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ПОРАЖЕНИЕ СЕРДЦА НА ФОНЕ ИШЕМИЧЕСКОГО ИНСУЛЬТА: ЧТО НОВОГО?

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Acute Cerebrocardial Syndrome: What's New?

Резюме

Ишемический инсульт и сердечно-сосудистые заболевания имеют множество общих факторов риска. Соответственно, пациенты, у которых имеются общие триггеры, подвержены высокому риску развития нарушений в оси «мозг-сердце». С 2018 года в структуре церебро-кардиальных взаимоотношений выделяется синдром «инсульт-сердце» (stroke–heart syndrome), включающий любые новые нарушения со стороны сердца или ухудшение имеющихся ранее заболеваний сердца, наблюдаемые в течение первых 30 дней после острого ишемического инсульта, пик развития которых приходится на первые 72 часа после неврологической катастрофы. Патогенетические механизмы этого синдрома в настоящее время активно изучаются. Основной причиной повреждения сердца на фоне инсульта считается дисфункция автономной нервной системы, которая проявляется в снижении парасимпатической и усилении симпатической активности, что проявляется в снижении вариабельности сердечного ритма и барорецепторного рефлекса. Инсульт также сопровождается активацией гипоталамо-гипофизарно-надпочечниковой оси и симпатоадреналовой системы, развитием системной воспалительной реакции и гиперкоагуляции. Недавние данные свидетельствуют о том, что в цереброкардиальном взаимодействии играют роль микро-РНК и кишечная микробиота.

В обзоре описаны современные представления о проявлениях данного типа острого цереброкардиального синдрома, возможностях его классификации и попытках фенотипирования, а также приведены современные эпидемиологические сведения. Обсуждаются диагностические возможности рутинных лабораторно-инструментальных обследований, а также перспективные методы, требующие дополнительных исследований.

Ключевые слова: *синдром «инсульт-сердце», инсультнокардиальный синдром, ось «мозг-сердце», ишемический инсульт, сердечная недостаточность*

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Abstract

Ischemic stroke and cardiovascular diseases have many common risk factors. Accordingly, patients with common triggers have high risk of developing brain-heart axis disorders. Since 2018, the stroke-heart syndrome has been distinguished in the cerebrocardial relationships structure. It includes any new heart disorders or worsening of existing heart diseases observed during the first 30 days after acute ischemic stroke, the peak of which

occurs in the first 72 hours after the neurological catastrophe. The pathogenetic mechanisms of this syndrome are currently being actively studied. The main cause of heart damage against the background of stroke is the autonomic nervous system dysfunction, which is manifested in a decrease in parasympathetic and an increase in sympathetic activity, which is presented as a heart rate variability and baroreceptor reflex decrease. Stroke is also accompanied by the hypothalamic-pituitary-adrenal axis and the sympathoadrenal system activation, the systemic inflammatory response and hypercoagulation development. Recent data indicate that microRNA and intestinal microbiota play a role in cerebrocardial interactions. The review describes current concepts of this type of acute cerebrocardial syndrome manifestations, the classification possibilities and attempts at phenotyping, and also provides current epidemiological data. The diagnostic capabilities of routine laboratory and instrumental examinations are discussed, as well as promising methods that require additional research.

Key words: *acute cerebrocardial syndrome, stroke-heart syndrome, brain-heart axis, ischemic stroke, heart failure*

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BNP — brain natriuretic peptide, NTproBNP — N-terminal fragment of the brain natriuretic peptide, SCD — sudden cardiac death, CAD — coronary artery disease, IS — ischemic stroke, SHS — stroke-heart syndrome, CA — cardiac arrhythmias, AMI — acute myocardial infarction, ACVA — acute cerebrovascular accident, HF — heart failure, CVD — cardiovascular diseases, TS — Takotsubo syndrome, TIA — transient ischemic attack, LVEF — left ventricular ejection fraction, AFib — atrial fibrillation, CHF — chronic heart failure, CCS — cerebrocardiac syndrome, ECG — electrocardiography

Background

831,600 people died from cardiovascular diseases in Russia in 2022. Coronary artery disease (CAD) (451,000) and cerebrovascular diseases (248,900) were predominant in the mortality structure [1]. According to the World Health Organization data, CAD was the first among leading non-infectious causes of death in 2021; however, when analyzing subgroups in higher and lower income countries, the stroke took over CAD concerning this parameter [2].

