

## STATE-OF-THE-ART REVIEW

# Annular Rupture During Transcatheter Aortic Valve Replacement



## Classification, Pathophysiology, Diagnostics, Treatment Approaches, and Prevention

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### ABSTRACT

Annular rupture is an umbrella term covering different procedural-related injuries that may occur in the region of the aortic root and the left ventricular outflow tract during transcatheter aortic valve replacement. According to the anatomical location of the injury, there are 4 main types: supra-annular, intra-annular, subannular, and combined rupture. Annular rupture is a rare, unpredictable, and potentially fatal complication. It can be treated successfully if it is immediately recognized and adequately managed. The type of therapy depends on the location of the annular rupture and the nature of the clinical manifestations. Treatment approaches include conventional cardiac procedure, isolated pericardial drainage, and conservative therapy. This summary describes theoretical and practical considerations of the etiology, pathophysiology, classification, natural history, diagnostic and treatment strategies, and prevention approaches of annular rupture. (J Am Coll Cardiol Intv 2015;8:1-9) © 2015 by the American College of Cardiology Foundation.

**T**ranscatheter aortic valve replacement (TAVR) or, transcatheter aortic valve implantation (TAVI) is reserved for patients not amenable to conventional valve procedure due to inoperability or very high surgical risk. However, TAVR possesses its own procedural complications (1-5). The most feared is annular rupture, a rare and unpredictable occurrence. If it does occur, it can jeopardize a patient's life immediately (1-8). Little is known about this serious complication, which is mostly considered difficult to treat. This summary describes theoretical and practical considerations of the etiology, pathophysiology, classification, natural history, diagnostic strategies, and treatment approaches of annular rupture.

### INCIDENCE

Annular rupture occurs in about 1% of all TAVR procedures (2-5,7,8). It is unknown how frequently this

complication occurs but remains undetected. The real incidence of TAVR complications including annular rupture is suspected to be somewhat higher than reported (7,8).

### TERMINOLOGY AND DEFINITION

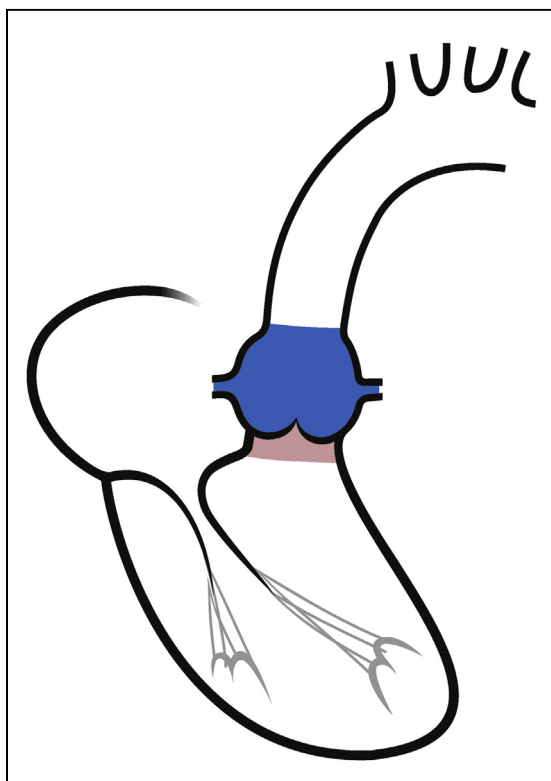
“Annular rupture” (or “annulus rupture”) is an umbrella term covering different procedural-related injuries that may occur in the region of the aortic root and the left ventricular outflow tract (LVOT) during transcatheter aortic valve replacement. The synonyms are “aortic root rupture” and “rupture of the device landing zone.” The latter is—from an anatomical point of view—more precise, as it includes all anatomical regions where possible rupture occurs (6,9) (Figure 1). According to the Merriam-Webster Dictionary, the term is also correctly written as “anular” using a single letter “n” (10).

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**ABBREVIATIONS  
AND ACRONYMS****LV** = left ventricle**LVOT** = left ventricular  
outflow tract**TAVR** = transcatheter aortic  
valve replacement**VSD** = ventricular septal defect**ANATOMY OF THE  
AORTIC VALVE ANNULUS**

The popularization of TAVR awakens an old myth about the aortic annulus (11). The annulus is neither circular nor oval, and is not even in a horizontal plane. Anatomically, it has a scalloped shape and looks like a crown. The annulus is a part of the fibrous skeleton of the heart and connects the left ventricle (LV) and the aortic root. It is a conjunctive area between the aortic valve on one side and the inter-ventricular septum, the anterior leaflet of the mitral valve, and the free myocardial wall of the LV on the other side. From a surgical viewpoint, the annulus is a fibrous structure of the insertion of the aortic valve leaflets on the aortic wall. “Annulus for TAVR”—measured by different imaging techniques—is a “virtual annulus,” a cross-sectional plane of the most distal part of the LVOT, at the level of the 3 nadirs of the aortic leaflets. This plane is subject to dynamic changes of geometry during a heart cycle.

