Review

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Chronic Kidney Disease and Myocardial Infarction

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ABSTRACT

The number of patients with chronic kidney disease (CKD) and end-stage renal disease is steadily growing. With the failure of kidney function, the risk of developing acute myocardial infarction (AMI) with severe coronary artery stenosis and hospital mortality is growing. Case management of CKD and AMI is a complicated task. This review reflects the distinctive features of the course of AMI in hemodialysis patients, patients with kidney transplantation, their diagnosis and treatment.

Keywords: chronic kidney disease, acute myocardial infarction, hemodialysis, kidney transplant, diagnosis, treatment

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ACB, aorto-coronary bypass
ACS, acute coronary syndrome
AF, atrial fibrillation
AH, arterial hypertension
AKI, acute kidney injury
AMI, acute myocardial infarction
AT, angiotensin
BP, blood pressure
CA, coronary artery
CABG, coronary artery bypass grafting
CFR, coronary flow reserve
CG, coronarography
CIN, contrast-induced nephropathy
CHD, coronary heart disease
CRF, chronic kidney disease
CRF, chronic renal failure
CVD, cardiovascular disease
CVE, cardiovascular event
CVM, cardiovascular mortality
DAPT, dual antiplatelet therapy
DES, drug eluting stent
DM, diabetes mellitus

ED, endothelial dysfunction
eGFR, estimated glomerular filtration rate
ESRD, end-stage renal disease
GFR, glomerular filtration rate
GI bleeding, gastrointestinal bleeding
HD, hemodialysis
HF, heart failure
hsTn, highly sensitive troponin
KT, kidney transplantation
LA, left atrium
LV, left ventricle
LVH, left ventricle hypertrophy
LVMI, left ventricular mass index
MINOCA, myocardial infarction with nonobstructive coronary arteries
MR, mortality risk
MRv, myocardial revascularization
NSTEMI, non-ST-elevation myocardial infarction
NT-proBNP, N-terminal brain natriuretic propeptide
OR, odds ratio
PCI, percutaneous coronary intervention
RF, renal failure
RskF, risk factor
SD, sudden death
STEMI, ST-segment elevation myocardial infarction
TnI and TnT, troponins I and T

INTRODUCTION

The aging of population, the prevalence of arterial hypertension (AH), diabetes mellitus (DM), and the implementing the parameter of the estimated glomerular filtration rate (eGFR) into practice, including update diagnostic techniques have increased the rate of identifying patients with chronic kidney disease (CKD) [1]. In some cases, clinically significant kidney damage is diagnosed late. The main causes are the absence of uremia symptoms and/or the development of complications or death of CKD patients from cardiovascular diseases (CVD) even before replacement therapy becomes required. CKD is determined by a decrease in the estimated glomerular filtration rate (eGFR) and/or the presence of albuminuria. The population of patients with CKD is heterogeneous: a significant part has isolated albuminuria, an isolated decrease in eGFR, or a combination of both. CKD can be diagnosed in eGFR ⩾90 ml/min/1.73m2 and abnormal albuminuria [2, 3]. The leading cause of death in CKD patients is CVD [4-5].

CKD is an independent risk factor (RskF) for the development of coronary heart disease (CHD) and adverse outcomes in patients with CVD. As renal function worsens, the prevalence of (CHD) the number of severe coronary artery (CA) stenosis cases increase. At decrease in eGFR from ~60 to 75 ml/min/1.73 m2, the likelihood of CHD increases linearly; and in patients with CKD stages 3a–4 (15-60 ml/min/1.73 m2) the mortality risk (MR) from CVD increases twice and three times compared to patients without CKD [4, 6]. The number of patients with end-stage renal disease (ESRD) and CA stenosis (defined as stenosis over 50%) ranges from 37% to 65%, which puts them at a high risk for an unfavorable short- and long-term prognosis [7-8]. A decrease in eGFR increases cardiovascular mortality (CVM), which reaches 50%, and in people younger than 25 years is equivalent to that of the 75 – 85-year-olds in the general population [5]. In patients undergoing hemodialysis (HD), sudden cardiac death (SD) makes 25%, and the mortality rates from pulmonary embolism, acute myocardial infarction (AMI), and stroke are 12, 11, and 8 times higher, respectively, compared to the general population [9-13].

CKD is diagnosed in 30-40% of patients with acute coronary syndrome (ACS), of whom 1-2% require dialysis [5]. After the ACS development, the risk of subsequent adverse cardiovascular events (CVEs) increases; after 2 years, the CVM is 50%, which is twice as high as in individuals with normal renal function [14]. In CKD, AMI is more likely to develop than stable angina, but if uremic cardiomyopathy develops, the cause of death is not AMI, but arrhythmia. The prevalence of infarction is 73.4%, and the risk of a 5-year all-cause mortality increases progressively with decreased renal function [15]. The incidence of recurrent AMI does not differ in the groups of patients with and without dialysis [16]. Patients with severe CKD, including those on HD, are more likely to develop cardiogenic shock, cardiac arrest, and heart failure (HF) and are less likely to receive an evidence–based cardioprotective therapy (45% of patients received reperfusion therapy, 67% received aspirin, and 57% received beta–blockers) [6, 17]. In CKD with DM, the eGFR threshold below which mortality increases is higher than in patients without diabetes (60 vs. 45 ml/min/1.73m2) [18]. Renal failure (RF) is a predictor of an unfavorable outcome in HD patients who sustained AMI. They had the worst hospital and long-term outcomes: mortality was more than 20% per year, of which half of the cases were related to cardiovascular causes, being 10-20 times higher than in the general population [17, 19-20].

