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Cardiovascular complications of burn disease (literature review)

Introduction. Burns are the fourth most common type of injury in the world after road traffic injuries, falls and interpersonal violence. Cardiovascular complications are mostly associated with severe burns, infectious complications with increased length of hospital stay and increased mortality.Patients who have survived burn trauma suffer from negative consequences for the health of the cardiovascular system. **Evidence of obtaining information.** A retrospective information search of literature sources was performed using the spatial-vector model of the descriptor system based on classifiers, and supplemented by manual search of the literature lists of included articles. **Materials and methods.** Using the information search engines Scopus, Google Scholar, CrossRef and PubMed, 40 relevant scientific sources were selected and analyzed, of which 97.5% were published in the last 10 years and 72.5% in the last 5 years. **Results and discussion.** Burn survivors demonstrate chronotropic incompetence. Arrhythmias and heart blocks can occur with any size of burn in approximately 35% of patients. Patients with severe burns have a high incidence of myocardial damage, which can be predicted by the total area of the burn. Heart failure plays a significant role in the clinical picture of burn disease. Diagnosis of myocardial infarction in burn patients can be quite difficult. Traditional markers of myocardial damage in the presence of severe burns are often unreliable. The incidence of deep vein thrombosis was 0.48%, pulmonary embolism – 0.18%, and venous thromboembolism – 0.61%. Patients with a history of burn injury are 50% more likely to develop cardiovascular complications, which can occur 3 years or more later. **Conclusions.** Cardiovascular disorders occur both immediately after burn injury and years after the episode. Hypodiagnosis of cardiovascular disorders in burn disease contributes to an increase in the incidence of delayed complications.

Key words: burns, burn disease, arrhythmias, myocardial infarction, diagnosis, thromboembolic complications.

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Серцево-судинні ускладнення опікової хвороби (літературний огляд)

Вступ. Опіки є четвертим за поширеністю в світі типом травм у світі після дорожньо-транспортних травм, падінь і міжособистісного насильства. Серцево-судинні ускладнення здебільшого пов'язані з важкими опіками, інфекційними ускладненнями зі збільшенням тривалості госпіталізації та зростанням смертності. Пацієнти, які пережили опікову травму, потерпають від негативні наслідків для здоров'я серцево-судинної системи. Пацієнти, які пережили опікову травму, потерпають від негативні наслідків для здоров'я серцево-судинної системи. **Докази отримання інформації.** Ретроспективний інформаційний пошук літературних джерел

виконано за просторово-векторною моделлю дескрипторної системи на базі класифікаторів, та доповнено шляхом ручного пошуку переліків літератури включених статей. **Матеріали та методи**. Із залученням інформаційних пошукових систем Scopus, Google Scholar, CrossRef та PubMed було відібрано та проаналізовано 40 актуальних наукових джерела, з яких за останні 10 років – 97,5%, за останні 5 років – 72,5%. **Результати та їх обговорення.** Пацієнти, які пережили опік, демонструють хронотропну некомпетентність. Аритмії та блокади серця можуть виникати при будь-якому розмірі опіку приблизно у 35% хворих. Пацієнти з важкими опіками мають високу частоту пошкодження міокарду, яке можна передбачити за загальною площею опіку. Серцева недостатність відіграє значну роль у клінічній картині опікової хвороби. Діагностика ІМ в опікових пацієнтів може бути досить складною. Традиційні маркери ураження міокарду за наявності важкого опіку часто є ненадійними. Частота тромбозу глибоких вен становила 0,48%, тромбоемболії легеневої артерії – 0,18%, а венозних тромбоемболій – 0,61%. В пацієнтів з опіковою травмою в анамнезі на 50% частіше, виникають серцево-судинні ускладнення, які можуть виникати через 3 роки і більше. **Висновки.** Серцево-судинні розлади виникають як безпосередньо після опікової травми, так і через роки після епізоду. Гіподіагностика серцево-судинних розладів при опіковій хворобі сприяє зростанню частоти випадків відстрокованих ускладнень.

Ключові слова: опіки, опікова хвороба, аритмії, інфаркт міокарду, діагностика, тромбоемболічні ускладнення.

Introduction. Burns are the fourth most common type of injury in the world after road traffic injuries, falls, and interpersonal violence [1,2]. Ukraine has one of the highest mortality rates for burn disease (BD) in the world – 4.55:100,000, which is second only to ten countries of the post-Soviet space and the African continent [3]. Cardiovascular complications (CVC) are mostly associated with severe burns, infectious complications with increased length of hospitalization and increased mortality [4].