**FIGURE 1** Simplified Schema of the “Device Landing Zone”

The aortic root (blue) and the left ventricular outflow tract (pink) form the “device landing zone.”

**PATHOPHYSIOLOGY OF ANNULAR RUPTURE**

Injury of the aortic root or LVOT may occur during balloon dilation of the native aortic valve, prosthetic valve deployment, or valve redilation for paravalvular leakage (1,4-7). Aortic rupture as a consequence of percutaneous balloon valvuloplasty for valvular aortic stenosis was already reported in the 1980s (12). Annular rupture is not associated with the access route chosen for implantation, but rather with the type of the implanted valve (1-6). It has been almost exclusively observed after the use of a balloon-expandable valve and only exceptionally after TAVR with self-expandable valves (1,2,5-8,13). In the latter group, it is mostly a result of overdistension of the prosthesis during reballoning to treat residual paravalvular regurgitation (4,6,8,14).

Aggressive oversizing ( $\geq 20\%$ ) of a transcatheter valve—a discrepancy between the size of the native annulus and the prosthesis—is thought to carry an increased risk for annular rupture (1,5-7,15). It occurs, however, only in the presence of 1 or more other factors, mainly calcification, which per se can cause rupture during TAVR (1,6,7,13). Rupture is, therefore, not necessarily related to discrepancy between the annular size and the new prosthetic valve (6,7,13,16). In an experimental model, no gross or microscopic damage was observed when balloon diameters were  $\leq 1.7$  times the aortic annulus diameter (17). However, it should be emphasized that overdistension of a healthy aortic annulus is a completely different process than tissue overdistension in patients with aortic valve stenosis and rather ubiquitous presence of calcification.

Anatomic characteristics that may predispose a patient to annular rupture are small aortic valve annulus ( $< 20$  mm), a narrow aortic root (with a small difference between the diameters of the annulus and aorta at the level of the sinuses of Valsalva), a large amount of calcification in the aortic valve leaflets (even in a large aortic root), calcification of the annulus (especially circular calcification), presence of calcium in the LVOT, calcification of the wall of the sinuses of Valsalva in the region adjacent to the annulus, heavily calcified bicuspid valve, short distance from a coronary artery to the annulus, severe asymmetric sub-aortic LV hypertrophy, and global LV hypertrophy in elderly, mostly female patients (with decreased LV compliance and fragile tissue) (1-8, 13-27).

Despite identification of possible factors for annular rupture, it is currently unknown—and therefore unpredictable—which specific combination of the factors will lead to rupture. Recent scientific

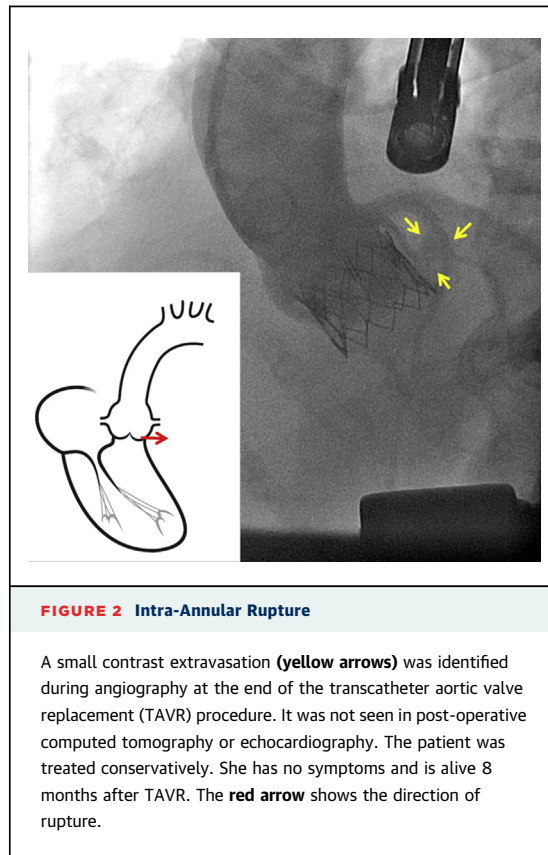
publications have attempted to identify real predictive features of this complication, specifying some potential factors such as a calcium score beyond the limit of safety, bicuspid heavily calcified valves, certain locations of calcium, or nodules over 4 to 5 mm (7,28). The cumulative analysis from 16 centers with a series of 31 consecutive patients who experienced annular rupture during TAVR with balloon-expandable prostheses revealed that patients with root rupture had a higher degree of LVOT calcification quantified by the Agatston score and a higher frequency of  $\geq 20\%$  annular area oversizing than comparable patients who did not experience rupture (7).

