Cryopreserved Aortic Homograft for In Situ Replacement of Infected Thoracic Stent Graft Associated With Distal Aortic Arch Rupture and Hematemesis

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Endograft infections present a potentially lethal complication of thoracic endovascular aortic repair (TEVAR). We report a case of a young male patient who was referred to our institution because of a stent graft infection that occurred 10 months after TEVAR. Contained distal aortic arch rupture and hematemesis were associated with the endograft infection. Emergent open surgical repair was undertaken with deep hypothermic circulatory arrest. After the removal of the infected endograft, the distal aortic arch and proximal descending thoracic aorta were replaced with a cryopreserved aortic homograft. Fifteen-month follow-up was uneventful. We discuss techniques and materials for replacement of the infected endograft. The article provides an outline of the potential benefit of cryopreserved aortic homografts within the setting of a complex thoracic aortic infection.


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tent graft infections remain among the most devastating and life-threatening complications after TEVAR. The possible causes of adverse outcome include progressive septicemia, aortic rupture, or bleeding from the aortoesophageal or aortobronchial fistula [1–3].

Our patient was a 21-year-old man who underwent TEVAR 10 months earlier. The patient was initially treated in another hospital for chest pain, fever, chills, fatigue, and weight loss. He received broad-spectrum antibiotics, but his condition progressively deteriorated within the following 3 weeks. Multidetector computed tomographic (MDCT) aortography revealed an aortic rupture 4 cm caudal from the origin of the left subclavian artery. A large surrounding hematoma was also identified. The Medtronic Valiant thoracic stent graft (26 × 150 mm; Medtronic, Minneapolis, MN) was then implanted successfully. The patient recovered and the hematoma resolved completely. However, 10 months after TEVAR and initial recovery, he was referred to our institution because of a high-grade fever (39.4°C), cough, hemothysis, and chest pain. At admission, the value of the C-reactive protein was 199.1 mg/L, leukocyte count was 13.83 × 10⁹ cells/L (with 89.5% of neutrophils), hematocrit was 29.4%, and prothrombin time was 53%. MDCT aortography performed at our institution showed a contained rupture of the distal aortic arch, with edema and air bubbles surrounding the endograft (Fig 1). A severe hematemesis occurred during diagnostic evaluation, necessitating urgent esophagogastro-duodenoscopy. The procedure revealed a bluish discoloration of the esophageal mucosa, indicating an underlying hematoma located 25 cm distal from the incisors. The patient was transported to the operating room, where median sternotomy was performed to enable conventional central cannulation. Additional anterolateral left thoracotomy through the fourth intercostal space was performed to achieve exposure of the distal aortic arch and proximal descending thoracic aorta. Extradural circulation with deep hypothermic circulatory arrest was established, and aortotomy was performed at the level of the aortic arch. A large aortic tear was identified at the aortic arch concavity, with surrounding hematoma and purulent debris. Proximal struts of the stent graft protruded well out of the aortic wall. The hematoma and debris were removed, and aortic tissue samples were taken for microbiological and histologic analysis. The endograft was removed and the distal aortic arch...
and proximal descending aorta were replaced with a cryopreserved aortic homograft measuring 22 mm in diameter. A proximal anastomosis was created caudal to the origin of the innominate artery, with the origin of the left carotid artery included within the beveled proximal aortic tongue. A caudal anastomosis was performed at the level of the left pulmonary hilum, using a continuous 3-0 polypropylene monofilament suture (Fig 2). Hemostasis, followed by mediastinal and thoracic drainage, was performed after decannulation. The nasogastric tube was left in place, and parenteral feeding was maintained for 2 weeks. The subsequent control esophagography showed normal findings, and oral feeding was started. Vancomycin was continued postoperatively because of the isolation of Staphylococcus aureus in tissue cultures, but was eventually replaced by linezolid because of the development of incipient renal insufficiency and neutropenia. The patient recovered completely, and he was discharged from the hospital 3 weeks after surgery in good general condition and with normal laboratory findings. Follow-up was uneventful and MRI angiography, performed 15 months after the operation, demonstrated a favorable position and patency of the cryopreserved thoracic aortic homograft (Fig 3).

