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CASE REPORT

Autopsy case of aortic dissection after transcatheter aortic valve implantation (TAVI)

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Accepted 26 February 2018

SUMMARY

Aortic dissection is one of the severe but rare vascular complications arising from transcatheter aortic valve implantation (TAVI). This paper presents an autopsy case of an 81-year-old male patient with delayed aortic dissection with underlying haemorrhages and acute inflammation 3 years after TAVI.

BACKGROUND

Transcatheter aortic valve implantation (TAVI), also known as transcatheter aortic valve replacement, is currently an interventional method for patients with significant aortic valve stenosis. Major and clinically significant complications related to TAVI include deaths in connection with the intervention, postsurgical aortic regurgitation, the need for pacemaker implantation, cardiac tamponade, myocardial infarction, stroke, severe vascular and bleeding complications, acute renal failure and infectious endocarditis, with vascular complications being the most common.^{1 2} The most widespread and approved valves used today are the balloon-expandable Edwards Sapien valve and the self-expanding Medtronic CoreValve System. The incidence of vascular complications was lower with Medtronic valve until the new generation of Edwards Sapien arose, resulting in similar frequency of incidence as shown in clinical studies.² The aortic dissection is infrequent (0.12%) and it usually occurs during the TAVI procedure.³ Some case reports about delayed aortic dissection were published but the clinical data about the incidence and the pathogenesis are limited. Some studies, including all aortic valve replacement methods, describe, as a risk factor for developing a dissection, the diameter of

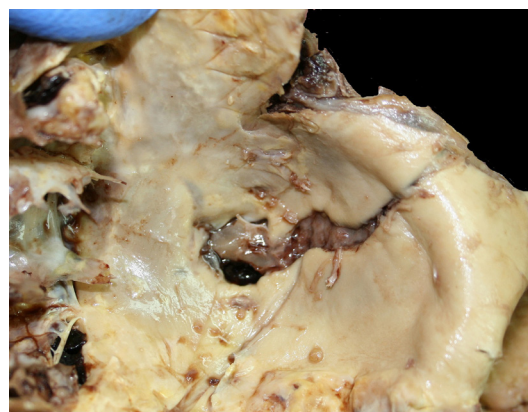


Figure 2 Fixed specimen after removal of the valve cage with underlying haemorrhages.

the ascending aorta (especially over 50 mm). Other characteristics such as sex, age, severity of aortic dilatation, presence of progression in diameter, left ventricular function and time interval after valve replacement were not helpful in predicting the risk for dissection.⁴

CASE PRESENTATION

We report a case of an 81-year-old male patient who was urgently admitted to a hospital due to fever and weakness. In blood cultures, *Staphylococcus aureus* was detected and hence an antibiotic treatment with amoxicillin and clavulanate was started. Three years before, the patient had received a TAVI Medtronic CoreValve (31 mm) due to a symptomatic aortic valve stenosis. Transthoracic

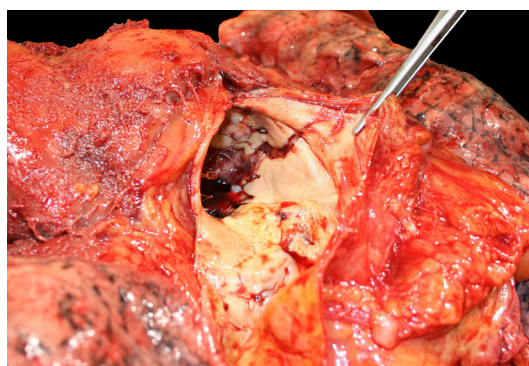


Figure 1 Aortic dissection in situ (transverse section of the aorta above the aortic valve).

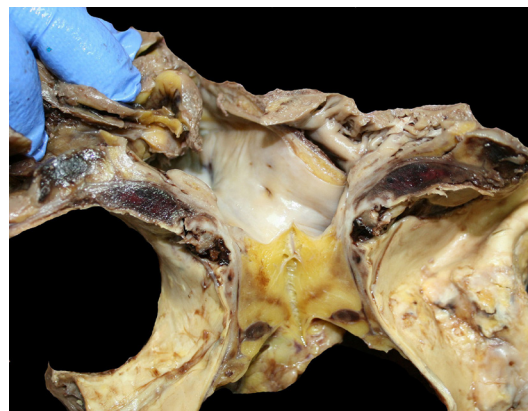


Figure 3 Fixed specimen after removal of the valve cage with underlying haemorrhages.



