TAVR as treatment of choice in severe aortic regurgitation with multivalve pathology and chronic kidney disease patient

Abstract: The combination of aortic and mitral regurgitation is a typical example of a frequent yet understudied multiple valve disease scenario. Transcatheter aortic valve replacement (TAVR) can be an effective option for high-risk Aortic Regurgitation (AR) patients. Potential risks for transcatheter aortic valve are associated with malpositioning due to inadequate sealing, valve embolization and significant residual paravalvular regurgitation. Patients with AR are usually referred for intervention when the disease is at an advanced stage, with irreversible reduction of left ventricular function and severe pulmonary hyper-tension. İn this case report patient has multivalve pathology and also suffers from CKD. AVR surgery was recommended to the patient, but taking into account comorbid status and high surgery risk (EUROSCORE – 11.17%), TAVR was decided as a result of the heart team consultation.

Key words: aortic regurgitation, TAVR, chronic kidney disease, multivalve pathology

Clinical Case: A 65 year old woman admitted to the hospital with worsening shortness of breath, weakness, dizziness. She had a long-standing history of hypertension and chronic kidney disease due to polycystic kidney disease (she has been receiving dialysis 3 times a week for 6 years).

Physical examination: There were single bibasilar crackles on lung auscultataion.

Cardiac: A crescendo-decrescendo mid systolic murmur was heard along the right 2nd intercostal space and radiated to the neck.

Vital signs: BP- 140/80 mmHg, HR 88 bpm, body temperature -36.6 C^{o}, RR -18 bpm, SpO2-98%

ECG: sinus rhytm, HR 76 bpm, in I,AVL,V4-V6 leads minimal ST depression.

Pre-op Echocardiography: LVEF-40%, LV spheric remodeled, biatrial dilation and elongation (LA 58x70, RA 39x59), AS gradient 36/22mmHg, severe aortic insufficiency (AR-severe, PHT -196 msec, jet/LVOT-0.9, a holodiastolic reverse flow in descending aorta was noted. Severe mitral insufficiency (MR-III). Moderate

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tricuspid insufficiency (TR-II). RV basal 42 mm, RV function (Tapse-27 mm, rvsm-16 sm/sec) is normal.

MPA is dilated (35 mm), SPAP 50 mmHg. İVC is dilated (22 mm) and its collapse is reduced.

TEE: LVEF 40%, LV hypertrophy, global hypokinesis, longitudinal muscles contractility better than radial muscles.A decrease in GLS is noted due to eccentric severe aortic insufficiency in the posterior and inferior segments.Aortic valve leaflets are retracted, RCC is calcific, severe aortic regurgitation due to coaptation defect. Valve opening is limited and asymmetric, JET/LVOT- 0.9, vk-1.4 sm. Pulmonary artery is dilated and SPAP is increased. İAS is intact, there is a spontan left-to-right flow-PFO.

