

**Case Report**

Unusual Perceval Sutureless Valve Dysfunction 2,5 Years After Implantation. Not Only Thrombosis.

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Abstract

We present a case of a Perceval S XL 25-27 dysfunction caused by an unusual thrombosis 2.5 years after implantation. The 71-year-old male patient was admitted to the heart surgery ward because of suspicion of prosthetic valve dysfunction. The echocardiographic examination confirmed the increased trans-prosthetic gradient and thickened prosthesis cusps with limited mobility. Computer tomography confirmed the structural valve deterioration. The patient was referred for urgent surgery. Intraoperatively, sandy-coloured deposits were found at the junction of the stent and the prosthesis leaflets, except for typical valve leaflet thrombosis. Pathological examination revealed atheromatous material with older fibrin and leukocytes. Typically, thrombi are localized on the aortic surface of the prosthesis leaflets, leading to thickening and gradual impairment of motion. The commissures, free leaflet margin, and prosthesis stent are not affected. In our case, additional deposits were found at the junction of the stent and the prosthesis leaflets. They were symmetric and equal in all three commissures. The cause and possible role of deposits in valve thrombosis remains unknown.

Keywords: Perceval; Prosthesis Thrombosis; Prosthesis Dysfunction; Suture less Prosthetic Valve.

Introduction

The Perceval (Cordis, London, UK) suture less prosthetic valve was first introduced in Europe in 2011 and in the US in 2016. Clinical valve dysfunction caused by thrombosis is reported extremely rare. To our best knowledge, only two cases have been reported till now [1,2]. We present a case report of a Perceval S dysfunction caused by unusual thrombosis 2.5 years after implantation.

Case Presentation

The 71-year-old male patient was admitted to the heart surgery

ward because of increasing fatigue, dyspnea during exertion, and anginal symptoms for a few weeks with suspicion of prosthetic valve dysfunction. Two and a half years before the hospitalization, Perceval S XL 25-27 valve was implanted because of symptomatic severe aortic valve stenosis. The operation was complicated with allergic reactions and conclusively cardiogenic shock after protamine injection. It was therefore necessary to apply a veno-arterial ECMO (Extracorporeal Membrane Oxygenation) and renal replacement therapy. In the further course, pneumonia was diagnosed, which was treated with intravenous Ciprofloxacin and Meropenem. Finally, the patient developed bilateral pleural effusion, which had to be punctured. The blood cultures were negative. The patient left the hospital after 30 days. Life-long single antiplatelet therapy (Aspirin 100 mg/day) was applied without

vitamin K antagonists. One month later, a DDD pacemaker was implanted because of AV block III. The patient's condition was stable till admission.

The echocardiographic examination on admission revealed a high trans prosthetic gradient (PPG 67 mmHg, MPG 43 mmHg), and thickened prosthesis cusps with limited mobility (Figure 1A, Video 1 and 2.). Computer tomography confirmed the diagnosis of structural valve deterioration (Figure 1B). The patient was referred for urgent surgery. Intraoperatively, typical valve leaflet thrombosis was detected (Figure 2, dashed arrow). Additionally, sandy-coloured deposits were found at the junction of the stent and the prosthesis leaflets (Figure 2, arrow). Pathological examination revealed atheromatous material with older fibrin and leukocytes at the junction of the stent and the prosthesis leaflets, except for the typical clot on the leaflets.

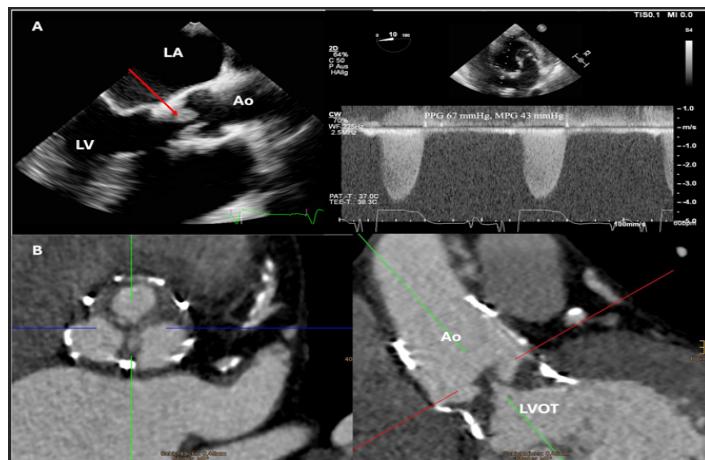


Figure 1: A. Transesophageal examination, thickening of the leaflet (arrow), and increased gradient through the valve are visible. B. Computer tomography. Thickening at the junction of the stent and the prosthesis leaflets (arrow) is visible. Ao - aorta; LVOT - left ventricular outflow tract; LA - left atrium; LV - left ventricle.

