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Research Article

STRUCTURAL AND FUNCTIONAL FEATURES OF THE MYOCARDIUM AGAINST THE BACKGROUND OF RENAL REPLACEMENT THERAPY

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ABSTRACT

Cardiovascular diseases are one of the most significant predictors of mortality in patients with end-stage renal disease treated with chronic hemodialysis, and cause at least a third of all hospitalizations [Eisner D. et al., 2001; Johnston N. et al., 2008]. According to the US Renal Disease Registry, the five-year survival rate in the general population of patients on hemodialysis is 33.4% [U. S. Renal Data System, 2009]. More than half of the deaths of patients on renal replacement therapy are due to cardiovascular pathology, the mortality from which in patients on

hemodialysis is 30-35 times higher compared to the general population [Zavy A.S. et al., 1998; Herzog S.A., 2003; U.S. Renal Data System, 2009]. A significant proportion of deaths in patients with end-stage renal disease receiving

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hemodialysis treatment are attributed to those associated with coronary heart disease [Foley R.N. et al., 1998a; Sarnak M.J. et al., 2003; Cheung A.K. et al., 2004]. The theoretical method of research was used. Many articles and dissertations by international scientists were analyzed, which were based on various books, dissertations, as well as electronic journals.

KEYWORDS

Cardiovascular diseases, hemodynamic syndialysis stress, hemodialysis-induced myocardial ischemia, hyperhydration, pulmonary hypertension, heart failure.

INTRODUCTION

The article presents up-to-date data on morbidity and mortality from cardiovascular diseases (CVD) in patients undergoing programmed hemodialysis (PGD). According to literature data, by the end of the first year of PGD, approximately one in five patients dies, CVD and their complications occupy a leading place in the structure of mortality. High cardiac mortality is a consequence of a combination of traditional and non-traditional risk factors for the development of CVD in the dialysis population of patients. Non-traditional risk factors are combination of toxic-metabolic and hemodynamic effects on the myocardium caused by both kidney damage and replacement therapy. From the perspective of a cardiologist, PGD-associated risk factors for CVD are of particular interest, among which the following deserve the closest study: hemodynamic syndialysis stress, hemodialysis-induced myocardial ischemia, hyperhydration, pulmonary hypertension, intra- and postdialysis hypotension, heart failure with high cardiac output and dynamic intraventricular obstruction. All of the above phenomena have a direct damaging effect on the myocardium and lead to aggravation of heart failure. The stated problems dictate the need to develop a specific methodology for assessing the initial cardiological status of patients on PGD and heart diseases are the cause of 40-50% of death in patients with stage 5 in chronic kidney disease (CKD 5) [13, 18, 26, 21]. The results of long-term population studies initiated in the early 70s of the last century and continuing to this may indicate that patients with CKD5 die mainly from cardiovascular diseases catastrophes [27, 43, 45]. In absolute terms, patients receiving renal replacement therapy (HRT) by dialysis methods have a 10-50 fold rise of cardiovascular death compared to the general population [19, 21, 58, 59]. It is becoming increasingly obvious that programmatic hemodialysis (HD) is associated with a number of independent risk factors for by the beginning of 2014, at least 2.25 million people in the world suffering from CKD5 were receiving PGD [24]. The number of patients receiving this type of renal replacement therapy, in most countries, including Central Asia is steadily increasing [29, 23, 1]. Among the causes of death of PGD patients, cardiovascular pathology occupies a leading place. It is important to note that, despite the revolutionary changes in hemodialysis technology and the continuous improvement of approaches to the treatment of patients with CKD 5, over the past 20 years, the share of cardiovascular pathology in the

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mortality structure of PGD patients has not changed and is 40-45% [60,59, 23, 3, 2]. At the same time, special attention is drawn to the increase in mortality in the first year of PGD, which is now increasingly regarded as a global phenomenon [16, 37, 44, 50]. It is particularly impressive that more than a third of deaths (about 35%) occur during the first three months of RRT [44], with more than half of the cases being sudden cardiac death [28]. The critical point of mortality of patients on PGD reaches 120 days and is 27.5 deaths per 100 patients per year, and the integral annual mortality is approaching 20% [14, 50, 55]. Thus, by the end of the first year of PGD, about one in five dies a patient, which is an undoubted challenge to the professional medical community. Data from the UK renal registry indicate that a patient who started HD at the age of 25 to 29 has an average life expectancy of only 18.5 years,