Investigations that can reliably detect clinically significant CHD in patients with advanced stages of CKD and ESRD are severely limited, as are strategies to reduce morbidity and mortality from CVD. Management of patients with CKD and ACS is challenging, as CKD is associated with an increased risk of both thrombotic

events and bleeding. Patients with CKD, especially those requiring renal replacement therapy, are generally excluded from clinical trials, and evidence for the efficacy and safety of AMI therapy is insufficient. Individuals with lower eGFR receive less aggressive treatment and are less likely to undergo revascularization after AMI compared to control patients [4, 16].

EPIDEMIOLOGY

Reduced eGFR predisposes to CA calcification and an increased risk of non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI) [21]. With a GFR of 15 to 59 ml/min/1.73m2, the risk of AMI in men and women increases 1.5 and 1.7 times, respectively [22]. According to the results of the Rotterdam study, among 4484 relatively healthy elderly people, the incidence of AMI increased as renal function worsened: a mean decrease in GFR by every 10 ml/min/1.73m2 increased the risk of AMI by 32% [23]. With low eGFR and/or albuminuria, the risk of AMI and CVM progressively increased.

Hospital mortality in individuals with AMI and normal renal function ranges from 1% (stage 1 CKD) to 12-35% in patients with severe renal dysfunction (stage 4-5 CKD) [14]. According to other researchers, intrahospital mortality reaches 21% in infarction patients with CKD, increases to 40% in ESRD (vs. 6-8% in general population) [24-25].

Hallan S. et al. [26], by studying the data from the Norwegian HUNT II study found that in eGFR lower 45 ml/min/1.73 m2 and a high albumin to creatinine ratio, the MR increased 6.7 times compared to patients with eGFR over 75 ml/min/1.73 m2 and a normal albumin to creatinine ratio. An isolated decrease in GFR increases the risk of NSTEMI development, and the combination of CKD and high albuminuria increases the risk of developing both types of AMI by more than 4 times [3]. Women with STEMI have a higher incidence of CKD. The eGFR reduced by 10 ml/min/1.73m2 increases the risk of the short-term and long-term mortality by 10% and 30%, respectively, for both genders. A recent study examined the outcome of patients with myocardial infarction with non-obstructive coronary arteries (MINOCA) and with impaired renal function: MINOCA was reported in every third patient (53: 178) with impaired renal function (eGFR lower 60 ml/min/1.73m2; 29.8%). Early and late prognoses with MINOCA and renal dysfunction are unfavorable, a 3-year mortality is comparable to AMI with significant CA stenosis and impaired renal function [27].

PATHOPHYSIOLOGY

In CKD and its end-stage, traditional (AH, dyslipidemia, age, smoking, and DM) and non-traditional (arteriosclerosis, vascular calcification, endothelial dysfunction and inflammation, hyperphosphatemia, hyperparathyroidism, hyperhomocysteinemia, and elevated levels of glycosylation end products that accelerate vascular calcification) CVD RskFs rule both CHD and non- CHD processes, such as left ventricular hypertrophy (LVH), diffuse fibrosis, and left ventricle (LV) dilatation [4]. In ESRD, traditional RskFs only partially explain the increased risk of coronary events. Wright J. et al. [28] believe that patients younger than 60 years of age with early CKD may benefit from CVD RskF correction. The unity of RskFs and pathogenetic mechanisms of CHD and CKD development is also reflected in the formation of systemic atherosclerosis. Many RskFs associated with uremia, such as inflammation, oxidative stress, and hyperhomocysteinemia, further exacerbate the atherosclerosis process [2].

According to the NEOERICA study, any CVD (congestive HF, CHD , or LVH) predicts a rapid decline in renal function when adjusted for baseline GFR [29]. Patients with CKD are usually older and have more comorbidities, and it is more difficult to recognize early AMI because the disease course is atypical, including electrocardiography results. Thus, comorbidity can be considered a RskF of functional decline in the kidneys, probably associated with both a sluggish inflammatory process and other factors.

CKD is a catabolic state. Alimentary insufficiency, inflammation, and atherosclerosis syndromes predominate in patients with stage 5 CKD (eGFR <15 ml/min/1.73m2), with constant activation of many acute-phase proteins and cytokines. Consequently, it is difficult to determine the impact of one particular factor on their increased risk of CVD and mortality. The mechanisms responsible for the progression of CVD and increased mortality in AMI in patients with CKD are complex [5]. The reasons for the worst AMI outcomes in patients with CKD can be attributed to a variety of factors. Possible explanations may include endothelial dysfunction (ED) and impaired hemostasis/homeostasis, atherosclerosis secondary to the accumulation of uremic toxins, and vascular calcification [30-31]. Dialysis status is associated with a generalized inflammatory state, secondary hyperparathyroidism, and electrolytic abnormalities, all associated with ED and accelerated

atherosclerosis, CA calcification leading to CHD complications. In patients with kidney diseases, vascular ED, LVH, decreased oxygen transport capacity, and the progression of atherosclerosis contribute to morbidity, AMI complications, and increased mortality [28].

ENDOTHELIAL DYSFUNCTION OF CORONARY ARTERIES AND INFLAMMATION

According to the study by Valdivielso J.M. et al. [2], the acceleration of atherosclerosis is associated with CKD progression, with the baseline state of atherosclerotic plaque, high levels of phosphates, uric acid, ferritin, and lower levels of vitamin D. An early stage of atherosclerosis and inflammation is ED, which causes vasospasm and disorders in the hemostatic system. Not only hemodynamically significant atherosclerotic changes in the CA, but also arteriolosclerosis and ED lead to impaired coronary microcirculation, LVH and myocardial fibrosis. Angiotensin (AT) II is a powerful growth factor that contributes to LVH and its fibrosis, as well as glomerular sclerosis through interaction with the AT1 receptor [2, 16, 30].