Comment

The cause of the initial aortic rupture in our patient remained undefined. The diagnostic evaluation did not confirm the preexisting aortic pathology. Our patient was febrile at the time of the initial admission and endograft implantation, but the presence of systemic bacteremia was not confirmed with positive hemocultures. Ten months after the initial TEVAR, the patient was referred to our institution because of the suspected graft infection. The most common causes of the thoracic stent graft infections include contamination during implantation, bacteremia, or progression of the infection from the surrounding tissue [1, 2, 4, 5]. Usually, the diagnosis of endograft infection can be established based on clinical signs of sepsis, blood tests, and blood culture findings. Imaging studies, such as computed tomography and positron emission tomography scan, and intraoperative findings can confirm an existing graft infection [2–4]. In this case, severe
hematemesis and esophagogastroduodenoscopy findings strongly suggested the existence of an aortoesophageal fistula. Intraoperatively, we identified a significant defect of the aortic wall in the concavity of the aortic arch, with metal struts of the stent graft protruding from the aortic wall into the surrounding hematoma. The protruding metal wires could have caused a discrete esophageal lesion. Such an event could have been facilitated by pulsatile movements of the aorta, superimposed by the increased aortic wall shear stress at the level of the distal aortic arch anulation \[1, 2\]. Hematemesis or hemoptysis may be the first signs of an aortoesophageal or aortobronchial fistula; however, occult esophageal lesions (discrete erosion, edema, or mucosal discoloration) may initially be asymptomatic and present later with fatal exsanguination \[4, 6\]. Early intervention should be performed to prevent such a catastrophic outcome. Several approaches have been used for treatment of an infected thoracic aortic endograft. The general recommendation is that such material be completely removed with the debridement of the surrounding tissue \[1–3\].

Self-made vascular tubes from xenopericardial tissue, rifampicin-bonded polyester grafts, and grafts coated with omental flaps have all been used for aortic reconstruction after graft removal \[2, 3, 5, 6\]. However, cryopreserved homografts appear to be more resistant to bacterial infection than other conduits are, and they are associated with a reduced immune response \[6–8\]. The definite role of those grafts in the treatment of complex aortic infections remains to be defined based on a larger volume of patients with extended follow-up.

References

Temporary Epicardial Pacing Wire Migrating to and Exiting From the Jaw

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A 63-year-old woman underwent cardiac surgery, with atrial and ventricular temporary epicardial pacing wires being placed at the end of the procedure. Four months after the operation, the patient experienced tooth decay and underwent a tooth extraction. Thereafter, the patient developed an infected, swollen neck; computed tomography revealed that one of the temporary pacing wires had migrated into her neck. The patient was readmitted for removal of the wire, but it spontaneously exited through the skin of the lower jaw; the infection was resolved with intravenous antibiotic therapy.

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During cardiac surgery, temporary epicardial pacing wires (TEPWs) are routinely inserted to prevent circulatory failure caused by bradycardia or arrhythmias. The pacing wires are often unnecessary after surgery, but rare, sometimes catastrophic, complications have been associated with the retention of TEPWs. Cardiac tamponade at the time of removal is one of the major complications associated with the use of wires, but retained wires also carry potential risks for infection and migration. We present the case of a patient with a retained temporary pacing wire that migrated into her neck and spontaneously exited from her lower jaw.

A 63-year-old woman presented with severe mitral valve regurgitation and paroxysmal atrial fibrillation which were treated by mitral valve plasty and the maze procedure. At the end of the procedure, we inserted atrial and ventricular TEPWs and fixed them to the right and left upper abdominal skin. The patient’s postoperative course was good, leading to the patient being discharged 3 weeks after the operation.

Before discharge, an attempt was made to remove the TEPWs, but both wires were resistant to the procedure. The wires were initially placed such that they would be easy to remove; however, they are occasionally resistant to removal due to binding by the fascial stitches. Therefore, they were cut at the skin level and left in place to avoid the possibility of bleeding and cardiac tamponade.

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