i.e. 33 years less than this indicator in the same age group in the general population without CKD5 [56]. In patients aged 65-74 years (the most typical age group for patients starting PGD in developed countries), according to the European Renal Registry, the average life expectancy is 5 years approximately 50% less than in the same age group in the general population without CKD5 [23]. Thus, as the dialysis population expands, the issues of diagnosis, prevention and treatment become more and more urgent cardiovascular diseases (CVD). It should be emphasized that chronic kidney disease (CKD) is itself a strong independent risk factor for the development and progression of CVD and, as CKD progresses, the incidence of CVD increases [13, 26, 27]. According to experts, the reason for this dependence is a combination of traditional and non-traditional risk factors for the development of CVD [37]. Traditional risk factors are well known, in the context of the problems discussed, the most important are: arterial hypertension (AH) with concomitant remodeling of the left ventricular myocardium (LV), left ventricular hypertrophy (LVH), hyperlipidemia, diabetes mellitus and anemia. The peculiarity of a patient with CKD5 is the presence of most or all of the traditional risk factors for the development and progression of CVD. Non-traditional risk factors are a combination of metabolic, toxic and hemodynamic features caused by both CKD and dialysis itself (CKD-dialysis-associated) [34, 37, 42]. The group of toxic and metabolic factors includes a significant increase in pro-inflammatory cytokines, homocysteine, hyperphosphatemia, hyper- and hypocalcemia, hyperparathyroidism, sodium, potassium and magnesium imbalance, oxidative stress and nitric oxide deficiency, insufficient water purification, etc. [22, 46, 52, 1]. The result of their action is both direct and indirect (through increased chronic inflammation) cardiotoxic effect, the development of micro- and macrovascular endothelial dysfunction, acceleration of atherogenesis and myocardial fibrosis, increased vascular stiffness and rapid calcification of intracardiac structures [15, 37, 41]. In addition, significant fluctuations in the levels of K+, Na+ and Ca2+ play a key role in the formation of electrophysiological mechanisms of ventricular tachycardia, ventricular fibrillation and complete transverse heart block The toxic effect of azotemia on the heart, leading to the formation of uremic fibrinous pericarditis and specific myocardial damage (uremic cardiomyopathy), should not be underestimated [41].An important risk factor for the development and progression of CVD is the presence of anemia, the genesis of which at the final stage of CKD is primarily associated with a decrease in erythropoietin production [26]. During the first year of RRT, a compensatory increase in cardiac output is noted against the background of anemia [51]. This compensation mechanism eventually leads to depletion of LV contractile function, its dilatation, cardiomegaly and ultimately, to the formation of

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severe congestive heart failure (HF) [36, 1]. Correction of anemia with drugs that stimulate erythropoiesis does not lead to a decrease in mortality from CVD, since an increase in viscosity is observed in parallel with the growth of hemoglobin blood and AG aggravation [53]. From a practical point of view, it is important to emphasize that in the dialysis population, complete correction of anemia should be avoided [26, 51, 53] – so, for today . However, most experts believe that hemoglobin targets should be in the range of 11-12 g/dl. This will compensate for hemic hypoxia of the myocardium and minimize the risks of CVD progression against the background of erythropoietin replacement therapy. It should be noted that the incidence of infectious endocarditis (IE) is certainly higher than in the general population [30]. Diagnosis of this pathology remains difficult in patients with calcified valvular heart defects, both due to the complexity of visualization of vegetations and due to the "blurring" of the clinical picture. A separate problem is the development of right-sided IE in patients with central venous catheters as a vascular access for RRT. From the perspective of a cardiologist working in a nephrological clinic, hemodialysis-associated CVD risk factors are of particular interest. The peculiarity of these factors is the inevitability and repeatability of the effect of the procedure itself on the heart muscle and the parameters of central hemodynamics (CGD) from dialysis to dialysis. We are talking about the formation of a kind of "hemodynamic swing", which ultimately leads to a persistent destabilization of the main compensatory mechanisms of the cardiovascular system as a whole. From our point of view, the following factors deserve the closest attention and study:

1. Recurrent hemodynamic stress during the HD procedure. The results of echocardiographic (Echo-KG) studies and positron emission tomography obtained during dialysis sessions indicate that during each HD procedure, myocardial perfusion decreases to one degree or another, causing its transient stun ("stunning"), which can eventually lead to fixed violations of local contractility and the development of myocardial fibrosis [12, 17, 5, 11]. The term hemodialysis-induced damage to the heart muscle has become legitimate. Moreover, transient hemodialysis-induced myocardial ischemia is a predictor of an increase in annual mortality in patients with PGD [12, 17].

Acute and/or chronic hyperhydration (volume overload).

It is well known that hyperhydration is a significant risk factor for further progression of hypertension, LVH and LV remodeling [32, 36, 61, 4, 6]. However, in the context of the discussed problems, it is extremely important to study the role of hyperhydration in the formation of congestive HF. Acute decompensation of CHF on the background and due to hyperhydration leads to the development of pulmonary edema and severe myocardial ischemia, which negatively affects the main determinants of prognosis. Of course, conducting HD critically reduces the degree of hyperhydration and its destructive effect on the CCC. But hemodynamic stress during the RRT procedure, especially in combination with excessive filtration volume, on the one hand, and an increase in the volume characteristics of the heart during the interdialysis period, on the other, will lead to further progression of LV myocardial remodeling processes, the formation of diastolic dysfunction and, ultimately, an increase in CHF phenomena.

