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Suicide Left Ventricle After Balloon Aortic Valvuloplasty

Abstract

Balloon aortic valvuloplasty (BAV) can be used as a bridge to definitive therapy– surgical (SAVR) or transcatheter aortic valve replacement (TAVR)–for treatment of aortic stenosis (AS) or as an option for symptomatic relief in patients who will not be a candidate for aortic valve replacement.

Keywords: Balloon aortic valvuloplasty; Aortic stenosis

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Introduction

To the best of our knowledge, this is the first case report describing Suicide Left Ventricle (SLV) during a BAV. In our case, the patient's demise was likely a result of dynamic LVOT obstruction that occurred after relief of fixed valvular stenosis. This phenomenon can lead to severe hemodynamic instability, often called "Suicide Left Ventricle" (SLV) [1].

Case Report

An 84-year-old male with a history of long-term nicotine abuse, hypertension, and chronic obstructive pulmonary disease presented with a 3-month history of worsening exertional dyspnea and chest pain. Examination revealed a mid-systolic ejection murmur suggestive of AS. EKG showed sinus rhythm and ST-depression in leads V4-V6 consistent with left ventricular hypertrophy (LVH). Troponins were elevated at 0.10 ng/mL. Transthoracic Echocardiogram displayed an Ejection Fraction (EF) of 60%, LVH (interventricular septum 1.4 cm; posterior wall 1.4 cm) with no systolic anterior motion (SAM) of mitral valve and severe AS (Aortic Valve area of 0.42 cm², mean gradient 45.8 mmHg, peak gradient 76 mmHg, and peak velocity 4.4 m/s).

Given the severity of his symptoms in light of his lung disease, the decision was made to do a combined heart catheterization with BAV **Figure 1 & 2**. After noting non-obstructive coronary artery disease, a BAV was performed which reduced the mean gradient from 77.74 to 21.24 mmHg and increased the aortic valve area to 1.56 cm² (**Figure 3A & 3B**). Post-BAV left ventriculogram showed mid-ventricular cavity obliteration in end-systole (**Figure 4A & 4B**). Immediately post-valvuloplasty, the patient developed hypotension and pulseless electrical activity (PEA). Resuscitation

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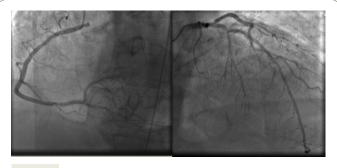


Figure 1 Non-obstructive Coronary Angiogram.

was initiated. Transoesophageal Echocardiogram revealed hyperdynamic left ventricular function with small cavity size and a trivial pericardial effusion that was apparent on pre-procedural imaging. An ascending aortogram showed no aortic dissection but showed mild Aortic Insufficiency. Epinephrine, atropine, and sodium bicarbonate were administered. A temporary pacemaker, intra-aortic balloon bump and peri cardiocentesis failed to

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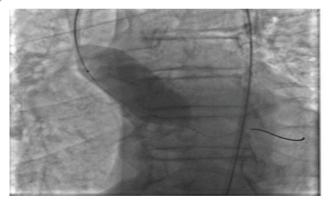
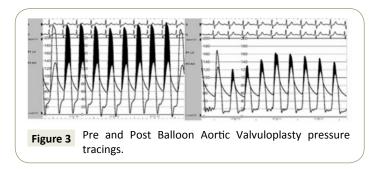
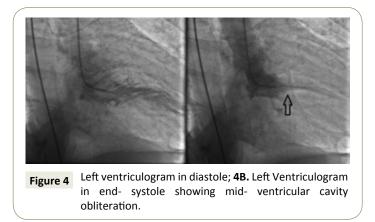


Figure 2 Balloon Aortic Valvuloplasty with Bard 26mm True Balloon.





improve the hemodynamic condition. Patient was declared dead after 20 minutes of resuscitative efforts.

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Discussion

Development of dynamic intraventricular gradient (DIG) after SAVR or TAVR is well-known [2]. Dynamic left ventricular outflow tract (LVOT) obstruction without SAM usually results from basal septal hypertrophy causing narrow LVOT. Sudden reduction in afterload post-valvuloplasty can lead to complete and forceful contraction of hypertrophied LV obstructing forward flow. In our case, the patient's demise was likely a result of dynamic LVOT obstruction that occurred after relief of fixed valvular stenosis. This phenomenon can lead to severe hemodynamic instability, often called "Suicide Left Ventricle" (SLV) [3].

Echocardiographic evidence of small LV end diastolic diameter, high EF, high valve gradients, asymmetrical hypertrophy of LV and small LV mass strongly predict the likelihood of developing DIG after a transcatheter valve position [2]. LVOT obstruction can be pronounced by profuse hypovolemia, administration of catecholamines (epinephrine, noradrenaline or dobutamine), administration of catecholamines (epinephrine, noradrenaline or dobutamine), and ventricular hyperkinesia [4].

All of these attributes were present in our patient contributing to the development and worsening of LVOT obstruction. Though administration of epinephrine potentially worsened the patients' LVOT obstruction, it was medically necessary due to PEA.

Development of SLV can lead to dire consequences necessitating immediate attention. Conservative treatments including intravenous fluids and beta- blockers have shown to be effective in non-emergent situations [3]. If unsuccessful, alcohol septal ablation could potentially relieve the hemodynamic collapse [5]. In emergent situations, extreme measures such as percutaneous left ventricular assist device placement or extracorporeal membrane oxygenation may be necessary to stabilize the patient.

Conclusion

The usage of BAV will continue to rise in order to relieve symptomatic severe AS in patients who are not immediate candidates for TAVR or SAVR. Developing this fatal complication warrants further research and careful consideration to minimize future morbidity and mortality.

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