CKD occurs with inflammation and atherosclerosis [2]. CKD is an independent predictor of the multifocal atherosclerosis development, which prevalence is about 7% in case of optimal GFR level, increasing to 17-48% with the deterioration of the renal function. The inflammatory process is the cause of the kidney dysfunction and LVH progression. In endogenous intoxication, an excess of products of normal or perverted metabolism accumulates in tissues and body fluids, which is caused by 1) products of normal metabolism: lactate, pyruvate, creatinine, uric acid, bilirubin; 2) products of impaired metabolism: ketones, ammonia, etc.; 3) products of cell and tissue breakdown or toxicants (lipases, lysosomal enzymes, cationic proteins, myoglobin, phenol) entering from the gastrointestinal tract in conditions of the impaired barrier functions of membranes; 4) immune-foreign products of cell breakdown, immune complexes. Under the action of reactive oxygen species, both the selective damage to one of the structures (brush borders of the renal epithelium, mitochondrial enzymes) and the structural and functional changes in several structures occur. Having sufficient stability, reactive oxygen species interact with various biomolecules, damaging them, and also provoking chain reactions of further formation of active radicals from lipids, amino acids, nucleic acids, etc. The consequence of tissue damage is necrosis, apoptosis, the fibrosis development in damaged organs, and neoplasia. In atherosclerosis and inflammation, in response to interleukin-6 secretion, hepatocytes enhance the synthesis and secretion of acute phase proteins (C-reactive protein [CRP], serum amyloid, haptoglobin, α1-proteinase inhibitor, lipoproteins (α), and fibrinogen) into the blood [2, 6].

Albuminuria is a marker of ED and/or AH [26]. Elevated albuminuria is a valuable and easily accessible test for predicting ACS [3]. With high urinary albumin excretion, the content of von Willebrand factor, fibrinogen, and tissue plasminogen activator increases. Impaired blood flow in small intramural resistive vessels or in the coronary capillary system, which cannot be visualized using coronary angiography (CAG), leads to a decrease in coronary microcirculation, which is largely associated with renal microcirculation [2, 5]. Thus, it was found that a decrease in the coronary blood flow reserve is associated with increased mortality in patients with CKD.

The complex interaction of cytokines together with the sustained process of active and chronic inflammation that occurs as CKD progresses in combination with malnutrition, leading to HF, atherosclerosis, and death, remains to be studied.

CORONARY ARTERY CALCIFICATION

CA calcification RskFs are age, DM, and CKD. Abnormal bone mineralization that occurs in the early stages of CKD and vascular calcification are interrelated [31]. As the CKD stage progresses, the incidence of vascular calcification increases, especially in patients with stage 5D CKD. CA calcification occurs in 13.9% of patients with CKD stages 1 and 2 and up to 83% in patients with CKD stages 3-5. CA calcification is an independent predictor of CVE leading to high CVM. Deterioration of kidney function correlates with high calcium levels. Vascular smooth muscle cells, probably induced by uremia, differentiate themselves into osteoblast-like cells (due to the uptake of phosphorus by cells via sodium-dependent co-transporters of phosphorus and a decrease in vascular calcification inhibitors) initiating the process of tissue mineralization. Vascular calcification leads to hemodynamic disorders, decreased coronary microcirculation, increased arterial stiffness, and increased LVH [4, 6, 32].

The main mechanisms of CA calcification include the increased levels of serum phosphates, parathyroid hormone, and fibroblast growth factor 23, as well as the decreased levels of active vitamin D [4]. Although these factors have a significant impact on the vascular calcification progression, the most important factor is

phosphate. The American Heart Association has presented a classification of CA atherosclerosis, according to which type 4 and type 5 atheromas include calcified lesions [4]. There are two types of vascular calcifications: atherosclerotic calcification, which occurs in the intima of the artery, and Mönckeberg's arteriosclerosis, which occurs in the medial layer of the artery. Although these types of vascular calcifications can be seen in patients with CKD, Mönckeberg's arteriosclerosis is more common [30]. In advanced stages of CKD, the percentage of necrotic nucleus and dense calcium increases and the necrotic nucleus/dense calcium ratio is higher in ACS patients than in stable angina. According to a study by Mori et al. [32], CA calcification is classified into "micro-" and "macrocalcifications". Microcalcification occurs mainly in young patients and is associated with the plaque inflammation and instability, leading to ACS. In contrast, macrocalcifications tend to occur in older patients with more stable and multivessel CHD. In a recent study, Beddhu S. et al. [33], who investigated the morphology of atherosclerotic plaque using magnetic resonance imaging, proved that it was not calcification itself, but the presence of a plaque necrotic core increases the risk of future CV events.

LEFT VENTRICULAR HYPERTROPHY

LV remodeling begins in the early stages of CKD (even in the absence of AH). The uremia duration exceeding 3 years leads to more pronounced structural and functional changes in the LV myocardium. Cardiac remodeling has a direct impact on the prognosis of dialysis patients [16]. LVH is an independent RskF of death from CVD. Even in the early stages of RF, the prevalence of LVH is higher than in the general population. In patients with creatinine clearance greater than 50 ml/min, 25-50 ml/min, and lower than 25 ml/min, the prevalence of LVH was 27%, 31%, and 45%, respectively [28]. The main predictor of LVH was systolic hypertension and anemia. In renal failure, cardiomyopathy develops due to the pressure and volume overload. Pressure overload results in a concentric LVH, and volume overload causes LV dilation. Thus, systolic and diastolic dysfunctions are the result of uremic cardiomyopathy.

