Cardiology in the Young

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Cerebral embolic protection during transcatheter stent expansion of restrictive extra-cardiac Fontan conduit

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Brief Report

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Abstract

We report a 20-year-old female patient (76 Kg/164 cm) with an extra-cardiac Fontan circulation who was referred to our institution for exertional dyspnoea and desaturation. The patient was diagnosed with a large calcified thrombus at the level of the Fontan fenestration, protruding inside the lumen of the conduit and reducing the diameter by half with a 3 mmHg pressure gradient. Transcatheter stent expansion of the obstructed extra-cardiac conduit was done with a 48 mm long XXL PTFE-covered Optimus-CVS® under temporary cerebral embolic protection with a TriGUARD-3™ deflection filter device (Keystone Heart). There was no procedural complication and the 3 months clinical outcomes are good.

Restriction in the dimensions of extra-cardiac conduits can occur over time and may deteriorate the function of the Fontan circuit.[1] Conduit stent expansion to nominal diameters and above can be safely performed to compensate for somatic growth.[1,2] This intervention can be riskier in the case of fenestrated conduits and central venous thrombus formations.[3] The TriGUARD-3™ (Keystone Heart) is a self-stabilizing deflection filter device that is designed to provide complete coverage of all cerebral arteries during transcatheter aortic valve replacements to allow adequate blood flow to the brain while diverting emboli downstream.[4,5] Here, we report a temporary cerebral protection from paradoxical embolism with TriGUARD-3 to perform a transcatheter stent expansion of an extra-cardiac fenestrated Fontan conduit that was partially obstructed with a large protruding calcified thrombus at the level of the Fenestration.

Case presentation

A 20-year-old female patient (76 Kg/164 cm) was referred to our institution for progressively worsening exertional dyspnoea and oxygen desaturation. The patient has a hypoplastic left heart and was palliated with a fenestrated extra-cardiac Fontan (18 mm Gore-Tex tube) at the age of five years. She has a history of spontaneous closure of the fenestration shunt during follow-up and the warfarin therapy was switched to daily oral antiplatelet therapy at the age of 14 years for treatment non-compliance. Cardiac 4D-Flow MRI showed a fistula originating from the innominate vein circulation and draining into the heart. The MRI also showed a moderate kinking of the extra-cardiac conduit and suspected a mild flow acceleration under the aortic valve. A diagnostic cardiac catheterisation was performed and showed low central venous pressure and no gradient pressure over the left outflow tract. Angiography showed a large calcified thrombus at the level of the Fontan fenestration, protruding inside the extra-cardiac conduit and reducing the inner diameter by half (Fig. 1a). There was an invasive gradient of 2 mmHg across the thrombus. We also identified an occlusion of the left jugular vein that was drained by a venous network into the coronary sinus. This fistula is draining the left cranial body part and thereby we did not close it although it was responsible for the oxygen desaturation.

The case was discussed during several multi-disciplinary team meetings and the patient was finally scheduled after 12 months for a conduit stent expansion to relieve the central venous obstacle under cerebral embolic protection. The delay from diagnosis to intervention was secondary to administrative and logistic issues. The right femoral vein and the left femoral artery were accessed with 7 and 8-Fr short introducers. Intravenous prophylactic antibiotics and heparin were given. Baseline aortography delineated the aortic arch anatomy. After 0.035-inch stiff wire positioning in the innominate vein, hemodynamic measurements, and caval angiography were done. The inner diameter of the conduit at the level of the thrombus was 7 mm (Fig. 1b). The invasive gradient across the thrombus was 3 mmHg. Over a 0.035-inch stiff wire, the TriGUARD-3 system was advanced sheathless from the left femoral artery into the

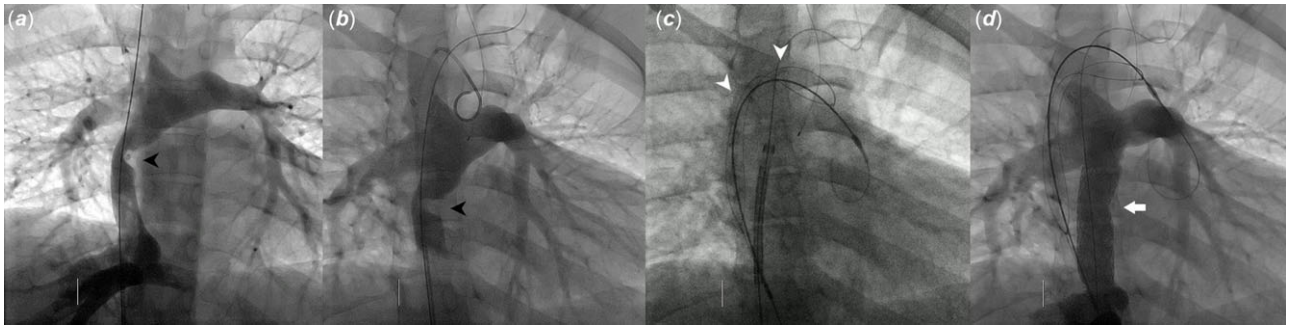

Figure 1. First caval angiography showing a large calcified thrombus at the level of the Fontan fenestration, protruding inside the lumen of the conduit and reducing the diameter by half (***a***). Caval angiography (12 months later) showing an increase in the size of the thrombus with an inner diameter of the conduit of 7 mm at the level of the thrombus (***b***). TriGUARD 3™ deflection filter device positioned in the aortic arch providing full coverage of all three major branches before the delivery of a 48 mm long XXL PTFE-covered Optimus-CVS® (***c***). Exit angiography showing no vascular lesion and good stent apposition to the vascular wall (***d***).

aortic arch under fluoroscopic guidance. The device was expanded and positioned, for the first attempt to accurately cover the ostia of the innominate, left common carotid, and subclavian arteries (Fig. 1c). The device was easily anchored by the device frame's circumferential apposition against the aortic arch.

