Vascular and Endovascular Surgery
47(1) 42-44
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DOI: 10.1177/1538574412463974
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Possible Genetic Component to the Etiology of Perigraft Hygromas

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Abstract

Perigraft hygroma is a known complication of prosthetic graft implantation. The specific etiology of perigraft hygromas is still unknown. We report 2 brothers who underwent open abdominal aortic aneurysm repairs with polytetrafluoroethylene grafts that developed progressively enlarging perigraft hygromas. This is the first case report of 2 brothers developing sac hygromas after open abdominal aortic aneurysm repair. This case demonstrates that there could be a genetic component associated with the development of perigraft hygromas and further investigation of genetic etiologies should be considered.

Keywords

abdominal aortic aneurysm, perigraft hygromas, endovascular stent

Introduction

Perigraft hygroma is a known complication of prosthetic graft implantation. This rare complication has been most commonly associated with the use of polytetrafluoroethylene (PTFE) grafts. The specific etiology of perigraft hygromas is still unknown. We report 2 brothers who underwent open abdominal aortic aneurysm (AAA) repairs with PTFE grafts that developed progressively enlarging perigraft hygromas.

Case Presentation

Patient 1

A 58-year-old male with history of hypertension presented with a ruptured abdominal aortic aneurysm in 1997 and underwent repair with an aortoiliac bifurcated PTFE graft. In 2005, his computed tomography (CT) scan showed a perigraft hygroma measuring 10.4 cm. In 2008, he developed marked left flank pain and was found to have an enlargement of his hygroma to the 15 cm range. Because he was symptomatic with pain, his PTFE graft was replaced with a Dacron graft. During this operation, he was found to have typical hygroma material surrounding the large sac and an area of hemorrhagic bulging. Cultures of this material were obtained and results were negative. Since the hygroma repair, he has had no recurrence and remains asymptomatic (Figures 1 and 2).

Patient 2

A 56-year-old male with a family history of ruptured aortic aneurysm presented with a 5.5 cm infrarenal AAA who underwent an elective repair of his abdominal aortic aneurysm in 2003. One year later, he was found to have a 6 cm perigraft hygroma on routine follow-up. The hygroma evolved with rupture on 2 occasions, was aspirated, and eventually regressed without further intervention. The aspirate was cultured and returned with no growth. This case has been previously reported.[1]

Discussion

Perigraft hygroma is defined as an expanding aneurysm sac without a false aneurysm or endoleak and contains translucent, fibrinous, and gelatinous fluid.[2] Perigraft hygromas only rarely occur after AAA repair. They have been reported in a variety of vascular procedures. Many etiologies have been proposed.

Blumenberg et al suggested the collection of fluid that accumulates around the grafts was due to an extravasation of serum ultrafiltration.[3-5] Based on their chemical and histological studies, they believed that this was due to poor graft incorporation near the anastomoses.[4] Bolton et al proposed that graft soilage by alcohol, betadine, tissue, fat, and blood can "wet" the graft causing an increase in porosity and permeability to the serum.

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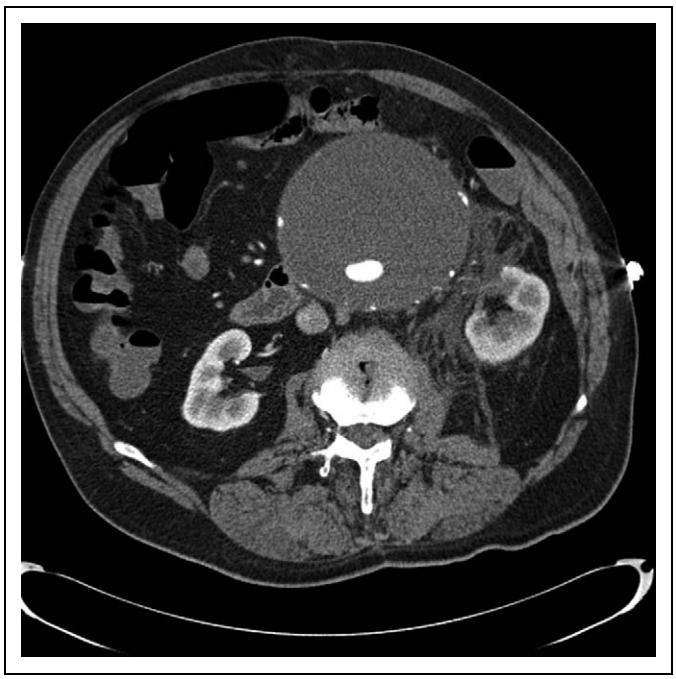

Figure 1. December 2008 sac enlargement to 15 cm.