In patients with CKD, LVH is manifested by a pronounced impairment of the microcirculatory bed and the development of interstitial fibrosis more often than in patients with the similar blood pressure level with preserved renal function. LVH and CKD are predictors of type 2 myocardial infarction, which develops as a result of an oxygen-energy imbalance associated with reduced coronary perfusion and inadequate oxygen delivery to the myocardium.

HEMOSTASIS FEATURES OF CHRONIC KIDNEY DISEASE

Patients with CKD have an increased risk of both thrombotic events and bleeding. In addition to changes in thrombin production and other blood clotting disorders, such patients have congenital platelet abnormalities, altered response to antiplatelet drugs, and abnormal endothelial function, leading to a decreased regulation of platelet activation, as well as an impaired platelet-vessel wall interaction [34]. Reduced glycoprotein Ib (GPIb) content in the platelets of uremic patients contributes to impaired binding to von Willebrand factor- and fibrinogen-activated platelets, thereby affecting primary hemostasis. In addition, elevated plasma levels of prostacyclin and nitric oxide derived from the endothelium additionally inhibit platelet adhesion. Finally, changes in calcium content, calcium mobilization, and cytoskeletal pathology contribute to impaired platelet secretory function in patients with uremia. All of the above abnormalities potentially contribute to an increased risk of bleeding in patients with CKD. Platelets express higher levels of platelet activation markers, P-selectin and CD-40 L. The incidence of major bleeding during hospitalization independently increased by 42% for each higher degree of renal impairment [35]. For comparison, Gibson C.M. et al. [37] showed that the incidence of major bleeding increased by 12% for every 10 ml/min/1.73m2 reduction in eGFR. Most anticoagulants recommended for use in AMI, if at least partially metabolized in the kidneys, can accumulate in the body, thereby increasing the risk of hemorrhagic complications.

CLINICAL PRESENTATION AND DIAGNOSIS OF MYOCARDIAL INFARCTION

The population of patients with CKD and AMI has not changed over time: as a rule, they are more often women, elderly people, people with CVD/RskF and with the signs of acute HF (Killip class 2-3) at admission [38]. A low functional capacity of CKD patients limits the manifestation of angina pectoris. Forty per cent of patients complain of chest pain in AMI; significantly more often, the main clinical symptoms are anginal equivalents (shortness of breath, fatigue) [15], arrhythmias, hemodynamic disorders, and LV dysfunction [18]. In AMI, the pain syndrome is seen in 44% and 68% of patients on dialysis and without dialysis, respectively.

They are characterized by the development of NSTEMI. Among patients with NSTEMI, 30-40% have CKD and are 2-5 times more likely to die than patients with normal renal function [38]. Most patients do not show typical electrocardiography (ECG) abnormalities in AMI, such as ST-segment elevation and abnormal Q wave [18]. SD is particularly common in CKD, which is caused by shifts in volume, electrolytes, and drug concentrations that can cause arrhythmia in patients with myocardial disease (LVH and HF). Finally, HD-specific intradialysis hypotension and stun syndromes increase mortality [4, 6].

In AMI, patients with CKD showed marked atherosclerotic CA lesions with a high incidence of multivessel CA lesions and changes in the composition of atherosclerotic plaque associated with decreased renal function [2], the spread of limited medial calcification, and signs of coronary plaque inflammation compared to control cases [4].

STEMI was observed in only 15.9% of patients with CKD progression, compared to 32.5% of patients without CKD. Shroff G.R. et al. [15] believe that ST segment ischemic changes in CKD are masked by frequently occurring non-specific ECG findings (associated with LVH or electrolyte disturbances); and the absence of transmural myocardial ischemia in most patients with RF can be attributed to fundamental differences in AMI pathophysiology. The prevalence of left bundle branch block increases with CKD progression in elderly patients with comorbidity. In the predialysis CKD stages, an extended PR interval was identified as an independent predictor of CVEs and CVM. However, the relationship between the extended PR interval and mortality is unclear. It is assumed that the PR interval is affected by an impaired water-electrolyte balance [39].

In CKD with AMI, an echocardiographic shortening of the time of early filling deceleration (peak E) is regarded as a marker of CVE. In STEMI with decreased GFR often shows enlarged left atrium (LA), LV with unchanged parameters of the right heart [40]. LVH is a frequent finding in late stages of CKD and ESRD. Up to 75% of patients have LVH after the initiation of maintenance dialysis, and an elevated left ventricular mass index (LVMI), which is associated with unfavorable CVEs and SD [41].

In end-stage renal disease patients, including those on dialysis, there is an increase in cardiac biomarkers (troponin T and I), focal fibrosis, and chronic myocardial damage [42]. Myocardial ischemia in late stages of CKD is diffuse [5, 43], and the pathophysiological mechanisms described above may be the main cause of the high incidence of congestive heart failure and type 2 myocardial infarction [5].

Huang H.L. et al. [42] evaluated the accuracy of troponin (Tn) assay for diagnosing AMI in HD patients and found that tracking dynamic changes in Tn levels in the short term significantly improves diagnostic accuracy. A dynamic change in Tn levels of 20% or more indicates ACS; measuring at regular intervals allows you to differentiate between acute or chronic elevation. TnI levels, less often than TnT, are elevated in patients with RF. The specificity of TnI assay for the diagnosis of AMI decreases with the increase in renal dysfunction severity, showing 93%-95% in normal function (eGFR≥90 ml/min/1.73 m2), 57%-61% in eGFR<30 ml/min/1.73m2, and 40%-41% in those on dialysis [44].

