Vascular homografts as bypass grafts for superior vena cava syndrome due to idiopathic fibrosing mediastinitis

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ABSTRACT

Fibrosing mediastinitis can lead to superior vena cava obstruction by generating a fibroinflammatory mass in the mediastinum. Surgical caval reconstruction with conduits could be indicated for cases of unsuccessful or technically unfeasible endovascular stenting and angioplasty. The use of cryopreserved vascular homografts seems to be better than prosthetic conduits for mid- and long-term patency, as was observed in the case we have described in the present report. (J Vasc Surg Cases and Innovative Techniques 2021;7:335-8.)

Keywords: Fibrosing medistinitis, Homografts, Superior vena cava syndromex

Fibrosing mediastinitis (FM) is a rare fibroinflammatory disorder that leads to compression of the mediastinal structures. Superior vena cava (SVC) syndrome can occur in >42% of patients with FM.[1] In the present report, we have described the case of chronic SVC syndrome due to idiopathic FM that was treated with vascular homografts for the reconstruction of the upper venous return to the right atrium (RA). Our patient provided written informed consent for the report of his case details and images.

CASE REPORT

A 52-year-old man had been referred to our institution for surgical advice regarding his recurrent SVC obstructive symptoms. His medical history was significant for a tumoral mass compressing the hilar structures of the right lung that had been revealed on a thoracic computed tomography (CT) scan performed after episodes of severe coughing and exertional dyspnea. Therefore, at 23 years old, he had undergone right pneumonectomy, with total excision of the hilar tumor mass. The histopathologic findings were consistent with fibroxanthoma, fibrosis, and granulomatous inflammation. His laboratory tests included fungal screening, parasitic panels, autoimmune serology tests, neoplastic investigations, tuberculosis screening, and syphilis serology. All the test results were negative, precluding a

potential etiology for FM. Corticosteroid treatment or any other treatment was not recommended because the etiology of FM was considered idiopathic. After an 11-year period with no remarkable symptoms, the patient suddenly began developing progressive upper extremity and fascial swelling with cyanosis and fatigue. Magnetic resonance venography revealed occlusion of the SVC (Fig, *A*). Thus, when our patient was 34 years old, a 10-mm polytetrafluoroethylene (PTFE) graft was selected to bypass the SVC obstruction between the innominate vein and the RA, through a standard median sternotomy. After this surgery, the patient had recovered completely and reported no recurrence of the fascial and upper extremity edema or fatigue for the next 12 months postoperatively. However, at the end of the first postoperative year, magnetic resonance angiography showed occlusion of the PTFE bypass graft, despite an effective postoperative anticoagulation and antiplatelet regimen (warfarin with an international normalized ratio of 2.5 to 3 and 100 mg/d of aspirin) and the presence of collateral vessels from the upper venous structures to the inferior vena cava (Fig, *B*). The patient remained asymptomatic despite the presence of mild fascial congestion until the age of 48 years when he reported some episodes of high-grade fever, followed by the sudden appearance of a cutaneous fistula at the suprasternal notch. Repeated cultures of an exudate specimen taken from the cutaneous end of the fistula were positive for *Staphylococcus epidermidis*, although those of blood samples remained negative. The findings from an ^{18}F-fluorodeoxyglucose positron emission tomography/CT scan were highly suggestive of a vascular PTFE graft infection, with extension along the right pretracheal space revealed by focal and heterogeneous uptake that corresponded to the projection over the bypassed SVC area and the fistula tract on the CT scan (Fig, *C*). Antibiotic treatment with trimethoprim/sulfamethoxazole and rifampicin was started. Concurrently, the patient became progressively breathless and anxious, with engorged neck veins and bilateral arm edema, and he was unable to sleep. Although the clinical condition of the patient prompted us to consider surgery, several teams declined to perform the surgical intervention, considering it

