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Coronary Artery Disease and Impaired Renal Function

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Abstract **Review Article**

Patients with chronic kidney disease (CKD) present with an accelerated form of coronary artery disease. Their vascular morbidity and mortality are significantly increased compared to the general population. In addition to traditional risk factors, there are also factors related to uremia and dialysis, which together induce an acute phase reaction via cytokine release and endothelial dysfunction. This chronic microinflammatory environment, combined with a defective immune system and disturbances in calcium and phosphate metabolism, leads to severe atherosclerosis. Coronary artery disease in chronic kidney disease. Patients with chronic kidney disease (CKD) present with a very early and severe form of coronary artery disease. Morbidity and mortality are extremely high. This is because, in addition to classic risk factors, uremia and dialysis induce cytokine release and changes in lipid metabolism, which creates a microinflammatory environment. This leads to an acute phase reaction and endothelial dysfunction. This reaction is accelerated by a defective immune system and disturbances in calcium and phosphate metabolism. The conservative treatment approach is similar to that for patients without kidney failure, but coronary artery bypass grafting is superior to PTCA ± stent implantation based on long-term follow-up. The use of arterial grafts significantly reduces morbidity and mortality, particularly in diabetics.

Keywords: Chronic Kidney Disease (CKD), Coronary Artery Disease (CAD), Uremia, Dialysis, Cardiovascular Mortality.

EPIDEMIOLOGY

Patients with kidney failure are considered a high-risk group for the development of coronary artery disease. This risk is already significantly increased in the pre-dialysis stage. In dialysis patients, cardiovascular mortality is 10 to 20 times higher than in the general population and amounts to approximately 9% per year [1]. The difference in mortality is particularly pronounced in the younger age groups [2]. Cardiovascular causes of death in dialysis patients are the most common cause (49%), followed by infections (15%), 6% of patients die from cerebrovascular events and 4% from malignant tumors. This does not mean that malignant tumors are rare in patients with renal failure, but rather that the life expectancy of these patients is so short that they often do not even experience their malignancy [3]. If a dialysis patient has suffered a myocardial infarction, their life expectancy is significantly reduced. Cardiovascular mortality is 40.8% after one year, 51.8% after two years, 70.2% after five years, and 83.3% after ten years.

Cumulative mortality is even higher, at 59.3% after one year, 73% after two years, 89.9% after five years, and 97.3% after ten years, i.e., 16 to 19 times higher than in the normal population [4]. After a myocardial infarction, overall mortality is directly correlated with the degree of renal impairment. In patients with mild renal impairment, known risk factors play a more important role than renal function per se. Only when creatinine clearance is less than 40 ml/min does the importance of uremia as a prognostic factor exceed other risk factors [5].

The question now arises as to why patients with chronic kidney disease have such high cardiovascular and overall mortality. Of course, many patients suffer from hypertension, hyperlipidemia, diabetes mellitus, or are smokers, but many have no history of such conditions. Dialysis patients often have normal or slightly elevated total cholesterol and LDL cholesterol (LDL-C). Low cholesterol is a risk factor for increased mortality and a marker of malnutrition [6]. HDL cholesterol (HDL-C) is low in 20–40% of patients, and

Triglycerides are elevated in 20–40% of them. Elevated triglyceride levels in patients with significant renal impairment are the result of increased production and accumulation of triglyceride-rich lipoproteins. Thus, ApoB, ApoC-III, and ApoE, all of which contain triglyceride-rich lipoproteins as components, are elevated [7]. As mentioned above, both HDL-C and apolipoprotein A-1 are reduced in dialysis patients. Reduced HDL-C metabolism is the result of reduced activity of cholesteryl ester transfer protein (CETP), but often also of lecithin cholesterol acyltransferase (LCAT), which is the key enzyme for the esterification of free cholesterol. The latter is therefore responsible for the reverse transport of cholesterol via HDL-C to the liver. The influence of altered LCAT activity on the progression of atherosclerosis is well known. Uremia induces an acute phase reaction in the body. This leads to changes in the composition of HDL-C, which shifts from an antioxidant to a prooxidant lipoprotein. This is primarily due to reduced paraoxonase activity in plasma and HDL-C. Paraoxonase inhibits the oxidation of HDL-C and the lipid peroxidation of LDL-C. Reduced activity of this enzyme, as in renal failure, leads to changes in the structure and function of HDL-C and LDL-C.