### CLASSIFICATION

According to the anatomical location of the injury, it can be classified into 4 types: intra-annular, subannular, supra-annular, and combined rupture (Table 1).

**INTRA-ANNULAR TYPE.** The native annulus is frequently calcified in patients with severe aortic valve stenosis. It can be speculated that small tears in the native annulus occur during TAVR, probably more often than is diagnosed. Theoretically, such annular lesions may be “sealed” by the transcatheter valve (4,6,23). A small, localized rupture may remain clinically nonrelevant. It is identified during TAVR as a slight contrast extravasation in the region that normally should not be perfused by contrast medium (2,6,15,20,22). It may not be detectable thereafter (Figure 2).

**SUBANNULAR TYPE.** Rupture is located below the native aortic valve annulus, in the LVOT. There are 3 main areas for subannular rupture: 1) the free myocardial wall of the LV; 2) the region below



**FIGURE 2** Intra-Annular Rupture

A small contrast extravasation (**yellow arrows**) was identified during angiography at the end of the transcatheter aortic valve replacement (TAVR) procedure. It was not seen in post-operative computed tomography or echocardiography. The patient was treated conservatively. She has no symptoms and is alive 8 months after TAVR. The **red arrow** shows the direction of rupture.

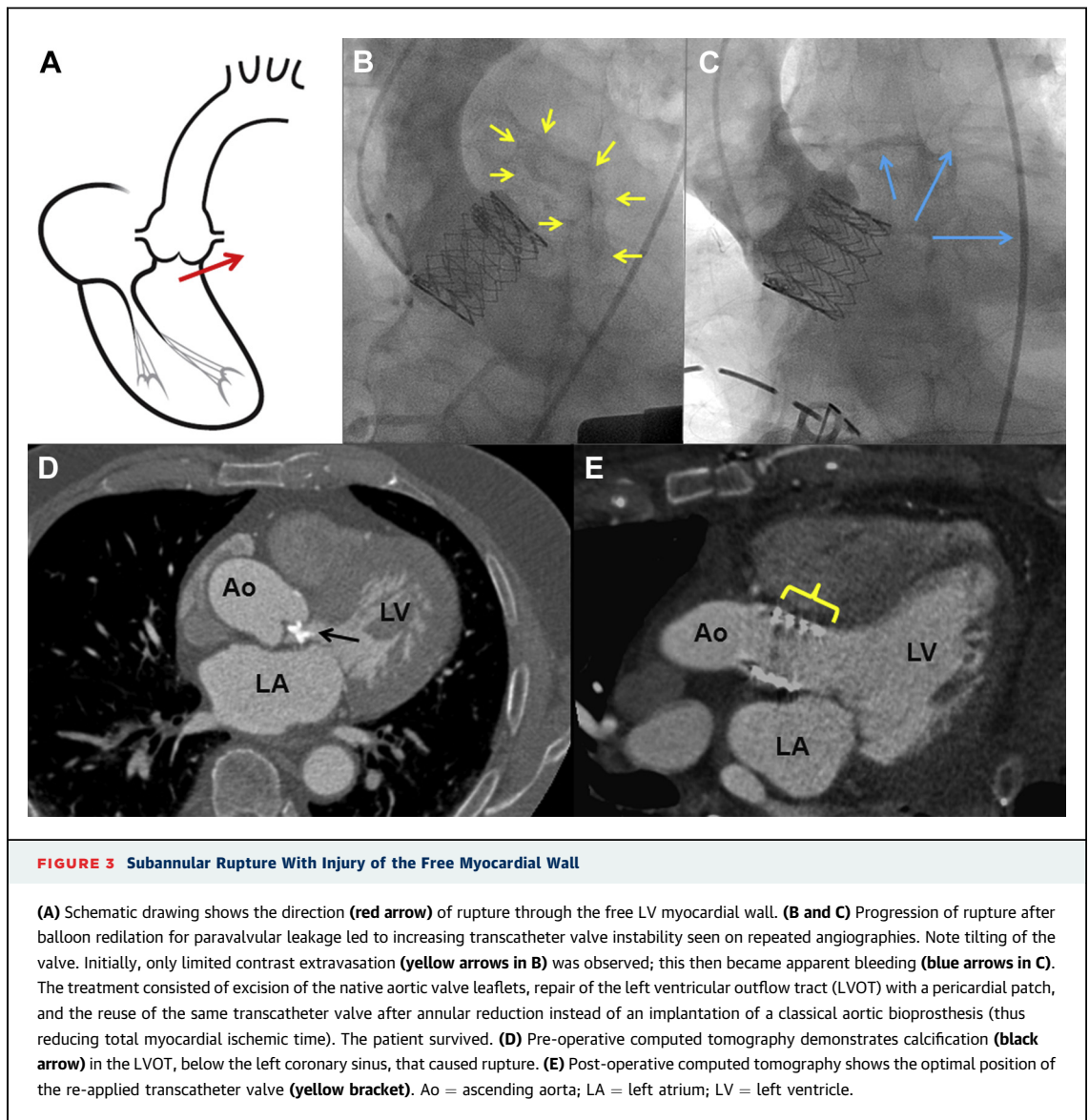
the noncoronary sinus of Valsalva (the fibrous conjunction between the sinus and the anterior mitral leaflet); and 3) the interventricular septum (1,5-7,14,19,20,22-24).

