Endograft Collapse after Standard Endovascular Aneurysm Repair Implantation: A Report of Two Cases

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Endovascular aneurysm repair (EVAR) has emerged as the preferred treatment for abdominal aortic aneurysm (AAA), offering advantages such as reduced mortality, faster recovery, and fewer complications compared to open repair (OR). However, concerns persist regarding long-term durability, and the need for reinterventions. Among all reported EVAR complications, endograft collapse poses a significant challenge, particularly when presenting with acute lower limb ischemia (ALI).
The aim of this report is to describe two cases of non-dissection-related abdominal endograft collapse manifesting as ALI, requiring urgent intervention, emergent conversion to OR and complete endograft removal.

Keywords: abdominal aortic aneurysm; acute limb ischemia; endograft collapse; endograft infolding; endovascular repair

Introduction

Endovascular aneurysm repair (EVAR) has become a valid alternative to open repair (OR) for the treatment of abdominal aortic aneurysm (AAA), with over >75% of AAA repairs performed endovascularly [1, 2, 3]. Undoubtedly, EVAR is associated with lower 30-day mortality and morbidity, faster discharge, and fewer complications than OR [4]. Some concerns remain regarding durability, the necessity for long-term follow-up and reinterventions [1, 4, 5, 6]. Endograft complications such as endoleaks, migrations, fractures and infections have been reported [7]. Among these, endograft collapse is an uncommon and rare event, mostly related to aortic dissection. In previous literature, only a few cases of acute (within 30 days) or late non-dissection-related endograft collapseoccurring after infrarenal EVAR have been documented [8, 9].

The primary aim of this study was to report two cases of non-dissection-related abdominal endograft collapse presenting with acute lower limb ischemia (ALI) and requiring urgent treatment. Both patients provided written informed consent for the report of case details and imaging studies.

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Case Report

Case One

Patient information: A 75-year-old man was admitted to our Emergency Department due to the sudden onset of acute renal failure and blue toe syndrome. His medical history included arterial hypertension, dyslipidemia, and a previous right internal carotid artery stenting implantation procedure, without a family history of cardiovascular disease or genetic disorders.
Clinical findings: Physical examination revealed a pulsating mass in the epigastric-mesogastric region. Femoral and popliteal pulses were detected bilaterally, whereas tibial pulses were not. Blood samples results, including complete blood count and coagulation were normal, except for increased creatinine (7.6 mg/dL) and blood urea nitrogen (BUN; 80 mg/dL).
Diagnostic assessment: Duplex ultrasound (DUS) revealed an infrarenal AAA with a maximum transverse diameter of 55 mm that was associated with severe left renal artery stenosis (Fig. 1). Diagnosis was confirmed using non-contrast computed tomography (CT). As a result of the development of an acute renal failure secondary to cholesterol embolism, the patient was treated with rehydration, bicarbonates and corticosteroids, resulting in decreased creatinine (2.7 mg/dL) and BUN (35 mg/dL) values.
Treatment interventions: Electively, the patients underwent a combined endovascular procedure of renal artery stenting plus EVAR.

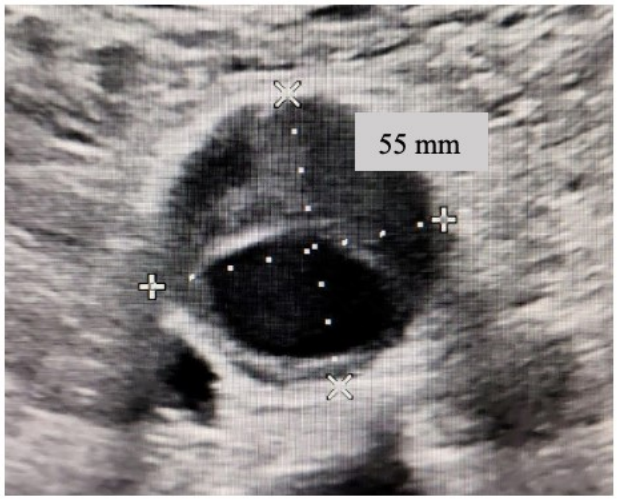

Fig. 1. Duplex ultrasound (DUS) revealed an infrarenal abdominal aortic aneurysm (AAA) with a maximum transverse diameter of 55 mm.

