

Transcatheter aortic valve implantation for tricuspid aortic valve with a calcium bridge between the cusps: a case report

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Background

Indications for transcatheter aortic valve (AV) implantation (TAVI) have been extended to patients with challenging anatomical features such as a bicuspid AV. However, no case reports of TAVI for tricuspid AV with a 'calcium bridge' have been published.

Case summary

We report the case of an 87-year-old woman with severe symptomatic aortic stenosis (AS). Pre-procedural multi-detector computed tomography (MDCT) showed a calcium bridge between the non-coronary and left cusps. Although these cusps appeared to be fused together in the centre by the calcium bridge, the commissure area between the fused cusps appeared to normally open. We cracked apart the calcium bridge from the commissure area using balloon aortic valvuloplasty, which was guided by transoesophageal echocardiography. Subsequently, we successfully implanted a balloon-expandable transcatheter heart valve (THV).

Discussion

To our knowledge, this is the first report of TAVI for a tricuspid AV with a 'calcium bridge' between the non-coronary and left cusps. In our patient, the unique structure of cusp fusing in the centre seemed to be caused by an acquired atherosclerotic process, as documented using a contemporary technique, i.e. MDCT. Cracking the calcium bridge with a balloon aortic valvuloplasty ensured that the THV adequately expanded. Severe AS caused by a calcium bridge may be treated with detailed evaluation of the AV morphology together with a procedural strategy planning.

Keywords

Aortic stenosis • Balloon aortic valvuloplasty • Calcium bridge • Transcatheter aortic valve implantation • Case report

Learning points

- This is the first report of a transcatheter aortic valve (AV) implantation for a tricuspid AV with a 'calcium bridge' between the non-coronary and left cusps.
- In our patient, the unique structure of cusp fusing in the centre seemed to be caused by an acquired atherosclerotic process, as documented using a contemporary technique, i.e. multidetector computed tomography.
- Severe aortic stenosis caused by the calcium bridge may be treated with detailed evaluation of the AV morphology together with a procedural strategy planning.

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Introduction

The indications for transcatheter aortic valve (AV) implantation (TAVI) have been extended to patients with challenging anatomical features such as a bicuspid AV (BAV).^{1,2} Although TAVI for tricommissural BAV (tricommissural AV with fusion of one commissure) has been performed,³ there have been no studies of TAVI for tricuspid AV with a 'calcium bridge' to date.

Timeline

2016	An 84-year-old woman was diagnosed with severe aortic stenosis (AS), but she refused surgical treatment.
April 2019	Three years after the initial diagnosis, the patient, now aged 87 years, was admitted to our hospital for acute decompensated heart failure. The diagnosis was severe AS [peak aortic valve (AV) velocity, 5.17 m/s; mean AV pressure gradient, 63 mmHg; and AV area, 0.56 cm ²]. The left ventricular function was preserved, and there was no other cause of heart failure (including obstructive coronary disease). The patient underwent successful transfemoral transcatheter aortic valve implantation (TAVI).
April 2020	At the 1-year follow-up visit after TAVI, the patient was well and exhibited good bioprosthesis function (peak AV velocity, 2.21 m/s; mean AV pressure gradient, 10 mmHg; and effective orifice area, 1.81 cm ²) with normal left ventricular function.

Case presentation

An 87-year-old female patient with a history of hypertension and diabetes was admitted to our hospital for acute decompensated heart failure. She had been diagnosed with severe aortic stenosis (AS) at another hospital 3 years before admission to our institution and had refused surgical treatment because she was asymptomatic. Subsequently, she has lived alone and has been independent regarding activities of daily living.

She presented to our hospital at midnight with tachypnoea and the following vital parameters: blood pressure, 219/111 mmHg; heart rate, 114 b.p.m.; and oxygen saturation, 89% in room air. Lung and cardiac auscultations demonstrated the presence of coarse crackles in both lung fields and a high-pitched systolic ejection murmur in the right upper sternal edge. She had both a distended internal jugular vein and bilateral pitting pedal oedema.