In CKD with atypical clinical manifestations of AMI, without significant myocardial necrosis, or in the absence of AMI, high levels of highly sensitive cardiac Tn (hsTn) are observed, which do not allow for accurate and rapid diagnosis of infarction [45]. The correlation of hsTnI with eGFR is weak, but being statistically significant at GFR at least 15 ml min (1.73 m2) and not exceeding 60 ml min (1.73 m2), the threshold values of hsTnI increased with a decrease in eGFR, but not in patients with AMI.

N-terminal brain natriuretic propeptide (NT-proBNP) levels have been shown to negatively affect the prognosis of CKD patients. High NT-proBNP values associated with reduced GFR lower 60 ml/min/1.73m2 may identify patients at a high risk of CVM [46].

ACUTE MYOCARDIAL INFARCTION IN DIALYSIS PATIENTS

Individuals on HD often do not have the classic signs and symptoms of CHD; they are less likely to be correctly and promptly diagnosed with ACS [6, 18]. The main causes are: low likeliness to display symptoms of coronary ischemia; a smaller proportion of patients with chest pain who do not have ST segment abnormalities; patients with ESRD and DM have a lower sensitivity to anginal pain, are physically less active and do not reach the physical activity threshold that would raise anginal symptoms or fatigue [19]. In addition, CHD symptoms may be erroneously associated with CKD-induced nephrogenic anemia rather than coronary pathology [16], and Tn levels in the absence of ACS will be chronically elevated [47]. Meanwhile, CHD occurs in 50% of patients over 65 years of age who are on dialysis [18].

Over the recent 15 years, the incidence of AMI (especially NSTEMI) in those on dialysis has abruptly increased [17]. CVD has become the main cause of CVM in patients with stage 5 CKD requiring long-term dialysis, with approximately 15% of these deaths being associated with AMI [47]. Gradaus F. et al. [48] reported a rapid progression of CA atherosclerosis in HD patients. After 30 months of dialysis treatment, 50% of patients had new hemodynamically significant CA stenoses of over 50% of the lumen diameter. AMI developed in the first 3 months of dialysis in 22.5% of patients, and in 37.5% within 1 year on dialysis. Of these, NSTEMI and STEMI were diagnosed in 62% and 37% of patients, having 80% atherosclerotic involvement of several CAs. RskFs of AMI development in dialysis patients were an old age, the previous history of CHD and DM as the cause of nephropathy. In-hospital fatality rate was 30%, which is significantly higher than in the general population. STEMI was less frequently diagnosed in patients on dialysis. At 1 year after index percutaneous coronary intervention (PCI) for STEMI, patients were 3.79 times more likely to die compared to those without CKD [18]. Ismail M.D. et al. [49] showed that patients with CKD accounted for a significant proportion of all STEMI cases undergoing PCI (23.1%). In patients with CKD, PCI can cause a number of complications. Poor prognosis is explained by a combination of difficulties in the diagnosis and treatment of AMI, a high risk of complications, and a lack of guideline-recommended knowledge on the management of this category of patients [10].

Age, diastolic BP, DM, previous history of CA disease, duration of dialysis therapy, high-density lipoprotein cholesterol, serum phosphate, hsTnT, serum creatinine, and low eGFR are positively associated with the risk of AMI. MR is higher in patients with elevated hsTnT ($P = 0.02$), and the degree of risk depends on the level of hsTnT increase [45]. An unfavorable outcome of CKD after sustained AMI is associated with the presence of more severe coronary artery involvement or comorbidities often associated with CKD [16]. Dialysis patients who experienced AMI, compared to those without dialysis, have several times higher mortality after AMI, they receive less invasive therapy and less secondary preventive treatment. Patients with AMI receiving HD had a 2-fold higher MR compared to the entire dialysis population [50]. The 18-year follow-up period showed that the decrease in 1-year mortality was about 13% and might have been associated with an improved general survival [10]. However, an increasing number of new studies indicate that over time, there is a significant increase in the use of invasive treatment strategies for patients on dialysis, which is consistently associated with their improved survival [51].

ACUTE MYOCARDIAL INFARCTION IN KIDNEY TRANSPLANT PATIENTS

Patients with kidney transplantation (KT) are at a high risk for CVD. CVD is the leading cause of death in recipients, accounting for 30% of all deaths in patients with a functioning graft [52]. The 30-day cumulative incidence of AMI in the post-transplantation period was 1.5%, and that of ischemic stroke was 0.3%. MR or the risk of serious CVEs in them was 6.4% higher than in the general population. In the study by Lam N. N. et al [53], serious CVEs and MR decreased throughout all years after transplantation and were low in the general population and high in the CKD patient population. The risk of developing AMI after KT is lower than in patients on dialysis. According to Kasiske B.L. et al. [54], the risk of AMI after KT is reduced by 17% compared to patients on dialysis. Although kidney transplants in patients with stage 5 CKD are associated with a reduced risk of developing CVD, they are still the main cause of morbidity and mortality in recipients [55]. Patients with graft rejection requiring dialysis have a more than 2-fold increased risk of AMI development [56]. The prevalence of hemodynamically significant CA stenosis (70% of lumen reduction and over) in patients referred for KT ranges from 25% to 59%, indicating an unfavorable outcome and increased CVM [18]. According to Awan A.A. et al. [57], at 1 and 10 years after transplantation, CVM was registered at 24.5% and 22.1%, respectively. There is a relationship between AMI and a delayed graft function [58]. Recovery of renal function through transplantation significantly reduces the risk of AMI and death; and a reduced GFR is a strong predictor of CVEs after transplantation. The CVE risk rate ranges from 3% to 5% [59]. Risk stratification of patients after KT can be based on traditional and non-traditional RskFs and cumulative risk assessments, the use of structural or functional parameters (LVH), clinical assessment (BP), and biomarkers (TnT, NT-proBNP, CRP) [60].

