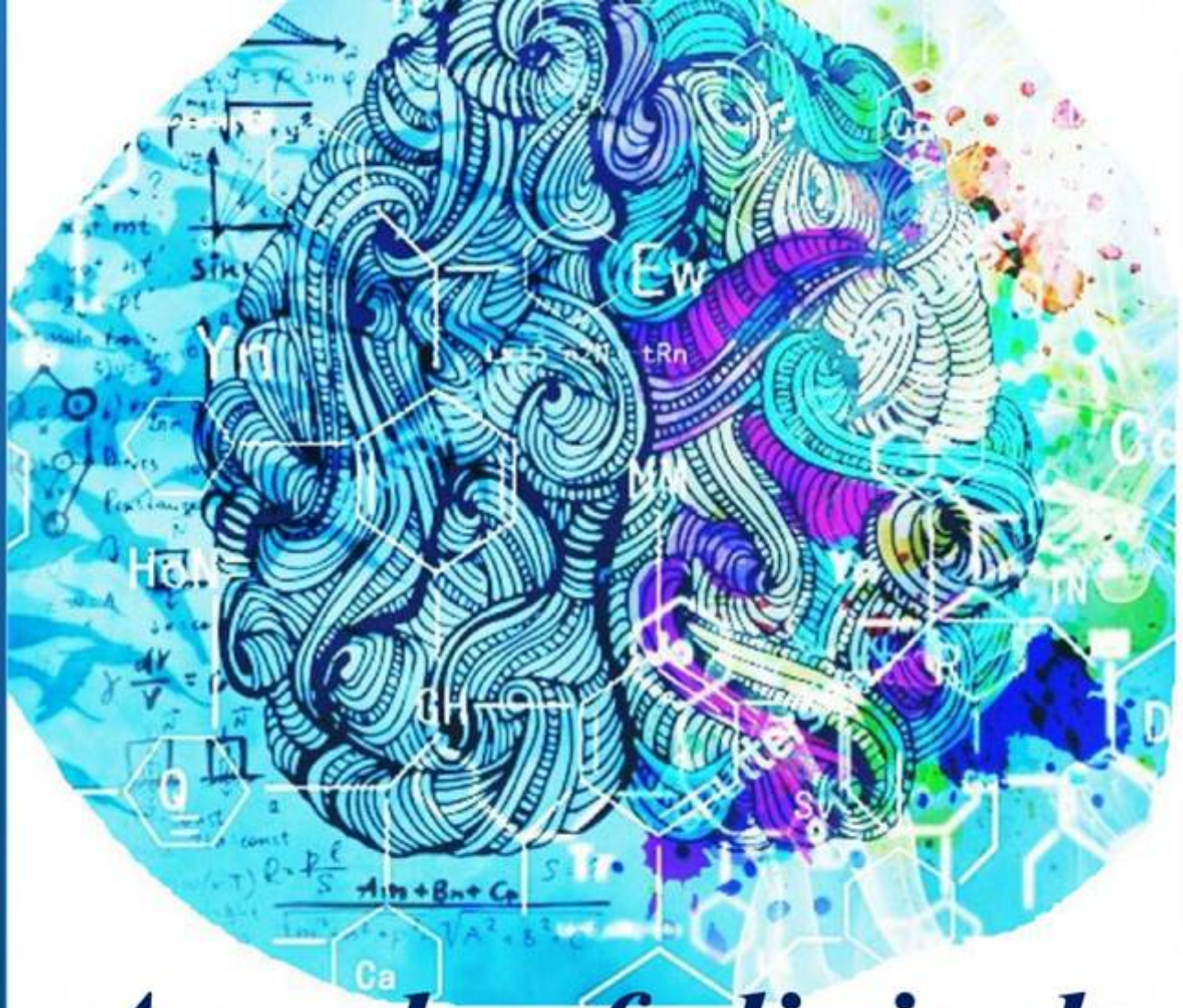

Annals of clinical disciplines

VOLUME 1, ISSUE 2

2024

https://tadqiqot.uz/index.php/spjacd

БУХОРО ДАВЛАТ ТИББИЁТ ИНСТИТУТИ

ISSN 0000-0000

DOI Journal 10.26739/0000-0000

ANNALS OF CLINICAL DISCIPLINE

1 ЖИЛД, 2 СОН

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН

ТОМ 1, НОМЕР 2

КЛИНИК ФАНЛАР ЙИЛНОМАСИ

VOLUME 1, ISSUE 2

ТОШКЕНТ-2024

ANNALS OF CLINICAL DISCIPLINE

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН | КЛИНИК ФАНЛАР ЙИЛНОМАСИ

№2 (2024) DOI http://dx.doi.org/10.26739/0000-0000-2024-2

© **Page Maker | Верстка | Саҳифаловчи:** Хуршид Мирзахмедов

О журнале
Журнал зарегистрирован в Агентство информации и массовых коммуникаций при Администрации Президента Республики Узбекистан
№ С-239963 от 14 марта 2024 года