Laboratory tests revealed a high B-type natriuretic peptide (218.6 pg/mL), no anaemia (haemoglobin (Hgb), 13.7 g/dL; haematocrit (Hct), 40.9%), and a moderately decreased renal function (creatinine (Cre), 1.18 mg/dL; estimated glomerular filtration rate

33.1 mL/min/1.73 m²). A mildly enlarged cardiac silhouette with pulmonary congestion and pleural effusion was observed on a chest X-ray. Electrocardiography demonstrated normal sinus rhythm and left ventricular hypertrophy with strain pattern ST segment depression in V4–6 leads. Transthoracic echocardiography (TTE) confirmed severe AS of uncertain AV morphology, with a peak AV velocity of 5.17 m/s, a mean AV pressure gradient of 63 mmHg, and an AV area of 0.56 cm². Mild aortic regurgitation (AR) was observed. Her left ventricular ejection fraction (LVEF) was 63% and her left ventricular end-diastolic diameter (LVEDd) was 53 mm.

Diuretic administration (intravenous furosemide, 20 mg daily) and non-invasive positive pressure ventilation relieved her symptoms. After considering the patient's age, a Society of Thoracic Surgeons risk score of 7.631 for surgical AV replacement, and the patient's preference to not undergo open-heart surgery, our heart team decided that TAVI was the best option for managing her condition.

A multidetector computed tomography (MDCT) scan demonstrated a severely calcified tricuspid AV. Although the non-coronary and left cusps seemed to be fused in the centre by a 'calcium bridge', the commissure area between the fused cusps appeared to normally open. This resulted in the formation of two small and large orifices divided by the calcium bridge (*Figure 1A–E*; [Supplementary material online, Video S1](#)). The Agatston calcium score for her AV was 2463.28. Mild calcification of the aortic annulus was detected. The variables of the aortic annulus were as follows: area, 395 mm²; perimeter, 71.4 mm; maximum diameter, 24.0 mm; and minimum diameter, 21.0 mm (*Figure 1F*). The mean diameter of the sinus of Valsalva (SOV) was 26.3 mm, and the heights of the right and left coronary arteries were 14.3 and 12.8 mm, respectively. There was no obstructive coronary disease. The patient's iliofemoral artery was suitable for transfemoral access. To acquire MDCT images, we used a computed tomography system with 320 detector rows (Aquilion ONE/VISION; Toshiba Medical Systems, Tokyo, Japan). To reconstruct images and measure morphological parameters, we used the Ziostation2 PLUS, version 2.9.3.0 software (Ziosoft, Tokyo, Japan).

To attain adequate transcatheter heart valve (THV) expansion, the calcium bridge should be managed beforehand. Our TAVI strategy was as follows: first, we passed the wire retrogradely through the small orifice. This was followed by a balloon aortic valvuloplasty (pre-dilatation) from the side once the THV was ready to be deployed. Finally, when expansion was adequate, we deployed a 23-mm-sized Edwards SAPIEN 3 (Edwards Lifesciences, Irvine, CA, USA) balloon-expandable THV. Regarding the THV type, we selected a balloon-expandable THV that could be quickly deployed, if required. Furthermore, the patient's SOV diameter was extremely small for the self-expandable THVs that are available in Japan. Although she was at high risk of cerebral infarction because of her significant calcium levels of the cusps, age, and gender, a cerebral protective device was unavailable because such devices are not approved in Japan.

We performed a transfemoral TAVI using transoesophageal echocardiography (TOE) guidance with the patient under general anaesthesia. Retrograde AV wire crossing was achieved via the small orifice as confirmed by TOE ([Supplementary material online, Video S2](#)). The wire was set at the centre of the aortic annulus even after setting the stiff wire in the left ventricle in the perpendicular view. We then performed a pre-dilatation using a 20 mm Nucleus-X balloon (B. Braun

