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Case Report and Review of the Literature

Case Report of a Heartmate II Outflow Graft Obstruction by Extrinsic Compression

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ABSTRACT

Left ventricular assist devices take an important place in the armamentarium of end-stage heart failure therapy. Outflow graft obstruction by extrinsic compression is a rare but possibly life-threatening complication of these types of devices. Pathophysiology is not completely elucidated but seems to be related to the common design of these devices, because of a porous Dacron graft surrounded within an impervious Teflon bend relief. In the absence of pump thrombosis, the pump replacement is not necessarily indicated, and only removal of the obstruction may be sufficient. Both surgical and interventional procedures are described. We reported a case successfully treated by angioplasty-stenting. We discussed the diagnosis, the pathophysiology, and the treatment of this specific complication of left ventricular assist devices. Finally, we open the discussion to the prevention of this complication.

Introduction

Left ventricular assist devices (LVAD) increase the survival of patients with end-stage heart failure, while improving their quality of life [1]. Outflow graft obstruction (OGO) is a rare complication, but increasingly reported in the literature [2, 3]. Due to its hemodynamic consequences, pump function and so patients' survival are compromised [3]. The main mechanism is the extrinsic compression by a "jelly" which accumulates between the graft and the bend relief [2]. With the increasing implantation of LVAD, it seems important to take an interest in it.

We reported the case of an OGO by extrinsic compression. Then, we discussed the pathophysiology, diagnostic and therapeutic modalities of this complication of LVAD. Finally, we open the discussion to the prevention.

Case Report

A 75-year-old man, with a HeartMate II ™ (Thoratec Corporation, California, U.S.A.) in destination therapy since 2015 for non-obstructive cardiomyopathy, presented with acute dyspnea evolving for 4 days due to left-sided heart failure.

Since the LVAD implantation, his medical history was marked by, first a transient stroke, then two episodes of upper gastrointestinal bleeding requiring red blood cells transfusion. Since this second bleeding event in 2019, aspirin was withdrawn, and his antithrombotic regimen was only warfarin. This was the first heart failure episode. The pump, set at 9,400 rpm since the implantation, never presented any alarm.

On admission, the blood pressure was 112/71 mmHg, mean of 84 mmHg. He was afebrile. The initial biological assessment showed the

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haemoglobin had dropped to 93 g/L, without reported bleeding but intravascular hemolysis with increasing serum bilirubin. The INR was 2.5 and no off-target variation was reported recently. Echocardiography showed left ventricular ejection fraction of 20% on a more dilated left ventricle (70 mm *versus* 64 mm previously) with increased moderate mitral regurgitation and stable mild aortic regurgitation. A ramp test from 8,800 to 10,600 rpm showed no change neither in left ventricular loading conditions nor valvular regurgitations. The pump parameters showed a slight decrease of consumption and flow rate. Computerized angio-tomography (CAT) showed the OGO by extrinsic compression on its proximal segment, within the bend relief (Figure 1a). The reviewing of a previous CAT, performed when the patient presented the transient stroke, showed this extrinsic substrate pre-existed and grown up to narrow the graft lumen more than 50% (Figure 1b).