The implemented preventive measures, advances in diagnostic and treatment methods, and (on the other hand) population aging affect the composition of the population of patients with cardiovascular diseases (CVDs). This is reflected in the predicted age-standardized CVD mortality decrease by 2050 with a relatively unchanged CVD prevalence. However, due to the same causes, one can expect the increase in the total CVD mortality in the nearest decades [3]. Besides, the stroke incidence has been increasing lately among people aged younger than 55 years [4].

Heart failure (HF) is the outcome of any cardiovascular disease. The prevalence of chronic heart failure (CHF) in Russia increased by 2.1 % within the period from 2002 to 2022. Patients with CHF have more comorbidities than patients with CVDs, but without CHF [5]. In particular, the stroke risk in patients with HF is 2–5-fold higher compared to the general population [6, 7]. The association of stroke prevalence and HF severity is ambiguous: some researchers have detected the stepwise association with the NYHA functional class, while others did not observe any associations, which is probably due to the features of analyzed groups. The elderly age and atrial fibrillation (AFib) undoubtedly play an important role in the pathogenesis of strokes [8, 9].

Currently inter-organ associations in CHF, including cardiorenal [10], cardiopulmonary [11], hepatocardial [12], cerebrocardiac [13] syndromes, are extensively studied. The latter one is usually considered as brain lesions in CVDs mainly associated with acute or chronic hypoperfusion of the central nervous system [13]. On the other hand, in neurological practice the cerebrocardiac syndrome (CCS) is considered as heart lesions associated with neurological accidents. CVDs are the second most likely cause of death among patients with strokes, giving way only to direct brain tissue lesions due to cerebral hemorrhages or infarctions [14]. A recent retrospective cohort study that enrolled 365,383 patients demonstrated that cardiac complications emerged in 27.6 % patients within 1 month after a stroke (including acute coronary syndrome (ACS), 11.1 %; AFib, 8.8 %; HF, 6.4 %; severe ventricular arrhythmias, 1.2 %; Takotsubo syndrome, 0.1 %) [15].

The primary cause and consequences cannot often be clearly distinguished in real clinical practice. Complex issues may be associated with the absence of significant history, lack of knowledge about pathogenetic interactions in the brain-heart axis, and the absence of an evidence-based method that could determine the sequence of lesions. Untimely diagnosis of cardiac lesions may be associated with the absence of clinical CVD manifestations due to the neurological deficit or impaired consciousness [16]. Cardioneurology, which became a separate science in the mid-20th century, analyzes the aforementioned questions.

Definition and background

In 1947 Byer E. et al. first reported their observations that cerebrovascular diseases may lead to cardiac arrhythmias and myocardial injury [17]. In 1949 N.K. Bogolepov described CCS as an apoplexy-like syndrome in acute myocardial infarction (AMI) [18]. In 1960-1970s he continued analyzing the issues of cardioneurology, e.g. he described clinical CCS variants (cerebrovascular impairment in AIM, cardiac impairment in cerebral accidents, simultaneously developing AIM and stroke); he also noted that CCS had different manifestations depending on the stroke location [18, 19]. Subsequently, cardioneurology differentiated into two specific branches, with the first analyzing cardiac diseases developing in the setting of brain lesions, and the second analyzing diseases of the central nervous system emerging in the setting of CVDs.

In 2018 Scheitz J.F. et al. define a stroke-heart syndrome in the structure of cerebrocardiac interactions, which included all new cardiac disorders or worsening pre-existing cardiac diseases emerging within the first 30 days after an acute ischemic stroke (IS). It was reported that almost 25 % patients with IS developed early cardiovascular complications, with their incidence peaking within the first 72 hours since the initiating event onset [20].