Impaired myocardial perfusion before KT is an independent predictor of CVEs after transplantation. The time between screening for myocardial ischemia and transplantation, as well as the frequency of screening, is a matter of debate [61]. The post-transplant period is associated with a high risk of AMI development, which incidence decreases after transplantation. In the first three months, the risk of AMI development reaches 45%

[62]. RskFs in the post-transplant period can be the age over 60 years, obesity, smoking, LVH, DM, AH and dyslipidemia, and non-traditional RskFs: male gender, time period on dialysis therapy before transplantation (more that 1 year), donor's history of AH, transplantation from an older donor, immunosuppressants. RskF correction and effective pharmacological therapy before surgery reduce the incidence of acute thrombotic events [4]. Preoperative CG can assess the risk of CVD in high-risk patients. In the study by Paizis I. A. [63], atherosclerotic involvement of one, two, and three CAs was seen in 26%, 22%, and 26% of patients with KT, respectively. Within 1 and 3 years after KT, 2.27% and 10% of AMI patients died. AMI (more often NSTEMI) is mainly diagnosed in young men (69%) with comorbidities (76% with AH; 39% with DM; 33% with dyslipidemia; 22% with HF; 13% with obesity; 11% with AF; 6% with heart disease). CHD, previous history of MI, and previous history of percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) were significantly more commonly seen in KT patients ($p<0,0001$) than among patients without KT. The mean time from KT surgery till AMI development was 0-3 months, and infarction was more common in patients with cadaveric kidney transplantation [53].

In kidney transplant recipients the combination of pathophysiological and clinical data explains an early onset of AMI: a high prevalence of CVD RskFs (AH, DM, dyslipidemia); CHD; inadequate efficacy of cardioprotective drugs or their inadequately rare prescribing to patients with CKD; the immediate postoperative period is associated with prolonged inflammation that may contribute to the destabilization of atheromatous plaques and ED; the acquired thrombophilia status after transplantation (the use of immunosuppressive drugs, viral infections, and discontinuation of anticoagulants in the first week after transplantation); anemia (known for its role in the development of type 2 NSTEMI) [55, 59-60, 63-64].

In transplant candidates who underwent myocardial revascularization (MRv) and KT, one-year and 3-year survival rates were 98% and 88%; in those without MRv and KT they were 75% and 37%; and 94% and 90% with MRv from the transplant waiting list [65].

Moreover, NSTEMI was more common in patients after KT than without transplantation, and an inverse relationship was observed in STEMI [66]. In patients after KT, STEMI had better outcomes compared to patients undergoing HD. Recipients are not affected by changes in intravascular volume, as are the patients undergoing maintenance dialysis. Therefore, the risk of SD and HF decreases after KT with a concomitant improvement in the metabolic status, absence of uremia, and the fluid balance return to normal [67]. Moreover, most patients have the LVMI, end systolic and diastolic LV diameters, and LA size normalized [68]. KT leads to a partial correction of metabolic disorders, potentially contributing to a reduced risk and improved outcomes after AMI [65].

Individuals who have had KT have a lower risk of developing CVD than patients with stage 5 CKD, but are more likely to develop CVD compared to the general population. A 2-year survival rate after AMI is 50% in patients with KT, and approximately 75% in the general population [66]. For patients after KT Soveri I. et al. [60] developed a formula for calculating the CVD risk and MR.

TREATMENT

Treatment of CKD patients with ACS is complicated by their characteristic high risk of both thrombotic conditions and bleeding [43]. The choice of medication between myocardial reperfusion for CKD is controversial. Most medications (acetylsalicylic acid, beta blockers, angiotensin-converting enzyme inhibitors, angiotensin II receptor antagonists, and nitroglycerin) used for ACS can be used in CKD patients, including those on HD, if they have no absolute contraindications. Statins can be administered to predialysis patients younger than 50 years of age [4]. As eGFR is decreasing, the benefit of statin prescription declines; there is no evidence of statin treatment in patients on dialysis therapy. At the same time, simvastatin and ezetimibe reduce the incidence of atherosclerotic CVEs in all patients, regardless of dialysis procedures.

High mortality and complications associated with recurrent stent thrombosis in patients with CKD should not exclude the use of appropriate antiplatelet therapy. An analysis of 28,320 randomly selected HD patients was conducted as part of the DOPPS study, which showed that aspirin therapy reduced the risk of cerebral circulatory disorders and did not significantly affect all-cause mortality [69]. According to McCullough P.A. et al. [70], CKD patients treated with aspirin in combination with beta-blockers experienced a decrease in the incidence of CVM compared to those who did not receive either drug. Recent prospective open-label randomized study that included 101 patients with CKD have not revealed any benefit of low-dose aspirin in reducing the combination of CCC, ACS, cerebrovascular diseases, HF or non-fatal diseases of peripheral

arteries, but they have found a statistically significant relationship with a reduction in the incidence of coronary events ($p = 0.014$) and an RF progression ($p = 0.043$) [71].

In CKD patients who underwent PCI, the antiplatelet response to antiplatelet agents is impaired. Park S.H. et al [72] showed a low antiplatelet effect of clopidogrel, and no effect of increasing the dose in patients with CKD and AMI. Patients with stage 4, 5, or 5D CKD who require PCI should receive antiplatelet therapy with clopidogrel. Ticagrelor and prasugrel cannot be recommended for CKD 5 and 5D [43]; when compared with clopidogrel, they reduce the incidence of thrombotic conditions, but increase the number of bleeding events. CKD patients have a higher risk of developing gastrointestinal (GI) bleeding than patients with preserved renal function. High rates of in-hospital GI bleeding in the dialysis group were caused by platelet dysfunction due to severe RF, heparinization during HD. In patients who has suffered AMI and are in predialysis stages of CKD, the cause of in-hospital GI bleeding may be associated with the age and comorbidities, rather than RF. The antiplatelet efficacy of aspirin is less than that of clopidogrel, but its use in combination with clopidogrel and an oral anticoagulant (OAC) increases the risk of serious bleeding. There was no statistically significant difference in the adjusted 1-year risk of bleeding with prasugrel and clopidogrel between patients with CKD and without it: 2.6% versus 3.5%, respectively [16]. According to the analysis of two registries, prasugrel and ticagrelor used in ACS patients with CKD, when compared to clopidogrel, were associated with lower MR and recurrent AMI incidence without increasing the risk of major bleeding [73].