3. Pulmonary hypertension (PH) in patients with PGD. LH in patients with PGD is a well-known fact, which is reflected in the modern classification. It is also known that the treatment of this condition is very problematic and expensive today. About 50% of patients on HD

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have signs of LH. It is believed that the development of LH almost 4 times increases the annual mortality rates in patients with CKD 5

[62]. The formation and/or progression of LV occurs both as a result of systolic LV dysfunction caused by the first two factors and as a result of a specific effect on the central hemodynamics of a functioning, both native arteriovenous fistula (AVF)

and AVF formed with the use of a vascular prosthesis [48].

4. Instability of blood pressure figures under the influence of the DG procedure.

The phenomenon of intradialysis and postdialysis hypotension is well known, which, in turn, is the cause of the development of repeated episodes of myocardial ischemia, and also contributes to the processes of LV remodeling [7, 8, 9, 20].

5. Heart failure with high cardiac output in patients with functioning AVF.

This condition is an interesting hemodynamic paradox: against the background of a detailed clinical picture of CHF, instrumental high rates of cardiac output are determined [38]. This naturally complicates the interpretation of the clinical situation, since sufficient LV ejection fraction (LVEF) is traditionally considered in most cases to be an indicator of a stable state of the cardiovascular system. It is not superfluous to recall that the diagnosis of HF with high cardiac output requires the exclusion of thyrotoxicosis and severe uncompensated anemia.

6. Dynamic intraventricular obstruction during a dialysis session, leading to arterial hypotension and increased myocardial oxygen demand [49]. The combined influence of traditional and non-traditional risk factors, as CPN progresses, leads to a decrease in global and regional LV contractility, i.e. to the development

of CHF proper. In the situation of the development of HF, the main compensation mechanism is the further activation of the body's pressor systems (sympathetic nerve system (SNS) and the renin-angiotensin-aldosterone system (RAAS), which allows you to maintain a cardiac output adequate to the needs of the body. However, maintaining the hemodynamic status has its price. Efferent vasoconstriction, which develops due to hyperactivation of SNS and RAAS, provides the necessary level of glomerular filtration, but high vascular resistance sooner or later leads to a decrease in renal blood flow. From a certain moment, the mechanisms of hypoxic damage to the tubules, apoptosis of nephrons are triggered,

development of renal replacement fibrosis. These processes lead to further progression of renal insufficiency due to persistent renal ischemia, which, in turn, activates SNS and RAAS, maintains a high blood pressure level and leads to the closure of a vicious circle of mutual aggravation of CKD and CVD. The described mechanisms underlie the formation of the cardiorenal continuum, which is a continuous series of sequential processes of damage and dysfunction of the heart and kidneys [34, 42]. Thus, in the predialysis period, specific prerequisites have already been formed for further exacerbation of HF – it is believed that about 35-40% of patients with CKD 5 have signs of CHF by the beginning of RRT [19, 30, 10]. After the start of HD in less than 2 years, the number of such patients increases by another 25%, i.e. every second will have signs of CHF [30]. As already mentioned above, the reason is more half of the cases of cardiovascular deaths during the first year of PGD are sudden cardiac death. In this contingent of patients, unlike the general

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population, acute coronary occlusion is not the leading cause of sudden cardiac death. On the contrary, sudden cardiac death is explained by the summation of the negative effects of LVH, prolonged arterial hypertension, hyperhydration, chronic inflammation, hyperactivation of the SNS, as well as a tendency to ventricular arrhythmias due to electrolyte disturbances – i.e. a fatal combination traditional and CKD/dialysis-associated CVD risk factors [29, 47].

CONCLUSION

Cardiovascular diseases and the high mortality rate caused by them remain one of the main problems of dialysis therapy. At the same time, despite the improvement of dialysis technologies, the share of cardiovascular deaths in the overall mortality structure of patients with CKD5 receiving RRT has remained unchanged over the past decades. Obviously, to change this situation, it is necessary to solve a number of problems concerning the optimal management tactics of patients comorbid for cardiovascular diseases. The stated problems dictate the necessity of developing a certain methodology for the initial assessment and subsequent monitoring of the cardiological status of nephrological patients, which will allow both objectifying the effectiveness of ongoing therapeutic measures and offering new ones in the future approaches to therapy. The solution of cardiac problems in nephrologia cannot be the prerogative of a cardiologist alone. Without counter–proposals and ongoing discussion of the tasks set by the nephrological community, it is impossible to achieve a common goal - to reduce mortality from CVD in the dialysis population of patients. None of the authors has any conflicts of interest.

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