



Non-Anastomotic Rupture of the Vascular Prosthesis Graft Detected Using Non-Obstructive Aortic Angioscopy: A Case Report

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Abstract

Non-anastomotic rupture of a vascular prosthesis graft in the thoracic region is extremely rare and difficult to diagnose. Non-obstructive general angioscopy can help monitor the aortic intima and detect the locations of abnormal findings, while aortic angioscopy can detect vulnerable plaques in the aorta, which are difficult to visualize using conventional diagnostic methods. Herein, we report the case of a patient with non-anastomotic rupture of a vascular prosthesis graft diagnosed using non-obstructive aortic angioscopy. An 85-year-old man, who had undergone total arch replacement 5 years prior presented to our institution with chest pain. Based on contrast-enhanced Computed Tomography (CT) and aortic angiography findings, we suspected extravasation of the thoracic vascular graft. Assessment of the vascular prosthesis graft in the ascending aorta using aortic angioscopy revealed a red vascular graft defect. Non-anastomotic rupture of the vascular prosthesis graft was diagnosed using aortic angioscopy. The patient underwent two debranching thoracic endovascular aortic repair (Zone 0) with right subclavian artery-left common carotid artery-left subclavian artery bypass. Completion angiography revealed disappearance of the extravasation from the graft rupture site, patent grafted vessels with flow, and no endoleak. Follow-up CT at 6 months postoperatively showed no extravasation. To our knowledge, this is the first report of non-anastomotic rupture of a vascular prosthesis graft detected using non-obstructive aortic angioscopy. Aortic angioscopy can help establish a definitive diagnosis in patients with disruption of the vascular prosthesis graft.

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Introduction

Disruption of vascular prosthesis grafts can be divided into two major categories: anastomotic and non-anastomotic failures. To date, most reports have focused on anastomotic failures associated with infections [1]. Non-anastomotic graft rupture in contemporary vascular prostheses that is caused by mechanical stress in areas such as the groin and subclavian regions has scarcely been reported [2]. Furthermore, it is extremely rare in a thoracic lesion, which makes it difficult to detect the site of disruption, especially during an emergency.

Although conventional imaging modalities, such as Computed Tomography (CT), Magnetic Resonance Imaging (MRI), and Transesophageal Echocardiography (TEE) have been used to diagnose aortic diseases, the ability to detect millimeter-sized plaques is limited because of the relatively poor spatial resolution [3,4]. In addition, very few modalities have the capability to completely detect intimal injury or vulnerable plaques of the aorta *in-vivo*.

Non-obstructive general angioscopy is known to help monitor the aortic intima and locations of abnormal findings in patients with a shaggy aorta and chronic type B aortic dissection [5]. Aortic angioscopy can also aid in detecting vulnerable plaques in the aorta [5], which are difficult to visualize using conventional diagnostic methods. Herein, we have reported the case of a patient with non-anastomotic rupture of a vascular prosthesis graft that was diagnosed using non-obstructive aortic angioscopy.

Case Presentation

An 85-year-old man with a history of total aortic arch replacement performed using a 26-mm; four-branched Triplex graft (Vascutek Terumo, Tokyo, Japan) for an aortic arch aneurysm 5 years back was admitted to the emergency department with chest pain. Blood investigations revealed anemia (Hb 9.4 mg/dl), elevated inflammatory markers (CRP 5.5 mg/dl), near normal renal