To cite: Losmanova T, Tosoni I, Fahrni S, et al. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-220105

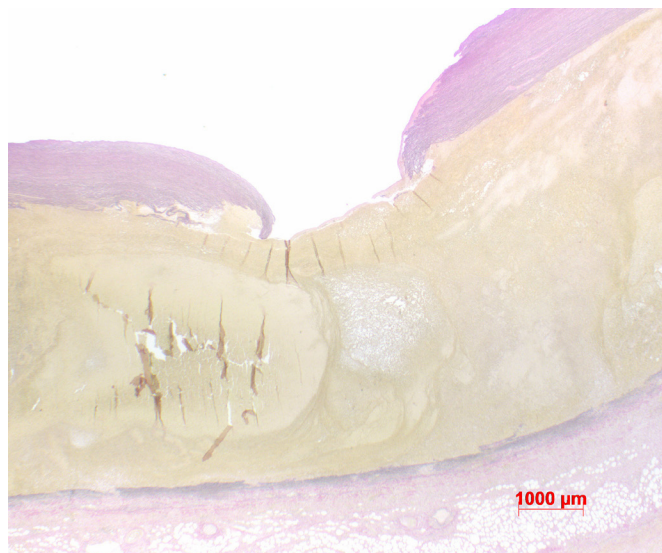


Figure 4 Histological view (Elastica van Gieson (EVG) stain): low-power cross section with wall defect.

echocardiography and transoesophageal echocardiography (TEE) were performed suspecting infectious endocarditis of the prosthetic valve. TEE showed no evidence of endocarditis but a dehiscence between the edge of the valve cage and the aortic wall, which was suspected to be caused by a wall abscess. With an increasing aggravation of the patient's condition, best supportive care therapy was initiated and the diagnostics were stopped, not performing any further imaging methods like a CT. The patient eventually died 2 weeks after hospitalisation. The patient was examined with TEE 4 days, 4 months and 14 months after the surgery, revealing a fully functional CoreValve with no signs of dissection or other pathology. For further investigation, an autopsy with macroscopic and microscopic examinations was carried out.

INVESTIGATIONS

The postmortem showed a wall defect of the ascending aorta (approximately 4×1×1 cm) directly adjacent to the upper

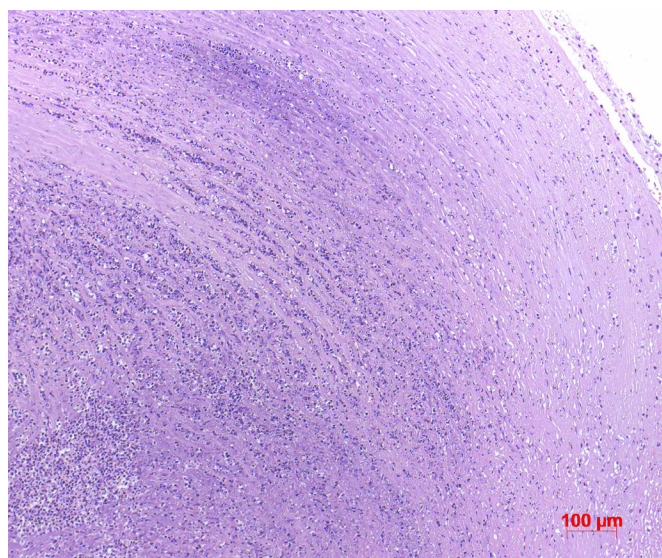


Figure 5 Histological view (H&E stain): transmurular granulocytic inflammation with focally abscess formation.

