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Following a Kidney Transplant, Pericardial Effusion

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Introduction

Following kidney transplantation, cardiac tamponade and pericardial effusion have been identified as two potentially significant side effects of using sirolimus for immunosuppression. Our research intends to examine the pericardial effusion caused by sirolimus's side effects. Recent research has shown cardiac tamponade and pericardial effusion as potentially significant side effects of sirolimus usage. The goal of our study is to examine the risk factors for the emergence of this condition [1].

Description

Between 2001 and 2014, patients who had kidney transplantation at our facility were examined and a connection between sirolimus exposure and pericardial effusion was found. Over the course of these 14 years, 19 out of 792 patients who had kidney transplantation experienced symptomatic pericardial effusion (determined by the need for pericardiocentesis or a pericardial window). Prior to transplantation, all patients underwent a cardiac evaluation that included an echocardiography but did not detect the existence of pericardial effusion. The majority of our cohort of patients are male (57.9%) and Caucasian (73.7%), which is typical of the transplant patients at our facility. When the transplant was performed, the mean age was 52.42 years. After a kidney transplant and while receiving sirolimus medication, symptomatic pericardial effusions appeared on average 5.06 years after the transplant [2]. Sirolimus levels were 5.19–7.47 ng/mL at the time of diagnosis. Following therapeutic intervention, including the stopping of sirolimus with or without pericardial drainage, no substantial pericardial effusion (leading in tamponade physiology) returned. The biggest single-center investigation on the potential link between sirolimus use and pericardial effusion in renal transplant patients has been conducted in this study. Clinicians must be alert for this possible cardiac hazard since sirolimus is commonly used in organ donation.

A 75-year-old man was hospitalised after experiencing 2 syncopal episodes and increasing dyspnea. Over the previous year, he had gained 6 kg in weight and had weakness and dyspnea. An echocardiography performed three months prior to admission revealed intact biventricular systolic function, normal aortic bioprosthesis hemodynamics (mean gradient, 7 mm Hg) and no constrictive physiology or pericardial effusion [3]. Right atrial and pulmonary capillary wedge pressures were both increased during a cardiac catheterization two weeks before to admission (22 mm Hg and 26 mm Hg, respectively). The right ventricular (RV) tracing showed an early-diastolic dip and plateau pattern and the RV end-diastolic pressure matched the wedge, right atrial (RA) and pulmonary artery diastolic pressures, indicating the possibility of pericardial constriction or limitation. At 39/23/29 mm Hg, pulmonary arterial pressures were just marginally increased. A coronary angiography revealed nonobstructive disease and a lower-than-expected cardiac index of 1.5 l/min/m^2 by thermodilution was found. An outpatient cardiac magnetic resonance scan was scheduled to check for pericardial constriction or infiltrative cardiomyopathy in light of the patient's symptoms and aberrant hemodynamics. Investigations into the lungs revealed consistent lung transplant performance [4].

Due to adhesions that connect the visceral and parietal pericardia, loculated pericardial collections are more common in individuals who have undergone prior instrumentation or trauma. The quantity of fluid or blood that can build anteriorly can be limited by postoperative adhesion of the right side of the heart and the anterior pericardium to the anterior chest. Given the post-procedure worsening, perforation cannot be ruled out even though the documented incidence of pericardial effusion and pericardiocentesis after combined left-sided and right-sided cardiac catheterization is extremely low (0.03% each). The results of the catheterization revealed aberrant hemodynamics that point to LV filling dysfunction. Constriction was a potential diagnosis due to the equalisation of end-diastolic pressures and the early diastolic dip and plateau pattern in the RV tracing; however, tamponade can also exhibit this equalisation of end-diastolic pressures. Even though localised effusions might present with unusual hemodynamics, the presence of fast early diastolic filling and the absence of pulsus paradoxus on the femoral artery tracing do not support the diagnosis of pre-existing tamponade. The symptoms and hemodynamic results might have been caused by an effusion that has been gradually worsening from the time of the heart surgery.

As seen in this instance, loculated pericardial collections can impair cardiac output by isolating and compressing distinct chambers differently. All the chambers would be compressed as a result of the circumferential pericardial effusion, resulting in RV and RA diastolic collapse as well as respirophasic alterations in mitral and tricuspid input velocities. The previously mentioned Doppler and 2-dimensional observations are frequently missing with localised pericardial effusion. There aren't many research that look at how localised LV pericardial effusion affects hemodynamics, but case studies and animal studies on the subject imply that LV diastolic collapse is a good indicator of LV tamponade. With no change in RA pressure or pulsus paradoxus, the diastolic collapse is frequently accompanied by a decrease in cardiac output and an increase in LV end-diastolic pressure.

Along with tamponade, our patient also displayed a blockage of the LV outflow tract and systolic anterior motion of the mitral valve as a result of distortion of the mitral apparatus. The hemopericardium made the LV cavity smaller and anterior mitral apparatus displacement made this patient more susceptible to extended contact between the interventricular septum and mitral valve. Our example indicates a mechanical mechanism of blockage, despite the fact that dynamic LV outflow tract obstruction has previously been reported as a physiological result of aortic dissection with pericardial effusion [5].

Conclusion

Pericardial effusion was common in our cohort of kidney transplant recipients and usage of sirolimus or sirolimus/tarcrolimus combination was linked to late-onset pericardial effusion. Further research is required to identify risk factors and raise awareness of pericardial disease in this group.

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Conflict of Interest

The author shows no conflict of interest towards this manuscript.

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