**Free myocardial wall.** Rupture of the free myocardial wall is always localized in the LVOT, below the left coronary sinus of Valsalva, and in particular, below that one-half of the sinus that is close to the commissure of the left and right aortic valve leaflets. (The LVOT below the other one-half of the left sinus is “protected” by the fibrous tissue of the attachment of the anterior mitral leaflet.) This is a dynamic process causing either acute rupture of the free myocardial wall of the LV with massive bleeding (1,4-7,19) (Figure 3) or a slowly growing pseudoaneurysm below the left coronary artery (15). The finding is similar to that shown in Figure 4. The consequences may be dynamic obstruction of the coronary flow or sudden death some weeks or months after TAVR.

Calcifications of the LVOT, especially those that protrude into the LVOT, may cause rupture as a sole initiating factor (1,6,7). The calcification is impressed into the myocardium during balloon inflation causing an endocardial and myocardial tear. Mechanical pressure of the blood during systole expands the

**TABLE 1** Classification of Annular Rupture According to the Anatomical Location

1. Intra-annular
2. Subannular
a. Injury of the free myocardial wall
b. Injury of the anterior mitral leaflet
c. Injury of the interventricular septum
3. Supra-annular
a. Injury of the wall of a sinus of Valsalva
b. Injury of the ostium of a coronary artery
c. Injury of the sinotubular junction
4. Combined
a. Intra-annular and supra-annular
b. Intra-annular and subannular
c. Intra-annular, supra-annular, and subannular

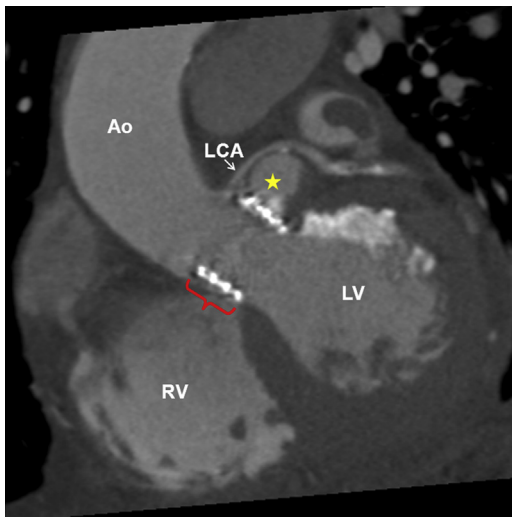


initial small tear, and the damage further propagates into the fragile posterolateral LV myocardium. Then, it penetrates through the whole thickness of the myocardium, toward the proximal parts of the left descending coronary artery and the circumflex artery, and is halted by the epicardium. At this stage, it is manifested as a localized subepicardial hematoma seen on the LV base, near the left appendage. Next, the hematoma may spread toward the LV apex by further subepicardial propagation of the pressurized blood. Finally, the epicardium ruptures and the bleeding becomes apparent (6,19) (Figure 3). Bleeding is usually seen at a place remote to the LV tear, frequently prompting a false and misleading initial

diagnosis, such as wrongly suspected iatrogenic injury of a coronary artery (6).

Rarely, rupture of the free muscular wall occurs even when there is no calcification in the LVOT. Overdistension of the LVOT during balloon inflation may cause a tear in the fragile myocardium, mostly in the hypertrophic heart with a small LV cavity and in older female patients (2,19).