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Fig. A, Magnetic resonance venogram of superior vena cava (*SVC*) and upper limb venous system showing complete occlusion of the SVC. **B,** Magnetic resonance angiogram of thorax showing complete occlusion of the SVC and left brachiocephalic vein (*white arrows*). The absence of the polytetrafluoroethylene (*PTFE*) graft suggested intraluminal obstruction. **C,** An ^{18}F-fluorodeoxyglucose positron emission tomography (*PET*)/computed tomography (*CT*) scan showing findings strongly suggestive of vascular polytetrafluoroethylene (PTFE) graft infection. **D,** Intraoperative photograph of the reconstruction between the left subclavian–left internal jugular vein confluence and the right atrium (RA) with a thoracic aortic homograft and the connection of the right internal jugular vein (*RIJV*) with an iliac artery homograft to the thoracic aortic homograft. *RV,* Right ventricle. **E,** Postoperative CT angiogram at 3 years of follow-up showing a patent upper venous reconstruction with the two vascular homografts.

highly risky. At 10 months after prolonged antibiotic treatment, which had led to complete resolution of the cutaneous fistula, the patient was referred to our institution for additional surgical advice. At surgery, cardiopulmonary bypass was initiated by cannulating the right femoral artery and vein before repeat sternotomy. The heart was tightly adherent to the sternum and was carefully released from the retrosternal and pericardial adhesions. An exudate-filled cavity along the entire right lateral wall of the ascending aorta, which was covered by a very thick fibrous tissue, was incised, the exudate was drained, and the PTFE graft was entirely resected. Extensive surgical debridement of the right upper mediastinum was then performed, and the area was irrigated with povidone-iodine and normal saline solutions. Next, vascular venous reconstruction was begun by obliquely cutting the patent confluence of the left subclavian vein with the left internal jugular vein and performing an end-to-end anastomosis with the proximal portion of a cryopreserved

thoracic aortic homograft (13 mm in diameter). The distal part of the homograft was obliquely cut 15 cm from the proximal anastomosis and then sutured to a large right atriotomy, which was created close to the right atrioventricular junction to avoid any kinking or compression of the homograft. The proximal patent part of the right internal jugular vein was prepared by extending the median sternotomy's skin incision to the right part of the neck, parallel to the anterior border of the sternocleidomastoid muscle. An end-to-end anastomosis between the proximal part of an iliac artery homograft (8 mm in diameter) and the proximal right internal jugular vein was performed. The iliac homograft was then anastomosed to the thoracic aortic homograft in an end-to-side fashion by placing a side-biting vascular clamp across the thoracic aortic homograft, 5 cm proximal to its anastomosis with the RA (Fig, *D*). The patient improved dramatically and was discharged home 10 days after surgery with a regimen of warfarin for 6 months. The results
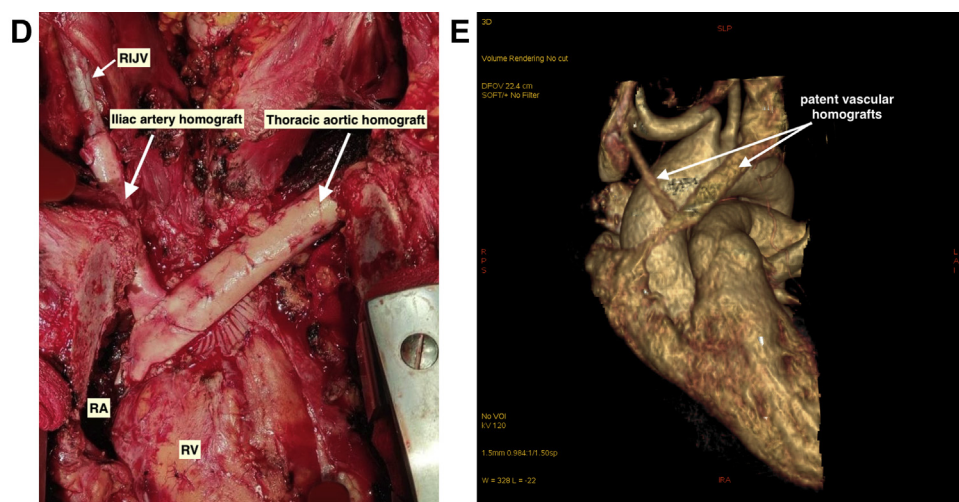
Fig. Continued.

of the fungal, aerobic, and anaerobic bacteria cultures of the collected intraoperative specimens were negative. Follow-up CT angiography at 3 years after surgery revealed satisfactory drainage through the arterial homografts into the RA (Fig, *E*), and the patient remained asymptomatic.