Under general anesthesia, via bilateral percutaneous access, a self-expanding 7 × 29 mm stent (Express RX; Boston Scientific, Marlborough, MA, USA) was deployed at the origin of the left renal artery (Fig. 2).

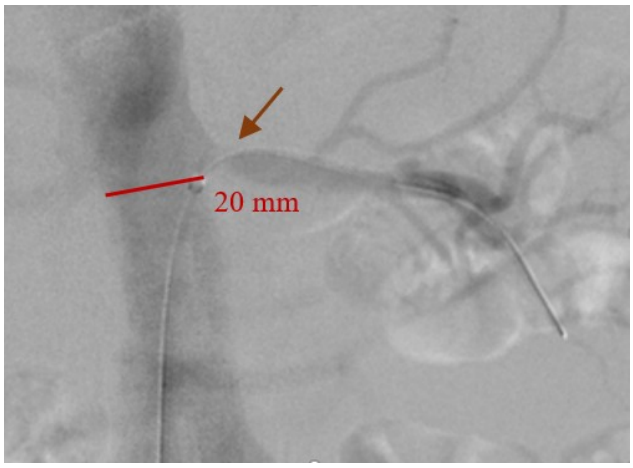

Fig. 2. Intraoperative super-selective digital subtraction angiography showing the proximal aortic neck diameter and the left renal artery stenosis (red arrow).

Subsequently, through the right common femoral artery, an AFX II 28–120/16–40 mm endoprosthesis (Endologix Inc., Irvine, CA, USA) was implanted. Procedure was carried out without any complication, and technical success was fully achieved without evidence of an endoleak, despite the presence of a conical proximal aortic neck (from 20 mm diameter at the lowest renal artery level to 25 mm in 15 mm in length). The post-operative course was uneventful, and the patient was discharged on the fourth postoperative day with decreased creatinine values (1.5 mg/dL).

Scheduled thirty-day CT-angiography (CTA) showed a complex type Ib and type II endoleak, resulting in a sudden increase in sac diameter (61 mm; Fig. 3).

Consequently, the patient underwent a further endovascular procedure consisting of transealing [10] type II endoleak exclusion via coil implantation (four detachable coils 16 × 60 cm, two 18 × 57 cm, and one 20 × 60 cm; Ruby Coil, Penumbra Inc., Alameda, CA, USA), and an iliac limb relining with balloon-expandable stent-graft 11 × 39 mm (VBX; WL Gore & Associates, Flagstaff, AZ, USA) implantation. Technical result was successfully achieved, and the patient was discharged on postoperative day II.

Two weeks after the reintervention, the patient was readmitted to our emergency department because of the acute onset of bilateral gluteal claudication with a free walking distance of less than 10 meters. On physical examination, the femoral and distal pulses were absent. CTA showed complete proximal collapse of the unibody device, aortic occlusion and no endoleaks (Fig. 4).

Therefore, a surgical intervention was performed in an emergency setting. Under general anesthesia, through bilateral subcostal incisions, complete endograft removal was followed by aorto-biiliac anatomical reconstruction with a 20 × 10 mm Dacron graft (Intergard Knitted; Getinge, Göteborg, Sweden). Intraoperative culture examination was negative.

Follow-up and outcomes: The post-operative course was complicated by transient acute renal damage, which spontaneously resolved on the seventh postoperative day. The patient was discharged with life-long antiplatelet (acetylsalicylic acid 100 mg per day) therapy and with an indication for periodic follow-up with DUS. At three years follow-up, the patient was alive in the absence of further complications (Fig. 5).

Case Two

Patient information: A 78-year-old man was admitted to our Emergency Department with acute onset of lower limb pain and hypothermia associated with IIA Rutherford acute limb ischemia. The patient's cardiovascular risk factors included hypertension, hyperlipidemia and remote smoking and family history was negative for abdominal aortic aneurysm. Thirty days earlier, at another hospital, the patient had undergone EVAR for a symptomatic 50 mm AAA (Fig. 6).