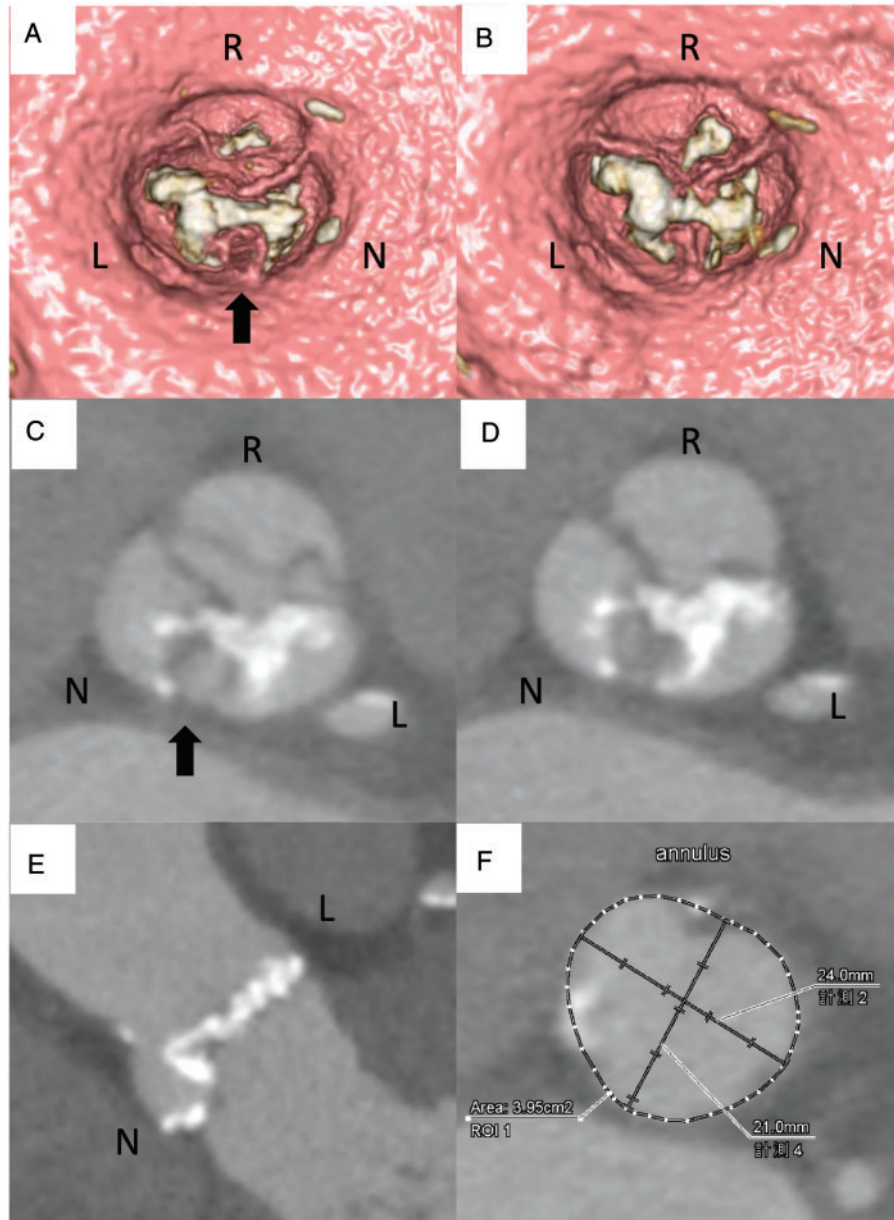


Figure 1 Pre-procedural computed tomography images. Virtual endoscopic image of the aortic valve from the ascending aorta in the mid-systole (A) and diastole (B) phases. Short-axis images of the aortic valve in the mid-systole (C) and diastole (D) phases, and long-axis image of the non-coronary and left cusps in the diastolic phase (E). A ‘calcium bridge’ between the non-coronary and left cusps and a small orifice (arrow) surrounded by the calcium bridge and the non-coronary and left commissure are clearly identified. (F) Aortic annulus measurement. L, left coronary cusp; N, non-coronary cusp; R, right coronary cusp.

Interventional Systems, Bethlehem, PA, USA). Initially, the balloon expanded in an hourglass shape at the level of the aortic annulus. However, after additional inflation to up to 2atm, we successfully cracked apart the fused calcium bridge between the cusps. After pre-dilatation, the wire route changed and attached to the greater curvature of the aortic annulus (Figure 2A–E; Supplementary material online, Videos S3 and S4). The patient’s haemodynamics after

the pre-dilatation remained stable without AR worsening. Subsequently, we deployed a 23-mm-sized SAPIEN 3 with a nominal volume of 17 mL. A TOE evaluation revealed residual moderate perivalvular leakage; therefore, post-dilatation with 1 mL more than the nominal volume was applied. Consequently, an optimal depth and adequate expansion with trivial perivalvular leakage were attained (Figure 2F and G).