These modified lipoproteins are not recognized by their receptors and therefore circulate longer in the plasma with a prolonged half-life until they are absorbed and metabolized via so-called "non-saturable scavenger receptor pathways." A prolonged half-life and circulation of these lipoproteins in plasma lead to their further modification by oxidative and glycosylation processes. The reason for this is the reaction of the aldehyde and ketone groups of carbohydrates with amino acids to form glycosylation end products (AGEs). These react with LDL-C, but also with endothelial cell receptors, thus triggering an inflammatory stimulus. In addition, impaired renal function leads to reduced renal clearance of AGEs [8]. Circulating AGEs can reduce the plasma clearance of native LDL-C and increase LDL-C oxidation. The consequences of this accumulation of altered lipoproteins are the formation of foam cells and the progression of atherosclerosis.

The increased uptake of ox-LDL-C by monocytes/macrophages results from increased expression of scavenger receptors on these cells in uremic patients [9]. This occurs via a peroxisome proliferator-activator receptor on these cells. The increased formation of oxygen free radicals in renal failure contributes to LDL-C oxidation. Ox-LDL-C has antigenic effects and leads to the formation of antibodies (AB). These antibodies against ox-LDL-C are known to play a pathogenic role in the development of atherosclerotic changes in dialysis patients [10]. Although LDL-C is often low overall in patients with renal failure, its subfractions differ significantly from those of the normal population.

In a study by Rajman *et al*., [11], LDL-C was divided into seven subfractions based on its electrophoretic mobility. Control patients, predialysis and dialysis patients, peritoneal dialysis patients, and kidney transplant patients with approximately the same LDL-C levels were examined. With increasing severity of renal failure, the percentage of "small dense particles" in total LDL-C increased. LDL-C became more atherogenic.

As mentioned above, morbidity and mortality due to cardiovascular disease are increased in dialysis patients. One of the first steps on the path to atherosclerosis is endothelial dysfunction. The consequences are increased adhesion and permeability of mononuclear cells to the endothelium and the release of cell markers and cytokines. Uremia is associated with elevated levels of pro-inflammatory cytokines, which can upregulate the expression and release of various adhesion molecules. These cytokines include: B. Interleukin-1 (IL-1) and tumor necrosis factor (TNF-α). Selectins are involved in leukocyte rolling along the endothelium, VCAM-1 (vascular cell adhesion molecule-1) and ICAM-1 (intercellular adhesion molecule-1) are of great importance for firm adhesion and transendothelial migration of leukocytes. Elevated levels of von Willebrand factor (vWF) and thrombomodulin are markers of dysfunction endothelial ent. The level of vWF is correlated with the extent of peripheral atherosclerosis. Thrombomodulin is an indicator of endothelial cell destruction. It is increased in diabetics, particularly in the presence of microvascular complications. Significantly increased levels of vWF and thrombomodulin are also observed in different severities of renal failure compared to control subjects. ICAM-1 and VCAM-1, representatives of endothelium-derived adhesion molecules, play a major role in the pathogenesis of atherosclerosis. sICAM-1 is often present several years before the clinical manifestation of coronary artery disease [12]. The same is true for sVCAM-1, sL-selectin, and MCP-1 (monocyte chemotactic protein-1). MCP-1 is produced by endothelial cells after stimulation with IL-1 and TNF-α and plays an important role in the migration and activation of monocytes, T cells, and smooth muscle cell proliferation. However, MCP-1 also increases during erythropoietin treatment [13].

In a study of dialysis patients, it was shown that ICAM-1, VCAM-1, and MCP-1 levels were already significantly increased before hemodialysis compared to control patients, but the dialysis process itself led to a further significant increase in these parameters [14]. In various studies, the magnitude of the increase was also associated with the bioincompatibility of the dialysis membrane and the purity of the dialysate. As vital as hemodialysis is, it contributes to the progression of atherosclerosis. Thus, in uremia, classic risk factors such as hypertension, hyperlipidemia, diabetes mellitus, and smoking, but also factors related to uremia and dialysis,

are responsible for the onset and progression of atherosclerosis. Factors related to uremia include acidosis, uremic toxins, elevated homocysteic acid, increased production of oxygen free radicals and oxidized LDL-C, recurrent infections, activation of the complement cascade, and a tendency toward platelet aggregation. During dialysis itself, the biocompatibility of the dialysis membrane plays a role, as do the purity of the dialysate, endotoxins, access site infections, and backfiltration. Together, these factors lead to a microinflammatory environment with an acute phase response via the release of proinflammatory cytokines and endothelial dysfunction. As modulation, positive acute phase proteins (APRs), such as C-reactive protein (CRP), serum amyloid A (SAA), fibrinogen, and haptoglobin, as well as negative APRs, such as transferrin, increase, and hypoalbuminemia develops. Against the background of a disturbed immune system and changes in calcium and phosphate metabolism, this systemic inflammatory reaction leads to accelerated atherosclerosis. CRP is not only a marker of atherosclerosis but also plays a causal role. Many years before a vascular event, CRP levels are already elevated; CRP activates the complement system, induces the expression of tissue factor and adhesion molecules, and binds to enzymatically degraded LDL particles [15, 16].