Адрес редакции: Республика Узбекистан, 200114, г. Бухара, ул. Гиждуван, 23
Телефон: +998(65)2230050
Сайт: https://tadqiqot.uz/index.php/spjacd
e-mail: abumkur14@gmail.com

МУНДАРИЖА | СОДЕРЖАНИЕ | CONTENT

SCIENTIFIC AND PRACTICAL JOURNAL
ANNALS OF CLINICAL DISCIPLINE
АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН
КЛИНИК ФАНЛАР ЙИЛНОМАСИ
ISSN: 0000-0000
www.tadqiqot.uz
BUKHARA STATE MEDICAL INSTITUTE

Ergashov Bobir Bahodirovich
Bukhara State Medical Institute, Bukhara, Uzbekistan.

CHEMOTHERAPY AND CARDIAC ARRHYTHMIAS

http://dx.doi.org/10.5281/zenodo.12788582

ANNOTATION

Arrhythmias are often detected in cancer patients receiving treatment. The proposed review examines rhythm disturbances by the type of tachyarrhythmias, most often found in cancer patients, as well as their features, characteristic directly for oncological diseases and emphasizes the importance of ECG monitoring for early diagnosis, treatment and monitoring of this cohort of patients who are more susceptible to the development of proarrhythmia. Oncologists should be fully aware of possible cardiac arrhythmias and close cooperation between cardiologists and oncologists will lead to better stratification of the risk of developing cardiovascular diseases, monitoring and treatment.

Keywords: tachyarrhythmia, oncology, chemotherapy, arrhythmogenic effect, supraventricular tachycardia, ventricular tachycardia, cardiooncology, cardiotoxicity.

Эргашов Бобир Баходирович
Бухарский государственный медицинский институт, Бухара, Узбекистан.

ХИМИОТЕРАПИЯ И НАРУШЕНИЯ СЕРДЕЧНОГО РИТМА

АННОТАЦИЯ

Аритмии часто выявляются у онкологических больных, проходящих лечение. В предлагаемом обзоре рассматриваются нарушения ритма по типу тахиаритмий, наиболее часто встречающиеся у онкологических больных, а также их особенности, характерные непосредственно для онкологических заболеваний и подчеркивается важность мониторинга ЭКГ для ранней диагностики, лечения и наблюдения за этой когортой пациентов, которые более подвержены развитию проаритмии. Онкологи должны быть полностью осведомлены о возможных нарушениях сердечного ритма и тесное сотрудничество между кардиологами и онкологами приведет к лучшей стратификации риска развития сердечно-сосудистых заболеваний, мониторингу и лечению.

Ключевые слова: тахиаритмия, онкология, химиотерапия, аритмогенный эффект, наджелудочковая тахикардия, желудочковая тахикардия, кардиоонкология, кардиотоксичность.

Ergashov Bobir Bahodirovich
Buxoro Davlat Tibbiyot Instituti, Buxoro, O'zbekiston.

KIMYOTERAPIYA VA YURAK RITMINING BUZILISHI

ANNOTATSIYA

Aritmiya ko'pincha davolanayotgan saraton kasalligida aniqlanadi. Taklif etilayotgan maqolada, ko'pincha, saraton kasalligida uchraydigan taxiaritmiya turi bo'yicha ritm buzilishlarini, shuningdek ularning onkologik kasalliklar uchun xarakterli xususiyatlarini o'rganilgan hamda proaritmiya rivojlanishiga ko'proq moyil bo'lgan bemorlarning ushbu kohortasini erta tashxislash, davolash va monitoring qilish uchun EKG monitoringining muhimligini ta'kidlaydi. Onkologlar sodir bo`lishi mumkin bo'lgan yurak aritmiyalaridan to'liq xabardor bo'lishlari kerak va kardiologlar hamda onkologlar o'rtasidagi yaqin hamkorlik yurak-qon tomir kasalliklarini rivojlanish xavfini yaxshiroq tabaqalashtirishga, boshqarish va davolashga olib keladi.