The intervention was performed via percutaneous access, implanting an Excluder endograft (28-14-140 mm main body plus 18–10 mm left leg limb and 12–140 mm right leg limb; WL Gore & Associates, Flagstaff, AZ, USA).

Clinical findings: On physical examination, no palpable pulses were detected in both legs. Blood tests revealed mild anemia (hemoglobin 9.0 g/dL), leukocytosis (13.1 × 10^3/μL) with neutrophilia (11.88 × 10^3/μL), elevated serum C-reactive protein level (27.47 mg/L) and procalcitonin (0.15 ng/mL) values.

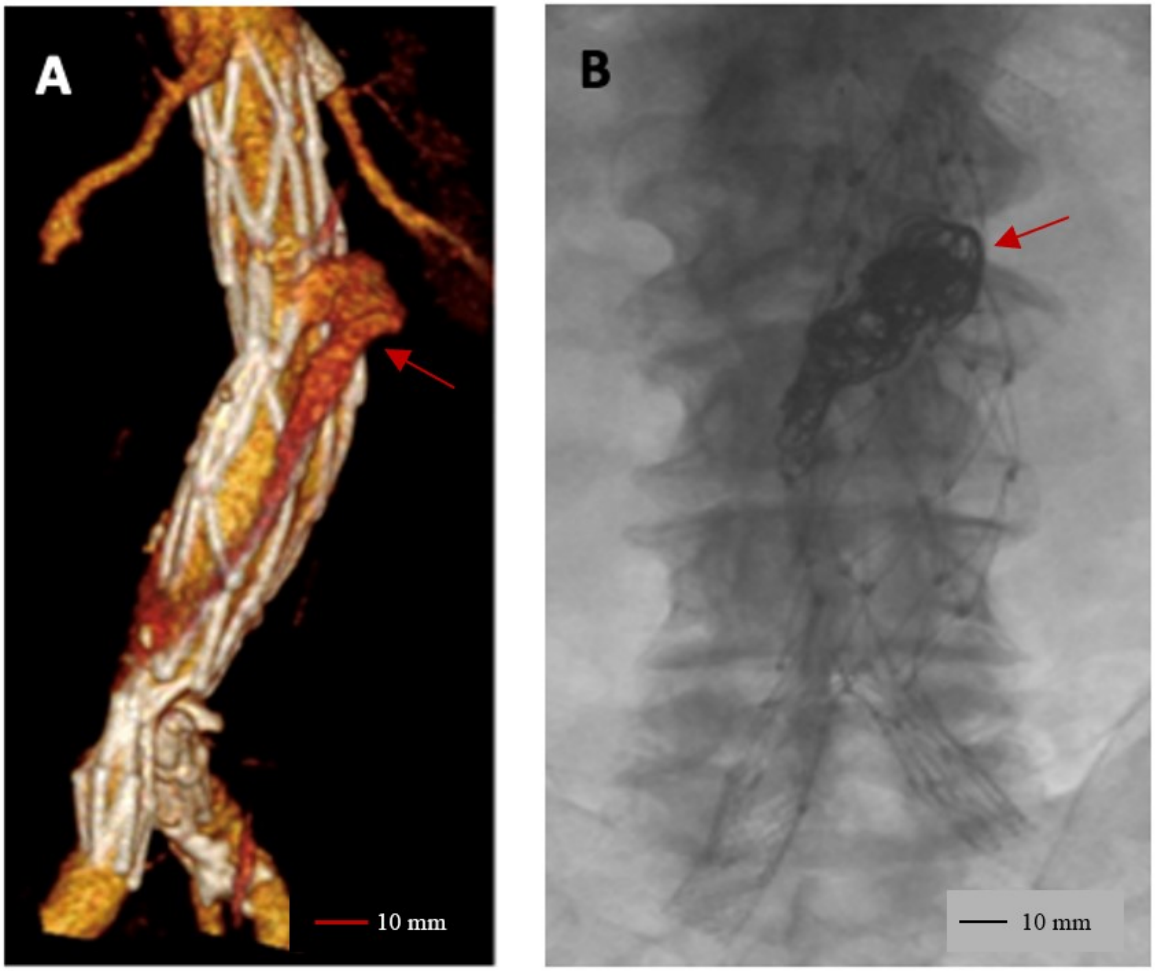

