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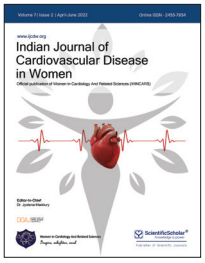

Cardiovascular *Case Report*

Cardiac Allograft Vasculopathy Evaluation by Optical Coherence Tomography

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ABSTRACT

Cardiac allograft vasculopathy (CAV) is a rapidly progressive form of atherosclerosis and it is the common cause of late allograft dysfunction and death in patients following orthotopic heart transplantation. After heart transplantation, patients lack adequate anginal mechanisms and may present with refractory heart failure and sudden cardiac death, so regular screening is required to detect CAV. CAV narrows the coronary arteries in diffuse concentric pattern, so detection by coronary angiogram is difficult. Intravascular imaging such as optical coherence tomography (OCT) and intravascular ultrasound is most sensitive diagnostic test for the detection of CAV. Once CAV is diagnosed, patients should be on statins, adequate immunosuppressive medications such as sirolimus, everolimus in focal lesions angioplasty, and stenting can be done, but in some patients with significant burden of CAV, retransplantation is the only available option. Once CAV is diagnosed, regular surveillance of heart function is mandatory. We are reporting a case of CAV evaluation by OCT.

Keywords: Atherosclerosis, Cardiac Allograft Vasculopathy, Optical Coherence Tomography (OCT)

INTRODUCTION

Cardiac allograft vasculopathy (CAV) narrows the coronary arteries in diffuse concentric pattern, unlike atherosclerosis which is focal and eccentric, so detection by coronary angiogram is difficult. Intravascular imaging such as optical coherence tomography (OCT)/intravascular ultrasound (IVUS) with fractional flow reserve plays a key role in detecting CAV.

CAV is known to occur in post-heart transplantation patients and it can lead to heart failure or occasionally sudden cardiac death. Hence, all post-cardiac transplant patients should undergo routine screening for detecting CAV.

During first 5 years post-transplant, annual coronary angiogram should be performed, along with OCT, as detecting CAV will be easy. If CAV is diagnosed, echocardiogram should be done to know left ventricular function and LV function should be monitored serially.

Patients should be on mammalian target of rapamycin (mTOR) inhibitors such as everolimus, sirolimus, and statins to prevent worse outcomes.

CASE REPORT

A 39-year-old male with history of diabetes mellitus, for 5 years on oral hypoglycemic agents, presented to emergency department with history of breathing difficulty for 2 months

and gradually progressed, and presently in NYHA class-4. He was admitted in ICCU, complete blood picture and renal function tests were normal, no significant ischemic changes in ECG. On further evaluation found to have dilated cardiomyopathy with severe lv dysfunction, LVEF-20%, mild mitral regurgitation and mild tricuspid regurgitation with Grade 3 diastolic dysfunction, IVC plethoric, and no pericardial effusion. After stabilization, he was subjected for coronary angiogram which revealed normal coronary artery. Right heart cath study showed PVR (pulmonary vascular resistance)-5.65 wood units, pa-81/36, (mean-46) mmHg. He was kept on guideline directed medical therapy such as diuretic agents, aldactone, carvedilol, dapagliflozin, atorvastatin, and other symptomatic medications.

HE was requiring, multiple admissions for heart failure. As he was remained symptomatic even on guideline directed medications, so listed for heart transplantation. Pre-transplant work up was done along with repeat right heart cath study-which showed-PCWP -13mmHg, pa-19/15/(mean-16), RV-15/4, PVR-0.92 wood units, SVR-23.92-Wood Units. Hence, PVR was decreased when compared to initial cath study and heart transplantation done on February 25, 2020 successfully.

Post-heart transplantation, he was stable and discharged on immunosuppressive medications along with other guideline directed management and he resumed his normal work. At end of 1 year, he was evaluated for CAV evaluation by OCT.

On routine evaluation, his complete blood picture and renal function tests were normal. ECG suggestive of normal sinus rhythm with the right bundle branch block [Figure 1]. Echo done found to have no LV RWMA, good LV and RV function, and Grade 1 diastolic dysfunction. He was subjected for coronary angiogram with OCT, which revealed normal coronary artery, [Figures 2 and 3] and OCT run from LAD to LMCA done, which showed distal LAD remodeling with increased mild intimal thickness, with no much significant narrowing of lumen, proximal LAD and LMCA diameter 3.6 mm and 5.4 mm, respectively [Figure 4]. OCT runs from LCx to LMCA done which showed normal coronary artery and proximal LCx diameter 3 mm. OCT runs from PDA to proximal RCA showed plaque erosion in proximal RCA, with no significant narrowing of RCA and PDA, and proximal RCA lumen diameter of 3.6 mm [Figure 5].