DISCUSSION

FM has many postulated mechanisms, with the most plausible an abnormal immunologic response to an infection or autoimmune process during which acellular collagen and fibrosis overwhelm the mediastinum,[2] leading to obstruction, compression, and compromise of the mediastinal structures (mainly the large airways, SVC, and pulmonary artery) in 98% of the patients according to Peikert et al.[1]

Because no curative treatment of FM is available, the medical, surgical, and endovascular interventions have focused on symptomatic patients and their clinical manifestations, comorbidities, and ability to tolerate treatment. Although endovascular management is emerging as the first-line treatment of SVC syndrome secondary to FM, some patients will still require surgical decompression because of the severity, extent of the obstructive process, unsuccessful or unfeasible stenting attempts, and evidence of restenosis after previous successful stenting.[3,4]

Previous reports regarding the use of arterial homografts to treat SVC syndrome resulting from other etiologies than FM are promising.[5-9] Aortic and arterial cryopreserved homografts represent an obvious advantage compared with prosthetic grafts owing to their low immunogenicity, thrombogenicity, lack of a need for long-term anticoagulation, and resistance to infection, such as for our patient with an infected vascular obstructed PTFE graft. Aortic homografts as large caliber bypass conduits with their more rigid wall compared

with venous homografts or autografts allow for the performance of larger anastomoses with less risk of kinking and thrombosis.[6] Furthermore, homografts might offer the possibility to perform more complex reconstructions starting from other obstructed head and neck veins using smaller arterial homografts, such as the use of an iliac artery homograft for the reconstruction of the right internal jugular vein in our patient. A spiral vein graft as an alternative biologic material compared with arterial homografts has proved susceptible to restenosis, probably owing to the interrupted endothelial lining generated by the long suture line.[9,10]

CONCLUSIONS

The superior potential long-term patency of aortic and arterial homografts has encouraged surgeons to consider them as attractive and more suitable biologic bypass conduits for SVC decompression in the setting of FM for which endovascular approaches are unfeasible.

REFERENCES

1. Peikert T, Colby TV, Midthun DE, Pairolero PC, Edell ES, Schroeder DR, et al. Fibrosing mediastinitis: clinical presentation, therapeutic outcomes, and adaptive immune response. Medicine (Baltimore) 2011;90:412-23.
2. Kauffman CA. Histoplasmosis. Clin Chest Med Rev 2009;30:217-25.
3. Sfyroeras G, Antonopoulos C, Mantas G, Moulakakis KG, Kakisis JD, Brountzos E, et al. A review of open and endovascular treatment of superior vena cava syndrome of benign aetiology. Eur J Vasc Endovasc Surg 2017;53:238-54.
4. Majumdar S, Shoela R, Kim DJ, Ramaswamy R, Mani N, Salter A, et al. Endovascular management of SVC syndrome due to fibrosing mediastinitis—a feasibility and safety analysis. Vasc Endovasc Surg 2018;52:202-6.
5. Gomez-Caro A, Martinez E, Rodriguez A, Sanchez D, Martorell J, Gimferrer JM, et al. Cryopreserved arterial allograft reconstruction after excision of thoracic malignancies. Ann Thorac Surg 2008;86: 1753-61.
6. Firstenberg MS, Blais D, Abel E, Go MR. Superior vena cava bypass with cryopreserved ascending aorta allograft. Ann Thorac Surg 2011;91:905-7.

7. Billing JS, Sudarshan CD, Schofield PM, Murgatroyd F, Wells FC. Aortic arch homograft as a bypass conduit for superior vena cava obstruction. Ann Thorac Surg 2003;76:1296-7.

8. Ohri SK, Lawrence DR, Townsend ER. Homograft as a conduit for superior vena cava syndrome. Ann Thorac Surg 1997;64:531-3.

9. Oyarzun JR, McCormick JR. Homograft and SVC syndrome. Ann Thorac Surg 1998;65:1836-7.

10. Dotty DB. Bypass of superior vena cava: six years' experience with spiral vein graft for obstruction of superior vena cava due to benign and malignant disease. J Thorac Cardiovasc Surg 1982;83:326-38.