Based on the Framingham study, heart diseases are a significant risk factor of IS, with the 2-fold risk in patients with CAD, 3-fold risk in patients with essential hypertension, 4-fold risk in patients with HF, and 5-fold risk in patients with AFib [21].

Although the stroke-heart syndrome (SHS) rate peaks during the first 3 days after the stroke, the risk of death increases with its later onset. ACS, myocardial injury, left ventricular dysfunction, and AFib are independently associated with death within 90 days after IS [22]. Besides, SHS is associated with an increased 1-year risk of dementia [23].

Stroke-heart syndrome variants

5 cardiac dysfunction types have been described in the literature in the setting of SHS [20, 24]:

1) Acute myocardial injury of ischemic or non-ischemic origin (with changing altered cardiac troponin levels; this is often asymptomatic)
2) Acute coronary syndrome (due to decreased coronary blood flow or coronary plaque destabilization)
3) Left ventricular systolic and diastolic dysfunction, including the post-stroke Takotsubo syndrome (TS)
4) Any ECG alterations, arrhythmias (including AFib) diagnosed after the stroke
5) Sudden cardiac death (SCD).

These variants may be isolated or combined in a single patient who often does not have a confirmed history of structural or functional heart changes [20].

Acute myocardial injury of ischemic or non-ischemic origin (with changing altered cardiac troponin levels; this is often asymptomatic)

Troponin levels may increase due to various coronary and non-coronary events [25]. Elevated troponin levels in neurogenic diseases have been demonstrated in many studies [26–31]. Concerning IS, the mechanisms of this marker elevation has not been fully analyzed. Main hypotheses include the sympathetic-adrenal system activation, HF, and chronic kidney disease [25].

Elevated high-sensitive troponin levels are associated with a high risk of stroke in the general populations and among patients with AFib [26], as well as with the unfavorable prognosis after the cerebral accident [27, 31].

In the study of Scheitz J.F. et al. (2021), primary endpoints (recurrent stroke, AMI, all-cause mortality) were more often among patients with elevated troponin T levels after the stroke vs. the non-elevation group (27.3 % vs. 10.2 %; adjusted odds ratio 2.0; 95 % CI 1.3-3.3) during the three-year follow-up [28].

During the retrospective comparison of troponin levels in 565 patients (including 73 with transient ischemic attacks (TIAs) and 492 with acute cerebrovascular accidents (ACVAs)), troponin levels were significantly lower in patients with TIAs, with the lowest troponin levels detected in TIA and lacunar stroke groups, while altered troponin levels (both elevated and decreased) were detected in over 30 % patients with any stroke variants (based on the TOAST pathogenetic classification), except for cryptogenic stroke. Besides, elevated troponin levels were associated with a more advanced age and higher NT-proBNP levels [32]. In the post-hoc analysis of the HEBRAS (HEart and BRain interfaces in Acute Stroke) study that analyzed the level of cardiac biomarkers and cardiac MRI parameters, elevated troponin T levels (in 21 % patients of 233) were associated with confirmed pathological alterations (focal fibrosis) during the gadolinium enhancement phase, with the decreased left ventricular ejection fraction (LVEF) and LV hypertrophy [29].

Detecting serial cardiac troponin levels is used to distinguish chronic myocardial injuries from acute ones. According to Scheitz J.F. et al. (2014), the latter ones are associated with higher short-term mortality, while the cut-off troponin level, after which the risk of unfavorable prognosis increased, was 16 ng/L [31].

However, the PRAISE-DZHK19 I DZNEB001 (Prediction of Acute Coronary Syndrome in Acute Ischemic Stroke) study demonstrated that serial alterations of highly sensitive troponin levels in patients with IS was not associated with AMI, while only highly sensitive

troponin levels 5–10-fold higher than the upper reference value were beneficial for the prediction of Type 1 MI [30].

Despite all controversy, the troponin test on admission is included into the US Stroke Treatment Guidelines [33]. However, its isolated elevation cannot confirm any specific cardiac lesion, requiring additional examinations and more thorough monitoring of such patients.