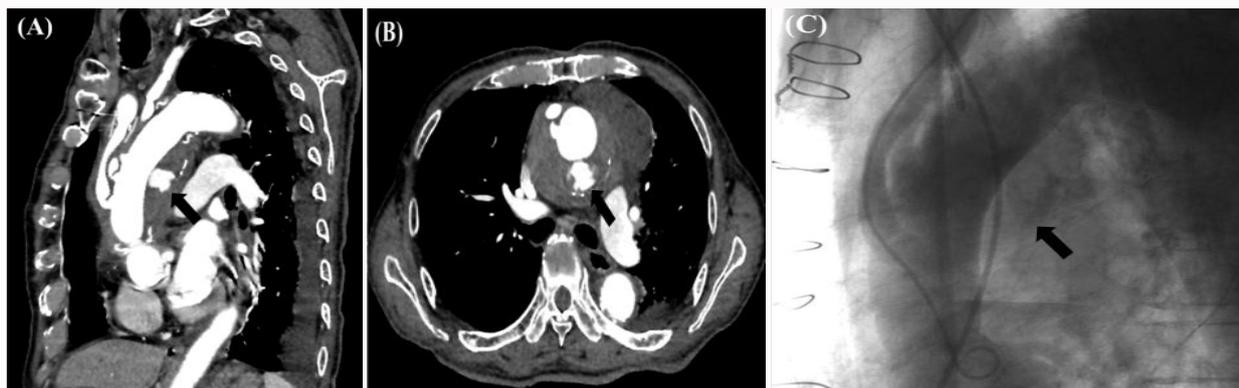


Figure 1: Computed tomography findings.

A) Sagittal view shows an intramediastinal hematoma around the vascular graft in the ascending aorta and extravasation on the dorsal side of the center of the vascular graft (black arrow).

B) Axial view shows an intramediastinal hematoma around the vascular graft in the ascending aorta and extravasation on the dorsal side of the center of the vascular graft (black arrow).

C) Preoperative angiography shows extravasation from the dorsal center of the vascular graft.

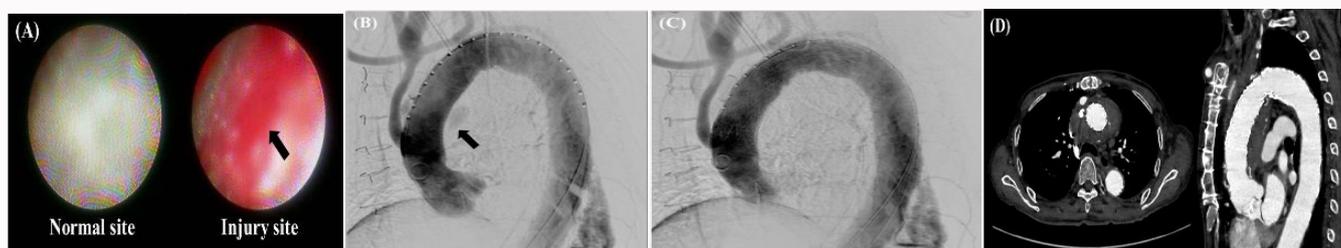


Figure 2: Aortic angiography and postoperative computed tomography findings.

A) Aortic angioscopy shows a red artificial blood vessel defect that matches the extravasation observed on angiography at the level of the contralateral left common carotid artery branching (black arrow).

B) Pre-deployment angiography shows extravasation.

C) Post-deployment angiography shows no extravasation.

D) Postoperative computed tomography shows no extravasation.

function, and an elevated total bilirubin level (total bilirubin 5.6 mg/dl, direct bilirubin 2.9 mg/dl). Contrast-enhanced CT revealed an intramediastinal hematoma around the vascular graft of the ascending aorta and extravasation on the dorsal side of the vascular graft (Figure 1A, 1B). Emergency angiography performed to explore the rupture site and vascular graft revealed that the extravasation had occurred from the vascular graft. We suspected a non-anastomotic rupture of the thoracic vascular graft (Figure 1C). However, the possibility of an anastomotic rupture at the various anastomotic sites could not be excluded. Therefore, aortic angioscopy was planned to identify and confirm the site of rupture in the vascular graft.

The operation was performed under general anesthesia in a hybrid operating room with an attached image guided interventional suite. The patient's hemodynamic parameters were stable. Assessment of the vascular graft in the ascending aorta using aortic angioscopy revealed a red vascular graft defect (Figure 2A) that corresponded to the site of angiographic extravasation at the level of the prosthetic Left Common Carotid Artery (LCCA) branch on the contralateral side (Figure 2B). We confirmed graft rupture and performed a two debranching thoracic endovascular aortic repair (zone 0). The LCCA and Left Subclavian Artery (LSCA) were revascularized *via* right subclavian artery-LCCA-LSCA bypass using an 8-mm T-shaped ring-enforced polytetrafluoroethylene graft. A cTAG stent-graft (34 mm × 150 mm; W.L. Gore and Associates, Flagstaff, AZ) was

deployed immediately distal to the first branch from the graft (the brachiocephalic artery) *via* a femoral approach. Finally, embolization at the LSCA was performed using a vascular plug (12 mm), and the proximal LCCA of the bypass anastomosis was closed using a Hem-o-lok XL ligation system (Research Triangle Park, NC, USA). Completion angiography revealed disappearance of the extravasation from the graft rupture site, patent grafted vessels with flow, and no endoleak (Figure 2C).