Picture1.A) PLAX aortic 2D view B)Severe aortic insufficiency

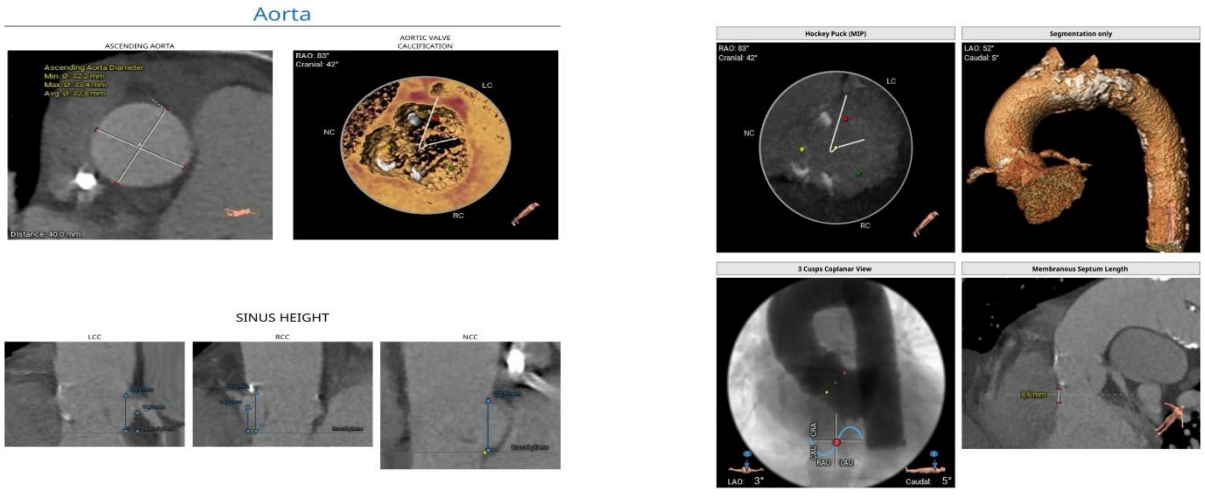
İnitial Diagnosis: Severe aortic insufficiency. Moderete Aortic stenosis. Heart failure NHYA class III. Pulmonary Hypertension type II. Hypertension class II. CKD

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.

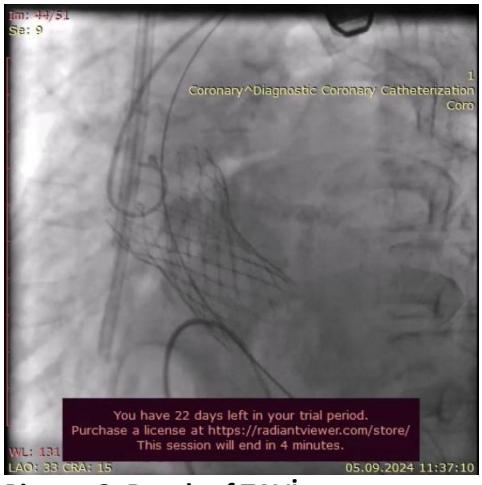
TAVİ CT: RV basal-31 mm, LV basal-69 mm, RA-35 mm, LA-59 mm.

Anulus diameter-30x23 mm, area-5.8 sm2.

Sinus Valsalva 32x32mm, area-8.5 sm2 Sinotubular junction 32x33 mm, area-9.4 sm2.



Picture 2. A)CT coronary height and ascending aorta, calcium B) Angle, Septal length



Picture 3. Result of TAVİ

CT angioqraphy: A long-segment eccentric calcific plaque causing 50-60% stenosis of the orifice in a 25-mm segment in the LAD-mid. Calcific plaque causing moderate orifice stenosis in a 13-mm segment of the LAD Diagonal branch. Cx-open, RCA-dominant, open.

Pre-op Analysis: WBC 6.56 10^3 u/L, HGB 12.5 q/dl, HCT 37.5 %, PLT 243 10^3/uL, NEUT 4.32 10^3/uL, LYMPH 1.42 10^3/uL, AST 35.8 U/L, ALT 34.2 U/L, Serum Creatinine 4.31 mg/dl, CRP 4.72 mg/l, Potassium 4.76 MMOL/L, PT- 10.6 san, İNR 1.01.

Treatment: Sefamezin 1.0 mg x2, Finerone 10mg x1, Clopidogrel 75 mg x1, Atorvastatine 40 mg x1, Clexane 0,4 mg x1, Lasix 20 mg x1, Pantap 40 mg x1 was started.

TAVİ: Femoral sheaths were placed into right femoral artery, left femoral artery and vein. A temporary pacemaker was implanted via left femoral vein. A pigtail catheter was placed in the noncoronary cusp, and a stiff Confida wire was placed inside the left ventricle. The 34 mm Medtronic Evolute PRO+ Valve was carefully advanced until the aortic annulus, positioned and deployed under rapid ventricular pacing. After deployment, rapid ventricular pacing stimulation was kept. Any hemodynamically significant leakage and gradient were not observed in valve.