It has also been shown that foam cells in atherosclerotic plaques stain positively for CRP and that CRP deposits are found in the fibroelastic and fibromuscular layers of the intima with activated complement. In the experimental model of infarction, CRP injection resulted in increased tissue death via complement-dependent mechanisms [17–19]. There are also significant associations between elevated CRP levels and hypoalbuminemia, malnutrition, erythropoietin resistance, and morbidity and mortality in dialysis patients [20]. In a four-year study, a significant direct association between CRP level and overall and cardiovascular mortality was demonstrated [21] (Fig. 1). Nitric oxide (NO) has a strong vasodilatory effect, is an inhibitor of platelet activation, inhibits platelet aggregation with prostacyclin, and induces platelet disaggregation. Increased production via inducible NO synthase (iNOS) leads to clinical symptoms and progression of atherosclerosis. The "interleukin hypothesis" supports the hypothesis of the importance of NO in intradialytic hypotension.

However, NO is also a cytotoxic molecule. It reacts with superoxide oxygen radicals, e.g., B, to form peroxynitrite, which causes severe oxidative damage to lipids, DNA, and proteins. Peroxynitrite induces breaks in the DNA molecule, activates the DNA repair enzyme poly(ADP)-ribosyltransferase and the tumor suppressor protein p53, which trigger apoptosis. One possible cause of increased NO production is the dialysis process itself. Blood flowing back from the extracorporeal circuit of the dialysis machine comes into contact with endothelial cells and induces the release of growth factors, cytokines, chemokines, and vasoactive substances. In addition, there is an increase in NOS activity with the expression of iNOS messenger RNA (mRNA). In addition to the bioincompatibility of dialysis membranes with cytokine release, the buffer solution used in dialysis also plays a role. Acetate-containing buffers increase NOS activity, while acetate-free buffer solutions practically do not show this reaction. Thus, hemodialysis leads to periodic stimulation of endothelial iNOS with an increase in NO, induction of generalized inflammation disruption of endothelial function leading to vasculopathy and atherosclerosis as well as vascular occlusion, but also apoptosis [10].

Clinical and Diagnostic

Patients with renal failure are at higher risk of silent ischemia and often present with clinically atypical symptoms of acute coronary syndrome. Furthermore, elevated troponin T (TnT) levels can be detected in approximately one-third of dialysis patients without clinical symptoms. With first-generation TnT assays, the values were even higher. The reasons for this are complex. Approximately 7% of TnT is present in the cytoplasm of myocytes in free form; the remainder is bound to tropomyosin and other troponins in the sarcomere in a complex form. The TnT content per gram of myocardium is twice that of troponin I (TnI). The TnI assay is less accurate in the lower range of the reference band than the TnT assay. Free and bound TnT are relatively large molecules with molecular weights of 37 and 77 kDa, respectively, which explains why there is virtually no renal clearance. This renal clearance of TnT is less than 0.01 ml/min in healthy kidneys [23].

While TnI values decrease after hemodialysis, TnT values increase (hemoconcentration?). The cause of elevated TnT levels in dialysis patients without clinical symptoms of acute coronary syndrome is ultimately unclear. Reexpression of TnT in skeletal muscle in the context of uremic myopathy has been suggested as a possible explanation. Since diabetics with renal failure and secondary damage often show increased TnT values, the increased occurrence of glycosylation end products has also been held responsible, particularly because they can induce gene expression [24]. In a multivariate analysis, elevated TnT levels were correlated with patient age, hypercholesterolemia, known coronary artery disease, left ventricular hypertrophy, weight gain between hemodialysis sessions, and overall mortality [25]. Elevated TnT levels are certainly a marker of subclinical myocyte death in dialysis patients without precordial symptoms. Clinically silent myocardial ischemia or unrecognized left heart failure may play a role. In any case, elevated TnT values define a group of at-risk patients for whom additional diagnostic and therapeutic measures are indicated.