**Below the noncoronary sinus of Valsalva.** Rupture in the region below the noncoronary sinus of Valsalva is caused by calcified deposits of the fibrous conjunction between the sinus and the anterior mitral leaflet, whereby the leaflet is mostly calcified and partially stiffened. The outward pressure on the



**FIGURE 4** Chronic Pseudoaneurysm

Routine post-operative computed tomography shows an incidental finding of a chronic pseudoaneurysm below the left coronary artery (yellow star) with dynamic obstruction of the coronary flow. The site of rupture is not clearly identifiable. During TAVR, the patient underwent redilation for paravalvular regurgitation and implantation of a second transcatheter valve for subsequent transvalvular insufficiency. Note "valve-in-valve" (red bracket). The patient had no symptoms and refused surgery. She died suddenly 6 months after TAVR. LCA = left coronary artery; RV = right ventricle; other abbreviations as in Figures 2 and 3.

deposits during balloon inflation might damage this region, resulting in an acute, frequently large shunt of blood from the LVOT into the left atrium (5,6) (Figure 5). Conduction disturbances are frequently combined (1).

**Interventricular septum.** Depending on the location of the calcification in the LVOT, the myocardial tear may be confined to the septum. The manifestations vary from conduction disturbances with atrioventricular block or hematoma in the interventricular septum, to fistula formation in the septum itself, or even to a ventricular septal defect (VSD) with left-to-right ventricular shunt (1,5,17). Calcifications squeezed into the membranous septum are a possible cause (23). The consequence is silent or apparent heart failure (1,2,14,22,23).

**SUPRA-ANNULAR TYPE.** Supra-annular rupture encompasses injuries of the wall of a sinus of Valsalva, damage of an ostium of a coronary artery, and/or injury of the sinotubular junction. Tear, rupture, or dissection is caused by balloon over-distension

during TAVR (1,4,6,7,13). When it occurs, it is usually in patients with narrow, calcified aortic root and/or sinotubular junction and with a cusp containing bulky and calcified material (1,6,7). The cusp is pressed toward the calcified aortic wall during valve deployment and causes damage (4,16,25,26). A tear of a calcified sinus is clinically presented as a hematoma in the aortic wall or localized dissection (7,13,20,26). It can result in apparent bleeding (Figure 6) (4,6,7,13) or chronic pseudoaneurysm (Figure 4). Rupture of the calcified left or right sinus of Valsalva is sometimes accompanied by injury or even destruction of the coronary artery ostium and acute disturbance of the coronary flow (6,25).

**COMBINED TYPE.** This form of rupture includes combined injuries of the native annulus and the supra-annular or subannular structures. Rupture of the native annulus may progress in a proximal or distal direction, especially when calcification extends from the annulus into the LVOT or the aortic wall. In the extreme case of extensive calcification, damage involves all levels of the device landing zone and is of a disastrous nature (1,5,6,24).