Acute coronary syndrome (due to decreased coronary blood flow or coronary plaque destabilization)

Based on a large meta-analysis including over 131,000 people, the risk of AMI after the stroke was 1.67 % per year, although recurrent IS was a more common cause of death rather than MI [34]. In another large-scale study, IS was independently associated with a higher MACE risk, especially during the first 30 days after the neurological accident (HR 25.0; 95 % CI 20.5–30.5), while subsequently decreasing within a year [35]. Coronary atherothrombosis due to the enhanced plaque instability in the setting of systemic inflammation is one of the potential post-stroke ACS mechanisms [36].

Left ventricular systolic and diastolic dysfunction, including the post-stroke Takotsubo syndrome

Animal studies (mice, rats) demonstrated that the experimental large IS was accompanied by the transient LVEF decrease for 2 weeks, as well as the short-term elevation of highly sensitive troponin levels, bradycardia, decreased myocardial mass, and signs of cardiomyocyte atrophy [37, 38]. Another analysis showed that signs of HF with a 15 % LVEF drop developed in rodents 8 weeks after the short-term occlusion of the middle cerebral artery vs. the control group, with a higher heart rate, increased end-systolic and diastolic LV volumes [39].

The prevalence of systolic and diastolic LV dysfunction in the post-stroke period in human studies significantly varies in different studies. For example, in the SIC-FAIL study the rate of HF detection in patients with the ischemic stroke was 5.4 % (HFrEF 4.35 %) [40]. The rate of HF detection with reduced LVEF ranged from 0.78 to 15 % [40, 41]. Based on various data, diastolic dysfunction is diagnosed in 23.3–59 % patients [40–42].

Currently it is unknown whether the LV dysfunction diagnosed in the post-stroke period is actually transient and whether it is gradually restored or leads to symptomatic CHF in the future [14]. For example, in the study of Sposato L.A. et al. (2020), the risk of HF 1 year after the first ACVA among patients without a history of CVDs was 3.3-fold higher than in a matched patient group without ACVAs (95 % CI 3.1–3.7) [35]. Many studies do not provide data about the presence or absence of CVDs before the stroke.

Prognostically significant risk factors for LV dysfunction development and its severity after ACVAs include the elderly age, a history of CVDs, elevated cardiac troponin [40], BNP and NT-proBNP levels [43], the focus size and its location [44]. In the study of Dieplinger B. et al. (2017), NT-proBNP levels were independently associated with the all-cause mortality within 3 months [45].

The post-stroke TS is a very rare finding, which is complex for the diagnosis and which risk is increased in Caucasians, females, and elderly persons [40, 46, 47]. According to different studies, its incidence is 0–0.42 % [15, 40, 46, 47]. This syndrome is usually considered as a transient LV dysfunction with its subsequent recovery within several months [48]. Large registry-based studies have demonstrated that acute neurological conditions form the largest group of physical TS triggers with the with the worst clinical outcomes and a slower LV function recovery [48].

The post-stroke TS is a predictor of poor prognosis, high mortality (including the in-hospital one), poor functional outcome [49, 50]; it is also associated with the risks of such complications as cardiogenic shock (OR 8.84, CI 4.07–19.17, $P < 0.001$), cardiac arrest (OR 3.17, CI 1.57–6.42, $P = 0.001$), venous thromboembolism (OR 1.68, CI 1.14–2.47, $P = 0.008$) [49].

Exact pathogenetic TS mechanisms have not been established. The most probable current hypothesis presumes the direct catecholamine-induced toxicity and associated spasm of the coronary artery microvasculature [49]. It has been established that in patients with this syndrome blood catecholamine levels are 2–3-fold higher than in patients with AMI and 20-fold higher than in the control group [50]. The post-stroke TS is often asymptomatic [51].

