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# Tackling 'Suicidal Left Ventricle' Following Transcatheter Aortic Valve Implantation : A Case Report

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**Abstract**

*Suicide left ventricle (SLV) is a state of profound haemodynamic collapse provoked by dynamic left ventricular outflow tract obstruction after a sudden afterload reduction. 'Suicidal' left ventricle after transcatheter aortic valve implantation (TAVI) is a rare occurance, with increased morbidity and mortality. Anticipating this devastating complication with timely intervention can prevent poor clinical outcomes. Early warning signs with possible hemodynamic compromise should be promptly recognized and readdressed . Here, we present a case report of 'suicidal' LV post TAVI and discuss it's early recognition and treatment options.*

**Keywords:** 'Suicidal' left ventricle, Transcatheter aortic valve implantation, fixed obstruction pressure overload, hemodynamic compromise.

## Background

The concept of transcatheter insertion of heart valves as a treatment option for valvular heart disease has been around since 1960s (Davies, 1965). It was not until 2000 that the first implantation of a transcatheter pulmonic valve in a human being was realized (Bonhoeffer et al., 2002). Cribier et al. (2002) described the first percutaneous transcatheter implantation of an aortic valve prosthesis in a 57-year-old patient with calcific aortic stenosis. However, this procedure is not without its inherent risks. TAVI can result in several life-threatening complications such as coronary obstruction, ventricular rupture, vascular injury, stroke, and death. Acute decompensated heart failure may also occur after TAVI from various causes, including LV dysfunction (due to coronary obstruction, "stunning" via rapid pacing, or stress-induced cardiomyopathy), arrhythmias, aortic annular rupture, pericardial effusion, mitral valve insufficiency and "suicide ventricle".

After a transcatheter aortic valve implantation (TAVI), the new valve instantaneously alleviates the fixed obstruction. As a result, the LV can eject more stroke volume; however, this can result in LV chamber collapse and hemodynamic instability leading to "suicide left ventricle." Risk factors include patients with preserved/high ejection fraction, small LV end-diastolic diameters, small LV masses, and high interventricular septum-to-posterior-wall thickness ratios. Given the increasing frequency of patients undergoing TAVI, it is important to highlight this under-recognized problem.

Here we describe, a rare case of 'suicidal' LV that occurred post TAVI, and discuss its early recognition with special emphasis on its management.

## Case Presentation

A 83-year-old male, known case of hypertension and diabetes, presented with the complaints of dyspnea on exertion (NYHA class III) and orthopnea for the last 3 months. Electrocardiogram (ECG) showed sinus rhythm with a heart rate of 80 beats/min and a left ventricular (LV) hypertrophy. 2D-Echo showed severe Bicuspid Aortic valve stenosis (Peak/Mean gradients of 110/68 mm of Hg) with aortic valve area (AVA) of 0.54 $cm^2$, concentric LVH with small LV cavity (septal thickness of 16 mm) and normal LV ejection fraction of 60%.

Computed Tomography (CT) of the aorta showed calcification in annulus, ascending arch and descending aorta with mild tortuosity. The aortic valve was Bicuspid (type with severe calcification. The following diameters were measured on CT scan (Figuress:1a-d): annulus (internal diameter) min/max 24/28.6 mm, perimeter of 83.2 mm, LVOT diameter of 26.9 mm, mid-sinus 35.6 mm, sino-tubular junction 35.8 mm, distance to the left coronary ostium 15.7 mm and to the right coronary ostium 15.6 mm. Both common iliac (9 mm) and femoral artery (7.6 mm) diameters were adequate. CT-coronary angiography showed only minor plaques in LAD and RCA (Figures: 2a-d).