Therapy

In addition to purely pharmacological treatment of coronary artery disease, which should be performed in

a manner similar to that used in patients without renal failure, percutaneous transluminal coronary angioplasty (PTCA) with stent implantation and coronary artery bypass grafting (CABG) are the methods of choice. PTCA without stent implantation shows poor short- and long-term outcomes in patients with renal failure. Gruberg *et al*., showed that even patients with a creatinine between 1.4 mg% and 3 mg% with equally good vascular opening rates using PTCA plus stent implantation had higher in-hospital cardiovascular and overall mortality. Vascular complications and major bleeding, as well as deterioration of renal function and the need for hemodialysis, also occurred more frequently. One year after the study, more patients with renal failure had died compared to the control group (17.4% vs. 5.1%), and the number of myocardial infarctions was higher [26].

Even in patients requiring dialysis, angiographic success was as good as in the control group. However, nine months after surgery, significantly more patients had died than in the control group (18% vs. 2%), and the same vessel required reintervention significantly more often (35% vs. 16%). The rate of intervention on other vessels was also higher (39% vs. 19%), and major cardiac events occurred more frequently in dialysis patients than in the control group (50% vs. 25%) [27].

Post-surgery mortality is directly related to the severity of renal failure. One-year mortality showed a relative risk of 1.46 for a creatinine clearance of 70 ml/min, a relative risk of 3.7 for a clearance of 30 ml/min, and a value of 8.91 for patients requiring dialysis [28]. In a comparative study of control patients with patients with a mean creatinine of 2 mg% and diabetics with a mean creatinine of 2.2 mg%, the mean survival time of control patients was 3.6 years, for patients with renal failure, it dropped to 2.7 years, and diabetics with elevated creatinine lived an average of 1.25 years after PTCA [29].

Very often, especially in patients with renal failure, coronary morphology is so severely impaired that only surgical interventions are feasible. Cardiac surgery often impairs renal function to varying degrees, especially when varying degrees of renal impairment are present preoperatively. Weerasinghe *et al*., examined patients with mildly impaired renal function preoperatively undergoing coronary artery bypass grafting (CABG) [30]. Even in-hospital mortality differed significantly within the groups. The group with a preoperative creatinine between 1.5 mg% and 1.7 mg% had a three-fold higher in-hospital mortality ($p = 0.045$) than the group with a creatinine below 1.4 mg%. If creatinine was greater than 1.7 mg%, mortality was increased sevenfold compared to a creatinine less than 1.4 mg% ($p < 0.001$). Other factors associated with higher mortality were a left ventricular ejection fraction less than 30%, longer cardiopulmonary bypass time, age greater than 70 years, and female gender.

Dialysis patients represent a high-risk group in terms of overall mortality. A recently published study presented the long-term outcomes of dialysis patients with coronary artery disease who were treated with PTCA, PTCA and stent implantation, and coronary artery bypass grafting [31]. After coronary artery bypass grafting, 56.4% of patients survived after two years and 37% after 42 months. After PTCA, 48.2% of patients were alive after two years and 28.6% after 42 months. The figures for PTCA with stent implantation were 48.4% and 29.4%. The comparison between CABG and PTCA regarding overall mortality (risk reduction [RR] = 0.80; $p < 0.0001$) and cardiac mortality (RR = 0.72; $p < 0.0001$) showed an advantage for CABG. Stent implantation also gave better results in terms of overall mortality (RR = 0.94; $p = 0.03$) and cardiac mortality (RR = 0.92; $p = 0.04$) compared with PTCA alone. The differences were even more pronounced in diabetics. Regarding overall mortality, the comparison of coronary artery bypass grafting with PTCA showed a risk reduction of 0.81 ($p < 0.0001$) and for cardiac mortality a risk reduction of 0.71 ($p < 0.0001$) in favor of surgery. In diabetics, there was no difference between PTCA and PTCA plus stent implantation in terms of overall and cardiac mortality.

In general, it has been shown, particularly in dialysis patients, that long-term outcomes are significantly better when using arterial grafts than vein grafts. The extent to which outcomes can be improved by the implantation of drug-eluting stents or the use of brachytherapy in favor of non-surgical procedures remains to be studied.

Patients with renal failure and coronary artery disease constitute a high-risk patient population. In addition to the above-mentioned problems with various types of interventions, hemorrhagic complications, infections, strokes, and further deterioration of renal function are also to be expected to a greater extent [32]. Postoperative intensive care measures are also required more frequently and prolong the length of hospital stay. Patients with renal insufficiency therefore represent a particular challenge for the treating physician.

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