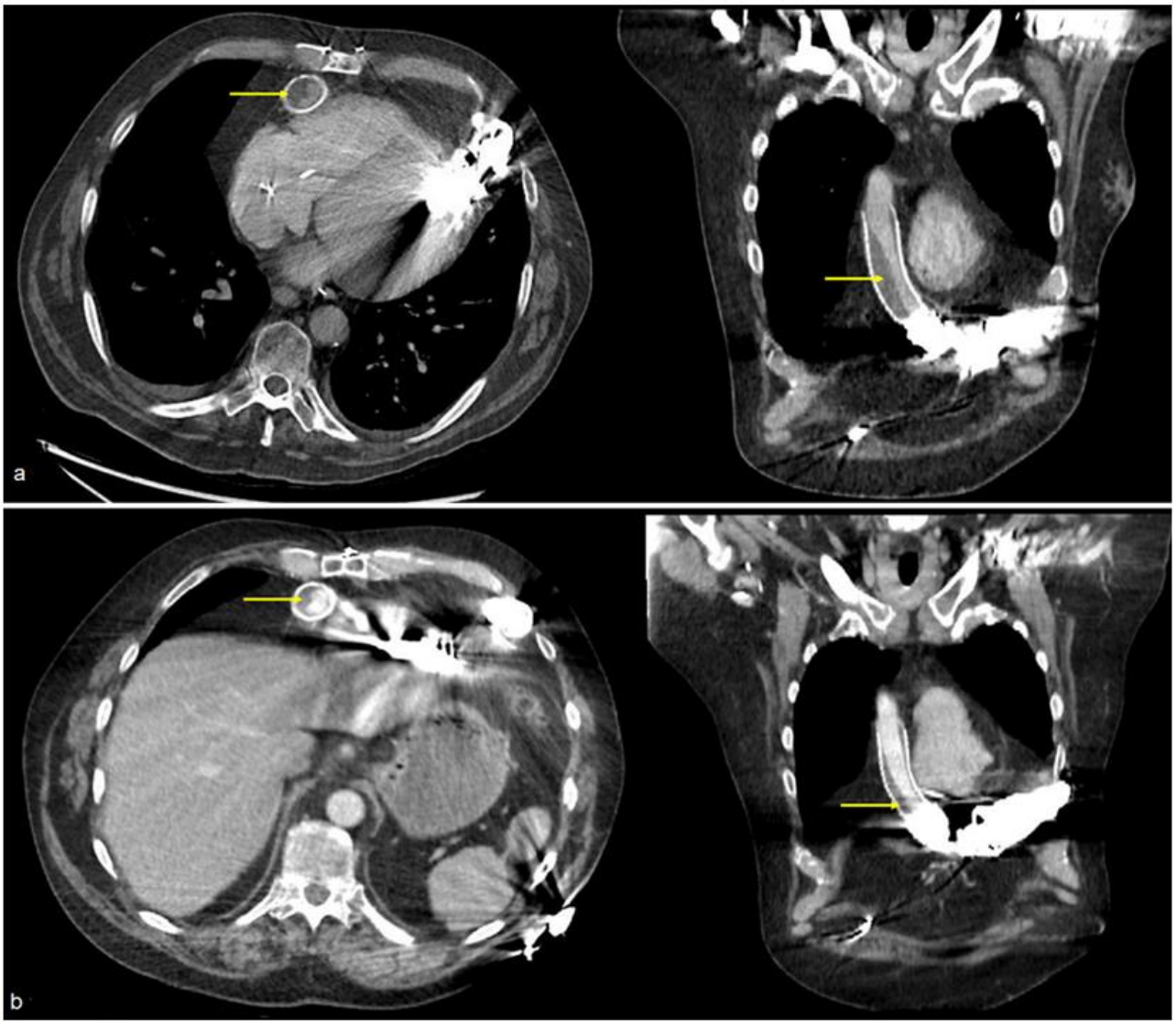

Figure 1: Outflow graft obstruction by extrinsic compression. **a)** Computerized angio-tomography performed for dysfunction pump assessment **b)** showed the extrinsic compression of the outflow graft by an increasing substrate (yellow arrow) within the bend relief, pre-existing for 4 years.

We decided on an interventional "graftoplasty". The procedure was performed under local anaesthesia with a percutaneous femoral approach without extracorporeal circulatory support. A 57 mm AndraStent® (Andramed Gmbh, Germany) endoprosthesis crimped on a 16 mm balloon was deployed in the graft. After angiographic control, we observed a "displacement" of the narrowing downstream. A second same endoprosthesis removed the obstruction definitively (Figure 2). During the procedure, the Abbott engineers were asked to reduce the rotation speed to 6,000 rpm (an inaccessible mode for physicians) to facilitate wires navigation and endoprosthesis deployment closer to the pump. The fluoroscopy time was 15 minutes.

During hospital stay, the patient did not present any event. Hemolysis decreased with serum bilirubin reached to normal range. At 1 month, the patient reported dyspnea only on exertion, stage I-II/IV of NYHA classification, with the haemoglobin spontaneously increased to 99 g/L. Echocardiography showed the mitral regurgitation reduced, and LVAD parameters were stable at 9,400 rpm: flow rate 5.5 L/min, consumption 5.7 watts, pulsatility index 5. At 3 months, CAT confirmed good patency of the stented graft (Figure 3).