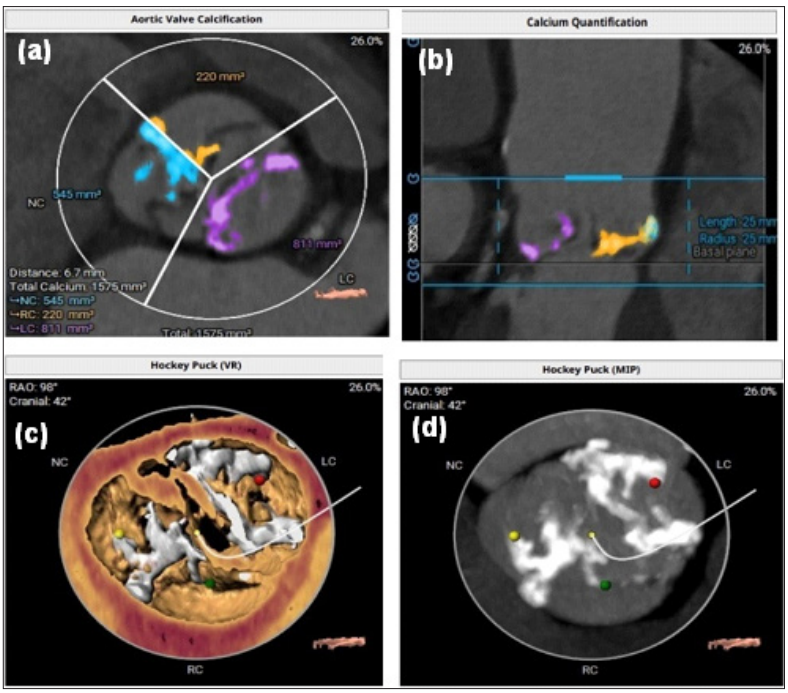


**Figure 1:** Computed Tomography (CT) aortogram (a,b) showing various dimensions of Bicuspid aortic valve with raphe , and CT-coronary aortogram (c,d) showing severe valvular calcification.

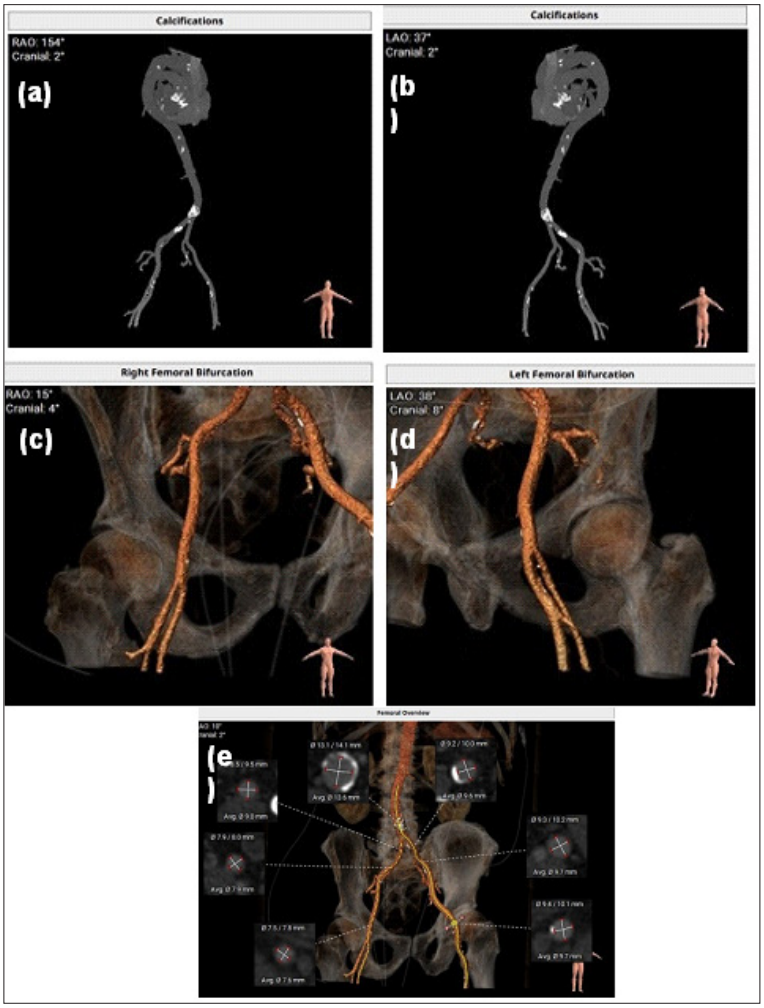


**Figure 2:** CT image showing (a-e) various dimensions of the peripheral arteries with calcification.

The patient's perioperative mortality rate was evaluated, and subsequently, he was classified into the intermediate- risk group with a Society of Thoracic Surgeons risk score of 6%. Surgical AVR and TAVI options were given to the patient in the presence of a multidisciplinary team comprising of cardiologists, cardiac surgeons, anesthesiologists, and the decision to perform TAVI was reached.