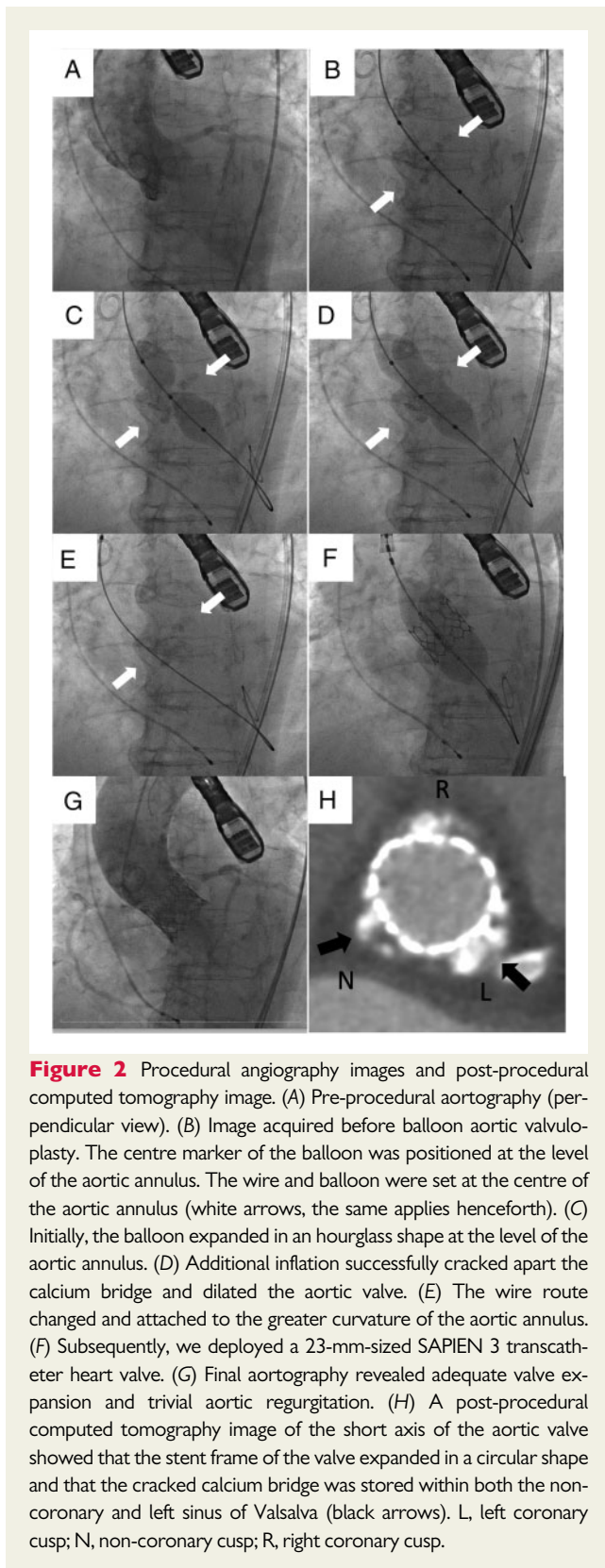


Figure 2 Procedural angiography images and post-procedural computed tomography image. (A) Pre-procedural aortography (perpendicular view). (B) Image acquired before balloon aortic valvuloplasty. The centre marker of the balloon was positioned at the level of the aortic annulus. The wire and balloon were set at the centre of the aortic annulus (white arrows, the same applies henceforth). (C) Initially, the balloon expanded in an hourglass shape at the level of the aortic annulus. (D) Additional inflation successfully cracked apart the calcium bridge and dilated the aortic valve. (E) The wire route changed and attached to the greater curvature of the aortic annulus. (F) Subsequently, we deployed a 23-mm-sized SAPIEN 3 transcatheter heart valve. (G) Final aortography revealed adequate valve expansion and trivial aortic regurgitation. (H) A post-procedural computed tomography image of the short axis of the aortic valve showed that the stent frame of the valve expanded in a circular shape and that the cracked calcium bridge was stored within both the non-coronary and left sinus of Valsalva (black arrows). L, left coronary cusp; N, non-coronary cusp; R, right coronary cusp.