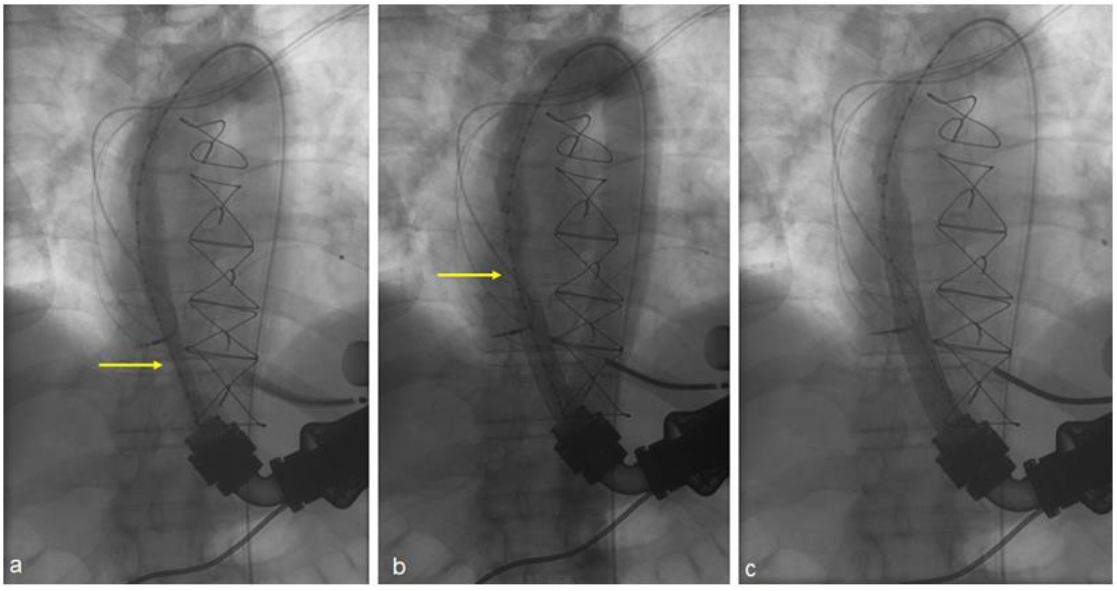

Figure 2: Outflow graft stenting procedure. **a)** The outflow graft obstruction (yellow arrow) was proximal. **b)** The stent ought to be placed closer to the pump. If necessary, the pump flow may be decreased with engineers' help to minimize the displacement of the stent during its deployment. The first stent removed up the « jelly » as « the toothpaste that comes out of its tube », displacing the narrowing. **c)** A second stent ended with a successful procedure.

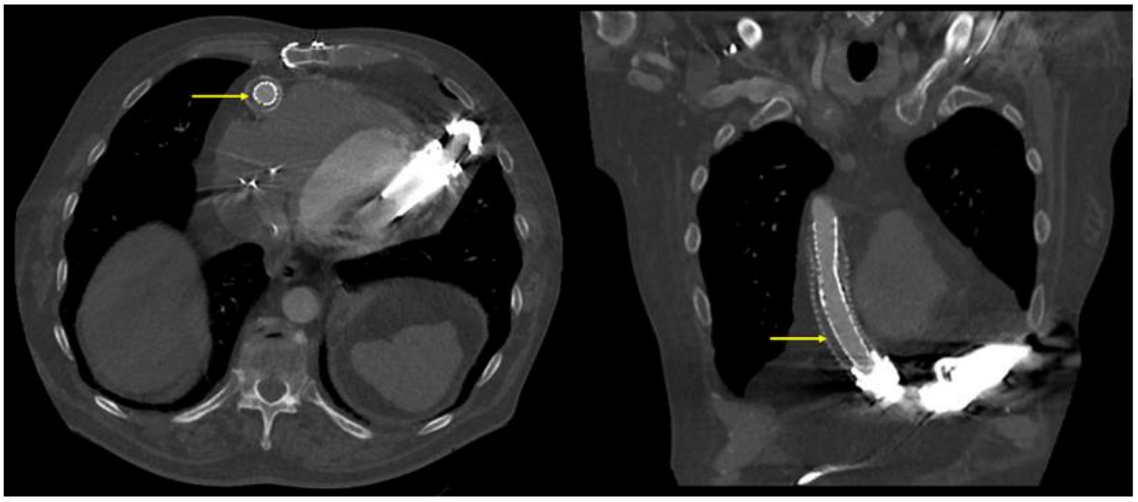

Figure 3: Computerized angio-tomography at 3 months. Although the « jelly » (yellow arrow) was not surgically removed, the stented graft no longer had stenosis.