Fig. 3. Postoperative images. Three-dimensional volume rendering CT-angiography (CTA) showing the concomitant presence of a type Ib and a type II endoleak (red arrow) after endovascular aneurysm repair (EVAR) (A); Direct fluoroscopy visualization after correction of EL type II and type Ib showing the present of the embolization coils (red arrow) in the excluded aneurysmal l sac and the normal endograft expansion (B).

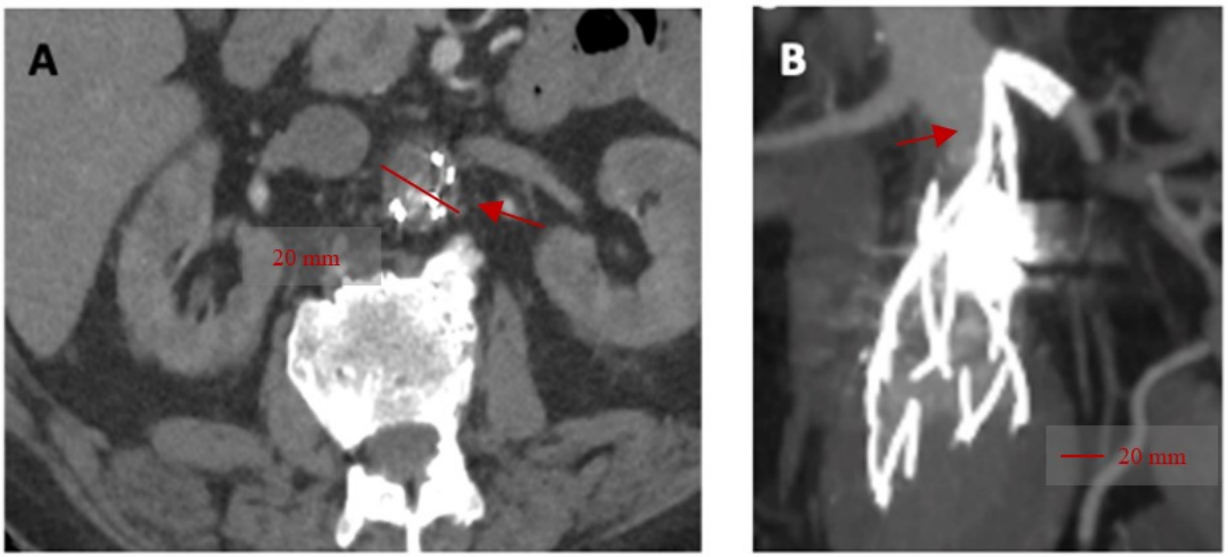

Fig. 4. CTA showing the endograft collapse (red arrows) in axial (A) and coronal (B) projections.

