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Initiation of hemodialysis and sudden cardiac death — a constant challenge on the threshold of new therapy

ABSTRACT

The risk of death from cardiovascular diseases in patients initiating hemodialysis therapy is 50% higher than in patients with stage 5 chronic kidney disease before starting renal replacement therapy. Sudden cardiac death is the leading cause of death within the first months after initiation of renal replacement therapy. Identification of risk factors for early mortality in patients with end-stage renal disease is important for their future care. Myocardial stunning, intradialytic hypotension, or pulmonary hypertension in patients with arteriovenous fistula have been known for years to cause deterioration in myocardial function. On the other hand, based on observational studies conducted so far, it is not clear whether diabetes promotes early mortality in patients treated with renal replacement therapy. In addition to standard tests performed routinely in dialysis patients, it is recommended to measure high-sensitivity troponin and natriuretic peptide as well as obtain an echocardiography study during both pre-dialysis care and after the initiation of renal replacement therapy for both prognostic and diagnostic purposes. However, the main focus should be on the dynamics of changes in those parameters rather than single measurements.

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Key words: chronic kidney disease, hemodialysis therapy, sudden cardiac death

SUDDEN CARDIAC DEATH IN DIALYSIS PATIENTS

In patients initiating hemodialysis therapy, the risk of death from cardiovascular diseases is 50% higher than in patients with stage 5 chronic kidney disease (CKD) before initiation of renal replacement therapy. Sudden cardiac death is the leading cause of death within the first months after initiation of renal replacement therapy [1]. The incidence of sudden cardiac death is more than three times higher compared to patients with stage 4 chronic kidney disease. At the same time, the incidence of acute coronary syndromes after initiation of hemodialysis therapy rises several times compared to earlier stages of chronic kidney disease [2]. It should be noted that ST-segment elevation myocardial infarction (STEMI) predominates during the first month of renal replacement therapy and that the incidence of non-ST-segment elevation myocardial infarction (NSTEMI) increases in subsequent months of hemodialysis therapy. While the risk of STEMI remains constant at a level higher than in the pre-dialysis period, the occurrence of sudden cardiac death (SCD) is characterized by different dynamics. Initially, the risk of SCD starts to increase in the second month of therapy and remains constant for three months, and after that period it drops down and stays at a level higher than in the pre-dialysis period [3]. Based on different dynamics of sudden cardiac death and myocardial infarction during the first months of dialysis therapy, it should be assumed that one of the elements of reducing SCD risk during early stages of dialysis therapy should be individual morphological and functional evaluation of the cardiovascular system in every patient enrolled in the renal replacement therapy program. Evaluation for cardiac and vascular disorders should start even earlier during pre-dialysis stages of chronic kidney disease. Research

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suggests that from a clinical point of view, the risk of heart disorders increases significantly when the glomerular filtration rate falls below 60 mL/min/1.73 m^2. With a further decrease in the glomerular filtration rate, there are harmful changes to the cardiovascular system, both quantitative and qualitative while the nature and degree of those changes vary significantly between individuals. Considering the heterogeneity of possible causes of heart damage, it can be explained to a certain degree why the clinical course of sudden cardiac death and myocardial infarction is so difficult to predict after initiation of renal replacement therapy [4].

According to some researchers, the issue of early death after initiation of dialysis therapy is not only underestimated but also requires further research as the number of elderly patients with significant comorbidities is increasing. In the context of the earlier considerations, several attempts have been made to estimate the risk of death among patients initiating renal replacement therapy. The importance of routine echocardiography for risk assessment in renal replacement therapy has been emphasized. Such a study should include a very diverse population of patients with chronic kidney disease [5]. The spectacular progress in hemodialysis therapy in the last decades including an improved dialysis technique and more effective pharmacotherapy has increased the life expectancy of hemodialysis patients. However, the problem of early mortality associated with initiation of hemodialysis therapy remains unresolved and bothering. Since it is still valid, there is a need for thorough analysis of the diagnosis and pathogenesis of sudden cardiac death in early stages of renal replacement therapy [6].

THE IMPACT OF DIABETES ON EARLY MORTALITY OF DIALYSIS PATIENTS

Results of studies carried out so far clearly indicate that older age and female sex are associated with a higher risk of early death in patients with end-stage renal failure receiving hemodialysis therapy. However, the hypothesis about the relationship between diabetes and the increased risk of early mortality in patients with end-stage renal failure receiving hemodialysis therapy is not confirmed. In several studies, early mortality was compared in the group of diabetic patients treated with hemodialysis and patients with a different cause of end-stage renal failure (case-control study). In the largest study, Wolf et al. compared a group of 50 patients who died prematurely with a matched control group of 750 individuals [7]. In both groups, diabetes was the cause of end-stage renal failure in 43% of patients. In turn, Khan et al. compared 42 patients who died within 90 days after starting hemodialysis with 42 individuals in the control group; the proportion of diabetic patients was the same in both groups (3/42 in each group) [8]. Those studies have not shown that diabetic patients have an increased risk of premature death. On the other hand, de Lima et al. obtained opposite results. The authors compared patients who died prematurely with a group of patients who survived hemodialysis treatment for more than 10 years. The percentage of patients with diabetes was 35% and 0%, respectively. This study showed a relationship between early mortality and underlying diabetic nephropathy [9].