On postoperative day one, the patient developed worsening jaundice and had elevated bilirubin levels. Imaging findings indicated cholecystitis; therefore, the patient underwent cholecystectomy on the same day. The blood cultures collected at the time of admission were negative. Follow-up CT at 6 months postoperatively showed no extravasation (Figure 2D). This report was approved by the Institutional Review Board, and informed consent was obtained from the patient for the publication of this case report and accompanying images.

Discussion

Disruption of the vascular prosthesis graft has been reported in some patients, and most reports demonstrate rupture of the anastomoses with or without infection [1]. In this case, blood test at the time of admission showed elevated inflammatory markers; therefore, infection could not be ruled out. Additionally, the possibility of

anastomotic rupture could not be completely ruled out because the bleeding site could not be identified on CT. Intrinsic rupture of the Dacron graft occurs in 0.5% to 3.0% of cases [4] in the subclavian and inguinal regions with high graft mobility with joint flexion and mechanical damage, but not in the thoracic region. The most common cause of a vascular prosthesis graft rupture in the thoracic region is infection, but there are reports of physical damage due to aging, suturing during previous surgery, calcification of the wrapped aorta, and contact with the ribs [6-9]. In this case, the rupture site of the vascular prosthetic graft was on the dorsal side of the prosthetic graft, and aortic wrapping had not been performed in the previous surgery. Thus, physical damage as described above was unlikely.

With respect to infection, there were preoperative findings of cholecystitis, but the blood culture was negative, and we could not prove a causal relationship between the rupture of the vascular prosthesis graft and the infection. Various grafts, such as woven and knitted grafts have been developed with the current third-generation Dacron graft (Triplex) having a three-layer structure. The inner layer is a standard uncoated woven Dacron graft (DuPont, Wilmington, DE, USA), whereas the outer layer is a standard expanded polytetrafluoroethylene graft. Both layers are fused together using the central layer that is a self-sealing elastomeric membrane [10], which contributes to the reduction of pericardial effusion. There have been reports on perioperative reduction in pericardial effusion and graft durability [3,10]. The literature search retrieved no case reports of disruption of the vascular prosthesis graft with the Triplex used in this study.

Although the first-line treatment for rupture of vascular prosthesis graft is surgical closure of the rupture site, open repair for non-anastomotic rupture of the vascular graft is expected to have a high perioperative mortality, especially in the thoracic aorta. Our patient was considered to have a higher risk of mortality and morbidity after open repair due to age and frailty. In contrast, endovascular stent grafting for aneurysmal disease is feasible, safe, and associated with acceptable outcomes. Advances in endovascular stent-grafting technology have enabled the use of these devices for the successful repair of Dacron graft disruptions [11,12].

In this case, the detection of the rupture site was crucial. When the rupture site is a proximal anastomosis, open surgery is the only treatment option despite the advanced age of the patient. In contrast, endovascular treatment can be performed for grafts or distal anastomoses. However, the site of rupture is often difficult to detect despite the use of CT or angiography. In our case, angiography was performed to determine the rupture site, which was visualized as a red thrombus on the vascular graft. Angiography has been performed in endovascular therapy to confirm the location of arteriosclerotic plaques to prevent embolisms during aneurysmal repair or to confirm the location of primary entry and re-entry in cases of aortic dissection [5,13]. No complications have been previously reported with aortic angiography for chronic type B aortic dissections and anastomotic pseudoaneurysms.

Conclusion

To our knowledge, this is the first report of non-anastomotic rupture of a vascular prosthesis graft that was detected using non-obstructive aortic angiography. Thus, non-obstructive aortic angiography can be a helpful modality in establishing the definitive diagnosis in patients with graft rupture.

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