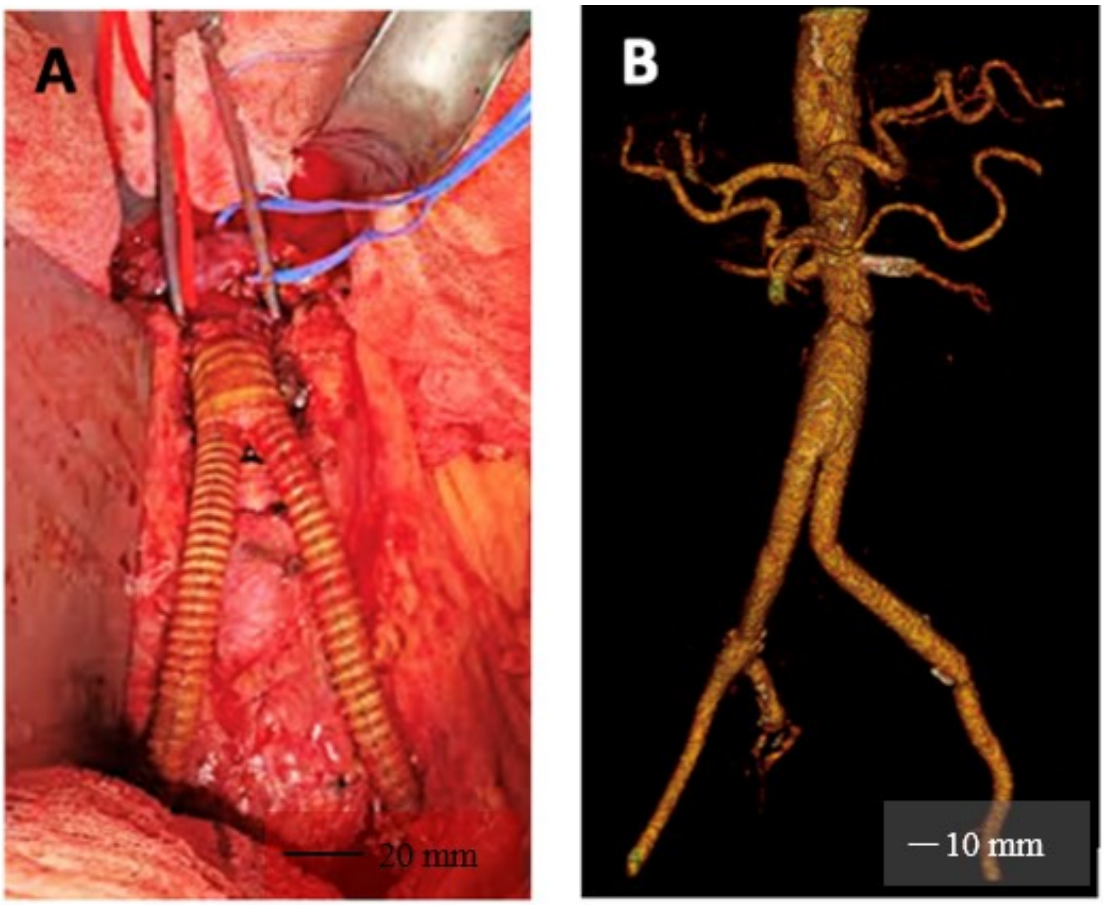

Fig. 5. Intraoperative and postoperative images. *In situ* reconstruction with a Dacron graft (A); Three-dimensional volume rendering CTA showing aorto-biiliac *in situ* reconstruction after endograft explantation (B).