## Procedure

During TAVI procedure, a 14 French sheath was inserted and sutured into place and procedure commenced with predilating the native bicuspid aortic valve with 23 mm balloon. A 34 mm EVOLUT PRO + Bioprosthetic valve was then precisely deployed. The deployed valve was also post dilated with a 25 mm balloon to give excellent final results (P/M gradients of 8/5mm of Hg) with no paravalvar leak (PVL) (Figures:3a-e). However, soon after the valve deployment, patient had severe refractory hypotension. 2D-ECHO showed no evidence of pericardial effusion or significant PVL, but LV showed hypercontractility and collapse (commonly referred to as 'kissing' LV) with increased Dynamic Intraventricular Gradient (DIG) of 135 mm of Hg (Figure: 4). Suspecting 'Suicidal LV' which occurs due to sudden reduction in afterload on a heart that was accustomed to aortic Stenosis for so many years, large amounts of crystalloids (to increase Preload) was started. However, hypotension persisted and peripheral vaso constrictors like phenylephrine (1 mcg/kg/min) and vasopressin (0.01U/min) (to increase afterload) were added. After rapidly transfusing nearly 2 litres of IV fluids over next one hour and 5 litres over next 12 hours, LV cavity slowly recovered. Vaso constrictors were slowly withdrawn after 4 hours. He was discharged after 3 days with normal LV function and no significant LV gradients.

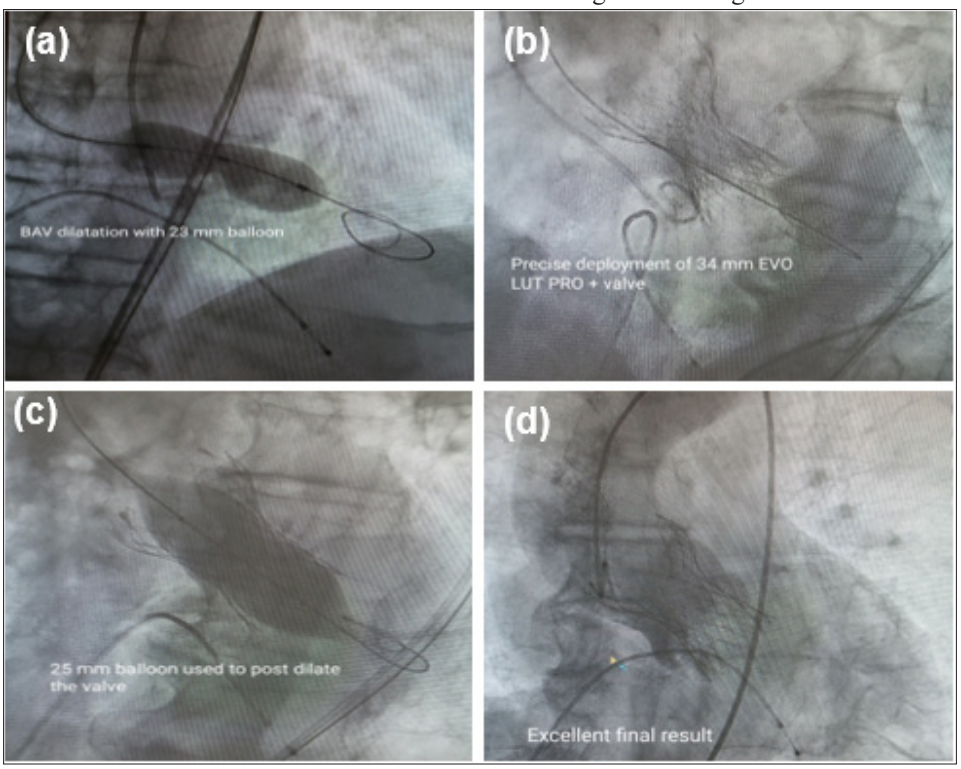


**Figure 3:** CAG showing: (a) Predilatation of the BAV with a 23 mm balloon, (b), Deployement of 34 mm EVOLUT PRO + Bioprosthetic valve, (c) Dilatation of deployed valve with a 25 mm balloon, and (d) Final image showing excellent deployment of the Bioprosthetic valve.