In the acute phase of burn disease, cardiac symptoms are observed in only one third of patients, the frequency of cardiac disease in pediatric burns is quite low [5], and intensive efforts of medical professionals are primarily focused on short-term goals [6]. However, patients who have survived burn trauma suffer from profound long-term pathophysiological changes, which may have long-term negative consequences for cardiovascular health [7]. It is likely that many cardiovascular diseases develop without dramatic manifestations and cardiac arrests in patients in this case series and, as a result, are not diagnosed. However, the problem of cardiac pathology in combustiology remains quite relevant and is due to the significant increase in CVC during the years after burn trauma [8].

Evidence of obtaining information. Articles of choice were included in the study if they (1) were published in Ukrainian, English and Chinese, (2) were related to cardiac complications of burn disease, (3) used an observational study design (cross-sectional or cohort). A retrospective information search of literature sources was performed using a spatial-vector model of a descriptor system based on classifiers, and supplemented by a manual search of the literature lists of included articles.

Materials and methods. The literature search was carried out using the information search engines Scopus, Google Scholar, CrossRef and PubMed, which was also supplemented by a manual search of the articles used using the search terms: burn disease; cardiac complications of burn disease; diagnosis of cardiac complications of burn disease. 40 relevant scientific sources were selected and analyzed, of which 97.5% were published in the last 10 years, and 72.5% in the last 5 years.

Results and discussion. Patients who have survived burns demonstrate chronotropic incompetence [9]. Arrhythmias and heart blocks can occur with any degree of burn in approximately 35% of patients [5, 10].

Heart rate (HR) in burns is always increased due to the fact that changes in heart rate are a universal adaptive response of the body to injury. With small lesions up to 10% of the total body surface area (TBSA), only moderate tachycardia occurs and an increase in blood pressure (BP) may be observed. With more extensive and deep burns (over 10–15% TBSA), shortness of breath, palpitations, chest pain, acrocyanosis, tachycardia and weakening of the first heart sound at the apex often occur in the first period of BD. BP increases slightly during the erectile phase of burn shock and decreases in the torpid phase [10, 11]. In severe burns, the HR was significantly increased. The average HR on admission is 170±4% of normal values and reaches a maximum in the fourth week – 181±7%. By discharge, the average heart rate decreases to 155±3% of normal. In some cases, indicating an unfavorable prognosis, refractory arterial hypotension develops in the early stages of BD. BP in burn shock (BS) usually drops to normal values and often remains at the same level during the subsequent period of the disease [10, 11]. In the toxemia phase, shortness of breath, tachycardia, palpitations and chest pain may remain. Systolic murmur at the apex, decreased blood pressure and dilation of the heart borders are determined. During the period of septicotoxemia, pericarditis or endocarditis often develops, which can cause the development and progression of heart failure (HF). In these cases, the use of large infusion volumes can worsen the condition of patients due to a significant increase in pre- and afterload [12].

Cardiac rhythm and intracardiac conduction disorders are usually associated with generalized pathophysiological disorders in the acute stage of burn injury and lead to damage to the structure and function of cardiomyocytes [10].

According to ECG data in BD, cardiac disorders were distributed as follows: sinus tachycardia – 76.2%; sinus arrhythmia – 23.7%; extrasystole 10.9%; paroxysmal tachycardia 2.6%; atrial fibrillation 0.6%; atrial flutter 0.6%; atrioventricular block – 2.7%; intraventricular conduction disorders – 16.0%; myocardial ischemia – 48.7%; cardiac overload – 22.4% [10].

Extrasystoles are supraventricular, ventricular, or combined, both single and frequent, and sometimes acquire complex forms. Paroxysms of supraventricular tachycardia occur at all stages of burn disease, they are short-lived and disappear with pathogenetic therapy of BD. But in the vast majority of patients they are transient and do not lead to the development of acute cardiovascular failure [10].

Among cases of atrial fibrillation, the maximum area of damage was 60% TBSA, the operation time was up to 400 minutes, and atrial fibrillation occurred due to extensive bleeding and insufficient rehydration, which caused excitation of the sympathetic system [12, 13].

Burn patients may be at higher risk of developing new-onset atrial fibrillation (NOAF) due to burn shock and

fluid resuscitation, inflammatory response to burn injury, multiple surgeries, and sepsis, among other risk factors. In burns with a mean total body surface area of up to 22%, NOAF occurred by day 10 of hospitalization. 65% of cases were perioperative NOAF, which occurred on average 4 days after surgery [12, 14].