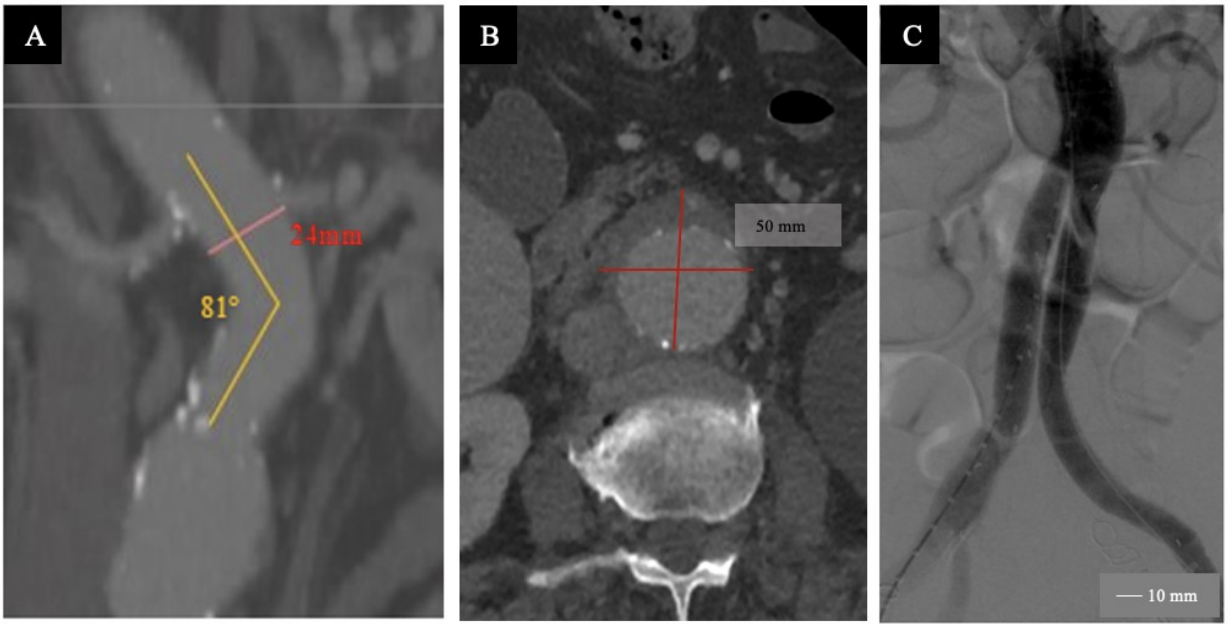

Fig. 6. Preoperative and intraoperative images. Preoperative CTA coronal view of the aneurysm showing the actual aortic neck dimension and its angulation (A); CTA axial view showing the aneurysm (B); Intraoperative digital subtraction angiography showing the complete aneurysm exclusion without evidence of endoleak (C)**.**

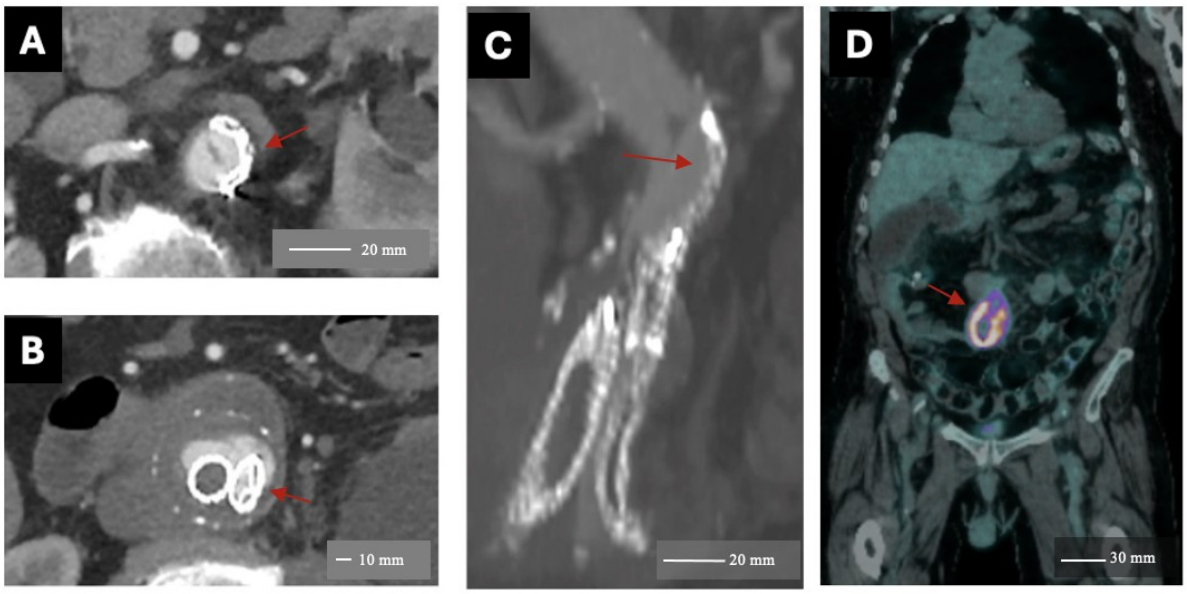

Fig. 7. Postoperative images. Axial (A,B) and multiplanar reconstruction (C) CTA images showing complete endograft collapse (red arrows) associated with periaortic fluid collection; (D) Positron emission tomography (PET) images showing a high and diffused 18F-fluorodeoxiglucose ([18F]FDG) uptake in the aortic wall (red arrow).

