients with VT have significant heart disease, particularly prior myocardial infarction or cardiomyopathy. Disorders of electrolyte metabolism (especially hypokalemia and hypomagnesemia), acidemia, hypoxemia, and side effects of drugs contribute to VT. Long QT syndrome (congenital or acquired) is associated with a special form of VT, torsade de pointes (TdP).

Key words: Ventricular tachycardia can be monomorphic and polymorphic, as well as unstable and stable.

Monomorphic VT: regular QRS complexes with a single pathological focus or reentrant mechanism and therefore identical in morphology

Polymorphic VT: several different foci or accessory pathways and therefore irregularly shaped QRS complexes

Nonsustained VT: Lasts < 30 seconds

Sustained VT: duration ≥ 30 seconds or less due to hemodynamic collapse

Catecholaminergic polymorphic ventricular tachycardia is a genetic disorder affecting intracellular calcium regulation in heart tissue. Patients are prone to atrial and/or ventricular tachyarrhythmias and sudden cardiac death, especially during periods of increased adrenergic activity.

VT often progresses to ventricular fibrillation and therefore leads to cardiac arrest.

Signs and symptoms of ventricular tachycardia

Short-term or slow-rate ventricular tachycardia may be asymptomatic. Sustained VT is almost always symptomatic, leading to palpitations, signs of hemodynamic compromise, or sudden cardiac death.

Diagnosis of ventricular tachycardia

Electrocardiography (ECG)

The diagnosis of ventricular tachycardia is determined by ECG (see picture of wide QRS ventricular tachycardia). Any tachycardia with wide QRS complexes (QRS ≥ 0.12 seconds) should be considered VT unless another tachycardia is proven.

Common sense and precautions

Any tachycardia with wide QRS complexes (QRS ≥ 0.12 seconds) should be considered VT unless another tachycardia is proven.

The diagnosis is confirmed by the dissociation of P waves, the presence of confluent complexes or supraventricular "seizures" in the ECG, the contrast of the T-wave vectors with the uniformity (concordance) of the QRS vectors in the precordial leads. Location of the QRS axis in the frontal plane in the northwest quadrant. The differential diagnosis is supraventricular tachycardia and bundle branch block or accessory pathway (see Fig. modified Brugada criteria for ventricular tachycardia). However, since some patients tolerate VT very well, it is a mistake to conclude that well-tolerated wide complex tachycardia can be supraventricular. In patients with VT, the use of drugs suitable for the treatment of supraventricular tachycardia (eg, verapamil, diltiazem) can lead to hemodynamic collapse and death.

Sustained stable VT can also be treated with intravenous class I or III antiarrhythmic drugs (see table of antiarrhythmic drugs). Lidocaine works quickly but is often ineffective. If lidocaine is ineffective, a class IV procainamide drug can be used, but the waiting time for its effect may last up to 1 hour. Intravenous amiodarone is often used, but it usually doesn't work as quickly. Failure of IV procainamide or IV amiodarone is an indication for cardioversion.

Nonsustained VT does not require immediate treatment until the racing is frequent or long enough to cause symptoms. In such cases, antiarrhythmics are used to treat stable VT.

Long-term treatment is not required when an episode of ventricular tachycardia occurs for temporary reasons (for example, within 48 hours after a myocardial infarction) or for reversible reasons (acid-base disorders, electrolyte disorders, proarrhythmic effects of drugs).

Unless there is a temporary or reversible cause, patients who experience a persistent VT episode usually require implantation of an implantable cardioverter defibrillator. Most patients with stable VT and severe systemic heart disease should also receive beta-blockers. If an ICD is not available, amiodarone may be the antiarrhythmic drug of choice to prevent sudden death.

Because unstable VT is a marker of increased risk of sudden death in patients with structural heart disease, such patients (especially those with an ejection fraction < 35%) should be evaluated further. Such patients need ICD implantation.