Kalit so'zlar: taxiaritmiya, onkologiya, kimyoterapiya, aritmogen ta'sir, supraventrikulyar taxikardiya, qorincha taxikardiyasi, kardioonkologiya, kardiotoksiklik.

Introduction. Cardiac arrhythmias are often noted in cancer patients. According to various estimates – in 16-36% of cases. Detectability depends on the thoroughness of monitoring the heart rhythm. Most of the arrhythmias, as a rule, are transient in cancer patients. A normal sinus rhythm, registered once on an ECG or with short monitoring, does not exclude the presence of hidden or even life-threatening disturbances of the rhythm and conduction of the heart. In the presence of clinical symptoms suspected of arrhythmia, but without explicit detection during hospitalization, it is necessary to make every effort to search for a particular cardiac arrhythmia. At the same time, it should be remembered that sometimes we are dealing with "pseudo-arrhythmia" by which we must understand artifacts or incorrect interpretations of ECG data. Chronic lung diseases, pleural or pericardial effusion, lung resection and radiation therapy of the chest or mediastinal organs, displacement of the heart during respiration and fibrosis can lead to positional changes in the electrical axis of the heart, RR intervals, QT interval, amplitude and width of the QRS complex, which can easily be mistaken for arrhythmia [1-4].

Arrhythmias can be classified by their electrophysiological mechanisms or by their underlying causes. Primary arrhythmias are caused by specific heart pathology, while secondary arrhythmias result from metabolic imbalances without obvious signs of structural heart issues. This classification is somewhat arbitrary, as a patient with a weakened heart due to illness or aggressive treatment may be more vulnerable to metabolic disturbances and environmental factors that can also trigger arrhythmias. Even though cardiac arrhythmias are most often classified according to their electrophysiological mechanisms, they can also be divided by etiological factors (the cause of arrhythmia). Arrhythmias of cardiac origin (primary) occur due to a particular heart pathology. The secondary genesis of cardiac arrhythmias, on the contrary, is due to metabolic imbalance, without obvious signs of structural pathology of the heart. This classification is rather conditional, because the heart of a patient weakened by a malignant neoplasm or aggressive treatment will be more sensitive to metabolic disorders and environmental factors, which can also induce cardiac arrhythmias.

In primary arrhythmias, the substrate is located in the cardiac and pericardial structures. This may be a limited pathological focus, for example, a necrosis site after a myocardial infarction, or a diffusely altered pathological zone in cardiomyopathies and infiltrative diseases, such as amyloidosis. Coronary artery disease, arterial hypertension and myocardial hypertrophy, dilated cardiomyopathy and fibrosis often lead to rhythm disturbances, both in patients with and without cancer. However, several diseases are more common in patients with cancer: these are primary and metastatic heart tumors, amyloid infiltration, pericardial pathology, and cardiomyopathy associated with chemotherapy. Radiation therapy of the chest organs can also contribute to the occurrence of arrhythmia as a result of the formation of fibrosis sites, inflammation of the endocardium, myocardium, or pericardium, and progressive damage to the coronary arteries [5-8]. Secondary arrhythmias occur without any obvious structural pathology of the heart. Predisposing factors may be toxic effects of drugs, increased sympathetic tone, surgical interventions, hypoxia, release of inflammatory mediators and vasoactive kinins, and other metabolic disorders. The formation of an arrhythmogenic substrate can also be caused by the disintegration of the tumor and the cardiotoxic

effect of chemotherapy. A carcinoid tumor can cause both primary and secondary arrhythmias: the production of metabolically active mediators leads to the formation of secondary arrhythmias, while the formation of endocardial infiltrates and valve damage is associated with the occurrence of primary arrhythmias [1,2].

Cardiac arrhythmias and conduction disturbances resulting from antitumor medications may be associated with damage to cardiomyocytes (considered primary) and changes in metabolism due to the therapy (causing secondary arrhythmias). In cases of significant disruption to internal balance, it is crucial to monitor heart rhythm closely. If potentially life-threatening arrhythmias occur, the antitumor therapy plan should be modified. It's important to note that not only antitumor agents can cause heart rhythm disturbances. Cancer patients may also receive antibacterial drugs, psychotropic agents, antiemetics, hormonal drugs, electrolytes, and radiation therapy, all of which can trigger various cardiac arrhythmias. In the following section, we will discuss some types of cardiac arrhythmias [9-12].