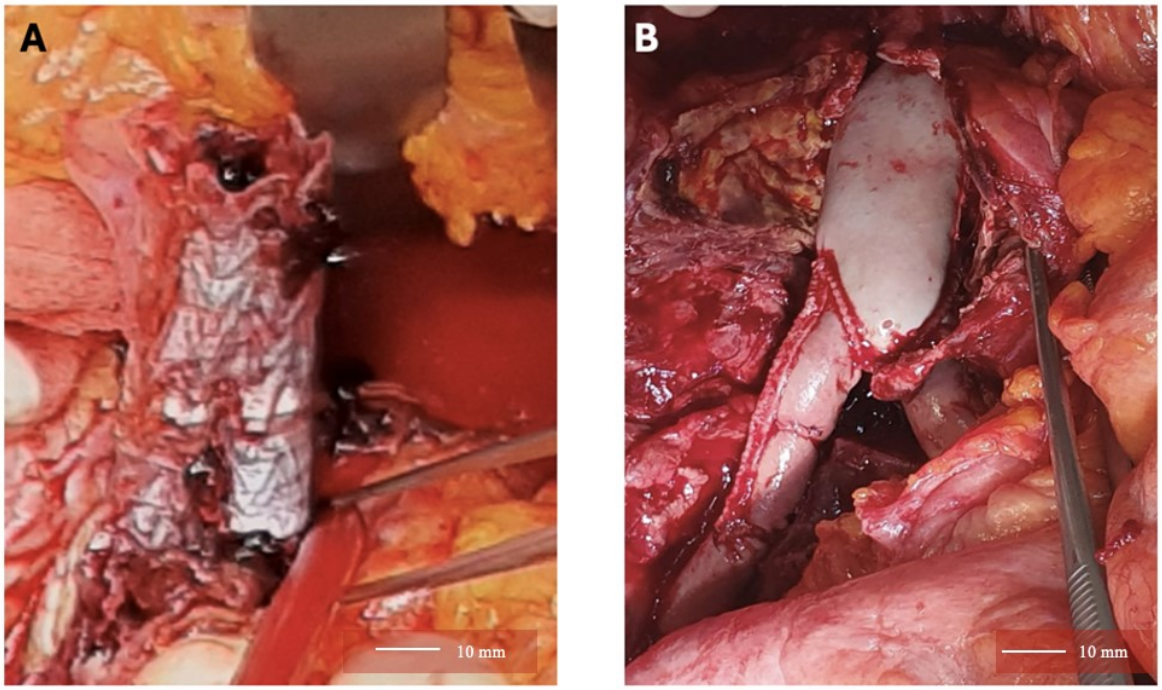

Fig. 8. Intraoperative images. (A) Macroscopic sings of endograft infection. (B) *In situ* reconstruction with a porcine pericardium graft.

Diagnostic assessment: Urgent CTA performed at Hospital admission showed a complete endograft collapse associated with periaortic fluid collection, and subsequent white blood cell scintigraphy and positron emission tomography (PET) with 18F-fluorodeoxyglucose ([18F] FDG) confirmed the early endograft infection diagnosis according to MAGIC criteria (Fig. 7) [11, 12].

Treatment interventions: Subsequently, the patient underwent an urgent endograft explantation. Under general anesthesia, through bilateral subcostal incisions, complete endograft removal was performed via *in situ* aorto-right-iliac-left-femoral reconstruction with a porcine pericardium graft 18 × 9 mm (No-React BioAortic; BioIntegral, Mississauga, ON, Canada; Fig. 8).

Intraoperative sample material culture was positive for *S. chromogenes*. The infection was treated with the administration of antibiotic therapy including ceftazidime/avibactam (2 g/0.5 g × 3/die) and teicoplanin (800 mg/die) for one month. The postoperative course was uneventful without complications.

Follow-up and outcomes: The patient was discharged on the 30th postoperative day with an indication to continue the same antibiotic therapy for another 6 months and with life-long antiplatelet therapy (acetylsalicylic acid 100 mg per day). At one-year follow-up, the patient was in good clinical condition with no laboratory or radiologic signs of persistent or recurrent infection.