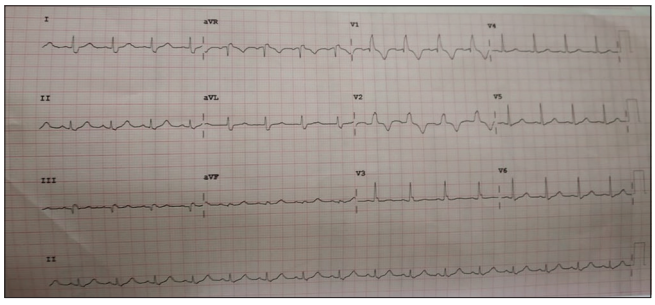

Figure 1: ECG-normal sinus rate, RBBB.

He was also subjected for endomyocardial biopsy from the right ventricle, which showed – single focus interstitial lymphocytic infiltrate, and no definite features of rejection seen. He was kept on tacrolimus, mycophenolate, wysolone, statin, antiplatelets, aldactone, metoprolol, dapagliflozin, insulin, and discharged in stable condition.

DISCUSSION

CAV is one of the long-term complications in post-heart transplant patients. The prevalence of CAV in heart transplant recipients at 1, 5, and 10 years was 8, 29, and 47%, respectively.[1] It is the chronic fibroproliferative rejection to the transplanted heart, laboratory investigations

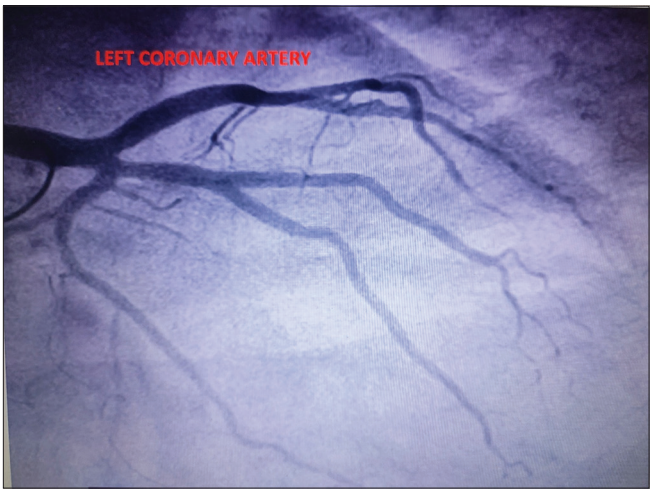

Figure 2: Coronary angiogram-left cranial artery, AP caudal view-normal coronaries.

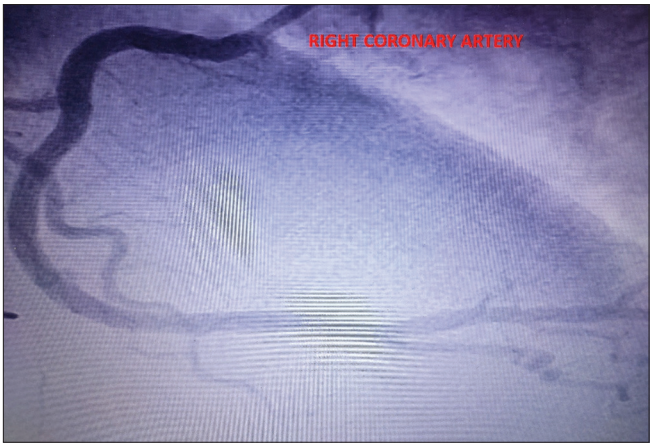

Figure 3: Coronary angiogram-right coronary artery, AP cranial view-normal coronaries.

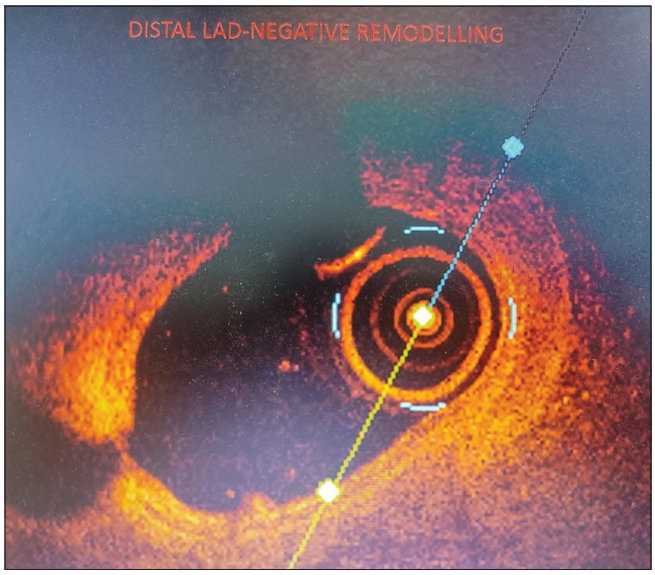

Figure 4: Optical coherence tomography-left coronary artery-distal lad shows negative remodelling (increased intimal thickness).