The 7-Fr venous introducer was exchanged with a 12-Fr Flexor® Check-Flo® sheath (Cook Medical, USA) that was positioned more cranially to the lesion site. We hand-mounted a 48 mm long XXL PTFE-Covered Optimus-CVS® (AndraTec, Germany) on a 20 mm large/50 mm long AltoSa-XL™ single balloon catheter (AndraTec, Germany). The balloon-stent unit was unsheathed and the stent was implanted. The stent was re-dilated using a 20 mm large/40 mm long Atlas® Gold (Bard Peripheral Vascular, Inc., USA) non-compliant ultra-high-pressure angioplasty balloon catheter to reduce the stent waist. Exit angiography showed no vascular lesion and good stent apposition to the vascular wall (Fig. 1d). The invasive gradient was abolished. The TriGUARD-3 system was easily removed. Access hemostasis was obtained with manual compression. The overall procedure time was 55 minutes and the fluoroscopy time was 20 minutes. The patient had uneventful post-operative care and was discharged after two days. The daily oral aspirin therapy was maintained. There was no vascular-access complication. There were no clinical signs of neurological complications, acute kidney injury, coronary artery obstruction, or aortic valve-related dysfunction. Three months of follow-up showed no complications.

Discussion

We describe the first reported use of the TriGUARD-3 in a congenital heart patient with a high risk of paradoxical thrombo-embolism during a transvenous intervention. Thrombus migration from the Fontan circulation across the fenestration into the left atrium and then directly into the ascending aorta was considered very likely to occur during balloon-stent expansion inside the Fontan conduit with a calcified thrombus at the level of the fenestration. We sought that it would be more reasonable and safer to control that risk and prevent stroke with the TriGUARD-3 deflection device.

Major stroke has been reported in 3–6% of patients during the first 30 days after transcatheter aortic valve replacement procedures.[6] Several pathological mechanisms are involved in the development of peri-procedural ischaemic stroke transient or ischaemic attack with the majority being due to embolism of calcified or atheromatous particles.[6,7] Cerebral embolic protection devices are designed to mechanically reduce the incidence of procedural cerebral thromboembolic events during these procedures.[4,5,8] These currently available devices can either capture (Montage, Claret CE Pro, Embol-X) or deflect (Embrella, TriGuard-3) embolic material.[8] The TriGUARD-3 is a temporary, retrievable, single-use, and self-expanding cerebral embolic protection device mainly during transcatheter aortic valve replacements.[4,5] The device is CE-marked and approved for investigational use only in the United States. It has a 74 mm large × 98 mm long radiopaque Nitinol frame with a dome-shaped polymer mesh deflector (nominal pore size 115 × 145 µm). Compared to its previous generation, TriGUARD-3 provides a large filtration surface that self-stabilizes and conforms to aortic arch anatomies without cerebral artery engagement. The safe use of the TriGUARD-3 during transfemoral aortic valve replacement procedures was shown according to the findings of the REFLECT II trial Phase II including 345 patients.[4] However, it remains unclear whether it improves patient outcomes. The pre-specified primary superiority efficacy endpoint was not met and the trial failed to show a significant reduction of procedure-related cerebral injury.[4] This finding has been more recently debated by the positive findings of a prospective single-centre study including 117 patients.[5]

In this case, we found out that the device was easy to handle. The preparation, delivery, deployment, and retrieval were relatively simple and did not require excessive training. It did not lengthen or complicate the procedure or unreasonably increase the radiation exposure. The device has an additional port in case a 5Fr pigtail catheter had to be placed for procedural guidance and pressure monitoring, eliminating the need for another arterial access. The device did not require extensive operator attention and we think it provided stable protection of the three cerebral arteries throughout the entire procedure. The device is indeed self-positioning, but it needs to be placed carefully with the wire being kept abutted into the north wall of the aorta. In this case, all manipulations were done transvenously and the device stability was not conditioned. However, it has been reported that there is a risk of TriGUARD-3 interference with other arterially delivered devices such as the valve delivery system during transfemoral aortic valve replacement procedures.[4] It is also important to keep in mind that TriGUARD-3 is a deflecting device and in case of thrombus migration, lower body part embolic events can still occur. Therefore, we performed this procedure with an interventional neuroradiologist, in case a cerebral or peripheral thrombo-embolic event had to be dealt with. We did not perform a head MRI

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nor an assessment by a neurologist to identify silent strokes.[5,8] However, the goal of a cerebral protection device is to prevent clinically relevant intra-procedural strokes. Therefore, good clinical post-operative outcomes can support the efficacy of the device in this reported case.

Conclusion

Transcatheter stent expansion of obstructed or restrictive extracardiac conduits can be a challenging and risky procedure in the case of fenestrated conduits and central venous thrombus formations. Temporary cerebral embolic protection devices can help divert potential paradoxical emboli downstream and allow adequate cerebral blood perfusion.

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Author contribution. RH collected data, designed illustrative material, and took the lead in writing the entire manuscript. All authors discussed the results, and have read and approved the final version of the manuscript.

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Competing interests. None.

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