Discussion

EVAR has become an established alternative to OR for infrarenal AAA treatment, primarily due to its lower 30-day mortality and morbidity rates, along with shorter hospital stays compared to OR [1, 2, 3]. However, concerns still exist about its long-term durability and effectiveness. Among EVAR specific complications such as endoleafks, migrations, and fractures, endografts collapses are an uncommon and lesser-known occurrence, mostly related to dissections [7, 8, 9].

The majority of endograft collapses (also called infolding) occur after thoracic aortic endograft implantation, rather than after EVAR. The causes of aortic endograft collapse remain somewhat uncertain, various anatomical, hemodynamic, and technical factors have been postulated as potential triggers for this uncommon occurrence [13].

Apart from those collapses related to aortic dissection in which the physiopathology is quite intuitive (a dissection extending into the aorta may be associated with a displacement of the graft from the false lumen compressing the true lumen), other causes responsible for graft collapse after EVAR remain unclear [9].

To the best of our knowledge, only four cases of non-dissection-related endograft collapses [9] have been reported, associated with different anatomical and procedural findings. Among the latter, an excessive oversizing in a reverse-tapered aortic neck or a severe aortoiliac tortuosity, associated with an elongated aortic neck, are factors that could contribute to enhancing the tension on the weaker aortic curve, resulting in poor adherence on the affected side and promoting collapse. Along with a bird beak following implantation, these features have all been postulated as potential risk factors for infolding, especially in thoracic cases [14].

The two cases reported in this study seem to confirm this hypothesis only partially. In the first case, the reintervention performed to exclude concurrent type Ib and IIb endoleaks may have initiated the unibody endograft dislodgment, resulting in malposition to the aortic wall. This compromised sealing, coupled with the endograft low radial force potentially led to collapse through a mechanism similar to that observed in dissection-related collapses.

Conversely, in the second reported patient, endograft collapse could have been triggered by severe aortic neck angulation (>80°). This angulation is probably associated with the slight oversizing of 8.4%, lower than the 10% suggested in the Instructions for Use, which may have contributed to the collapse. Notably, it could be speculated that a concurrent high-grade infection may have played a role in infolding through undiscovered factors, such as increased blood viscosity.

Regardless of its origin, endograft infolding requires immediate surgical or endovascular treatment. Although several endovascular procedures like balloon dilation, covered stent, or proximal aortic cuff deployment have been proposed as feasible treatment options, surgical repair via endograft removal seems to be the best available option [9, 15]. In our experience, both cases were successfully treated by surgical conversion and *in situ* aortic reconstruction due to the sudden onset of collapse associated with lower limb ischemia. Moreover, one patient had already undergone multiple endovascular procedures, and the other presented with associated endograft infection, strongly discouraging the use of a further endovascular procedure.

Conclusions

This case report shows two rare cases of endograft collapse after EVAR, unrelated to dissection, an extremely unlikely event. Understanding the predictive factors associated with collapse is crucial for enhancing clinical outcomes and patient safety in EVAR procedures. Careful preoperative case planning, with attention to potential anatomical complexities that may predict an endoleak, such as the presence of conical necks, very short necks, or neck angulation >80 degrees, could reduce the incidence of collapse. Additionally, studying the structural characteristics of the endograft (radial force, columnar strength) can guide the operator in selecting the best designed device for the patient's anatomy. Therefore, establishing a prospective multicenter registry could prove beneficial for assessing the actual incidence of this rare complication.

Availability of Data and Materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Author Contributions

MT and PS designed the study. MP, NS, MT, PS treated the patients and acquired the data. GC, AS, LR treated the patients and analysed the data. MP and PS wrote the manuscript. All authors revised the manuscript critically for important intellectual content. All authors read and ap-

proved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Helsinki Declaration, and both patients provided written informed consent for the report of their case details and imaging studies. The study was exempted from ethical approval by Sant'Andrea Hospital of Rome.

Acknowledgment

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Conflict of Interest

The authors declare no conflict of interest. Maurizio Taurino is serving as one of the Guest Editors of this journal. Pasqualino Sirignano is serving as one of the Editorial Board Members and Guest Editors of this journal. We declare that they had no involvement in the peer review of this article and had no access to information regarding its peer review.

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