Supraventricular arrhythmias. The substrate of supraventricular arrhythmia (SVT) is positioned above the level of the atrioventricular node (AV node). SVT can be either permanent or paroxysmal. Symptoms are influenced by the frequency of ventricular contractions, the duration of arrhythmia, and the extent of decrease in cardiac output during tachycardia. SVT can present with palpitations, pulsation in the neck, chest discomfort, shortness of breath, increased sweating, progressive heart failure, dizziness, and fainting. These complaints are frequently misattributed to panic attacks prior to the identification of supraventricular rhythm disturbances.

Paroxysmal supraventricular tachycardia (SVT) is quite common in cancer patients, especially as atrial fibrillation (AF). SVT mainly occurs in older patients with a history of arterial hypertension and in cases of disrupted homeostasis, such as multiple organ pathology, hemodynamic and metabolic imbalances, and increased catecholamine levels. Aggressive chemotherapy and stem cell transplantation have also been linked to the occurrence of SVT. Additionally, radiation therapy administered outside the heart's location, as seen in patients with cervical cancer, can provoke SVT [3,7].

Sinus tachycardia is the most common type of tachycardia in cancer patients, although it is often overlooked. In sinus tachycardia, an ECG will always show a P wave with a morphology identical to the normal sinus rhythm. The key characteristic of sinus tachycardia is its gradual onset and cessation, which helps to differentiate it from supraventricular tachycardia (SVT). The reasons for the development of this arrhythmia in cancer patients are typically related to factors such as pain, anxiety, fever, anemia, intoxication, hypovolemia, hypotension, and pulmonary embolism. As sinus tachycardia is a physiological response of the body to stress, treatment with medications like beta-blockers is generally not justified and can potentially worsen the patient's condition. It is crucial to identify the cause of sinus tachycardia in an oncological patient and address it, rather than simply trying to manage the elevated heart rate.

Supraventricular extrasystole (SE). Supraventricular extrasystole is the most common type of supraventricular arrhythmia. In most cases, these arrhythmias are harmless and asymptomatic, and do not require treatment unless there are complaints of palpitations. They are common in the population and can occur after chemotherapy, during stress, with anemia, arteriovenous shunting of blood in a tumor, or thyrotoxicosis. They may also be associated with the development of a hyperadrenergic condition.

Paroxysmal supraventricular tachycardia (SVT) is a broad group of arrhythmias with various mechanisms of occurrence. Unlike sinus tachycardia, they are characterized by sudden onset and cessation, without the phenomenon of "warming up" and "fading". In this type of arrhythmia, the frequency of ventricular contractions typically ranges from 100 to 300 beats per minute with equal R-R intervals.

Reciprocal paroxysmal supraventricular tachycardias (SVTs) include atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reentrant tachycardia (AVRT), and occasionally sinus node reentrant tachycardia and atrial tachycardia. These tachyarrhythmias occur due to an abnormal conducting system, which involves two or more pathways with different pulse rates and

refractory periods. An essential condition for inducing the arrhythmia is the ability to conduct the pulse forward along one pathway and backward along another, leading to the formation of a re-entry mechanism.

These pathways can be anatomically separated (macro re-entry), as seen in Wolff-Parkinson-White syndrome (WPW), or localized in one anatomical zone (micro re-entry), such as in the AV node in AVNRT.

Arrhythmias are often triggered by premature heartbeats occurring when one pathway is still in its refractory period while the other has recovered and can depolarize. In the presence of a substrate for SVT, an increase in catecholamine levels can lead to more frequent arrhythmia episodes due to changes in the electrical properties of the re-entry pathways and an increase in premature heartbeats. Therefore, SVT may first manifest in oncology patients after major surgery or during intensive chemotherapy. The most common SVT is AVURT. The re-entry chain in this type of arrhythmia is localized inside or in the immediate vicinity of the atrioventricular node. The frequency of ventricular contractions in arrhythmia is 150-250 beats per minute and Fig. 2. Supraventricular tachycardia negative P waves are visible on the ECG in the terminal part of the QRS complex (usually in the II lead).

AVRT is a type of abnormal heart rhythm with two pathways, one through the AV node and the other through an additional atrioventricular junction (DAVS). Normally, impulses can only travel in a retrograde direction, which means they cannot be detected on a regular ECG during normal sinus rhythm because the impulses from the atrium to the ventricles typically travel through the AV node (concealed pathway). However, if the impulse travels in an antegrade direction through the pathway, it can cause ventricular preexcitation, which shows up on the ECG as a widened QRS complex due to a delta wave, indicating overt pathway involvement. In this situation, tachycardia may cause retrograde P waves to appear after ventricular complexes.