Similarly ambiguous results were obtained in cohort studies comparing mortality in hemodialysis patients with and without a history of diabetes. Some studies showed an increased risk of premature death in patients with diabetes, while other studies did not show it. Robinson et al. observed a group of 86 886 new patients initiating renal replacement therapy. Among those who died within the first 120 days after initiating hemodialysis, 8.1% had diabetic nephropathy. In comparison, 9.2% of patients who died at the same time had a different etiology of kidney disease. The conclusions from this study were that diabetes does not affect early mortality in dialysis patients. However, another large study showed the opposite outcome. In their study, Tsakiris et al. (n = 78534) reported early mortality at 5% in diabetic patients and 6% in non-diabetic patients. Other studies, depending on the methodology, did not show higher early mortality in diabetic patients receiving dialysis therapy compared to non-diabetic patients although superficial analysis may suggest higher mortality in diabetic patients [10].

CARDIAC STUNNING, INTRADIALYTIC HYPOTENSION

Myocardial stunning, which is manifested by a transient impairment of the heart's contractility, is a well-known condition associated with changes in blood flow in the coronary vessels. However, the return of muscle activity to

the previous level can range from a few hours to even a couple of days. Repeated episodes of myocardial stunning may exacerbate heart failure or trigger arrhythmias. In addition, more and more data from animal studies, supplemented by clinical observations, indicate that cardiac stunning is accompanied by several systemic disorders that may influence further prognosis regarding the course of the disease or the patient's survival.

In pre-dialysis stages of chronic kidney, several systemic disorders gradually lead to myocardial damage. Systemic inflammation and gradually increasing fluid overload are substrates facilitating occurrence of cardiac stunning. It seems that in some patients with chronic kidney disease, even in pre-dialysis stages, cardiac stunning may occur as a consequence of the following: anemia, poorly controlled hypertension, or calcium-phosphate metabolism disorders. The initiation of dialysis treatment in such patients creates additional conditions for more frequent occurrence of cardiac stunning as well as a larger area of cardiac dysfunction. It cannot be ruled out that early deaths after initiation of renal replacement therapy occur exactly in such patients. Therefore, it seems reasonable to implement appropriate diagnostic workup to identify patients at higher risk of cardiac stunning at early stages of their disease and to initiate effective pharmacotherapy. However, due to a moderate number of studies evaluating the efficacy of pharmacotherapy of cardiovascular diseases in patients with chronic kidney disease, there is limited possibility of therapy based on reliable clinical trials [11]. Considering the earlier doubts, the role of β-blockers in the prevention of cardiovascular diseases in hemodialysis patients is an interesting topic. Despite more than forty years of experience in pharmacotherapy with e.g., selective β-blockers, their use in dialysis patients still raises many doubts such as which β-blockers should be used in patients with intradialytic hypotension, whether they should be used in hemodialysis patients at all, and which β-blockers should be avoided in such patients.

Cardiac magnetic resonance imaging during a hemodialysis session in chronic hemodialysis patients clearly confirmed the occurrence of myocardial stunning. At the same time, the presentation of those changes in the heart varied greatly. In some patients, the changes appeared already within the first hour of the procedure and remained until it ended. In others, they persisted after the hemodialysis session, and it is difficult to clearly determine for how long. The evaluation of myocardial contractility using resonance imaging was performed only for 30 minutes after the end of the procedure. Even in those patients in whom the myocardial contractility improved at the end of the dialysis session, this beneficial change did not apply to the entire area of the examined muscle. The improvement was gradual and affected the previously stunned area of the heart to a varying degree. A gradual decrease in stroke volume and cardiac output was observed throughout the hemodialysis session with a maximum decrease in those parameters at the end of the third hour of the session. The cause of those changes was thought to be both dehydration resulting from the hemodialysis procedure and cardiac stunning. However, the influence of those two factors on stroke volume varies between patients. This is confirmed by our experience in our daily clinical practice [12].