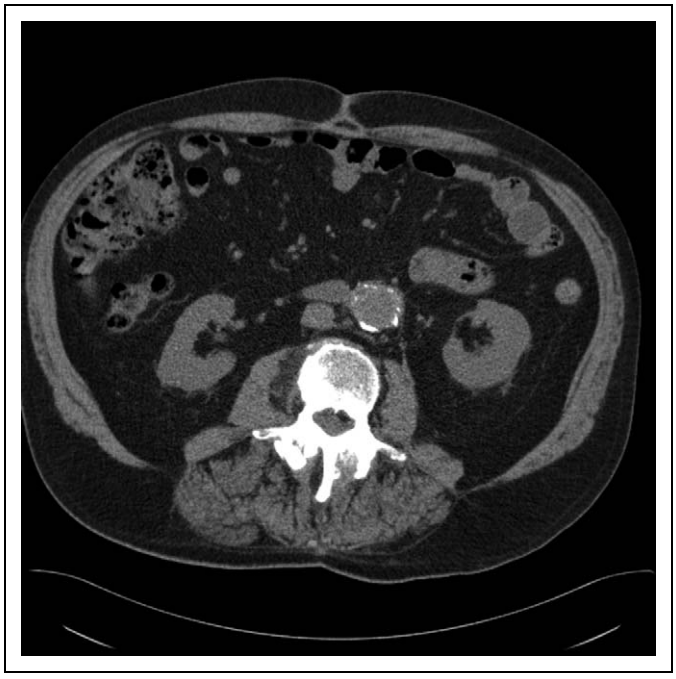

Figure 2. August 2009 computed tomography (CT) scan showing an interval decrease in the size of sac status post Dacron graft placement.

Another theory is related to fibroblast inhibition.[6] Sladen et al and Ahn et al demonstrated the presence of humoral fibroblast inhibitor in the serum that prevented maturation and proliferation of perigraft fibroblasts, thus preventing good graft incorporation.[5-7]

Another theory on sac hygromas was proposed by Towne et al who reported patients with sac hygromas had biofilm infections around the grafts. He characterized them as "an absence of systemic sepsis, a fluid-filled cavity surrounding the graft, a draining sinus tract, and microorganisms that must be removed from the fabric prosthesis for bacterial cultures."[8]

There have been theories of patients having an immunologic reactions to the graft material.[5] A survey conducted by Blumenberg demonstrated that the increased incidence of seromas were found in knitted Dacron and PTFE grafts (88%).[4] Many case reports describe different types of interventions for sac hygromas including replacing grafts with a different type of graft that would support patient-specific tolerance for certain grafts.

Lastly, one of the more recent theories for the development of sac hygromas is proposed by Risberg et al. They proposed that local hyperfibrinolysis/coagulation may promote rebleeding, liquefaction, and continue expansion of sac hygromas.[2] van Nes et al supported this theory by finding high levels of fibrin degradation product (FDP) and/or D-dimers in these sac hygromas, suggesting that the hyperfibrinolysis can lead to a hyperosmotic state leading to fluid accumulation and sac hygromas.[9]

We believe this is the first case reported of 2 brothers developing sac hygromas from open AAA repair. Both patients had negative microbiology cultures from their fluid collection which makes an infectious etiology less likely. Inherited characteristics in the immune system or fibrinolytic system may predispose individuals to perigraft hygroma. This case demonstrates that there could be a genetic component associated with the development of perigraft hygromas and further investigation of genetic etiologies should be considered.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

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