CASE REPORT

Sealing capacity of the ventricular muscle band after iatrogenic left ventricular perforation during transcatheter aortic valve implantation

Kerstin Piayda, Katharina Hellhammer, Verena Veulemans, Tobias Zeus

Division of Cardiology,
Pulmonology and Vascular
Medicine, Medical Faculty,
Heinrich Heine University,
Düsseldorf, Germany

Correspondence to

Dr Kerstin Piayda,
kerstin.piayda@med.uni-
duesseldorf.de

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SUMMARY

Left ventricular (LV) perforation accompanied by acute cardiac tamponade is a rare but one of the most feared complication during transcatheter aortic valve implantation. Few cases with the need of emergent surgical repair are described in literature. Handling of this uncommon but possible life-threatening event requires well-considered action by the implanting team and is associated with substantially increased intrahospital mortality. We present a unique case of LV perforation management with percutaneous pericardiocentesis only. As a possible underlying physiological mechanism, we identified the movement of the ventricular muscle band which possibly sealed the perforation side due to transverse and circumferential muscle contractions.

BACKGROUND

Left ventricular (LV) perforation is a seldom (0.15%¹) but feared complication during transcatheter aortic valve implantation (TAVI) and usually requires emergent surgical repair.² Handling of this rare but possible life-threatening event requires well-considered action by the implanting team and is associated with substantially increased intrahospital mortality and impaired outcome. We present a unique case of LV perforation management with pericardiocentesis only, with special focus on underlying physiological mechanisms such as the sealing capacity of the ventricular muscle band.

CASE PRESENTATION

An 86-year-old woman with symptomatic severe aortic valve stenosis was referred to our clinic. She presented with progressive shortness of breath (New York Heart Association class III), chest pain (Canadian Cardiovascular Society class I) and fatigue for over 6 months. Her medical history included a stable combined mitral valve lesion (moderate stenosis and mild regurgitation), severe pulmonary hypertension (systolic pulmonary artery pressure: 47 mm Hg), coronary sclerosis and a porcelain aorta (figure 1A). A pacemaker implantation was necessary in 2015 because of persistent atrial fibrillation with symptomatic bradycardia. Prevalent cardiovascular risk factors were arterial hypertension, non-insulin dependent diabetes mellitus and obesity grade 2.

INVESTIGATIONS

Physical examination revealed discrete peripheral oedema and a prominent S2, accompanied by a systolic fusiform murmur (grade 3) with maximum intensity at the right second parasternal intercostal space. Transthoracic echocardiography (TTE) confirmed a severe paradoxical low-flow low-gradient aortic valve stenosis (aortic valve area: 0.6 cm², mean pressure gradient: 21 mm Hg, maximum velocity: 2.9 m/s) with normal ejection fraction (63%) and reduced stroke volume index of 28 mL/m². The low-flow low-gradient aortic stenosis was confirmed via three-dimensional transoesophageal echocardiography. Figure 1B shows an x-plane view of the tricuspid aortic valve with considerable sclerosis, calcification and restricted opening of the tricuspid valve. According to the current European Society of Cardiology guidelines on the management of valvular heart disease,³ we did not perform a dobutamine stress echo. Instead, we assessed the calcium score by CT which accounted 1822 HU (Agatston method). Laboratory workup showed N-terminal probrain natriuretic peptide levels of 2648 pg/mL (<125 pg/mL).

Taking all clinical and diagnostic findings together, a true severe low-flow low-gradient aortic stenosis was highly likely and required consecutive therapy.

TREATMENT

The case was discussed in our interdisciplinary heart team, and we determined an interventional approach because of high perioperative risk (log EuroSCORE 25%), age >85 years and contraindications for surgical valve replacement (porcelain aorta).

During preparation for transfemoral TAVI with local anaesthesia only, retrograde aortic valve crossing with a 6 French AL1 guiding catheter and a straight, soft-tipped wire (0.35-inch Fixed Core Wire, Cook Medical, USA) was performed. Contrast medium is routinely administered at our institution to confirm the correct position of the AL1 in the LV cavity. In this case, the contrast agent was first seen intramyocardial in the anterolateral LV wall (figure 2A) and then spilled over in the pericardial space (figure 2B). Subsequently, the AL1 catheter was pulled back into the aorta, the procedure was interrupted and the patient's vital signs and her pericardial space were continuously monitored.

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Figure 1 Porcelain aorta (displayed with 3mensio Structural Heart-Software, Pie Medical Imaging, Maastricht, The Netherlands) (A). X-plane view of the tricuspid aortic valve with considerable sclerosis, calcification and restricted opening (B).