Patients with atrial fibrillation (AF) had hypertension, significantly more severe burn severity, and larger burn area than the non-AF group, which are now recognized as independent predictors of perioperative AF in BD ($p < 0.001$) [13].

Risk factors for atrial fibrillation are divided into modifiable (dyshydria, acid-base imbalance, hypotension) and unmodifiable (ischemic heart disease, myocardial infarction). The majority (73%) of patients with NOAF had at least one modifiable risk factor [12, 13].

Common risk factors for AF include steroid use (49%), vasopressors/inotropes (43%), presence of vascular catheters (38%), electrolyte abnormalities (59%), renal dysfunction (43%), and sepsis (65%) [14].

The risk of NOAF in patients with sepsis, severe sepsis, and septic shock is 8%, 10%, and 23%, respectively. This may be due to the systemic inflammatory response induced by sepsis, and inflammatory factors are involved in the initiation and maintenance of atrial fibrillation. In 42.8% of cases, AF occurred within 2 weeks after the burn. During this period, due to the presence of a necrotic scab and low anti-infective capacity, accompanied by reabsorption of edematous fluid with absorption of a large number of bacteria and toxins, a predisposition to sepsis is formed [12, 14]. Paroxysmal tachycardia and AF attacks mostly occurred with sepsis and endocarditis on the 30th – 68th day after the burn [10]. Patients with perioperative AF had longer artificial lung ventilation and a longer hospital stay [13].

Among the cardiac complications, dystrophic changes in the myocardium and intracardiac blockade were registered in 45.1% of patients [10]. In a clinical study, 79.2% of burn patients with cardiac pathology had an increase in the end-systolic diameter of the left ventricle 3 months after burn injury. Radiologically, this increase was small but statistically significant ($p < 0.015$) [15, 16]. On the 30th day after burn injury, pathomorphological experimental study revealed myocardial wasting ($p < 0.05$), significant histological hypotrophy of cardiomyocytes ($p < 0.05$), significant accumulation in the myocardium of molecular structures associated with cardiodepressive injury, and infiltration by granulocytes and monocytes, as well as significant left ventricular fibrosis and persistent changes in markers of cardiac dysfunction consistent with significant histological hypotrophy of cardiomyocytes [17, 18]. Exercise tolerance has been assessed in young adults who had sustained severe burns 5 to 15 years earlier in early childhood. Their findings have demonstrated persistent systolic and diastolic dysfunction, as well as evidence of myocardial fibrosis [16].

Myocardial dysfunction is usually characterized by delayed isovolemic relaxation, impaired contractility, and decreased left ventricular diastolic compliance. This dysfunction is often manifested by decreased cardiac output, which may be a consequence of hypovolemia and cardiac stress after burn injury [20]. 62% of pediatric patients with severe burns demonstrate evidence of systolic dysfunction during their stay in the intensive care unit (ICU). This is associated with an almost double duration of hospitalization compared with burn patients without systolic dysfunction [16]. More than half of patients with BD on day 1–2 of burn shock show evidence of circulatory insufficiency, which in the BS stage is associated with tachycardia of 96–150 beats/min. In the following periods, myocardial ischemia is determined in 42.9% of patients with localization in one or several areas of the heart. The duration of hypoxic myocardial disorders is from several days to 1–2 weeks, which in some cases requires observation by a cardiologist to exclude myocardial infarction. The contractile capacity of the heart ventricles decreases from the first hours after the burn and is maximally suppressed in the toxemia stage. Hypodynamia of the right ventricle myocardium is mainly observed due to increased resistance to blood flow in the pulmonary artery system [10].

Heart failure (HF) plays a significant role in the clinical picture of BD. In severe burns, electrolyte imbalance (hyper- or hypokalemia), ischemia and myocardial infarction, myocarditis or underlying cardiac pathology can cause a decrease in myocardial contractility, various conduction, rhythm, and HF disorders. These changes occur within 6 to 8 hours after injury, so the sooner measures are taken to prevent and compensate for them, the more likely a favorable course of BD and a decrease in the incidence of complications will be [10, 11].

Patients with severe burns have a high incidence of myocardial damage, which can be predicted from the total area of the burn. They are more likely to suffer from a decrease in effective blood volume, impaired tissue oxygenation, and shock damage to other organs [20].