If prevention of VT is important (usually in patients with an ICD and frequent episodes of VT), antiarrhythmic therapy or transcatheter or surgical ablation of the arrhythmogenic substrate is required. Any - Ia, Ib, Ic, II or III - antiarrhythmic drugs can be used. Due to their safety, beta-blockers are the number 1 drug in the absence of contraindications. If additional drugs are needed, sotalol is usually used, followed by amiodarone.

Transcatheter ablation is often performed in patients with VT with certain syndromes (eg, right ventricular outflow tract VT or left ventricular septal tachycardia [Belhassen tachycardia, verapamil-sensitive VT]) and in patients with otherwise healthy hearts. is used.

Basic rules

Any wide complex tachycardia (QRS ≥ 0.12 seconds) should be considered ventricular tachycardia (VT) unless proven otherwise.

Patients without a pulse should be defibrillated with a cardioverter.

If the patient is stable, synchronous cardioversion or antiarrhythmic drugs can be used.

Patients who experience an episode of persistent VT without a transient or reversible cause usually require implantation of an implantable cardioverter defibrillator (ICD).

Atrial fibrillation is a rapid, irregular atrial rhythm. Symptoms include: heart palpitations and sometimes weakness, decreased exercise tolerance, shortness of breath, and presyncope. Often there is the formation of blood clots in the atrium, which are an important risk factor for embolic stroke. Diagnosed by electrocardiography. Treatment includes rate control drugs, thromboembolic prophylaxis with anticoagulants, and sometimes restoration of sinus rhythm with drugs or cardioversion.

Atrial fibrillation is caused by several waves of chaotic repetitive excitation in the atria. However, in most cases, an excitatory ectopic focus in the venous structures adjacent to the atria (usually the pulmonary veins) plays a role and is responsible for causing and maintaining atrial fibrillation. In atrial fibrillation, the atrium does not contract and the AV junction is bombarded with many electrical impulses, the conduction of which causes an irregular ventricular rhythm, which is usually in the tachycardia range.

Atrial fibrillation is one of the most common arrhythmias, affecting 2 to 6 million adults in the United States. Men and Caucasians are more likely to suffer from AF than women and blacks. Prevalence

increases with age; Almost 10% of people over the age of 80 have AF. Atrial fibrillation usually occurs in patients with existing heart disease.

Complications of atrial fibrillation

Absence of atrial contraction predisposes to the formation of blood clots; the annual risk of cerebrovascular embolic events is approximately 7%. The risk of stroke is higher in elderly patients and patients with rheumatic valvular disease, mechanical prosthetic heart valves, hyperthyroidism, hypertension, diabetes, left ventricular systolic dysfunction, or previous thromboembolic events. Systemic embolism can also cause necrosis of other organs (heart, kidneys, gastrointestinal tract, eyes) or limbs.

Atrial fibrillation can also reduce cardiac output; the absence of atrial contraction can reduce cardiac output by about 10% during normosystole. This reduction is generally well tolerated, unless the ventricular rate is increased, the rhythm is very rapid (eg, > 140 beats/min), or patients have a borderline or reduced cardiac output at baseline. In such cases, heart failure may develop.

CLINICAL CALCULATOR

Atrial fibrillation grade CHA(2)DS(2)-VASc for predicting stroke risk

Etiology of atrial fibrillation

The most common causes of atrial fibrillation are:

Arterial hypertension

Cardiac ischemia

Cardiomyopathy

Heart valve defects: mitral stenosis, mitral insufficiency, tricuspid insufficiency

Hyperthyroidism

Excessive alcohol consumption (cardiac holiday syndrome)

Less common causes of atrial fibrillation include:

Pulmonary embolism

Atrial septal defects and other congenital heart defects

Chronic Obstructive Pulmonary Disease (COPD)

Myocarditis

Pericarditis

Idiopathic primary atrial fibrillation - atrial fibrillation without a clear cause in patients under 60 years of age.

