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Research Article

TAVR in a Bicuspid Aortic Patient with Stage 4 Cardiac Damage Severe Aortic Stenosis and Ascending Aortic Aneurysm

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Abstract

Aortic stenosis is a common pathology with congenital etiology-bicuspid or due to age-related calcific degenerative. The disease is divided into 4 stages according to its clinical and morphological signs. Although compensatory LV hypertrophy occurs at the initial stage, in the late period, extracellular volume increases, then apoptosis in cardiomyocytes as a result of widespread fibrosis, thinning of LV wall thickness and cavity dilatation occur. This already means irreversible and macroscopic myocardial damage and a serious decompensation stage of the process. In this final stage, biventricular heart failure, serious arrhythmias occur and the risk of the disease in terms of mortality increases significantly.

Keywords: TAVR, severe aortic stenosis, bicuspid aort, miokardial damage, ascending aortic aneurysm, right heart failure

Introduction

A 65-year-old male patient applied to our clinic with complaints of severe shortness of breath,chest pain , fainting and weakness. In the anamnesis, HT, DM, CKD and ischemic heart disease were reported [1]. The patient had previously undergone PCI of the LAD artery. A cardiosurgery consultation was performed, and invasive intervention was advised due to the high risk of the patient being severely symptomatic and serious comorbidities. The patient was evaluated for TAVI operation due to severe aortic stenosis (Euroscore 18.4%) [2,3].

Physical examination

Midsystolic was heard over the aorta and carotid arteries. Wet rales were heard in the middle and lower parts of the lungs. Jugular venous distention, + peripheral edema are observed.

AT 141/80mmHg, pulse-82 bpm, THS -22, SpO2-90%

EKG: Sinus rhythm, left bundle branch block with grade I AV block

Echocardiography: EF 20-25% МЧ-2-3, АЧ2-3 ТЧ -2, SPAP -93 mmHg Biventricular dilatation, remodeling, PA dilatation, IVC plethora

X-ray: decrease in pneumatization in the middle and lower parts on both sides

İnitial Diagnosis

Severe aortic stenosis. Heartfailure NHYA class III-IV. Pulmonary Hypertension typ II. İschemic Heart disease, Hypertension class II. Diabetes typ II II ip.

Clinical course

The patient was hospitalized. The patient was treated for heart failure. Before the operation, a CT scan was performed and the coronary vessels were checked. Prepared for TAVI operation.

Analyzes were evaluated.

HGB-13.6, HCT -39.3, WBC-7.72, RBC -4.53, PLT-212

Kreatinin 1.29mg/dl, K- 4.37, ALT-12.7, AST -17, İNR-1.04

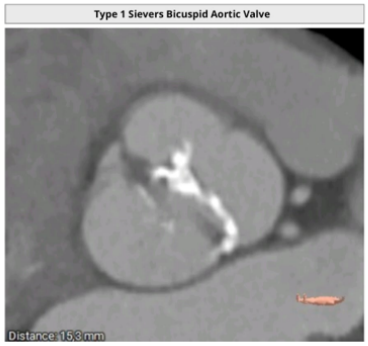
Furosemid infusion, Sprinolakton 50 mg x1, Clexane 0.6mlx2, Sacubitril/Valsartan 24/26 mg x2, Empagliflozin 10mg x1, Rozuvastatin 40 mgx1

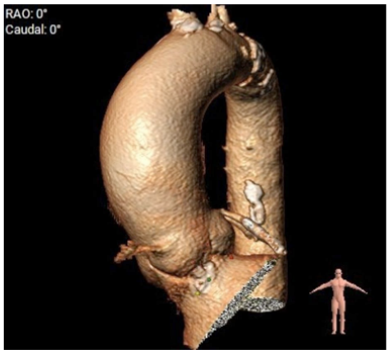
TAVİ CT: RV -46mm, LV -60mm, RA -57mm, LA-43mm

LAD and Cx originate from the left sinus through a separate coronary ostium. SVS diameter is 38x44mm area 13.2cm2, stj 45x48mm, area-15.9cm2, proximal ascending aorta is 49mm. Aortic root and ascending aortic aneurysm are noted.

Pulmonary conus was measured to be 29mm. MPA and its branches were monitored for dilatation.

-52mm of pleural fluid is observed in the left hemothorax.

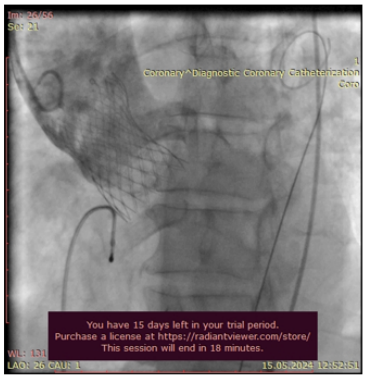


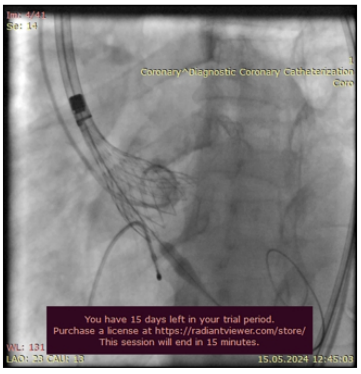


Coronary Angiography: RCA-mid 40%, Cx-N, LAD-mid stent was followed open. Medical treatment decision was made.