Myocarditis in burn disease is found in 20–40% of cardiac pathologies [10]. Some cases of burn fulminant myocarditis are accompanied by cardiogenic shock and require extracorporeal membrane oxygenation and renal replacement therapy [21].

In severe burns, long-term cardiac dysfunction and reversible cardiomyopathy may develop [5, 22]. Rare cases of dilated cardiomyopathy have been reported [11]. Left ventricular systolic dysfunction, usually characterized by Takotsubo syndrome or regional wall motion abnormalities, can most often be observed in patients with burn sepsis and is associated with an increased risk of death [11, 23].

Signs of myocardial ischemia were found in 48.7% of all cardiac complications of BD patients aged 14 to 75 years. At the same time, patients under 45 years of age accounted for almost 2/3 of all cases of cardiac ischemia (more often trauma), and only 1/3 were over 60 years of age [10]. The burn cohort in the long term had higher rates of hospitalization for ischemic heart disease by 21%, heart failure by 129%, and cerebrovascular disease by 57% than the general population [11, 24].

A relatively rare but very serious complication of BD is acute coronary syndrome, including the development of myocardial infarction (MI), the frequency of which in BD is low, but is associated with high mortality [25]. Vasoconstriction of coronary arteries and arterioles is caused by stress factors and increased activity of sympathetic tone. Left ventricular systolic and diastolic function deteriorate, and coronary flow velocity reserve decreases, leading to microvascular dysfunction in the absence of coronary artery obstruction or stenosis

[11]. Several cases have been documented in which acute coronary syndrome occurred within 5 months of burn injury [26]. In addition to preexisting ischemic heart disease, burns to the trunk and airways, as well as lesions > 20% TBSA, have been associated with a higher incidence of MI in BD. Extensive burns are accompanied by a hypermetabolic state, manifested by hyperthermia, multiorgan dysfunction, increased oxygen consumption, and the release of inflammatory markers. In patients with airway burns, exposure to carbon monoxide can lead to myocardial damage. In addition to hemic hypoxia, carbon monoxide leads to increased platelet aggregation. The higher incidence of MI in trunk burns is due to two factors: first, they have a higher TBSA; second, they are associated with a higher incidence of inhalation injuries [25].

Compared with patients who did not develop MI, burn patients with MI were typically older men and had a higher prevalence of cardiovascular risk factors, such as hypertension, diabetes mellitus, AF, HF, chronic kidney disease, ischemic heart disease, peripheral arterial disease, and a history of acute stroke [26]. The presence of underlying cardiopulmonary disease increases the risk of myocardial infarction in burn patients by more than 6-fold.

The diagnosis of MI in burn patients can be challenging because they may not report chest pain because it is masked by analgesics or because they are distracted by the pain of the burn.

A high index of clinical suspicion and prompt electrocardiogram acquisition are essential for timely diagnosis and treatment, especially in patients with pre-existing coronary artery disease (CAD) and latent decompensation [25]. Routine 12-lead ECG and cardiac monitoring in patients with chest burns is complicated by at least three factors: inability to palpate due to increased sensitivity and pain in the affected areas; difficulty with electrode attachment and removal and changes in tissue impedance; and the likelihood of additional mechanical damage and infection of the affected areas. Electrode detachment can also be quite painful if they are tightly attached to the skin. It is recommended to use a sterile gel; the electrode itself can be made less “sticky” by wetting it with sterile water. Skin grafting staples can be used as percutaneous sensors [16, 27].

In BD, X-ray examination is most often used to assess the location of cardiac catheters and for primary pulmonary diagnosis (inhalation injury, pulmonary edema, or pneumonia). Cardiac magnetic resonance imaging (MRI) provides unique imaging resolution for assessing cardiac structure, myocardial perfusion, and myocardial viability and is recognized as the gold standard for assessing left and right ventricular volumes and ejection fractions [16].

Transthoracic (TTE) and transesophageal echocardiography (TEE) are excellent tools for distinguishing between cardiac and noncardiac causes of hemodynamic instability. Echocardiography (either TTE or TEE) rapidly provides multiple parameters, including atrial and ventricular size, wall thickness, left ventricular systolic function (ejection fraction), diastolic indices (early and late mitral flow peaks, E and A waves, respectively), and verifies other structural abnormalities (valve, shunt, and presence of exudates) over time [5, 16]. Measurement of heart rate variability (HRV) is a reliable biomarker of CVD risk. HRV abnormalities have been consistently associated with a wide range of adverse outcomes, including metabolic syndrome, psychological stress, and systemic inflammation. In OX, lower HRV has been recognized as a useful adjunctive marker of injury severity and sympathetic dominance and is associated with poorer outcomes, including all-cause mortality [28].