The haemodynamic condition deteriorated acutely with a rapidly declining blood pressure. TTE showed an expanding pericardial effusion of 12 mm, and we decided to perform pericardiocentesis (figure 2C): a needle was inserted from subxiphoidal and advanced under fluoroscopic guidance and contrast staining until it reached the pericardial space. The correct position was confirmed by fluoroscopy and application of a small amount of contrast medium into the pericardial space. With Seldinger technique, a pericardial pigtail catheter was inserted, and the blood successfully drained of the pericardial space; 200 mL of blood were removed and reperfused into the femoral vein. Afterwards, the haemodynamic situation stabilised instantly. During the next 10 min, only 15 mL of blood were aspirated. Vital signs remained stable, and activated clotting time was kept in a low therapeutic range (150 s). After interdisciplinary consensus, we continued the procedure because of inoperability and severe, symptomatic stenosis. A CoreValve Evolut R 26 mm prosthesis was successfully implanted (figure 2D) as quickly as possible without intensifying the anticoagulation strategy. One day after the procedure, a slight circular pericardial effusion (4 mm) without haemodynamic relevance was detected, and we discontinued the drain.

OUTCOME AND FOLLOW-UP

Echocardiography showed correct positioning and function of the aortic valve prosthesis, and the patient was discharged home 6 days after the initial procedure. At 30-day clinical follow-up, the cardiopulmonary capacity was substantially increased, and TTE revealed excellent haemodynamic results and no pericardial effusion.

DISCUSSION

LV perforation during TAVI occurs infrequently and is one of the most serious complications. It demands thoughtful action by the implanting team and is a very time-sensitive matter. Since the introduction of TAVI in 2002, the incidence is low and reliable data on possible risk factors and optimal management strategies are not available. Potential predictors for the occurrence of LV perforation during TAVI are a hypercontractile state, a thin muscular wall and a small LV cavity.⁴ Guide-wire injuries with subsequent LV perforation are valve independent and the leading cause for emergent cardiac surgery during TAVI (28.3%) with an intrahospital mortality rate of over 50% (EuRECS-TAVI registry¹⁵). Only a few cases demonstrate the feasibility of percutaneous management of LV perforation with pseudoaneurysm and sealing through closure devices.^{6 7}

If LV perforation accompanied by acute cardiac tamponade occurs, the most important step is to identify the location, mechanism of injury and extent of bleeding immediately. Annulus or aortic rupture during balloon valvuloplasty and LV perforation during retrograde guide-wire and catheter placement are the most common causes. Different management strategies exist: usually venous bleeding from the right atrium or right ventricle can be managed with percutaneous pericardiocentesis whereas arterial bleeding requires immediate surgical intervention in almost all events.⁸

In our case, percutaneous management of LV perforation was a considerable and successful option because the LV lesion caused by the guide-wire and the 6 French diagnostic catheter was punctual, circular and possibly sealed due to transverse and

Figure 2 Fluoroscopic image of the left ventricular perforation with intramyocardial blush (A) and subsequent contrast agent accumulation in the pericardial space (B). Instantly, pericardiocentesis was performed: a needle was inserted from subxiphoidal with fluoroscopic guidance and contrast staining and advanced until it reached the pericardial space. The correct position was confirmed by fluoroscopy and application of a small amount of contrast medium into the pericardial space (arrow). With Seldinger technique, a pigtail catheter was inserted and the blood successfully drained of the pericardial space (C). Successful implantation of a CoreValve Evolut R 26 mm prosthesis and remaining pigtail catheter (*) after percutaneous pericardiocentesis (D).

Figure 3 Illustration of the ventricular myocardial band. A continuous muscle band stretches from the pulmonary artery (PA) to the aorta (Ao). It provides the anatomical backbone of the helical muscle configuration and results in two loops at the cardiac apex and base which results in transverse and circumferential muscle contractions of descending and ascending segments (adapted from Song⁹).

circumferential muscle contraction of the ventricular muscle band (figure 3⁹). Based on the anatomical model of Torrent-Guasp,¹⁰ both ventricles consist of a single myofiber which stretches from the pulmonary artery to the aorta. During embryological development, it is formed into a double helical coil which builds the backbone for the dynamic rearrangement of myocardial layers and allows transverse and circumferential muscle contractions of descending and ascending segments.

However, this strategy cannot be chosen in every patient. It is restricted by perforation size and morphology because passive self-sealing capacity of the left ventricle is limited to punctual injuries and fails in longish lacerations. Constant haemodynamic and echocardiographic assessment is essential and conversion to open heart surgery still remains an option.

Additionally, several precautions can be taken to avoid guide-wire injury: all steps should be performed with extreme caution, the soft-tipped straight guide-wire should only be advanced a few centimetres in the centre of the LV cavity, the AL1 should be pushed forward slowly to a stable central LV position and especially the anterolateral twitch has to be avoided. As soon as possible, the AL1 may be replaced by a standard J-shaped wire which is further advanced and subsequently exchanged for an atraumatic pigtail catheter.

In conclusion, LV perforation due to wire injury during TAVI is a seldom, but possible, life-threatening complication. Our case proved that percutaneous management may be a considerable

option, depending on the haemodynamic impact of pericardial effusion, perforation size and morphology. In some cases, the circular structure of the left ventricle with transverse and circumferential muscle contractions can support perforation site sealing.

Learning points

- ▶ Percutaneous management of left ventricular perforation due to guide-wire injury during transcatheter aortic valve implantation is a considerable option under certain circumstances.
- ▶ Understanding of mechanism, location and extent of the injury is crucial.
- ▶ The helical muscle configuration of the left ventricle with resulting transverse and circumferential muscle contraction of descending and ascending segments may support perforation site sealing.

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