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Clinical Case Report

Left ventricular outflow tract rupture during transcatheter aortic valve implantation: anatomic evidence of the vulnerable area☆,☆☆

Giuseppe Tarantini [1], Cristina Basso *,[1], Luca Nai Fovino, Chiara Fraccaro, Gaetano Thiene, Stefania Rizzo

Department of Cardiac, Thoracic and Vascular Sciences, University of Padua, Padua, Italy

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1. Introduction

Transcatheter aortic valve implantation (TAVI) has become the standard of care in inoperable or high-risk patients with aortic valve stenosis [1], and an ever-growing number of intermediate-risk patients are now being treated with TAVI worldwide [2]. The expansion of the use of TAVI to a younger, lower risk population mandates a reduction in procedural complications (i.e., stroke, acute coronary occlusion, aortic rupture, conduction disturbances with pacemaker implantations, paravalvular leak and vascular complication). Among these, rupture of the device landing zone, although rare (0.5–1.1%) [2], is an unpredictable occurrence associated with mortality as high as 50% [3]. Predisposing factors are the presence of severe and bulky calcifications of the left ventricular outflow tract (LVOT), excessive prosthesis area oversizing, aggressive balloon pre- and post-dilatation, and the use of a balloon-expandable rather than a self-expandable valve [4–7]. Herein we report a case of device landing zone rupture during TAVI together with a review of the literature of similar cases, aiming to find the pathophysiological connection between pre-procedural characteristics and postmortem findings.

2. Case report

An 82-year-old man with a history of severe chronic obstructive pulmonary disease and percutaneous coronary intervention on the left anterior descending and right coronary arteries was referred to our center for symptomatic severe aortic valve stenosis (mean aortic gradient 45 mmHg, aortic valve area 0.9 cm^2). Because of multiple comorbidities and high surgical risk (STS score morbidity/mortality 14.5%), he was scheduled for TAVI by the local Heart Team.

Pre-operative multi-detector computed tomography (MDCT) images are shown in Fig. 1. The aortic annulus maximal and minimal diameters were 30.9 and 22.4 mm, respectively; valve area was 5.46 cm^2; LVOT diameters were 22.2 and 18.3 mm, respectively. Accordingly, an Edwards Sapien 3 29 mm prosthesis was selected (area oversizing 21%). Notably, the aortic annulus was quite elliptic (eccentricity index of 28%) and a bulky calcification of the LVOT was detected just below the left-coronary cusp.

During TAVI procedure, when the prosthesis was fully inflated, aortic angiography showed a leak of contrast medium in the pericardial space (Online Videos 1 and 2). A small pericardial effusion with no sign of tamponade was detected at control transesophageal echocardiography. Contained intramural hematoma was suspected and the patient was sent to the intensive care unit. One hour later, he suddenly went into irreversible electromechanical dissociation, with transthoracic echocardiography showing large pericardial effusion.

At postmortem, a massive cardiac tamponade was found, together with massive hemorrhagic infiltration of the soft LVOT myocardium spreading into the subepicardial fat of the proximal left atrioventricular groove, the transverse pericardial sinus and surrounding areas, behind the pulmonary artery trunk and adjacent to the base of the left atrial appendage and the first segment of the circumflex coronary branch (Fig. 2).

After prosthesis removal, dystrophic calcification of the three native aortic valve cusps was evident, together with massive calcification of mitro-aortic fibrous continuity and of the right and left fibrous trigones, the latter in close continuity with the epicardial fat area. A perforation was visible at the nadir of the left coronary cusp, in correspondence of a calcific nodule, which was outward displaced during the TAVI procedure.

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* Corresponding author at: Cardiovascular Pathology, Department of Cardiac, Thoracic and Vascular Sciences, Via A. Gabelli, 61, 35121 Padova, Italy. Tel.: +39 0498272286; fax: +39 0498272285.
E-mail address: cristina.basso@unipd.it (C. Basso).

[1] The Authors contributed equally to the work.

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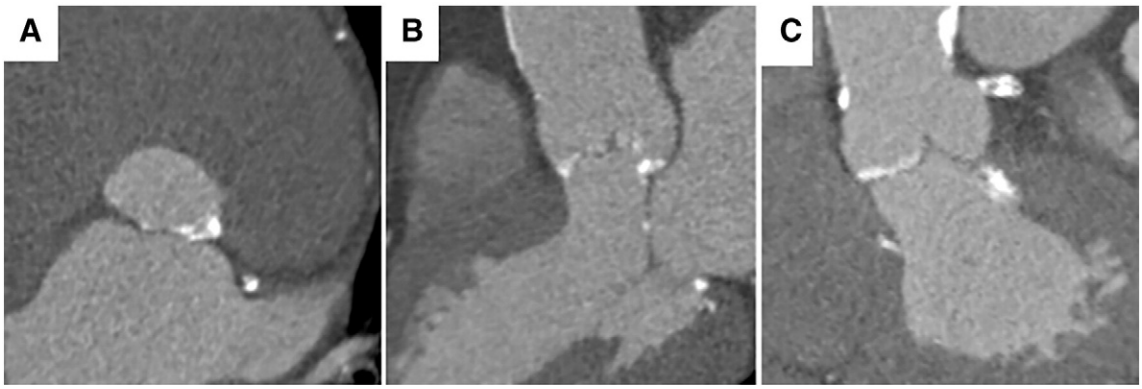

Fig. 1. Pre-operative MDCT double-oblique transverse (A), sagittal-oblique (B) and coronal-oblique (C) reconstructions. Notably, extensive LVOT calcifications are seen below the left-coronary aortic cusp. Aortic annulus measurements: max diameter 30.9 mm, min diameter 22.4 mm, eccentricity 28%, area-derived diameter 26.4 mm; valve area 5.46 cm^2; annular area oversizing 21%.