During intraop TEE, no residual AR was observed and decrease in mitral regurgitation severity was noted immediately after procedure. The patient was admitted to the intensive care unit. Dialysis was performed, 1200 ml of fluid was withdrawn during dialysis.During the patient's rehabilitation period, control analyzes were reviewed-06.09.2024. Hgb-10.8 q/dl, HCT -32.8, Serum Creatinine- 6.20 mg/dl, CRP- 16.82 mg/l.

Post-op ECG: sinus rhytm, HR 77 bpm, single atrial extrasystoles. On post-op ECG single atrial extrasystoles are seen, and given the patient's complaints of palpitations, Concor 2.5 mg x1 was added. Medical treatment was corrected. **Post-op Echocardiography:** LVEF- 45%, MR-II, TR-I-II, SPAP- 30 mmHg. During follow-up, the patient had considerable clinical improvement, with better quality of life and NHYA II.

Discussion points:

- **CKD and HF.** CKD is generally assosiasted with an increase risk of mortality, cardiovascular events and readmission for heart failure; this supports the concept of a cardio-renal syndrome (CRS). Our patient has end-stage renal disease with GFR 11 mL/min and dialysis require but with minimal daily free diuresis along with CRS type II clinic. CRS type II (chronic cardio-renal syndrome) portray venous congestion along with considerable reductions in cardiac output as a result of cardiac dysfunction, leading to a reduction in the glomerular filtration rate (GFR). Studies have additionally disclosed that angiotensin II has a direct effect on renal tubular cells and cardiomyocytes and is also known to promote fibrosis, cellular hypertrophy, and apoptosis.
- **AR and pulmonary hypertension.** In patients with aortic and/or mitral valve disease the presence of pulmonary hypertension (PH) indicates a decompensated state of the disease

with left ventricular and left atrial dysfunction and exhausted compensatory mechanism, i.e., a state of heart failure. In patients with ≥moderate AR, PHT is associated with a progressive risk of mortality, even at mildly elevated levels.

- **AR and multivalve pathology.** Chronic kidney disease (CKD) is a major risk factor for valvular heart disease (VHD). Varying degrees of MR may result from remodelling, shape distortion and impaired function of the left ventricle (LV) associated with severe AR, thereby restricting mitral valve motion. Both AR and MR increase left ventricular (LV) preload, whereas only AR has a significant effect on afterload, as a result of the impact of the increased total stroke volume ejected on systemic vascular resistance, and of the resulting systolic hypertension. Valvular heart calcification is common in patients with chronic kidney disease (CKD), especially in those receiving hemodialysis therapy, and it is associated with poor prognosis. Premature AVC and MAC is frequent in dialysis patients, which commonly lead to valvular stenosis and regurgitation and appears to be related to abnormal calcium and phosphate metabolism and to increased mechanical stress on the valve cusps. In a small but important group of patients on long-term dialysis, premature valve calcification is severe and produces aortic stenosis, or less frequently, mitral stenosis. VHD, especially mitral regurgitation and aortic stenosis, is associated with significantly reduced survival among CKD patients. Progressive sclerosis and calcification of the valves and valvular annuli are major components of the etiology. CKD is also associated with left ventricular remodelling and dysfunction, which might contribute to increase the risk of heart failure and death in patients with VHD. One concomitant valve lesion can modify the clinical effect of another. In our clinical case, with the resolution of aortic regurgitation by TAVİ, we see a decrease in the level of mitral regurgitation and a decrease in the patient's complaints.

Conclusion: TAVR is a promising option in AR. Our patient has Stage 3 (tricuspid valve or pulmonary artery vasculature damage) cardiac damage. This patient had a high surgical risk and poor prognosis if any intervention wouldn’t performed. In our clinical case, taking into account that the patient had end-stage renal disease, we decided that primary aortic valve insufficiency appeared, then other valvular pathologies appeared on the background of AR, and after AR was resolved by transaortic valve replacement, we observed an improvement in other valvular pathologies severity and the patient's clinical condition.