In a subanalysis of the PROMETHEUS trial, patients with CKD compared with those without CKD had a significantly high 1-year risk of serious adverse CVEs (1.27, $p < 0.001$) and bleeding (1.46, $p < 0.001$). In CKD without PCI, clopidogrel therapy was associated with reduced safety and no anti-ischemic benefit: in a repeated analysis of the CHARISMA study, clopidogrel+aspirin therapy compared with aspirin was associated with increased overall mortality and CVM [75]. There are no specific indications for clopidogrel dose adjustment in RF. Prasugrel dose adjustment is not required in patients with RF, including patients with ESRD and/or dialysis-dependent patients. Similarly, no dose adjustment of ticagrelor is required in patients with RF. Finally, due to the lack of a reliable evidence base, all current recommendations (ACC/AHA, ESC, RCS) suggest choosing P2Y12 inhibitors for patients with ACS and comorbid DM and/or CKD, similar to the recommendations for patients without these diseases. There are limited data on determining the duration of dual antiplatelet therapy (DAPT). At advanced stages of CKD, the extended duration of this therapy may be associated with an excessive risk of bleeding. Based on the update from the ESC, it can be assumed that at stages 3 and 4 CKD (eGFR 15-59 ml/min/1.73 m2) with no other comorbidities, the risk of ischemia is weightier than the risk of hemorrhage, whereas in stage 5 and 5D CKD (eGFR <15 ml/min/1.73 m2), the risk of hemorrhagic events increases, weighting the risk of bleeding. Extension of DAPT for more than 12 months after ACS with PCI is possible if the eGFR is between 30 ml/min/1.73 m2 and 60 ml/min/1.73 m2. Clopidogrel (or aspirin IIaA in the ESC update) should be continued for 12 months after PCI [43]. "Triple therapy" is not safe in the population with CKD severe stages. While there are no studies supporting the use of new OAKs in patients in stage 5D CKD, a 2018 meta-analysis of five observational studies found that among patients with severe stages of CKD, apixaban use was associated with a lower risk of major bleeding compared to warfarin. Apixaban is safer than warfarin in terms of bleeding. In addition, there are no studies on the use of antiplatelet and anticoagulant therapy in patients with CKD and/or ESRD with AF who have undergone PCI [4, 16, 43].

The optimal MRv strategy for patients with CKD remains unclear, as there are no results from randomized clinical trials. Patients with CKD are less likely to receive early coronary interventions, as shown in the CRUSADE study [76], and only 45% have been treated with invasive procedures [43]. It is difficult to decide which treatment strategy is best. CKD is often considered to be the cause of patients's refusal from MRv, as they are at a higher risk of procedural complications such as acute kidney injury (AKI), major bleeding leading to a new AMI case, and death [4]. Short-term procedural risks are higher among patients with CKD. The incidence of AKI in people who have had KT is higher (21.2%), given their CKD status and the use of nephrotoxic immunosuppressive drugs [6]. The risk of complications such as contrast-induced nephropathy and bleeding is likely to further exacerbate the adverse outcome. This is relevant in patients with ESRD (eGFR <15 ml/min per 1.73m2). There is no benefit from PCI in this group of patients, even when using the latest generation of stents.

Regardless of the intervention method, CKD patients with ACS should be treated with MRv. All types of MRv are associated with a high risk of morbidity and mortality in CKD compared to the general population.

The approach to MRv should include consideration of the issues such as the risk of restenosis, stent thrombosis, and bleeding. One should also consider the risk of the revascularization being able of permanently excluding patients from transplantation or delaying it. MRv is necessary for patients with affected left CA trunk, proximal anterior descending branch stenosis, and multivessel CA involvement [4].

CKD is a powerful predictor of hospital mortality in the ACS patients without ST-segment elevation treated with PCI. Shaw C. et al. [77] studied the results of MRv in patients with NSTEMI and GFR lower 60 ml/min and demonstrated that the revascularization strategy reduced a 1-year all-cause mortality by 30% compared to conservative treatment. In NSTEMI (odds ratio [OR] 0.5 for stage 3 and 4 CKD, and 0.52 for ESRD, $p<0.01$, statistically significant), PCI resulted in lower hospital mortality. Similar results were confirmed by Young-Altahan R. et al. [78] who showed that early MRv was associated with a reduced risk of nosocomial complications, 30-day and 1-year all-cause mortality compared to the group of patients without intervention. Patients after KT or on dialysis who had received MRv had a good survival rate, while those who refused invasive treatment had a low survival rate (1- and 3-year survival rates were 75% and 37%, respectively). The mortality rate of patients with critical CA stenosis who underwent MRv without PCI was 52%. In other studies, there is insufficient evidence for the benefit of MRv in patients with ESRD, with the exception of those with three-vessel CA involvement. Interestingly, in patients with CKD progression, early intervention was associated with lower mortality rates [39, 43], re-emphasizing the crucial role of adopting this strategy. Based on these studies, evidence suggests that among CKD patients suffering from NSTEMI, an early intervention with PCI reduces both short- and long-term mortality compared to conservative treatment alone. Recent studies have shown that among carefully selected patients, some of the treatment benefits appear to outweigh the risks. The benefit of early MRv has been shown in patients with creatinine clearance between 30 and 60 ml/min. Data from the SWEDEHEART registry have demonstrated that patients with NSTEMI and RF of mild to moderate severity, early MRv associated with greater 1-year survival, however, the benefit decreases with the decrease in renal function and less statistically significant in patients with eGFR<15 ml/min per 1.73 m2 or on dialysis (RR 1.61, 95% CI 0.84 to 3.09, $p=0.15$) [79].