Discussion

Heart failure is a major Public Health problem and LVAD are part of the treatment for end-staged patients, increasing their survival and quality of life while waiting for transplantation or in destination therapy both. Like all implantable devices, specific follow-up by a dedicated team is needed to manage specific complications [1]. Among these complications, some concern the outflow graft [2, 3]. As a reminder, the graft is a Dacron tube anastomosed to the thoracic aorta. It is sheathed over its first 7 centimeters by the bend relief, a Teflon tube secondarily connected to the pump, whose function is to prevent kinking, adhesions, and abrasions. Four mechanisms of OGO are described: stenosis at the aortic anastomosis site, kinking/twist, extrinsic compression, and intraluminal thrombosis [3].

Extrinsic compression receives particular attention in the literature [2, 3]. Indeed, it is the most reported mechanism of OGO. In their series, Tranckle *et al.* showed this was the only mechanism in their series of 15 cases, out of 251 with LVAD. This phenomenon is not only described for the HeartMate II ™, but also concerns other LVAD, testifying a probable relation with the common design of these devices [3, 4]. In fact, the high prevalence of reported case in Heart Mate II ™ may be explained because it is the most implanted LVAD worldwide. When LVAD was explanted, some authors observed a thrombofibrotic "jelly" between the graft and the bend relief [2]. Pathophysiology is not elucidated, but the macroscopic analysis of the graft did not find any tear. This phenomenon seems to occur between 6 months and 5 years post-implantation [2, 3]. The risk factors are non-ischaemic heart diseases and age [3]. Due to the rheological modifications induced by the change in geometry of the graft, intraluminal thrombosis may appear and extend to the pump [3]. Also, maybe the stroke which occurred could be related to

this lesion, even if it did not seem hemodynamically significant. At this time, we found only a slight underdosed vitamin K antagonist (one only INR of 1.9).

If the lesion involves exclusively the graft, the parameters of the pump may fail or warn of a drop in flow or consumption, and the waveform analysis can be helpful [3, 4]. The diagnosis is based on a series of arguments in which multimodal imaging plays a central role, with CAT as the gold standard [3]. However, the distinction between extrinsic compression and intraluminal thrombosis can sometimes be difficult and intravascular ultrasound can be used [5]. The symptomatology being poor, its incidence is probably underestimated, since OGO was searched only for patients who have a specific imaging [3].

Unlike pump thrombosis, where surgical replacement of devices or heart transplantation is necessary, the removal of the obstruction on the graft is sufficient for isolated extrinsic compression. The procedure can be performed surgically, and the intervention consists of splitting the bend relief to extract the "jelly" [3]. This procedure does not require extracorporeal circulation but exposes the patient to the risks of a redo procedure. Cases of minimally invasive approaches by anterior thoracotomy have been described [6]. More recently, angioplasty-stenting of the graft has given results with satisfactory long-term survival and seems to be favoured in the literature [7]. In our elderly patient with LVAD for destination therapy, we preferred this endovascular approach. Some authors advise the use of intravascular ultrasound during angioplasty to confirm the correct position of the stent [7]. The main risk is the failure due to displacement of the stent during deployment, which should be placed as close to the pump as possible. To facilitate this, engineers may be asked to provide the pump flow rate below the usual setting range.

Finally, even if the mechanism is not completely elucidated, it seems strongly related to the design of the devices, because of a porous Dacron graft within an impervious Teflon bend relief. This supposition could be accessible to a change by manufacturers. Meanwhile, some authors suggested a "handmade" fenestration of the bend relief to allow the jelly spread out [8].

Conclusion

OGO by extrinsic compression is a rare complication, but potentially compromising LVAD function. More and more cases are reported. This seems to occur during the mid-term post-implantation period. An interventional "graftoplasty" seems to be secure and favoured rather than the surgical removal, while the pump replacement may not be needed. Pathophysiology seems related to the common design of these devices. At a time when LVAD are increasingly implanted, this potentially avoidable complication deserves our interest to optimize the outcomes, and the preventive fenestration of the bend relief might be suggested. This case is not the first, but probably will not be the last. A multicenter register with follow-up by CAT could be helpful to screen the real incidence of this complication.

Conflicts of Interest

None.

Funding

None.

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