In some patients, a decrease in blood pressure associated with hemodialysis can be compensated by parenteral fluid infusion. In some patients, this brings the desired effect, in other patients the benefit resulting from such treatment is negligible. In the first case, the decrease in stroke volume is probably due to volume depletion; while in the second case, the decrease in myocardial contractility results from cardiac stunning due to impaired myocardial perfusion with preserved blood flow in large epicardial vessels. Cardiac stunning associated with hemodialysis is a risk factor for death and cardiovascular diseases, which is already noticeable within the first three months of dialysis therapy. One of the clinical signs suggesting hemodialysis-related cardiac stunning is a decrease in blood pressure during the procedure. This symptom is particularly evident during the first two to three hours of hemodialysis when the systolic blood pressure values can decrease by 0 to 30 mmHg, which also means that intradialytic hypotension should be diagnosed in those patients according to most criteria [13].

The relationship between intradialytic hypotension and cardiac stunning remains an open question, as the drop in blood pressure may be one of the symptoms of cardiac stunning. On the other hand, a decrease in blood pressure caused by inadequate pharmacotherapy of hypertension or excessive dehydration can compromise myocardial perfusion, which is probably reflected by cardiac stun-

ning or other irreversible changes. Therefore, it is increasingly accepted that the relationship between intradialytic hypotension and heart pathology, such as cardiac stunning, forms a vicious circle. Both of those factors, initially occurring independently of each other, can lead to a state in which each of them is increasingly intertwined with the other, intensifying each other' symptoms and sequelae simultaneously. Understanding this phenomenon is crucial for reducing mortality in chronic hemodialysis patients at different stages of renal replacement therapy.

Not only can cardiac stunning cause life-threatening hemodynamic disturbances, but it may also lead to cardiac arrhythmias. During the hemodialysis session, there are changes in the internal environment of the patient not only in terms of the volume of circulating fluids but also in the composition of the electrolytes. Therefore, monitoring those components of the patient's changing inner environment and their modification when needed is an important element of the prevention of sudden death among chronic hemodialysis patients. It is known that during the first 15 to 30 minutes of the hemodialysis session, several electrolyte disorders, such as hypomagnesemia or hypokalemia, can occur [14]. Since changes in the concentrations of the electrolytes can be found shortly after the procedure, it should be assumed that they appear already during the hemodialysis session. Two of the following electrolyte disorders, hypomagnesemia and hypokalemia, which coincide with cardiac stunning, present an additional risk of life-threatening cardiac arrhythmias. It is worth mentioning that disturbances of magnesium, potassium, and other electrolytes persisting for about 30 minutes can be missed in everyday practice because, in patients undergoing chronic hemodialysis, it is extremely rare to run such tests immediately after the procedure. As the proportion of patients with different types of arrhythmias increases in the population of chronic hemodialysis patients, it is necessary to assess the concentration of electrolytes during hemodialysis in real time. The first study using a laser beam or supramolecular chemistry has produced very promising results [15].

PULMONARY HYPERTENSION AND VASCULAR ACCESS TO HEMODIALYSIS

Pulmonary hypertension in patients with chronic kidney disease occurs much more often than in the general population. The World Health Organization has proposed the classification of pulmonary hypertension based on common clinical characteristics and etiology of the disease. Chronic kidney disease treated or not treated with dialysis is classified as group 5 of the clinical classification of pulmonary hypertension, i.e., pulmonary hypertension with an unclear, multi-factorial mechanism [16]. It is an independent risk factor for death in hemodialysis patients. At the same time, the risk of death is almost the same when comparing the group of patients with pulmonary hypertension diagnosed before initiating hemodialysis with hemodialysis patients in whom pulmonary hypertension developed during renal replacement therapy [17]. From a clinical point of view, it is very interesting that in patients with pulmonary hypertension diagnosed before initiation of dialysis therapy, adaptive mechanisms are efficient enough to make it possible to perform at least basic everyday activities to a limited extent. It should be assumed that adaptation of the body to new hemodynamic conditions progresses at a rate proportional to the progression of the kidney disease, and therefore in most cases, it is a relatively long process [18]. On the other hand, the initiation of extracorporeal therapy violates this balance for a long time, leading to deterioration of heart function and potential death. The occurrence of symptomatic pulmonary hypertension in patients who started renal replacement therapy without symptoms of pulmonary hypertension is, in most cases, associated with creation of vascular access i.e. arteriovenous fistula. As many reports point out, this situation poses a certain dilemma because the initiation of renal replacement therapy affects patients' survival. The risk of pulmonary hypertension and associated increased risk of death suggest that there is a need for other methods of renal replacement therapy such as organ transplantation or peritoneal dialysis. It is known from everyday practice that, unfortunately, in many patients, alternative methods of renal replacement therapy other than hemodialysis are not possible. Therefore, taking into account the adverse effects of vascular access, i.e. arteriovenous fistula, it is proposed to choose the first vascular access depending on the severity of heart failure. Broadly speaking, in patients with New York Heart Association (NYHA) functional class IV, the first choice of access should be tunneled catheter; for NYHA class I to III, the first choice of access should

be arteriovenous fistula from the patient's blood vessels, keeping in mind the risk and possible sequelae of the central line.