However, patients with CKD progression and STEMI were less likely to undergo PCI and had a higher hospital mortality rate than patients with normal renal function. Among patients with stage 3 and 4 CKD, STEMI has a higher hospital mortality rate, almost twice that of patients with NSTEMI [80]. In the absence of clinical studies, patients with STEMI need an invasive strategy, as do patients from the general population [4].

CA calcification is another problem for coronary intervention [43]. In patients undergoing dialysis, there is a marked calcification of blood vessels, multivessel CA involvement, difficulty in delivering the stent to the affected site. CKD is an independent predictor of worse outcomes, including post-PCI mortality. Fujii H. et al. [6] suggest the following indications for PCI: (1) emergency case, (2) early-to-mid-stage CKD, (3) high risk associated with surgery, (4) short life expectancy, and (5) contraindications for CABG (single-vessel or two-vessel involvement, except for the anterior interventricular branch and trunk of the left CA); and CABG should be performed in patients receiving dialysis if their condition is relatively stable. However, the investigators note that MR significantly outweighed the risk of further eGFR decline and progression to ESRD. More importantly, there were no differences in long-term renal function in patients who underwent CG with or without PCI. Bangalore S. [81] studied the efficacy of an invasive strategy in patients with progressive CKD and stable CHD. There was no evidence that the initial invasive strategy compared to the initial conservative strategy reduced MR or the incidence of non-fatal MI. The initial invasive strategy reduced the incidence of non-procedural AMI and increased the incidence of procedural AMI. The incidence of stroke, death, and dialysis initiation was higher in the invasive treatment group. However, there was no difference in mortality between the invasive and conservative strategies. Among the patients with clinically stable CHD who are scheduled for major vascular surgery, a long-term mortality after preventive CA revascularization was similar to that in optimal drug therapy (23% vs. 22%, $p = 0.92$) [4].

Early guidelines of the Association of Cardiothoracic Surgery recommended CABG instead of PCI for patients with moderate to severe CKD with multivessel CA involvement, provided life expectancy was >1 year [82]. These studies were conducted when only bare metal stents or first-generation drug-eluting stents were available for PCI. Next-generation drug-coated stents are the preferred treatment option for patients with CKD.

PCI and CABG in CKD bring about a high risk of AKI. There are no randomized controlled trials comparing CABG and PCI in HD patients [16]. The rates of surgical mortality, AMI, and stroke did not differ for patients undergoing CABG or PCI. However, when compared with PCI in patients after CABG, the risk of repeat

revascularization, 5-year mortality was lower and the long-term prognosis improved [83]. The postoperative 1-, 5-, and 8- year survival rates of HD patients who underwent CABG were 81.5%, 72.0%, and 68.4%, respectively. Death RskFs after CABG in patients with ESRD compared to patients without ESRD were AH, DM, previous history of AMI, stroke, and COPD [84]. CABG significantly reduced mortality and the incidence of AMI and repeated revascularization in patients who did not receive dialysis compared to PCI and drug therapy [85]. In CABG, compared with PCI, patients on dialysis had a high short-term MR and stroke risk. After PCI, a long-term high MR, the risk of AMI, and repeated revascularization were higher than in the general population. A retrospective study of patients on a maintenance dialysis with or without ACS compared the outcome between PCI and CABG; compared to CABG, the PCI with drug-eluting stents (DES) was associated with higher mortality in patients with ACS, but not in patients without ACS, and the PCI with bare-metal stents, compared to CABG or DES, yielded higher mortality both in ACS and non-ACS cohorts [81]. Patients with CKD are more likely to have a multivessel involvement, CABG provides complete revascularization and thus may lead to a better long-term survival. Therefore, a timely treatment of AMI in patients with CKD is critical to reducing both morbidity and mortality.

CONCLUSION

Chronic kidney disease is an independent risk factor for the development of CHD and adverse outcomes in patients with cardiovascular disease. Reduced eGFR predisposes to calcification and multivessel coronary artery involvement, and an increased risk of non ST-segment elevation myocardial infarction and ST-segment elevation myocardial infarction. The incidence of acute myocardial infarction in dialysis patients and after kidney transplantation s high. Acute myocardial infarction is one of the main causes of death in patients on hemodialysis and in the postoperative period of kidney transplantation. The pathophysiology of acute myocardial infarction in patients with chronic kidney disease CKD, especially in the end-stage renal disease, is multifactorial. Regardless of the coronary heart disease severity, kidney transplantation contributes to increased survival in the long-term period.

Chronic kidney disease at severe stages is generally excluded from clinical trials, and evidence for the efficacy and safety of the therapy for acute myocardial infarction is insufficient for them. In coronary artery by-pass grafting, the hospital mortality and stroke were higher among patients on dialysis compared to those in percutaneous coronary intervention. Patients with percutaneous coronary intervention have a high MR, the risk of AMI, and re-revascularization in the long-term. The standard AMI treatment protocol is not acceptable for patients with chronic kidney disease. The decision on the MRv for patients with chronic kidney disease should be made after considering the clinical and laboratory-instrumental characteristics of the patient.

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