The use of a pulmonary artery catheter (PAC) provides continuous monitoring of central venous pressure, cardiac output (CV) or cardiac index (CI), systemic vascular resistance index (SVRI), pulmonary capillary wedge pressure, and oxygen delivery and consumption, but its invasive nature with a high incidence of cardiopulmonary complications limits its clinical utility [16].

Transpulmonary thermodilution (TPTD) is a less invasive alternative for measuring hemodynamic parameters such as CV, CI, and SVRI. The extravascular lung water index and total end-diastolic volume provide independent measures of fluid overload and preload. The latter two methods provide additional information but are not included in the diagnostic standard for BD because they have not been shown to significantly improve treatment outcomes [6,16]. Most biomarkers of myocardial injury that were primarily developed for the diagnosis and prognosis of acute myocardial infarction include cardiac troponin I or T, cardiac myosin light chain-1, or creatine phosphokinase (CPK). Previously, biomarkers such as CPK, lactate dehydrogenase, and creatine phosphokinase-MB have been used as indicators of cardiac injury. However, they lacked specificity for clinical use due to the extensive muscle and soft tissue damage in burn injuries [16, 19].

Thus, cardiac troponin-I (cTnI), a regulatory contractile protein specific for cardiac muscle, is a valid biomarker for detecting cardiac dysfunction. cTnI levels are detectable within the first two days after burn injury and again from the fifth day onwards, with peak values observed between 7 and 13 days after burn injury, which is likely related to early burn wound infection [16, 19, 25]. Patients with burns with more than 15% TBSA have elevated cardiac troponin-I levels as early as 3 hours after injury. A positive troponin test within 24 hours of burn injury is strongly correlated with an increased risk of acute CVC and mortality [16, 29]. However, elevated cardiac biomarker levels may not be specific for acute coronary syndrome due to plaque rupture (type 1 MI) and may instead reflect an imbalance in oxygen supply and demand (type 2 MI) or other causes [25]. Traditional markers of myocardial injury in the presence of severe burns are often unreliable because they are either too nonspecific or of uncertain clinical significance [30, 31]. In major burns, no correlation has been found between troponin levels and TBSA, third-degree burn area, age, delay in intensive care, and the degree of myocardial dysfunction at BD. Thus, in burn patients, troponin I or T can only be used as a nonspecific marker of cardiac injury, with the former being much more dependent on changes in renal function [16, 25].

Brain natriuretic peptide (BNP) is a marker of acute and chronic heart failure. BNP levels in burn patients show a “peaking pattern”, with BNP continuously increasing until reaching a peak in most patients approximately 1 week after burn injury and decreasing until day 14 of burn injury. BNP levels during the first 2 weeks correlate with the severity of burn injury. An increase in plasma BNP >400 pg/mL is a reliable marker of cardiovascular risk. Peak BNP levels correlate with the amount of intensive care within the first 24 hours, length of hospital stay, and mortality [16].

A panel of 12 metabolites, including urea cycle intermediates, aromatic amino acids, and quinolinic acid, is detected in children with burn injury at significantly higher concentrations than in adults [32].

Proinflammatory mediators such as tumor necrosis factor (TNF-α), nuclear factor-κB (NF-κB), myogen-activated protein kinase p38, and macrophage inhibitory factor (MIF) may be partly responsible for the development of organic myocardial injury in BD [30, 31].

Although many of the above-mentioned biomarkers are currently used as gold standards, they have certain limitations in their specificity and sensitivity for different populations, severity, and time after injury [19].

The occurrence of MI is associated with a 4.5-fold increase in hospital mortality among burn patients. Burn patients with MI who underwent revascularization had a lower in-hospital mortality [25].