The post-procedural course was uneventful without any neurological sequelae. MDCT demonstrated a well-expanded THV stent frame and retention of the cracked calcium bridge within both the

non-coronary and left SOVs (Figure 2H). TTE showed a peak AV velocity of 2.37 m/s, a mean AV pressure gradient of 9 mmHg, an effective orifice area of 2.28 cm², and trivial paravalvular leakage. Her LVEF was 79% and the LVEDd was 48 mm.

At the 1-year follow-up visit, the patient was well. Repeat TTE revealed a peak AV velocity of 2.21 m/s, a mean AV pressure gradient of 10 mmHg, an effective orifice area of 1.81 cm², and mild paravalvular leakage. Her LVEF was 77% and the LVEDd was 42 mm.

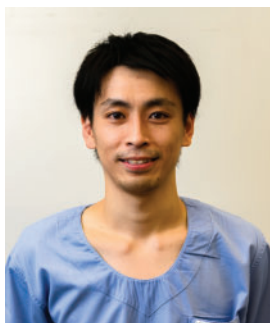
Discussion

We described a unique morphology of the tricuspid AV with a calcium bridge managed with successful TAVI. MDCT is the gold standard for pre-TAVI analysis of the AV complex and vascular access.⁴ In our patient, we clearly observed the 'calcium bridge' and two orifices in 2D and 3D virtual endoscopic images of the AV. A novel TAVI-directed classification of BAV morphology specifies three types.³ Although the patient's AV seemed to be a tricommisural or functional BAV, it lacked important features of commissural fusion. It can be argued that the fusion of the tips is part of the atherosclerotic change; in rheumatic valvular disease, in contrast, the fusion occurs at the base of the cusps. The unique structure of cusp fusing in the centre observed in our patient seemed to have been caused by an acquired atherosclerotic process that was documented using a contemporary technique (i.e. MDCT).⁵

TAVI without pre-dilatation has been used to simplify the procedure and avoid potential pre-dilatation-related complications.⁶ However, pre-dilatation played an important role in this situation. Our greatest concern was whether the THV would sufficiently expand. Although pre-dilatation from either the small or large orifice would have cracked the calcium bridge, pre-dilatation from the small orifice exhibited the advantage of ensuring its cracking. Alternation of the wire route from the centre of the aortic annulus to the greater curvature (Figure 2B and E) confirmed that the two orifices had been communicating with each other. If the wire had passed through the large orifice, it would have stayed at the greater curvature in the perpendicular view (Figure 2E), regardless of whether the calcium bridge was cracked or not. Other concerns included the development of pre-dilatation-related complications, particularly acute AR and aortic annulus rupture.⁷ The aortic annulus is a tough tissue and no calcium was present at the non-coronary and left commissure; therefore, we considered that calcium bridge cracking or leaflet tears should happen before the occurrence of annulus rupture. We prepared a loaded balloon-expandable THV before the pre-dilatation for an acute AR.⁸ Even if the SOV had been sufficiently large for deploying a self-expandable THV, we would have preferred the balloon-expandable THV because it can be quickly deployed.

This is the first report of TAVI for a tricuspid AV with a 'calcium bridge' between the non-coronary and left cusps. The detailed evaluation of the AV morphology together with a procedural strategy was crucial for a successful outcome.

Lead author biography



Masaki Nakashima received the MD degree from Kanazawa University School of Medicine, Kanazawa, Japan in 2012. He began clinical training in Kobe city medical centre general hospital, Kobe, Japan in 2017. Currently, he is undergoing structural heart disease intervention training in his current institution.

Supplementary material

[Supplementary material](#) is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: N.T. is a clinical proctor for Edwards Lifesciences and Medtronic. All the other authors have no conflict of interest to disclose.

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