The WPW syndrome requires special attention for two unique reasons. Firstly, during AVRT, if the pulse passes forward along the pathway, the QRS complex widens and a broad-complex tachycardia (antidromic AVRT) will appear on the ECG, which can be mistaken for ventricular tachycardia. Additionally, when atrial fibrillation occurs, the rapid forward passage of pulses along the pathway (non-incremental conduction) can cause an excessively high rate of ventricular activation, leading to ventricular tachyarrhythmia (even ventricular fibrillation) and sudden cardiac death. In this case, drugs that inhibit conduction in the AV node, such as digoxin or beta-blockers, are not recommended, as they may paradoxically increase the frequency of the ventricular response due to the shortening of the refractory period of the pathway. The preferred drugs for managing arrhythmia in this situation are Novocainamide, Amiodarone, or electrical pulse therapy [9,10].

Several cases of Wolff-Parkinson-White (WPW) syndrome have been reported in pediatric patients with tuberous sclerosis (Bourneville's disease), leading to the development of cardiac rhabdomyomas. If these tumors can conduct impulses similar to cardiomyocytes and are situated between the atrium and ventricle, crossing the fibrous ring of the heart, it can create conditions for the formation of macro re-entry. While the theory linking ventricular preexcitation to rhabdomyoma is debated, a correlation has been found between the tumor's location and the measured pressure during intracardiac electrophysiological examination. It's worth noting that the elimination of AVRT (atrioventricular reentrant tachycardia) occurred with both tumor resection and radiofrequency ablation of the DAVS (dual atrioventricular nodal pathways), and there were also instances of spontaneous cessation of arrhythmic syndrome.

Multifocal atrial tachycardia (MAT) is characterized by an irregular heartbeat of 100-300 beats per minute with 1:1 ventricular conduction and at least three different morphologies of the P wave, along with the absence of a dominant P wave. Differential diagnosis is carried out with sinus tachycardia with frequent supraventricular extrasystoles and atrial fibrillation. All these rhythm disturbances are due to an increased threshold of excitability of atrial tissue. It's worth noting that MAT is often transformed into atrial fibrillation. However, it is necessary to clearly distinguish

between these two types of arrhythmias, as the approaches to their treatment are completely different.

Ectopic atrial tachycardia is a rhythmic tachycardia with an atrial rate of 100-220 beats per minute and a P wave morphology different from the normal sinus rhythm. Cases of ectopic atrial tachycardia with atrial leiomyosarcoma and with the use of ifosfamide are described.

Atrial flutter and fibrillation . The cause of atrial fibrillation (AF) and atrial flutter (AFL) is an electrical and mechanical imbalance of the atria. Although these arrhythmias have different clinical aspects and treatment strategies, they share many similarities. As mentioned earlier, these arrhythmias are common in cancer patients. During both the occurrence of the arrhythmia and after the restoration of the sinus rhythm, the atria are unable to work effectively (there is no atrial systole). This leads to blood stagnation in the atria, creating conditions for the formation of atrial thrombi and subsequently resulting in thromboembolic complications. The most frequent site for the formation of blood clots is the left atrial appendage.

On an ECG, a typical atrial flutter is represented by regular atrial activity (F waves) with a frequency of about 300 beats per minute. The fluttering waves are asymmetric and have a sawtooth configuration, which is most clearly seen in the second lead.

Atrial fibrillation (AF) can be easily identified on an ECG. It is characterized by irregular atrial and ventricular activity. The ventricular response frequency depends on the conduction in the atrioventricular node and can vary widely.

The clinical symptoms of AF are diverse in both cancer patients and those without cancer. It can be an incidental finding during an examination in asymptomatic cases, or it can lead to severe hemodynamic instability and acute heart failure. Symptoms typically depend on the frequency of ventricular contractions and the presence of atrioventricular dissynchrony. AF episodes are usually accompanied by palpitations and shortness of breath.

Atrial fibrillation and flutter are usually a manifestation of underlying heart disease. Conditions that cause an increase in atrial volume can lead to increased atrial pressure, resulting in atrial arrhythmias. Other risk factors include age, hypertension, lung diseases, hyperthyroidism, surgical interventions, and other conditions associated with elevated catecholamine levels. In cases of first-time AF or atrial flutter in cancer patients, it is important to rule out other potential causes of arrhythmia (such as pulmonary embolism, acute or chronic pericarditis, infection, hyperthyroidism, or other metabolic disorders), regardless of the presence of underlying heart disease.