Due to forced immobilization, multiple surgical interventions, and repeated use of indwelling venous catheters, all of the P-triad factors are detected in burn patients. Top: stasis due to immobilization after transplantation procedures and prolonged hospitalization, major general surgery (risk increases with increasing duration of procedure > 45 min); endothelial damage due to persistent inflammation, multiple surgeries, and vascular catheterization; hypercoagulability: burns induce a hypercoagulable state caused by circulating clotting factors and platelet aggregation combined with endothelial dysfunction [33, 34]. The incidence of deep vein thrombosis (DVT) in burn patients ranges from 0.9% to 53% if anticoagulant prophylaxis is not administered. This incidence is reduced to 0.25–2.4% when prophylactic measures are taken, but the pathological process can still reach 23% in the presence of concomitant trigger factors [33, 35]. The reported mean rates of deep vein thrombosis were 0.48%, pulmonary embolism (PE) 0.18%, and venous thromboembolism (VTE) 0.61%. When the burn size exceeds 10% of the TBSA, the risk of DVT increases to 0.92%, PE to 0.38%, and VTE to 1.2%. Among burn patients admitted to the ICU, the incidence of venous thromboembolism was 1.2%, and this rate was significantly higher compared with burn patients who did not require admission to the ICU. These data emphasize the importance of considering the risk of DVT in burn patients, especially those with a larger TBSA [33]. Only 50% of DVT cases occurred on the burned extremity, so the distribution of burn wounds is thought to be less involved in the development of DVT in BD than other factors of the Virchow triad. [33].

All patients with VTE in BD had Caprini scores above 8 with a mean score of about 12 [33, 35]. Mechanical prophylaxis and early activation are recommended for patients with contraindications to anticoagulant prophylaxis and predisposing factors [36, 37].

Cardiovascular system dysfunction does not reflect loss of circulating volume, and volume fluid resuscitation does not always improve left ventricular function [19, 30]. Multiple organ failure syndrome (MOFS) triggers a pathophysiological "domino effect" where failure of one organ causes morphofunctional failure of others [19].

Systemic reactions that occur during BD affect the cardiovascular system and cause adverse outcomes both at the onset of the disease and in the long term. Patients with burn injuries older than 45 years were 1.46 times more likely to be hospitalized for CVC and their hospitalization time was almost 3 times longer [24].

The results of a 33-year study have shown that patients with a history of burn injury are 50% more likely than non-injured individuals to develop CVC, which can last up to 3 years or more. This prolonged time period distinguishes burn injuries from other types of injury [15, 38, 39].

Male gender, time to hospitalization after injury, and total burn area were independent risk factors associated with myocardial damage in patients with odds ratios of 2.608; 3.620 and 1.030; ($p < 0.01$). Members of the burn cohort also spent 103% more time in the hospital for ischemic heart disease. The incidence of myocardial injury in patients with less than 50% TBSA was 38.2%; 50–80% TBSA was 54.2%; and >80% TBSA was 61.0% ($p < 0.05$) [15, 20].

Recent studies in Iraq and Afghanistan have shown that the psychophysiological consequences of combat burn injury are associated with increased systemic inflammation, arterial stiffness, metabolic syndrome, hypertension, and ischemic heart disease. Furthermore, in these studies, the cardiovascular risk profile was more unfavorable with increasing injury severity [28].

Patients with a history of burns had an 11% higher rate of long-term cardiovascular mortality [11, 24, 38]. 40% of pediatric patients who died from burn shock had cardiovascular failure. A 15-year retrospective study at a large pediatric burn center identified heart failure as the most common cause of death in infants with severe burns [11, 40].

Many cardiovascular diseases in burn injury develop without dramatic manifestations or cardiac arrest and, as a result, are not diagnosed. In the acute phase of severe burn injury, intensive medical interventions are mainly focused on resuscitation and short-term goals [6], and clinical signs of cardiac pathology are masked by the pain and metabolic response to the burn injury [25], not to mention toxemia and septicemia. Therefore, it is quite likely that a certain number of cardiovascular complications of burn injuries remain out of the field of view of clinicians, are not timely prevented, diagnosed and treated, which undoubtedly contributes to the increase in the frequency of cases of delayed CVC, which cause a large number of repeated hospitalizations, deterioration of the quality of life of patients and increased material costs for treatment.

Conclusions

1. Cardiovascular complications of burn disease affect about a third of burn patients.
2. The severity of cardiovascular disorders depends on the area of the affected surface and associated trigger factors.
3. Cardiovascular disorders occur both immediately after a burn injury and years after the episode.
4. Diagnosis of cardiovascular disorders in burn disease is difficult primarily due to the masking of their symptoms by pain syndrome and the action of analgesics.
5. Underdiagnosis of cardiovascular disorders in burn disease contributes to an increase in the frequency of delayed complications.

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Personal contribution of the author:

Kravets O.V., Yekhalov V.V.– conceptualization, writing the original text;

Gorbuntsov V.V.– editing, translation.

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