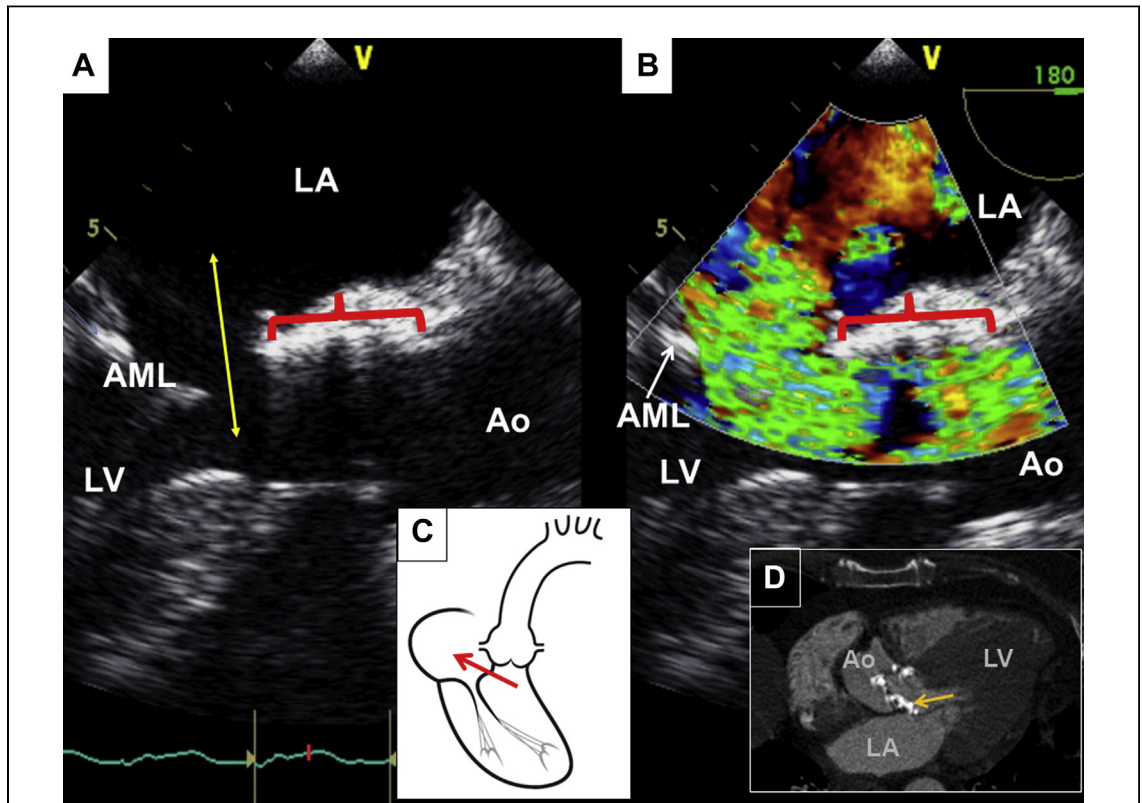
## PRESENTATION

Clinical symptoms and the time of presentation after rupture depend on the location and extent of the injury (1-7). Rupture may already be apparent during TAVR (1,2-7,13,25), or its clinical manifestations are delayed and not seen until some hours after the procedure or later (2,4-6,22,29). Small injuries frequently remain silent or even unrecognized (2,6,13,20,22).

Annular rupture can range from asymptomatic to an immediate catastrophic event (2-7,13,15). The acute signs are either aggressive, such as pericardial tamponade with hypotension, or subtle, such as pericardial effusion, subepicardial hematoma at the heart base, periaortic hematoma, new aortic wall thickening, hematoma between aorta and pulmonary artery, new onset of mitral and tricuspid regurgitation, atypical shunt, VSD, local or extended aortic dissection, contrast extravasation, conduction disturbances, and electrocardiographic changes (1-7, 13,15,16,18-29). Subtle signs might initially be overlooked. The most important clinical presentations are bleeding, hemodynamic instability, and acute myocardial failure (1-7,14).

## DIAGNOSTIC STRATEGIES

In acute situations, immediate suspicion of possible annular rupture is crucial (1,6). The principal rule is



**FIGURE 5** Subannular Rupture With Injury of the Anterior Mitral Leaflet

(A and B) Transesophageal echocardiography shows a large rupture (yellow double arrow) of the calcified fibrous region between the non-coronary sinus of Valsalva and the calcified anterior mitral leaflet (AML). Progressive severe pulmonary edema, hemodynamic instability, and atrioventricular block grade III occurred. Note the large shunt from the LV to the LA below the transcatheter valve (red brackets in A and B). (C) A schematic drawing shows the direction (red arrow) of rupture. (D) Pre-operative computed tomography demonstrates calcifications extending from the annulus into the anterior mitral leaflet (AML) (orange arrow). Mitral valve replacement and composite aortic valve graft implantation were performed. Extracorporeal membranous oxygenator was applied during the first post-operative day due to difficult ventilation. The patient died on the 8th post-operative day due to pneumonia. Abbreviations as in Figure 3.

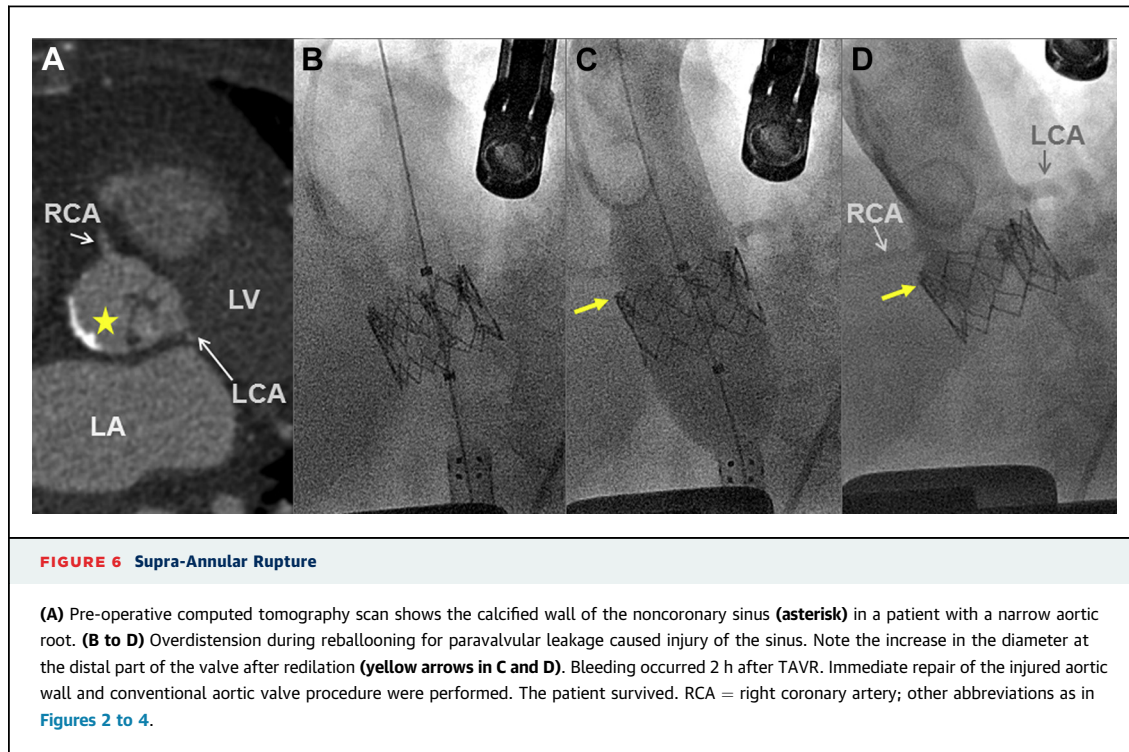
that any arterial bleeding in the pericardium with no identifiable cause should be considered as suspected annular rupture (1,6). Intraprocedurally, the examinations should be continuously performed and cautiously interpreted until the diagnosis is established or definitely excluded (2,6). Other possible causes of hemodynamic instability must be considered, such as occlusion of a coronary ostium, coronary artery embolization, injury of the LV or right ventricular myocardium or mitral chordae, malposition of the valve, aortic dissection, or retroperitoneal hemorrhage (1,2,4).