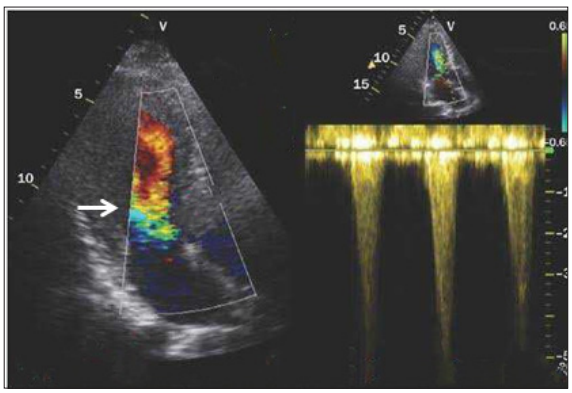

**Figure 4:** TTE image showing LVOT obstruction with LV collapse (arrow) and high Dynamic intraventricular gradient (DIG) of 135 mm of Hg.

## Discussion

Transcatheter aortic valve implantation is a widely used treatment modality for severe aortic stenosis. Though, complication rates of the procedure have reduced over time, owing to the improvements in procedural skills and development of devices, several rare but serious complications can still occur. Suicide left ventricle (SLV) is one such rare complication which can occur after surgical or transcatheter aortic valve replacement. The term SLV was first used due to its similarities with suicide right ventricle, seen after a pulmonic valvuloplasty (Suh et al., 2010). Patients at highest risk for SLV include those with normal to hyperdynamic LV function, asymmetrical septal or LV hypertrophy and small LV cavity. The mechanism behind SLV stems from increased LV unloading, leading to dynamic intraventricular gradients (DIG) in patients post TAVI who have severe LVH combined with a small LV cavity. The prevalence of high DIG ranges from 14% to 25% following SAVR (Bartunek et al., 1996). This is the physiology we often see with Systolic Anterior Motion (SAM) of the anterior mitral leaflet in hypertrophic cardiomyopathy (HCM).

SLV remains a underdiagnosed cause of haemodynamic compromise following a TAVI. Sudden after load reduction from the removal of the stenotic valve exposes the underlying hypercontractile left ventricle, resulting in excessive unloading of the LV cavity, leading to dynamic outflow tract obstruction and heart failure. The physiologic compensatory tachycardia paradoxically worsens the obstruction and subsequently leads to refractory hypotension. The management strategies for cardiogenic shock due to SLV have to be prompt. The cornerstone in management of SLV is volume loading, to expand the ventricular cavity and beta-blockers for their negative inotropic and chronotropic effect, leading to a increase in the diastolic filling time of the left ventricle. Vasoconstrictors such as phenylephrine (1-2 mcg/kg/min) or vasopressin (0.01-0.02 U/min) could also be added as they increase systemic vascular resistance, and prevent LV collapse post valve deployment.

Risk of SLV in patients with small LV cavity, asymmetrical septal or LV hypertrophy with normal ejection fraction, can be minimized by pre-hydration before TAVI with normal saline (1.0 to 1.5 ml/kg/hr) for at least 8 hours. This will increase the cardiac preload and prevent sudden LV collapse after the valve deployment.

Extra Corporeal Membrane Oxygenation (ECMO) can also be attempted as a last resort, in medically refractory cases of SLV. Surgical myectomy or alcohol septal ablation are reserved when all other measures fail, to remove the LV outflow obstruction. Avoidance of inotropes and diuretics is advised as they decrease the end-systolic volume and worsen the LVOT obstruction.

## Conclusions

Suicide left ventricle (SLV) in a post TAVI patient occurs as a result of chronic pressure overload from fixed obstruction that is acutely relieved after the valve deployment. Risk factors include hypertension with asymmetrical septal or LV hypertrophy and small LV cavity size. Though the treatment appears counterintuitive, early recognition and initiation with fluid resuscitation, Beta blockers with the addition of phenylephrine or vasopressin, may mitigate the dynamic obstruction and symptoms of cardiogenic shock. Pre-hydration before a TAVI procedure, with intravenous fluids in all at-risk patients can alleviate SLV.

## Conflict of Interest

The authors declare no conflict of interest.


## Acknowledgement

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