Any ECG alterations, arrhythmias (including atrial fibrillation) diagnosed after the stroke

ECG alterations after ACVAs emerge in 46–79 % cases based on various authors [52, 53]; however, the majority of ECG deviations are transient and disappear within 14 days [53]. The most common anomalies include QTc interval prolongation (20–65 %), altered T wave morphology (16–40 %) and altered ST segment changes (15–25 %), arrhythmias (28–36 %) [52–54]. The majority of patients have at least two pathological alterations [52]. Some data confirm that ECG anomalies correlate with outcomes. Thus, prolonged QTc interval based on the results of the regression analysis and ECG signs of ischemia ($p=0.044$) were considered independent predictors of poor prognosis during the acute stroke phase [55]. During the long-term evaluation based on the results of multifactorial analysis, the prognostic 90-day mortality factors included AFib, atrioventricular block, ST segment alterations, and T wave inversions [56].

Arrhythmias constitute approximately one third of all electrocardiographic alterations [53], with the highest probability of those detected during the first day after the stroke [57]. Some data demonstrate that the elderly age and a high National Institute of Health Stroke Scale (NIHSS) score became the predictors of arrhythmias within the first 3 days after the stroke [57]. It has been observed that arrhythmias with tachycardia occur more often than bradycardias [52, 57]. The rate of AFib after IS, based on different data, ranges from 5 to 32 % [52–54, 57, 58].

A special term AFDAS (atrial fibrillation detected after stroke and TIA) is used in English sources. This category includes AFib emerging during or after the stroke, as well as probably pre-existing AFib, but detected only after the stroke [59]. Patients with AFib detected after the stroke, based on several data, have a lower risk of recurrent stroke and have less chances of having a history of CVDs [60, 61].

It is known that 50 % of all AFib cases in patients with ACVAs and TIAs are first detected after neurological events, and in the majority of cases these arrhythmias are asymptomatic, while every second paroxysm lasts less than 30 seconds [59]. This requires prolonged ECG monitoring in the early post-stroke period.

Some data demonstrate the stroke located in the right insular lobe is associated with a higher rate of AFib than in the left one (39 vs. 4 %) [62].

AFib detected after IS may have a cardiogenic, neurogenic, or mixed origin. Cardiogenic factors include pre-existing CVDs and atrial remodeling, AFib episodes not detected earlier. Neurogenic mechanisms are associated with the left atrial changes in the setting of systemic inflammation and autonomous dysfunction as part of ACVA [63].

Despite the fact that the majority of ECG alterations are transient, AFib after the stroke may transform into persisting or permanent forms [64].

Fan X. et al. (2024) have concluded that one should define phenotypes of patients with AFib after IS with a variable risk in order to define the requirements for anticoagulant therapy and dosing in those patients. Further biomarker studies and neuroimaging patterns have to be arranged with this purpose [64]. Several prognostic models for the post-stroke AFib have been currently developed [65, 66], as well as the multimodal approach including imaging, ECG, and biomarkers (NT-proBNP, galectin 3, SR2, osteoprotegerin) [58]; however, further studies are required to clarify their prognostic significance.

Sudden cardiac death (SCD) is a non-violent death emerging instantaneously or within one hour from the moment of acute alterations in the patient's clinical status. Theoretically SCD is one of the SHS types. However, sudden cardiac and neurogenic deaths can be practically distinguished only after complete pathological examination of the brain and heart, as well as if the ECG data have been recorded. Cases with SCD after the stroke may be omitted from observations due to the study design features [24].

SHS pathogenesis

The stroke and CVDs have common risk factors [67]: hypertension, atherosclerosis, lipid metabolism disorders, diabetes mellitus, obesity, smoking, alcohol abuse, low physical activity level, stress, etc. Consequently, patients with common triggers have a higher risk of disorders in the brain-heart axis.

Pathogenetic SHS mechanisms are still being currently investigated (Fig. 1). Both true cardiomyocyte injury and reactive, potentially reversible alterations in their structure and function occur in IS.