Classification of atrial fibrillation

Paroxysmal atrial fibrillation is atrial fibrillation that typically lasts < 1 week and resolves spontaneously or with intervention before normal sinus rhythm is restored. Situations can be repeated.

Persistent atrial fibrillation - persistent atrial fibrillation lasting more than 1 week.

Long-term persistent atrial fibrillation lasts more than 1 year, but restoration of sinus rhythm is still possible.

Persistent atrial fibrillation cannot be converted to sinus rhythm (including patients in whom a decision has been made not to attempt to convert to sinus rhythm). The longer the atrial fibrillation, the lower the probability of spontaneous restoration of sinus rhythm and the lower the effectiveness of cardioversion, atrial remodeling (changes in atrial electrophysiology due to rapid atrial rhythm, which are mainly reduced atrial refractoriness related to and may include). increased spatial distribution of atrial refractoriness, slowing of atrial conduction, or all changes together).

Signs and symptoms of atrial fibrillation

Atrial fibrillation is often asymptomatic, but many patients have palpitations, chest discomfort, or symptoms of heart failure (weakness, dizziness, shortness of breath), especially if the ventricular rate is very high (140-160 beats/minute). . Symptoms may also mimic acute stroke or other organ damage due to systemic embolism.

With the disappearance of jugular venous pulse waves, the pulse is irregular. Pulse failure (peak rate faster than the pulse rate palpated at the wrist) can occur because the left ventricular stroke volume is insufficient to generate a peripheral pressure wave to beat immediately after the previous beat.

List of used literature:

1. Sarkisova, V., Lapasova, Z., & Shernazarov, F. (2023). O. Rakhmanov INFLAMMATORY DISEASES OF THE PELVIC WOMEN ORGANS.
2. Shernazarov I., Shernazarov F. Problems of translation of features related to the way of life of peoples //Science and innovation. – 2023. – T. 2. – №. B1. – C. 80-83.
3. Jalalova, D., Axmedov, A., Kuryazov, A., & Shernazarov, F. (2022). COMBINED DENTAL AND EYE PATHOLOGY. Science and innovation, 1(8), 91-100.
4. Shernazarov F., Zuhridinovna J. D. Microcirculation disorders in the vascular system of the bulbar conjunctiva in the initial manifestations of cerebral blood supply deficiency //Science and innovation. – 2022. – T. 1. – №. Special Issue 2. – C. 515-522.
5. Solovyova, Y., Prostyakova, N., Sharapova, D., & Shernazarov, F. (2023). The relevance of psychotic disorders in the acute period of a stroke. Science and innovation, 2(D12), 212-217.
6. Rotanov, A., Karshiyev, Z., Sharapova, D., & Shernazarov, F. (2023). Diagnosis of depressive and suicidal spectrum disorders in students of a secondary special education institution. Science and innovation, 2(D11), 309-315.
7. Shernazarov F. COMBINED DENTAL AND EYE PATHOLOGY //Science and Innovation. – 2022. – T. 1. – №. 8. – C. 91-100.