14.05.2024 Creatinine -1.18mg/dl

TAVİ - A sheath was placed in the right femoral artery, left femoral artery and vein. A temporary pacemaker was implanted. A 34mm Medtronic valve was implanted. A TAVI operation was performed successfully. EXOCQ was performed in the perioperative period. Valve function was evaluated as normal





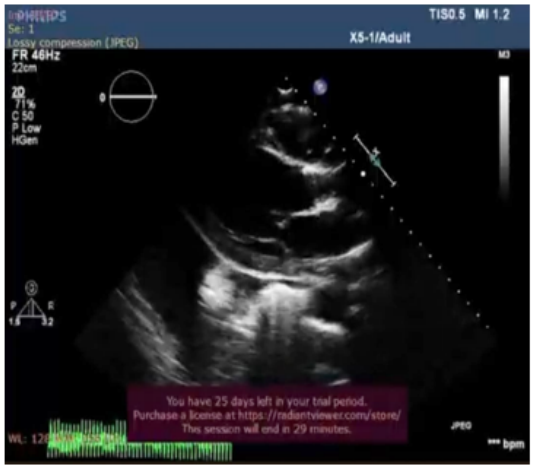
The patient was admitted to the intensive care unit. During the patient's rehabilitation period, control analyzes were reviewed.

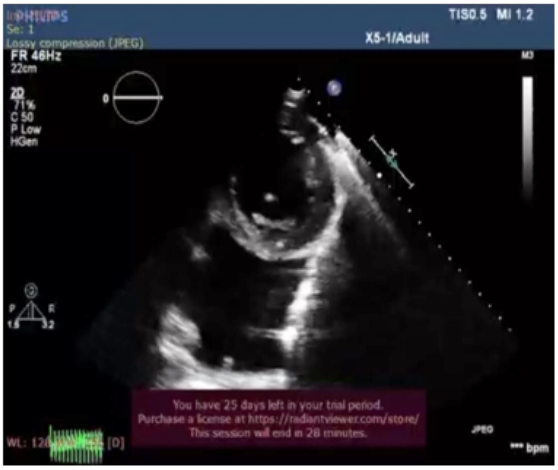
HGB-13.4, HCT -38.9, ÜBC-14.56, RBC -4.55, PLT-148

Creatinine-1.5mg/dl,

EXOKQ examination was performed. Valve function was observed to be normal and PVL was noted to be minimal. Pleural fluid was observed in the field of view.

Consultation of a nephrologist and pulmonologist was requested. Outpatient treatment was prescribed.





20.05.2024 Control Echocardiography shows 12 cm of fluid in the pleural space. Pleural puncture was performed on the patient by a pulmonologist. 1000ml of pleural serous fluid was removed, diuretic treatment was continued (treatment was corrected based on GFR-30 value, renal protective therapy was selected). Furosemide 40 mg x3, Torasemid 10 mg x1, Clopidogrel 75mg x1, Aspirin 100mg x1, Rosuvastatin 40mg x1, Empagliflozin 10mg. x1

10.06.2024. In the control examination, X-ray and echo examination? potassium and creatinine analysis were controlled. The patient's medical treatment was corrected and the follow-up continued.

20.06.2024 The patient's kidney tests, Echocardiography, X-ray examination were checked. NHYA I-II f.s. In the Echocardiography examination - LVEF 28%, MI-2, AI-min1, TI 1-2, sPAP -50mmHg, fluid is observed in the left pleura in the LV apical projection.

In the X-ray examination, a low amount of fluid is observed. The patient is clinically relieved and follow-up is ongoing.

Discussion and Learning points

1. Bicuspid aortic morphology in the patient - type 1 (lateral-medial with raphe) is consistent with eccentric opening and asymmetric coaptation defect. Mixed valvular pathology is observed with both severe stenosis and moderate-severe insufficiency due to coaptation defect. Aortic root is asymmetric - Anterior-posterior measured 44.5mm, Lateral-medial measured 47mm.Bicuspid leaflets are subjected to serious calcific degeneration in the mid and posterior portions at the hinge points, serious systolic-diastolic restriction is observed in this area of the leaflets. As a result of eccentricity of the inflow falling on the proximal segment of the aorta, laminar flow is disrupted and asymmetric pressure drop on the aortic wall in the inflow leads to the progression of aortic aneurysm. In this case, pressure-related dilatation is observed in a certain segment, unlike aortopathy.

2. İs LV remodeling secondary to LBBB and DCMP? or because of the last stage of aortic stenosis, the walls were thinned and the ventricle dilated?

- In remodeling with LBBB-DKMP etiology, serious damage is mainly to the mid and apical segments and asynchric movement in the septum "Septo-lateral delay" is observed. In this patient, "Apical sparing" is a typical injury symptom of forward aortic stenosis

3. When the patient is surgically suitable for Bentall's operation, valve implatation and aortic root replacement, in terms of the duration and technical difficulty of the operation, the lack of ventricular reserve increases the mortality risk of the operation many times (inoperable). Along with severe left ventricular failure, right heart failure and pulmonary hypertension, the symptomatic condition of the patient is very difficult to manage with either surgical or medical treatment. Invasive intervention is the most effective method for optimal results.

Conclusion

Compensatory management with optimal multidisciplinary treatment is the primary option in the subsequent medical follow-up of the patient. A decision was made to monitor aortic dilatation with annual CT scan and plan TEVAR in case of risk.

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