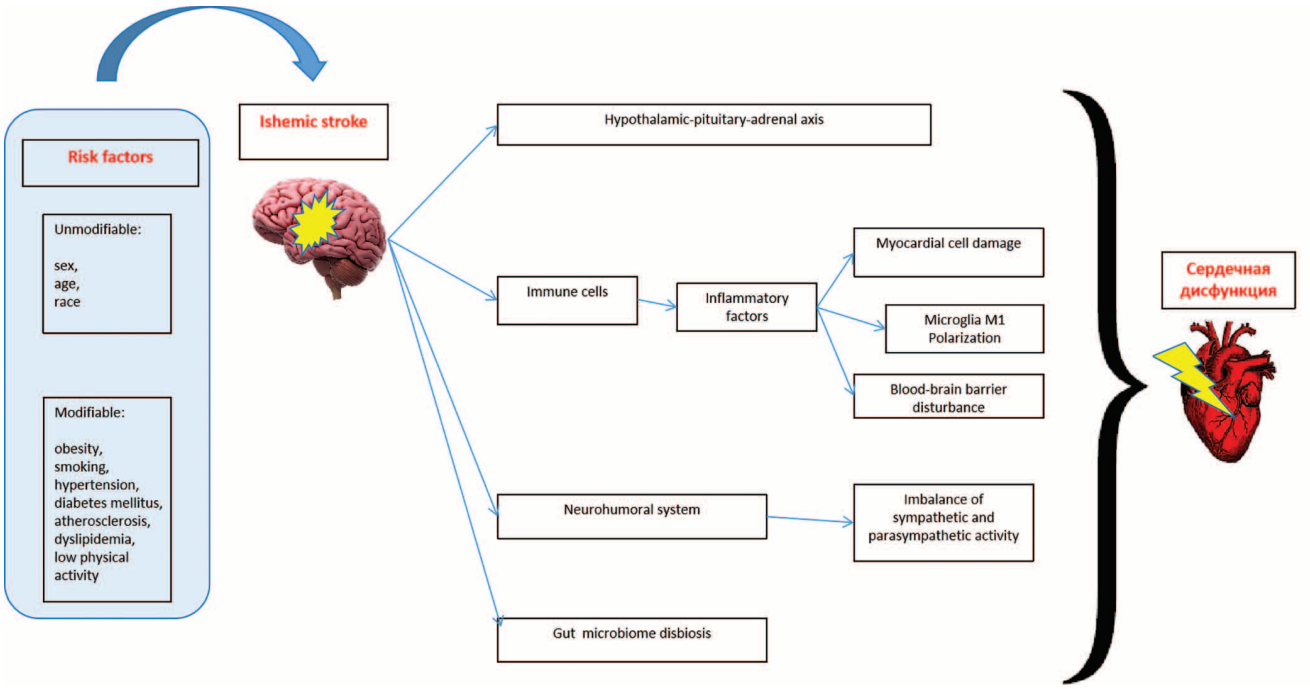
Autonomous dysfunction manifesting with the decreased parasympathetic and increased sympathetic activity is considered the main cause of cardiac lesions in the setting of a stroke. With that, parasympathetic dysfunction is more significant rather than sympathetic hyperactivity. These alterations develop in the acute stroke phase and are preserved for up to 6 months [68]. The autonomous balance shift towards sympathetic domination in the acute ischemic stroke phase manifests with the decreased cardiac rhythm variability and baroreceptor reflex [68], enhancing myocardial oxygen requirements.

Due to the enhanced sympathetic cardiac effects, catecholamines are released, which mediates electrolyte and metabolic disorders in cardiomyocytes with worsening relaxation processes [20]. In experimental animal stroke models the most significant myocardial lesions were detected in the left atrial region around pulmonary veins (area of the largest sympathetic effects) [69], which may promote AFib. Other murine stroke models demonstrated that the enhanced peripheral sympathetic activity was associated with SHS of the HFrEF type and LV dilation for 2 months [39].

ACVA is accompanied by the activation of the hypothalamic-pituitary-adrenal axis and sympathetic-adrenal system, which leads to elevated levels of stress hormones, including catecholamines and cortisol [68]. It has been demonstrated that the serum cortisol level correlates to the stroke severity and prognosis (both short-term and long-term) [70].

Both enhanced sympathetic activity and the systemic inflammatory reaction may activate hypercoagulation process, enhance the activity of platelets and neutrophils, lymphocyte apoptosis, and endothelial dysfunction [71–73].