Atrial fibrillation (AF) is linked to an increase in the level of C-reactive protein, indicating a potential role of systemic inflammatory processes in the development of an arrhythmia substrate in the left atrium. This theory was directly demonstrated in cancer patients. Examination of patients with colorectal cancer, in the absence of significant structural heart pathology and other risk factors, revealed a threefold increase in the incidence of AF compared to the control group. After elective colectomy, AF developed in 4.4% of patients, with a higher percentage in the group with laparotomic surgical access. Additionally, an elevated level of neutrophils on the first day after surgery was an independent predictor of AF development. The presumed mechanism for the high frequency of AF may be systemic inflammation.

Furthermore, many chemotherapeutic drugs increase the likelihood of this arrhythmia. For example, gemcitabine, docetaxel, alemtuzumab, 5-fluorouracil, doxorubicin, cisplatin, and melphalan have been identified. While their usage in the genesis of arrhythmia may involve a cardiotoxic effect, the role of systemic inflammation is also significant [11,13].

Ventricular arrhythmias are a group of rhythm disorders that include ventricular extrasystoles, trigger, and reciprocal (by the mechanism of re-entry) ventricular tachycardia. These arrhythmias are characterized by wide QRS complexes on the ECG.

The risk of ventricular arrhythmias increases with cancer. Patients in this group often have structural heart disease and additional risk factors, such as the use of cardiotoxic antitumor drugs, which may be arrhythmogenic. Hormonal and metabolic imbalances can also lead to ventricular arrhythmias in these patients.

Ventricular extrasystole (VE) is the most common ventricular arrhythmia. It is often found in healthy individuals without structural heart disease and does not pose any danger in such cases. However, in cancer patients, the number of VE may increase due to a disruption in homeostasis. Only when there is an underlying heart disease, VE is associated with the development of life-threatening ventricular arrhythmias. Clinically, VE may be asymptomatic, detected incidentally during an ECG or XM-ECG, or present as palpitations or dizziness, particularly with frequent extrasystoles leading to a decrease in the number of effective ventricular contractions. Previously, VE was considered a dangerous rhythm disorder requiring treatment. However, recent studies have shown that aggressive attempts to suppress ectopia may be more dangerous than the arrhythmia itself. Therefore, VE does not always require specific therapy, except for patients with poor arrhythmia tolerance. Beta-blockers are the drugs of choice in such cases, as they are generally effective and do not have a proarrhythmic effect [13,15].

Ventricular tachycardia (VT) is a type of fast heart rhythm with wide QRS complexes, occurring at a rate of more than 120 beats per minute (as shown in Figure 7). It originates in the ventricular myocardium. Ventricular tachycardia can disrupt normal cardiac output and may lead to sudden cardiac death, making it a more life-threatening rhythm disturbance than supraventricular tachycardia (SVT). Another type of VT is called Torsades de pointes, which has specific risk factors, different ECG morphology, and requires different treatment strategies. VT typically starts with a premature ventricular contraction (PVC) during a vulnerable period of the cardiac cycle.

Several chemotherapy drugs increase the risk of gastrointestinal tract issues, such as interleukin, doxorubicin, rituximab, trastuzumab, and thalidomide.

Ventricular fibrillation (VF) is identified by chaotic, low-amplitude electrical activity on an ECG. During this arrhythmia, the heart does not have any mechanical activity, which means that organ perfusion stops. If the sinus rhythm is not restored within a few minutes (either through spontaneous relief of the arrhythmia or electrical cardioversion), complete hemodynamic collapse and death are inevitable. Risk factors for VF are similar to those for ventricular tachycardia (VT), and it's important to be aware of the potential transformation of stable high-frequency VT into VF in cases of coronary circulatory insufficiency or severe metabolic disorders (such as severe hypoxia). Treatment for VF follows the protocol for stable hemodynamically significant VT. Unfortunately, the effectiveness of resuscitation in cancer patients is lower than in the general population.

Tachycardia of the "Pirouette" type is a unique form of polymorphic ventricular tachycardia (VT) that is linked to the lengthening of the QT interval, whether due to congenital or acquired reasons. Torsades de pointes, which translates to "twisting of the points," refers to the constantly changing shape, size, and direction of the ventricular complexes, appearing as if they are twisting around a central point. Although this arrhythmia typically stops on its own, it can potentially transform into a stable VT or ventricular fibrillation (VF), making it a life-threatening rhythm disorder. The ventricular contractions occur at a frequency of about 200-250 beats per minute.