Annular rupture can be identified clinically, by echocardiography and/or by angiography (1,2,6,13). Transesophageal echocardiography combined with angiography is important for the early diagnosis in the initial phase of a suspected rupture

(1,2,6,13,14,20). Angiography is useful for detection of supra-annular problems but is of less value in identifying infra-annular injury if there is no relevant paravalvular or valvular aortic regurgitation (6). Computed tomography in combination with repeated echocardiography is valuable for follow-up surveillance (15,20,22).

#### TREATMENT APPROACHES

The treatment depends on the type of annular rupture and its clinical manifestations. Therapeutic approaches include conventional cardiac procedure, isolated pericardial drainage, and a conservative strategy (1-7,13,15,18,20,22). The decision made in favor of conservative therapy or sole pericardial drainage should always be measured against the



standard surgical way of thinking, “if in doubt, open the chest and explore” (5,6).

In the acute situation, between the onset of rupture and subsequent hemodynamic deterioration, there is a timeframe for action (2,4,6). Importantly, the initial symptoms may be mild or unclear, with stable hemodynamics. However, the clinical situation can quickly deteriorate as the dynamic process of rupture continues. In acute situations, the primary therapeutic goal is to maintain or restore hemodynamic stability to secure adequate coronary and cerebral perfusion. To maintain hemodynamic stability, immediate institution of cardiopulmonary bypass (CPB), and sternotomy with surgical correction of the rupture site should be considered (5,6). CPB is placed as normothermic femoro-femoral or—in the case of severe peripheral artery disease—central CPB through median sternotomy. CPB is performed during simultaneous volume substitution and inotropic support, and active search for a possible problem. This type of resuscitation requires close coordination between the members of the heart team (6,16). The correct diagnosis is established by echocardiography, aortography, and/or coronary angiography, or clinically by direct exploration through a median sternotomy (1,2,5,6).

**CONVENTIONAL SURGICAL STRATEGY.** Surgical strategy and the type of surgical treatment depend on the type of the annular rupture (Table 2). In general, repair of the lesion and aortic valve replacement are performed. Removal of the TAVR prosthesis and excision of the native aortic valve are standard parts of this repair (1,2,4-7,21,25,27). Additionally, pacemaker implantation and/or an intra-aortic balloon pump are sometimes necessary. Alternative (13,21,24) or bailout treatments, such as placing a second transcatheter valve to seal an annular tear for isolated rupture of the annulus or to close a VSD (4,7,23), may not always be successful and are generally not recommended (4).

**ISOLATED PERICARDIAL DRAINAGE.** Pericardial drainage and observation are applied in patients with mild pericardial effusion, aortic wall hematoma, or wall thickening (1,2,4,5,7,13). Some survive without sequelae (2,13), but the definitive outcomes during follow-up remain uncertain (2,4).