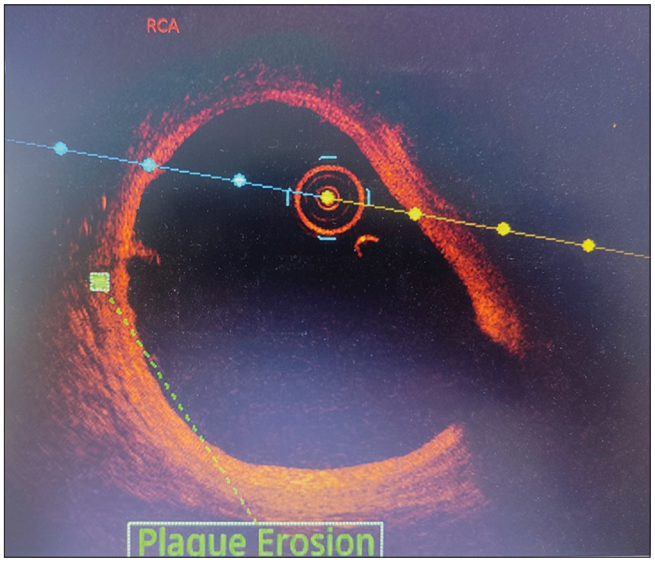

Figure 5: Optical coherence tomography-right coronary artery-shows plaque erosion in proximal RCA.

such as elevated CRP levels help in diagnosing CAV and allograft dysfunction.[2] It is pan arterial disease and limited to allograft. Factors contributing in development of CAV include diabetes, hypertension, hypercholesterolemia, and Cytomegalovirus infection.

CAV causes diffuse and concentric narrowing of coronary artery and also extends to microvasculature. Immunohistochemistry of affected vessels shows infiltration of macrophages and T lymphocytes into intima, media, and adventitia. Unlike atherosclerosis, calcium deposition is not prominent finding even in severely stenosed artery.[3]

As transplanted heart is denervated, patients usually not complain of chest pain, and they may present with nonspecific complaints such as fatigue, dyspnea, nausea, and sometimes may be asymptomatic.[4] Graft dysfunction can present as acute or chronic heart failure or sudden cardiac death if arrhythmia occurs. In such cases, it is vital to differentiate CAV from rejection reaction. Endomyocardial biopsy helps in diagnosing rejection reactions. In post-transplant patients, once CAV is diagnosed and if presents with severe LV dysfunction, prognosis is usually poor. Hence, heart transplant recipients should be monitored closely for detecting CAV early because treatment can be initiated at earliest to prevent significant LV dysfunction.

In CAV, coronary arteries are narrowed in diffuse and concentric pattern unlike atherosclerosis which is focal and eccentric, so coronary angiogram can easily detect atherosclerotic lesions unlike CAV lesions. Hence, intravascular imaging such as IVUS and OCT with FFR helps in detecting CAV lesions.[5,6] OCT helps us in knowing plaque morphology and lesion length.[6]

When CAV is diagnosed immunosuppressive therapy with mTOR, inhibitors should be augmented to halt progression of CAV.[7] In addition, the risks of increased immunosuppression, including infection and malignancy, may negate these supposed benefits. In transplant recipients, statins are shown to decrease CAV.[8] If it is limited to few coronary segments, percutaneous angioplasty can be performed, but restenosis is a long-term complication. In such restenosis cases, directional coronary atherectomy plays a role.[9] In selected group of cases, coronary artery bypass surgery has been done with good medium-term outcomes. For patients with significant burden of CAV,[10] re-transplantation is the only definitive treatment option.

All post-cardiac transplant patients should undergo routine screening for detecting CAV and during first 5 years of post-transplant, annual coronary angiogram should be performed, if renal function is normal.

CONCLUSION

CAV causes diffuse luminal narrowing of coronary arteries so detection by coronary angiogram may be difficult, so intravascular imaging such as OCT plays a key role in detecting CAV.

CAV can cause heart failure, so annual screening for detecting CAV should be done in post-transplant patients.

Patients should be on immunosuppressants and statins to halt progression of CAV and LV and RV function should be monitored regularly.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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