The QT interval is a measurement taken from one of the ECG leads, representing the duration from the beginning of the QRS complex to the end of the T wave, located on the isoline. Since the QT interval varies with heart rate, a corrected QT interval (QTc) is used to estimate it: the QT interval divided by the square root of the RR interval measured in milliseconds. Additionally, the normal value of the QT interval depends on the patient's gender (typically 340-450 msec for women and 340-430 msec for men). Prolongation of the QT interval to more than 500 ms, irrespective of the patient's gender, is associated with a high risk of developing pirouette tachycardia. An increase in the QT interval by 60 msec compared to the initial measurement also increases the risk of life-threatening arrhythmias.

When using drug therapy, if the QT interval is prolonged to more than 500 msec, it is crucial to assess all the risks and adjust the therapy. This may involve prescribing an alternative drug, correcting hypokalemia, and evaluating the interaction of all drugs being used.

The risk factors for pirouette tachycardia and prolonged QT interval are similar. This article does not cover the congenital forms of prolonged QT interval, which are channelopathies. Prolongation of the QT interval is common in cancer patients, often occurring when chemotherapy drugs are prescribed. It's important to note that concomitant pathologies such as diabetes mellitus, bradyarrhythmias, myocardial ischemia, heart failure, infections, cerebral circulation disorders, cachexia, hypothermia, electrolyte imbalance, and polypharmacy of drugs make these patients particularly susceptible. As a result, patients with cancer and concomitant cardiovascular pathology are at high risk of developing life-threatening tachyarrhythmias.

When prescribing the drugs listed in the table, it's important to monitor the ECG over time. Prolongation of the QT interval to more than 25% of the initial value or more than 500 msec is associated with a high risk of pirouette tachycardia. The exception to this is Amiodarone, which almost always leads to an extension of the QT interval without contributing to the occurrence of VT.

Implantable cardioverters-defibrillators.

Implantable cardioverter defibrillators (ICDs) are necessary for primary and secondary prevention of SCD, including ventricular tachyarrhythmias. The data of modern studies have demonstrated a decrease in mortality when using ICD in patients with ischemic and non-ischemic cardiomyopathy as part of the primary prevention of SCD. Indications for implantation of these devices are described in detail in the recommendations of the American College of Cardiology/ American Heart Association (AHA-ACC) and the European Society of Cardiology (ESC). For the relief of frequent ventricular tachyarrhythmias, ICTs are successfully used in patients with heart neoplasms, such as rhabdomyoma and lipoma. In general, ICD recommendations do not differ for oncological and non-oncological patients, however, there are several features [3-8].

Firstly, studies that included patients with cardiomyopathy and the NYHA IV FC heart failure clinic did not demonstrate the benefits of ICD therapy as part of the primary prevention of SCD in mortality.

This category of patients has an extremely unfavorable prognosis and the probability of death from heart failure and comorbid pathology is higher than from ventricular tachyarrhythmias. Similar results can be expected in patients with oncological pathology; even if there are indications for implantation of a cardioverter defibrillator, if the expected life expectancy is less than 1 year, the probability of dying from SCD due to ventricular tachyarrhythmias becomes unlikely. Also, with the terminal stage of the oncological process and a probable life expectancy of more than 1 year, ICDs will practically not affect the quality of life, so they are rarely used in this cohort of patients.

Secondly, several chemotherapeutic drugs contribute to a decrease in the left ventricular ejection fraction during treatment, which can recover in the future (for example, when trastuzumab is prescribed). Potentially reversible causes of LV dysfunction should be evaluated, because in some cases the use of ICD may be justified (for example, if anthracycline therapy was carried out several years ago, and severe progressive left ventricular dysfunction is currently taking place).

Thirdly, the presence of an ICD complicates the diagnosis and treatment of cancer patients. A particularly urgent problem in this group of patients is the inability to perform MRI after implantation of the device. But modern technologies allow us to overcome this barrier: implantation of MRI-compatible devices is currently possible. Also, special precautions are necessary when performing electrocoagulation during surgical manipulations, and radiation therapy.

Conclusion. Cardiovascular diseases and cancer are still the leading causes of death, but advances in diagnosis and treatment have significantly reduced mortality rates for both. New therapies have turned many cancers into chronic conditions, leading to a growing number of cancer survivors. However, some cancer treatments like chemotherapy and radiation can damage the heart. This makes cardiology particularly important in cancer care. Initially, the focus was on heart failure caused by chemotherapy, but it's also important to consider heart rhythm and conduction issues in these patients.

Cardiologists play a crucial role in identifying patients at high risk of developing heart problems before they start potentially harmful cancer treatments. Continuous monitoring of heart

health is essential for timely preventive and therapeutic interventions for cancer patients receiving chemotherapy.