**CONSERVATIVE STRATEGY.** Conservative therapy, including optimization of the coagulation status, is possible for small injuries in patients without any clinical signs of rupture or in patients with aortic wall hematoma without pericardial effusion (1,2,6,7,22,25,26). Close surveillance with repeated computed tomography examinations is prudent

**TABLE 2 Surgical Treatment According to the Type of Annular Rupture**

Type of Rupture	Treatment
1. Intra-annular	Repair* of the lesion + AVR
2. Subannular	
a. Injury of the free myocardial wall	Reconstruction of the LVOT from inside the LVOT with a pericardial patch using transaortic approach† + AVR
b. Injury of the anterior mitral leaflet	Repair with a pericardial patch ± MVR‡ + AVR
c. Injury of the interventricular septum	Repair* + AVR
3. Supra-annular	
a. Injury of the wall of sinus of Valsalva	Repair* of the lesion + AVR or composite valved graft
b. Injury of a coronary ostium	Composite valved graft or repair of the lesion + AVR ± stenting of a coronary ostium/CABG
c. Injury of the sinotubular junction	Repair* of the lesion ± AVR or supracoronary aortic tube graft replacement ± AVR
4. Combined	
a. Intra- and supra-annular	Repair* of the lesion + AVR or composite valved graft
b. Intra-annular and subannular	Repair* of the lesion + AVR ± MVR
c. Intra-annular, supra-annular, and subannular	Composite valved graft ± MVR or repair* of the lesion + AVR ± MVR

\*Using pericardial patch or pledgeted sutures. †No attempts should be made to close the rupture from outside the left ventricle by using U-stitches because bleeding stops when the LVOT is reconstructed. The danger of damaging the coronary arteries by myocardial sutures from the outside is very high; this would lead to myocardial infarction and unsuccessful weaning from cardiopulmonary bypass. ‡Performed via a transaortic approach with additional incision of the left atrial roof and reconstruction of the intervalvular fibrous body using 2 pericardial patches.

AVR = aortic valve replacement; CABG = coronary artery bypass grafting; LVOT = left ventricular outflow tract; MVR = mitral valve replacement.

(15,20). Transthoracic echocardiography may underestimate the finding seen on transesophageal echocardiography (20,22). Some patients treated conservatively die during the following post-operative hours (6) or after several days (4,20). It is not known how many and which of these patients will survive without surgery. Intrapericardial adhesions after previous heart procedure are thought to be a favorable factor for conservative treatment by stopping propagation of the rupture (25).

## OUTCOME

Treatment of the first reported cases of annular rupture was unsuccessful, but later papers clearly show that it can be successfully managed if it is immediately recognized and adequately handled (1-7,13,16,21,27,29). The mortality of patients requiring emergency cardiac procedure is higher than in patients undergoing uneventful TAVR procedures (2-8). Presently, at least one-half of the surgically-treated patients will survive (3-8,16). The long-term results of conservative or nonsurgical therapy are not known. Some of these patients die suddenly during follow-up (4,20) (Figure 4). More experience is necessary to define the optimal treatment of detected but asymptomatic annular rupture.

## PREVENTION

Awareness of possible procedural complications and advanced understanding of the anatomical and pathophysiological relationships during TAVR are crucial in preventing complications. These require training of the heart team to recognize unexpected events and choose the best strategy in unusual situations. Safe anchoring of the valve should be balanced between using a larger valve to eliminate paravalvular leakage or a smaller valve to prevent possible annulus rupture. Therefore, precise pre-procedural analysis of the “device landing zone” is mandatory. It includes determination of the size and morphology of all anatomical structures of the aortic root and LVOT, meticulous identification of possible factors for annular rupture, and correct interpretation of the findings. Although time consuming, this analysis is the most important part of a TAVR procedure, and the heart team can plan a tailored strategy for the individual patient. Improvements in devices, procedural techniques, and imaging tools may simplify TAVR and additionally reduce possible complications.

Concrete preventive measures can be summarized as following: 1) identify whether the patient is at high risk for annular rupture by searching for predictive factor(s) (such as a very high calcium score, severe LVOT calcification, heavily calcified bicuspid valves, specific locations of calcium, or nodules over 4 to 5 mm); 2) consider modification of the therapeutic strategy in regard to the choice of the size of transcatheter valve diameter (such as choosing a smaller valve, accepting less than classical “2-mm oversizing” of the annulus diameter); 3) consider incomplete inflation of the balloon during valve deployment (such as 2- to 3-ml less inflation); 4) modify implantation plan (such as higher valve positioning during valve deployment in order to avoid protruding valve deposits in the LVOT); 5) avoid performing valve reballoning for paravalvular leak in the presence of significant calcification; 6) consider the use of other type of valve rather than a balloon-expandable valve; 7) rethink conventional surgery instead of TAVR; 8) reconsider the option of only conservative therapy; or lastly, 9) consider transfer of the patient to a high-volume center with extensive expertise in TAVR.

## CONCLUSIONS

Annular rupture is a rare and potentially extremely dangerous complication of TAVR. Prevention, early recognition, and immediate appropriate management are crucial in reducing the deleterious effects of annular rupture.



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