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Kidney Transplantation's Effects on Valvular Heart Disease

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Abstract

Hypervolemia frequently results in elevated cardiac output in patients with end-stage renal failure, particularly in those who are underdialyzed and those who have cardiac decompensation. This investigation looked at how kidney transplantation affected valvular heart conditions. 180 adult patients (n=180) who underwent kidney transplantation at the Division of Organ Transplantation, University of Debrecen, Hungary, between February 2015 and June 2018 were included in this retrospective data study. This investigation looked at the echocardiographic parameters and lab findings both before and after surgery (at 6 and 12 months). The Kruskal-Wallis analysis of variance test and 2/Fisher exact tests were used to conduct statistical studies. It was deemed important if P .05.

Keywords: Aortic stenosis • Chronic kidney disease • End-stage kidney disease • Mitral annular calcification • Valvular heart disease

Introduction

One of the main risk factors for valvular heart disease is chronic kidney disease (CKD) (VHD). Patients with CKD frequently have mitral annulus and aortic valve calcifications, which frequently result in valvular stenosis and regurgitation as well as comorbidities such conduction system abnormalities and endocarditis. Patients with CKD who have VHD, particularly mitral regurgitation and aortic stenosis, have significantly lower survival rates. Given that CKD patients have a high frequency of concomitant illnesses, a heightened risk for periprocedural complications and death, knowledge about VHD in the general population may not always apply to CKD patients.T By outlining knowledge gaps, points of contention and research objectives, this Kidney Disease: Improving Global Outcomes (KDIGO) review of CKD and VHD aims to enhance understanding of the epidemiology, pathophysiology, diagnosis and management of VHD in CKD.

Description

In patients receiving hemodialysis (HD), peritoneal dialysis and renal transplantation, the prevalence of diagnosed VHD was reported to be 14%, 12% and 7.4%, respectively. Additionally, patients with VHD and ESKD had 2-year survival rates that were >30% poorer than those of people without these disorders. It's interesting to note that valve calcification detected by echocardiography in 35% to 40% of patients with ESKD was independently linked to poor cardiovascular outcomes, even in the absence of severe valvular dysfunction.

The most common valvular issue among ESKD patients is aortic stenosis. Patients with ESKD showed faster advancement of this issue and its prevalence was discovered to be between 6 and 13 percent. In most studies, ageing and the length of dialysis exposure are predictive of the prevalence of AV illness, with a connection with elevated calcium and phosphate levels rather than lipid problems. In the Action in Diabetes and Vascular disease: PreterAx and DiamicroN Controlled Evaluation (ADVANCE) trial, aortic calcification increased by 52% over the course of a year among HD patients with secondary hyperparathyroidism who had calcification at baseline and mitral calcification increased by 54% over the course of a year among HD patients with secondary hyperparathyroidism who had calcification at baseline. The annual incidence of aortic stenosis in 110 HD patients throughout a 7-year follow-up was 3%. The development of aortic stenosis was significantly predicted by older age, higher phosphate levels, calcium-phosphorus product and vitamin D levels.

Valvular stenosis and/or insufficiency secondary to CKD and ESKD are characterised by calcification of the interstitial cells of the valve leaflets (as well as the annulus and subvalvular apparatus of the mitral valve) on a pathological level. Numerous and intricate factors contribute to the calcification of valves in CKD. Although there are several components (hyperphosphatemia, calcium-phosphate product, parathyroid hormone and 2-microglobulin), it is still unclear how much each one contributes specifically and how they work together. These patients are likely predisposed to developing valvular calcification due to abnormal calcium and phosphate metabolism. Low vitamin D levels have also been linked to secondary hyperparathyroidism in older individuals with aortic stenosis, renal insufficiency and low vitamin D levels. Low vitamin D levels are linked to vascular calcification. Although epidemiologic data in humans have mostly focused on the association with arterial calcification, excessive vitamin D administration is also linked to valve calcification in animal models. Patients on warfarin who are undergoing HD are at risk for vertebral fractures and mortality and warfarin use is linked to calcification of the valves, peripheral vasculature and coronaries. Warfarin use is linked to higher mortality in ESKD patients, which may be as a result. The buildup of amyloid protein in calcific AVs may be a contributing cause. The material that is now accessible largely focuses on high-income areas where degenerative calcification predominates; information from areas where rheumatic heart disease is prevalent is relatively scarce. Furthermore, calcification and stenosis might advance more quickly in congenitally defective valves such a bicuspid AV. About a third of young (19- to 39-year-old) patients with childhood-onset renal disease who needed dialysis or a kidney transplant had valve and aortic calcification, which frequently coexists and can happen at a young age, frequently in conjunction with coronary artery calcification. The number of calcified valves was linked to both all-cause mortality and cardiovascular death in patients receiving long-term dialysis; 1-year all-cause mortality was 57% in people with both aortic and mitral valve calcification, 40% in people with either calcification and 15% in people with neither calcification [1-5].

Conclusion

Patients with CKD, particularly those with ESKD, have a higher prevalence of VHD than those in general. Additionally, when compared to the general population, people with CKD/ESKD experience a quicker progression of VHD.

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Valve calcification, along with the calcification of related structures, is the main pathogenic mechanism. Although the link between VHD and higher mortality is widely known, it is still unclear how to best manage the additional CV risk brought on by valvular heart disease in severe CKD and ESKD rather than coronary artery or cardiomyopathic heart disease.

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Conflict of Interest

The author shows no conflict of interest towards this manuscript.

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