References

1. Abdullaeva U.K., Sobirova G.N., Karimov M.M., Aslonova I.J The prevalence and possibilities of prevention of noncardial gastric cancer in the Bukhara region // American journal of medicine and medical sciences. 2020. Vol. 10. Iss. 9. P. 679-681.
2. Abdullaeva U.K. Predicting the risk of atrophic transformation in chronic gastritis using serum pepsinogen // World journal of pharmaceutical research, Faculty of Pharmacy Medical University, Bulgaria. 2019. Vol. 8. Iss. 13. P. 219-228.
3. Abdullaeva U.K., Shadjanova N.S. Using the OLGA system in chronic atrophic gastritis // New day in medicine. 2020. Iss. 13. P. 9-12.
4. Abdullayeva U.K. The value of interactive teaching methods in improving the level of clinical knowledge of students // Medical education and professional development. 2019. Vol. 1. Iss. 33. P. 29-33. (in Russian)
5. Aslonova I.Z., Tulyaganova F.M., Karimov M.M., Sobirova G.N., Abdullaeva U.K. Possibilities of serological diagnosis of atrophic processes of the gastric mucosa // European Journal of Molecular & Clinical Medicine. 2021. Vol. 7. Iss. 11. P. 2955-2960.
6. Bahodirovich, Ergashov Bobir A CAUSAL RELATIONSHIP OF ANTICANCER DRUGS WITH SPECIFIC ARRHYTHMIAS // Asian journal of pharmaceutical and biological research 10.2 (2021).
7. Bahodirovich, Ergashov Bobir. "TREATMENT AND PREVENTION OF ARRHYTHMIAS ASSOCIATED WITH ANTICANCER THERAPY." Asian journal of pharmaceutical and biological research 10.2 (2021).
8. Ergashov, B. B. A causal relationship of anticancer drugs with specific arrhythmias. Asian journal of Pharmaceutical and biological research 10.2 (2021): 55-55.
9. E.B. Bahodirovich, MR Barraevich, QL Kholmurodovich Modern concept of clinic and diagnosis of cardiovascular complications of anticancer therapy // British Medical Journal, 2021. Volume-1, No 2: 175-180.
10. Karimov M.M., Sobirova G.N., Abdullayeva U.K. Chronic gastritis and carcinogenesis issues // Herald of Pancreatic Club. 2021. Vol. 45. Iss. 4. P. 65-70.
11. Karimov M.M., Sobirova G.N., Abdullaeva U.K., Aslonova I.Z, Tulyaganova F.M. Possibilities of Serological Diagnosis of Atrophic Processes of the Gastric Mucosa // Annals of the Romanian Society for Cell Biology 2021. Vol. 25, Iss. 1. P. 6168-6174.
12. Mirzaeva D.B., Abdullaeva U.K., Boboeva R.R. The importance of interactive teaching methods in improving the level of clinical knowledge of students // Central Asian Problems of Modern Science and Education. 2019. Vol. 4, Iss. 2. P. 159-166.
13. Shamsutdinov A.S., Abdullaeva U.K., Akhmedova N.Sh. Determination of the level of pepsinogens in patients with chronic h. pylori associated gastritis // ACADEMICIA: An international multidisciplinary research journal.2021. Vol. 11, Iss. 2. P. 919-924.
14. Sobirova G.N., Abdullaeva U.K., Nosirova M.S., Aslonova I.J. Evaluation of the gastrointestinal mucosa by the OLGA system in chronic atrophic gastritis // Journal of Critical Reviews. 2020. Vol. 7. Iss. 2. P. 409-413
15. Sobirova G.N., Abdullaeva U.K. Chronic gastritis and carcinogenesis issues // Central Asian Problems of Modern Science and Education. 2019. Vol. 4. Iss. 2. P. 159-172.

Tadqiqot uz

БУХОРО ДАВЛАТ ТИББИЁТ ИНСТИТУТИ

ANNALS OF CLINICAL DISCIPLINE

1 ЖИЛД, 2 СОН

АННАЛЫ КЛИНИЧЕСКИХ ДИСЦИПЛИН

ТОМ 1, НОМЕР 2

КЛИНИК ФАНЛАР ЙИЛНОМАСИ

VOLUME 1, ISSUE 2

Научно-практический журнал по всем
направлениям медицины
основан в 2024 году
Бухарским государственным
медицинским институтом
Выходит один раз в 3 месяца
Учредитель Бухарский государственный
медицинский институт