MARKERS

In order to assess the risk of death in the early 90-day period of hemodialysis therapy, much hope was pinned on the use of some biomarkers in the blood. However, in 2016 the European Cardiac Society pointed to the limited usefulness of biomarkers in certain clinical scenarios in individuals from the general population. The conviction about the limited usefulness of biomarkers in cardiac diagnosis based on observations of the general population seems to be even more justified in relation to chronic hemodialysis patients [20]. Elevated serum levels of high-sensitivity troponin are of significant prognostic importance because chronic hemodialysis patients showed higher mortality in a few to several months. However, in a few hours' perspective, in the context of an acute coronary syndrome diagnosis, the diagnostic usefulness of troponin was significantly limited. The growth dynamics of troponin in the first hours of an acute cardiac event in patients treated with renal replacement was highly variable, both in terms of time and concentration.

Similar controversies concerning assessment of the total risk of death or cardiovascular events occur in relation to the changes in N-terminal prohormone of brain natriuretic peptide (NT-proBNP) in hemodialysis patients. Some observations suggest that elevated levels of this marker are a reliable indicator of an increased risk of mortality in the first three months after initiation of renal replacement therapy. At the same time, this risk increases in proportion to the plasma concentration of this peptide. However, the cut-off point of NT-proBNP concentration associated with occurrence of a real threat to health and life is much higher than in the general population defined as a group of individuals with normal renal function. The determination of serum natriuretic peptide concentration, at which more intensive diagnosis and, possibly, treatment should be instigated would be of colossal importance for daily clinical practice. Previous attempts to solve this clinical problem are unsatisfactory and are similar to the previously discussed scenario of troponins' usefulness in the diagnosis of acute coronary syndrome in chronic hemodialysis patients [21]. Difficulties in determining the threshold level of NT-proBNP that requires more intense therapy stem from the fact that plasma concentrations of this peptide increase in proportion to the decrease in glomerular filtration rate. This process occurs already in early stages of kidney disease, even before the initiation of renal replacement therapy. At the same time, it was observed that in chronic kidney disease patients, the natriuretic peptide concentration is an independent prognostic factor for worsening heart failure symptoms, regardless of the glomerular filtration rate [22].

Another limitation of NT-proBNP use in accurate prognosis of adverse cardiovascular events is due to the very high intra- and inter-individual variability of peptide behavior in patients undergoing hemodialysis treatment. As one of the proposed causes of this phenomenon, the direct impact of hemodialysis treatment was suggested. However, the analysis of several studies seems to undermine the contribution of hemodialysis as an important factor in determining the change in serum NT-proBNP concentration. Additional information indicating the low reliability of this marker in the diagnosis or evaluation of progression of heart failure has been provided by observations of the NT-proBNP level in the same patients at two-month intervals for six months. It turned out that in the group of patients without clinical signs of heart failure, in the majority of patients over the six-month period, the concentration of the peptide changed showing either an increase or a decrease. Similar changes in concentration were observed in the vast majority of hemodialysis patients; moreover, a similar time-varying nature of the concentration occurred regardless of whether the patient had previously had heart disease or not. The clinical message of this study may include a suggestion that serum NT-proBNP concentrations may be affected by factors other than cardiac dysfunction. Further observations may support such views. There was a significant relationship between the concentration of NT-proBNP and high-sensitivity troponin, hydration status, inflammation, or protein-calorie malnutrition [23]. Therefore, if we assume that the concentration of NT-proBNP is a biomarker indicating not only heart dysfunction but also, or perhaps above all, adverse systemic changes in chronic hemodialysis patients, then the risk of complications and death is proportional to an increase in the peptide concentration and seems easier to interpret. This is because the increase in

NT-proBNP concentration is a marker of the severity of systemic changes leading to impairment of the functions of many organs and systems, including the heart muscle.

SUMMARY

Identification of risk factors for early mortality in patients with end-stage renal failure is important for their future care. We hope that this will lead to the development of a management strategy to reduce the risk of early mortality in this group of patients. Myocardial stunning, intradialytic hypotension, or pulmonary hypertension in patients with arteriovenous fistula have been known for years to cause deterioration in myocardial function. In turn, observational studies show that no higher early mortality was observed in patients with diabetes included in the dialysis program compared to patients without diabetes. For prognostic and diagnostic purposes, in addition to routine tests performed in dialysis patients, it is recommended both during the pre-dialysis period and after inclusion in the renal replacement therapy program to determine the concentration of high-sensitivity troponin, natriuretic peptide (NT-proBNP), and to perform an echocardiographic study. Given the imperfection of those diagnostic methods in patients with end-stage renal disease, attention should be paid to the dynamics of changes in those parameters rather than assessment of single measurements.

CONFLICT OF INTEREST

None to declared.

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