3. Discussion

Rupture of the "device landing zone" is a devastating complication of TAVI [5]. Under this umbrella, all procedure-related injuries that may occur in the region of the aortic root and the LVOT during balloon dilatation of the native aortic valve, prosthetic valve deployment and prosthesis post-dilatation for paravalvular leak, are reported. However, from an anatomical viewpoint the rupture should be better described as supra-annular, annular or sub-annular [8].

The term supra-annular rupture includes injuries of the sinus of Valsalva and the sinotubular junction, is rare and can evolve in aortic wall haematoma or localized dissection that might impair coronary flow [5].

The annular rupture involves the fibrous portion of the aortic annulus and is considered an underreported complication, since a small annular tear can be sealed by the prosthesis, with no impact on clinical outcome [4].

The sub-annular rupture is the most frequent and, as in our case, is located in the LVOT below the aortic valve cusp attachment [5]. The LVOT, a thin-wall structure in direct contact with the prosthesis, is composed of a fibrous and a muscular part. While the fibrous component is formed by the strong and distensible tissue of the mitro-aortic fibrous continuity, the muscular component includes a portion which is underneath the right coronary cusp and the leftward portion of the left coronary cusp. Under the right coronary cusp there is the "anatomically protected muscular LVOT", with the muscular interventricular septum externally overrided by the right ventricular outflow tract. On the other hand, the small segment of muscular LVOT located between the left/right commissure and left fibrous trigone is the only portion of "anatomically unprotected muscular LVOT", which has not external support and thus represents the most vulnerable region at risk of external rupture. Injury of the muscular LVOT is a dynamic process and can account for either acute rupture with massive bleeding leading to cardiac tamponade, as occurred in our case, or a pseudoaneurysm formation below the left coronary artery.

There are few reports in the literature depicting the exact location of the sub-annular rupture, mostly part of surgical series of TAVI procedures emergently converted to open-chest surgery (Table 1) [9–11].

In the series by Girdauskas et al., sub-annular calcification in the muscular LVOT between the left fibrous trigone and the left/right commissure was detected by MDCT in 83% (5/6 patients) of cases [9]. Following removal of the prosthesis, the typical LVOT injury in the sub-annular region between the left fibrous trigone and left/right commissure was found in all. All had unsuccessful surgical attempt and died intra-operatively due to deep laceration. Notably, these patients were mostly female with important asymmetric left ventricular hypertrophy. Unic et al. reported a case of successful surgical repair of a tear between the left coronary ostium and the commissure between the left and non-coronary cusp of the aortic valve [10]. Hayashida et al. suggested the presence of a vulnerable "epicardial fat area" after *ex-post* MDCT examination of two patients who suffered aortic rupture after TAVI [11]. Of note, no sub-annular rupture occurred in the absence of LVOT calcifications. It must be acknowledged that all patients in the published case series received a balloon-expandable prosthesis, but landing zone rupture after self-expanding device implantation has been also reported in the literature, although less frequently [12].

Our case is the first to provide the anatomical - ex vivo - proof of the correlation between LVOT calcification displacement and device landing zone rupture. In this regard, the comparison between pre-operative MDCT images and postmortem findings sheds new light on the pathophysiology of this complication. The patient's aortic valve had an elliptical shape and carried a bulky LVOT calcification under the left-coronary aortic cusp, as in most cases reported in the literature. Therefore, the vulnerable area of the LVOT appears to be corresponding to the non-coronary and left-coronary cusps attachment, an area which is in close contact with the epicardial fat of the transverse sinus, without intervening cardiac structures. When the calcified nodules are mechanically compressed and displaced by the fully expanded prosthesis, this area acts as *locus minoris resistentiae*. The calcification is impressed into the myocardium during balloon inflation causing an initial small endocardial and myocardial tear, which can be further expanded by the mechanical pressure of the blood during systole, if the prosthesis frame does not seal the rupture site. Noteworthy, the TAVI prosthesis selected for our patient was a balloon-expandable one, quite aggressively oversized, and the location of the calcification at MDCT corresponded exactly to the sub-annular rupture site found at postmortem.

When planning a TAVI procedure, the selection of the perfect valve size depends on balancing the risk of paravalvular leakage — which is correlated with worse long-term outcome [11] — and that of landing zone rupture. The case herein reported suggests that in the presence of bulky calcifications located in close proximity of the anatomically unprotected muscular LVOT (i.e., under the left-coronary cusp), a higher rate of paravalvular leakage appears to be acceptable [13] in order to minimize the risk of a catastrophic complication such as device landing zone rupture.