The results of articles that underscore the role of inflammatory reactions in the emergence of SHS have



Picture 1. *Heart-stroke syndrome pathogenesis.*

been published recently. Besides, the macrophagal infiltration of the myocardium significantly enhances in IS, while levels of inflammatory macrophage-associated cytokines increase, which finally leads to myocardial fibrosis and hypertrophy [74, 38].

The systemic immune inflammation index (SII, equation: [(neutrophils × platelets/lymphocytes)/1000]) which has proven itself as an independent cancer predictor is currently actively analyzed as a new marker of systemic inflammatory reaction [76]. In a recent study of Hao X. et al. (2024) this index based on the multifactorial logistic regression results demonstrated the predictive value for SHS (adjusted OR 5.089 (95 % CI 1.981–15.74, p = 0.002) [77]. Meanwhile, in the study of Weng Y. et al. (2021) the SII index in ischemic stroke was associated with the functional outcome [78]. However, a larger number of studies is required to confirm its prognostic value for SHS.

The recent data also demonstrate that micro-RNA [79] and the intestinal microbiome [80] play a role in cerebrocardiac interactions. Data have been obtained that micro-RNA-126 deficiency is closely associated to HF, AFib, IS, and (probably) post-stroke cardiac lesions [81].

The systemic inflammation increases the intestinal permeability and promotes the bacterial translocation and endotoxins into the blood flow, which worsens the systemic inflammatory reaction and the risk of myocardial disorders. Severe intestinal dysfunction in patients with IS manifests with the increased counts of opportunistic bacteria and decreased probiotic levels. Nevertheless, the exact mechanisms of intestinal microbiome effects on SHS still remain unclear [64].

SHS risk stratification

Accounting for the burden of cardiovascular diseases and neurological accidents, the issue of post-stroke cardiac lesions and changes in the progression of pre-existing CVDs remains very significant. Despite the currently accumulated knowledge concerning the interaction of the brain-heart axis, many issues remain unsolved and require a large number of original studies. Besides terms and classifications, risk stratification and prognosis, it is important to describe specific phenotypes among patients with the developed SHS.

A retrospective study using the VISTA electronic database is interesting. That included more than 12,000 patients. Using the hierarchic cluster analysis, they were divided into 5 profiles according to the pre-existing SHS risk factors and the early mortality risk after ACVA: Profile 1 (“elderly patients with AFib”); Profile 2 (“young smokers”); Profile 3 (“young patients”); Profile 4 (“patients with concomitant CVDs”); Profile 5 (“patients with essential hypertension and atherosclerosis”). Profiles 4 and 1 demonstrated the highest SHS risk (adjusted HR (95 % CI) 2.01 (1.70–2.38) and 1.26 (1.05–1.51), respectively, vs. Profile 3). Profiles 5 and 2 had a moderate risk, while Profile 3 had the lowest risk. Profile 5 had the highest risk of cardiac and respiratory arrest (adjusted HR (95 % CI) 2.99 (1.22–7.34)), while the highest 90-day mortality risk was observed in Profiles 5

and 4 [82]. Such patient division into profiles or phenotypes may help to develop diagnostic algorithms and treatment approaches, although it requires a significant evidence base, continuing works in this direction.

Conclusion

Thus, the publication data underscore that stroke is not an isolated brain lesion, but rather a multisystemic problem requiring a complex approach in each specific case.

Currently multiple issues concerning worsening cardiac function in the setting of IS remain unsolved (no unified terms, no approved classification). Besides, scales for risk stratification have to be developed, and treatment algorithms based on various SHS phenotypes have to be determined. A larger number of works with a prolonged follow-up period is required, as it's not clear in which cases SHS is time-limited and regresses, and when it progresses with severe complications, e.g. symptomatic CHF. SHS probably can be divided into acute and chronic types (just like the cardiorenal syndrome).

To remove the knowledge gaps and advance the management tactics of patients with cardiac lesions after the stroke, collaborative research initiatives guided by interdisciplinary cardiologist and neurologist teams are required.

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All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

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