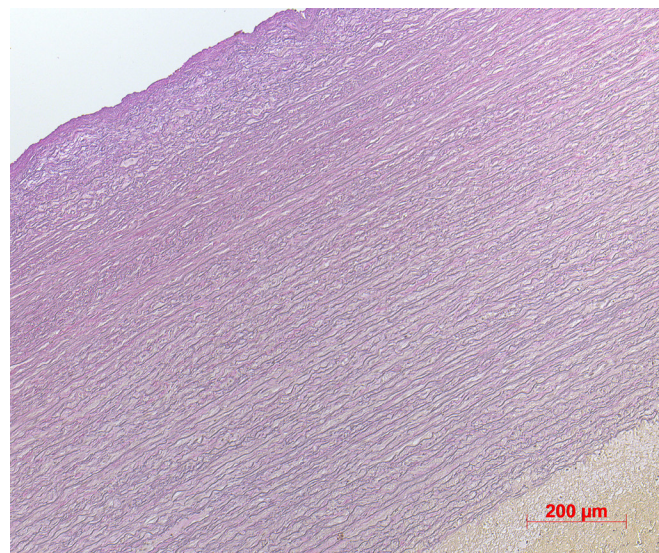


Figure 6 Histological view (Elastica van Gieson (EVG) stain): regular layered pattern of elastic fibres in tunica media, undamaged aortic wall.

edge of the valve cage (figures 1 and 2). Under the defect there was a circumscribed cavity filled out with fresh haemorrhages (figure 3). Macroscopically, an additional inflammation could not be excluded. The microscopical examination revealed a dissection reaching from the aortic lumen into the lower part of the tunica media (figure 4). In the area of the wall crack, the aortic wall showed signs of a granulocytic inflammation with destruction of the wall structures (figure 5) and underlying fresh haemorrhages with acute inflammation. Unfortunately, the exact determination of the age of the dissection was histologically not possible. The aortic wall in other areas of the thoracic aorta showed parallel running intact elastic fibres (figure 6) with no pre-existing medical condition such as Erdheim-Gsell medial necrosis or inflammation composed of macrophages or T lymphocytes as seen in the aortic aneurysms. In addition, thrombotic-polypoid endocarditis of the prosthetic aortic valve and pronounced pericarditis with haemorrhagic pericardial effusion were discovered.

DISCUSSION

The long-term complications that arise from TAVI include paravalvular and central valve regurgitation, valve thrombosis, prosthetic valve endocarditis and late bleeding. Aortic dissection is rare but it is a severe complication. In the medical literature, there have been reports about delayed aortic dissection after TAVI and its influence on the prediction of late mortality of this procedure, but the pathogenesis data are rare.^{5 6} The major predisposing factors for developing dissection are those which increase the aortic wall stress—such as aortic diameter or hypertension, or decreased wall strength—such as defects of the aortic wall (eg, in Marfan syndrome).⁷ In our case, the patient was treated for arterial hypertension, the measurements in the echocardiography were normal and the histological examination excluded any pre-existing defects of the aortic wall. The dissection, as seen in figures 1 and 2, appeared directly under the inlaying valve cage within the area of acute inflammation. Aortitis is one of the known risk factors of aortic dissections or ruptures,⁸ therefore we hypothesise that the mechanical stress of the valve cage together with the negative influence of the endocarditis on the endothelium could have been the cause of

the development of the dissection. However, the time when the dissection happened is difficult to determine. Another differential diagnosis, as Gerber *et al* discussed, could be that the aortic dissection or intimal tears occurred during the procedure of the TAVI, and that they could have been overlooked until the hospitalisation. Guidewire delivery system manipulations, valve repositioning retrieval or retraction, frequently in the setting of suboptimal aortic measurements and/or visualisation, could have been the cause of the late aortic dissection. Also an intimal tear due to atherosclerosis could be taken into account. However, there were no atherosclerotic changes in the area of the dissection and these changes usually appear in the descending part of the aorta, not in the ascending.⁹

Learning points

- ▶ Aortic dissection with possible abscess formation should always be taken into account as a late-onset complication of transcatheter aortic valve implantation.
- ▶ The pathogenesis of the dissection was not confirmed finally. We hypothesise that the mechanical stress together with the endocarditis was mainly responsible for the dissection.
- ▶ However, whether the inflammation was the cause of the aortic dissection or the secondary change caused by the dissection cannot be definitively concluded in our case.

Contributors All authors contributed to refinement of the article. This case report was designed, directed, funded and coordinated by PEB and corrected by IT. The manuscript was written by medical practitioner TL with SF, who contributed to the clinical findings. The manuscript was commented on by all authors.

Funding This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Next of kin consent obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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