8. Madaminov M., Shernazarov F. Breast cancer detection methods, symptoms, causes, treatment //Science and innovation. – 2022. – Т. 1. – №. D8. – С. 530-535.
9. Jalalova, D., Axmedov, A., Kuryazov, A., & Shernazarov, F. (2022). СОЧЕТАННАЯ СТОМАТОЛОГИЧЕСКАЯ И ГЛАЗНАЯ ПАТОЛОГИЯ. Science and innovation, 1(D8), 91-100.
10. Prostyakova, N., Solovyova, Y., Sharapova, D., & Shernazarov, F. (2023). Issues of professional ethics in the treatment and management of patients with late dementia. Science and innovation, 2(D12), 158-165.
11. Jalalova D., Raxmonov X., Shernazarov F. РОЛЬ С–РЕАКТИВНОГО БЕЛКА В ПАТОГЕНЕЗЕ СОСУДИСТЫХ ЗАБОЛЕВАНИЙ ОРГАНА ЗРЕНИЯ У БОЛЬНЫХ АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ //Science and innovation. – 2022. – Т. 1. – №. D8. – С. 114-121.
12. Pogosov S. et al. Psychogenetic properties of drug patients as risk factors for the formation of addiction //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 186-191.
13. Sedenkova M. et al. The possibility of predicting the time of formation and development of alcohol dependence: the role of genetic risk, family weight and its level //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 173-178.
14. Shamilov V. et al. Disorders of decision-making in the case of depression: clinical evaluation and correlation with eeg indicators //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 198-204.
15. Малявская С. И., Лебедев А. В. Метаболический портрет детей с ожирением //Российский вестник перинатологии и педиатрии. – 2015. – Т. 60. – №. 6. – С. 73-81.
16. Ишкабулова Г. Д. и др. Влияние димефосфона на основные функции почек новорожденных, рожденных от матерей с хроническим пиелонефритом с сочетанным ОПГ-гестозом //Вестник науки и образования. – 2018. – №. 14-2 (50). – С. 51-55.
17. Гарифулина Л. М., Ашурова М. Ж., Гойибова Н. С. Состояние здоровья детей с различными типами ожирения //Молодежь и медицинская наука в XXI веке. – 2018. – С. 35-37.
18. Гойибова Н. С. и др. Функция почек у недоношенных новорожденных, родившихся от матерей с преэклампсией //Достижения науки и образования. – 2019. – №. 10 (51). – С. 59-63.
19. Гойибова Н. С., Гарифулина Л. М. Функции почек у детей с ожирением //Вопросы науки и образования. – 2020. – №. 26 (110). – С. 51-57.
20. Uskov A. et al. Modern methods of therapeutic fasting as a way to overcome the pharmacoresistance of mental pathology //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 179-185.
21. Prostyakova N. et al. Strategy for early diagnosis with cardiovascular diseaseisomatized mental disorders //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 166-172.
22. Tahirova, J., Roziklov, N., Mamatkulova, F., & Shernazarov, F. (2022). Insomnia problem causes of sleep disorder, help measures at home. Science and innovation, 1(D8), 521-525.
23. Qizi T. J. I., Farrukh S. Treatment of myocardial infarction and first aid //Science and innovation. – 2022. – Т. 1. – №. D3. – С. 317-320.
24. Rotanov A. et al. Elderly epilepsy: neurophysiological aspects of non-psychotic mental disorders //Science and innovation. – 2023. – Т. 2. – №. D12. – С. 192-197.

25. Abdurakhmanov I., Shernazarov F. SPECIFIC ASPECTS OF TREATMENT OF CHRONIC RHINOSINUSITIS IN CHILDREN //Science and innovation. – 2023. – T. 2. – №. D10. – C. 164-168.
26. Tahirova J. et al. Neurose causes and mechanisms of development, symptoms, treatment, prevention //Science and innovation. – 2022. – T. 1. – №. D8. – C. 515-520.
27. Tahirova J., Shernazarov F. Symptoms of hymoritis, treatment, methods of folk medicine, prevention //Science and innovation. – 2022. – T. 1. – №. D8. – C. 983-990.
28. Shodiyeva D., Shernazarov F. Analysis of the compounds providing antihelmitic effects of chichorium intybus through fractionation. Science and innovation, 2 (D2), 64-70. – 2023.
29. Jalalova D., Normatova N., Shernazarov F. Genetic markers for the development of diabetic retinopathy //Science and Innovation. – 2022. – T. 1. – №. 8. – C. 919-923.
30. Abdukodirova S., Shernazarov F. SPECIFIC CHARACTERISTICS AND TREATMENT OF ACUTE OBSTRUCTIVE BRONCHITIS IN CHILDREN OF EARLY AGE //Science and innovation. – 2023. – T. 2. – №. D11. – C. 5-8.