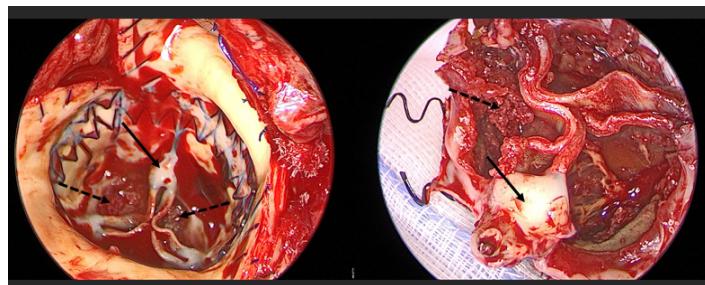


Figure 2: Explanted prosthesis, typical thrombosis is visible (dashed arrows). Additionally, sandy-coloured deposits at the junction of the stent and the prosthesis leaflets (solid arrows) are visible.

Video 1: Transesophageal examination, 2D. Thickening of the leaflet of the valve, as well as limited mobility, is visible.

Video 2: Transesophageal examination, 2D and colour Doppler. Limited mobility of the valve and flow acceleration is visible.

Discussion

The dysfunction of biological prostheses can be caused by gradual, time-related degeneration or thrombus formation. The incidence of clinically apparent Perceval prosthesis degeneration causing structural valve dysfunction requiring reoperation is relatively low. However, performed cardiac computed tomography (CT) in asymptomatic patients after Trans catheter aortic valve replacement (TAVR) showed reduced leaflet motion in 40% of patients without clinical manifestation, regardless of the type of prosthesis [3]. Hypo attenuated leaflet thickening (HALT) with or without reduced leaflet motion (RLM) could be detected by CT in many patients without clinically apparent valve dysfunction, not only after TAVR but also after surgical aortic valve replacement SAVR [4,5]. There is very limited data concerning suture less prosthetic valves. The only study of Perceval prosthesis was performed at a median of 491 days after implantation and showed the presence of HALT in 38% and RLM in 28% of patients. No difference was found in the mean Trans prosthetic gradient between the patients with and without RLM [6]. The actual number of patients with HALT and RLM in patients after the implantation of the Perceval prosthesis is not known because of a lack of routine CT examination. Clinically evident thrombosis of Perceval's prosthesis is reported to be extremely rare. Only two cases of Perceval prosthesis thrombosis and one case of HALT have been reported till now [1,2,7].

Typically, thrombi are localized on the aortic surface of the prosthesis, leading to leaflet thickening and gradual impairment of leaflet motion. The commissures, free leaflet margin, and prosthesis stent are not affected. Similar changes were found in both earlier reported cases. In our case, additional deposits were found at the junction of the stent and the prosthesis leaflets. They were symmetric and equal in all three commissures. The sequence of events leading to valve dysfunction needs to be clarified. The formation of deposits could be the reason for the primary limitation of the leaflet motion, which led to clot formation. The presence of leukocytes and atheromatous material in the deposits revealed during pathological examination in our case may indicate an inflammatory process as the cause of the deposits. The opposite scenario, where immobilized clotted leaflets lead to the formation of deposits, is also possible. Our patient was treated only with antiplatelet agents. It is difficult to state how far the lack of vitamin K antagonist (VKA) was the likely cause or trigger of valve thrombosis. The incidence of HALT was much more frequent in patients who were not treated with VKA. Moreover, the treatment with VKA led to regression of HALT [3]. The under expansion

of the prosthesis was also proposed as a possible cause of leaflet motion impairment leading to HALT [8]. Similarly, oversizing was a reason for increased trans-prosthetic gradients [9]. We did not observe either under expansion or under oversizing in our case.

Conclusions

In our case, the cause and possible role of deposits in valve thrombosis remains unknown. Such deposits may be resistant to anticoagulation therapy. Lack of improvement of pressure gradient and leaflet mobility under anticoagulation should raise suspicion of prosthesis degeneration, old organized thrombosis, or fibrin deposits. In such cases, surgery should be considered.

Ethical Statement: Written patient consent was obtained.

Conflict of Interest: All authors declare no conflict of interest.

Data Availability Statement: The data underlying this article will be shared on reasonable request to the corresponding author.

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