Journal of Clinical Case Reports
ISSN: 2165-7920
Salami, J Clin Case Rep 2017, 7:7
DOI: 10.4172/2165-7920.1000999
Case Report
OMICS International

Comparative Effectiveness of a Regional Virtual Tumor Board vs. Standard Care for Patients with Hepatocellular Cancer in a Regional Health Network

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Abstract

Endovascular aneurysm repair with visceral artery incorporation using fenestrated devices has gained widespread acceptance as a feasible alternative to the traditional open repair for the management of juxta-renal abdominal aortic aneurysms. However, complications with this endovascular technique continue to emerge. We present a patient who developed *de novo* inflammatory aortic aneurysm 3 years following endovascular repair of a juxta-renal AAA using a fenestrated stent graft.

Keywords: Endovascular; Abdominal aortic aneurysm; Fenestrated graft; Inflammatory aortic aneurysm

Introduction

Endovascular Repair (EVAR) with visceral artery incorporation using fenestrated devices is widely accepted as a feasible alternative to the traditional open repair for the management of juxta-renal Abdominal Aortic Aneurysms (AAA). Several studies have shown high technical success, low morbidity and mortality rates with fenestrated stent grafts in carefully selected patients [1-4]. However, complications with this endovascular technique continue to emerge [5]. We present a patient who developed inflammatory abdominal aortic aneurysm (IAAA) three years after EVAR with a fenestrated stent graft.

Case Report

A 75-year-old woman presented with an asymptomatic, expanding, 50 mm juxta-renal AAA. Her cardiovascular risk factors were hypertension, hyperlipidemia, mild aortic valve stenosis with severe regurgitation, and hypothyroidism. She had a 50 pack-year smoking history, but quit 1-month prior presentation. Her other medical comorbidities were a history of chronic obstructive pulmonary disease, cryptogenic cirrhosis with portal hypertension, Sjögren's syndrome, and polyclonal hypergammaglobulinemia. Based on an increased risk for rupture, a decision was made to repair her aneurysm electively.

Preoperative laboratory tests and imaging were obtained. Complete blood counts were found to be within normal limits. C-reactive protein (CRP) was normal at 3.7 mg/L (normal range: <8 mg/L), while erythrocyte sedimentation rate (ESR) was elevated at 55 mm/hr (normal range: 0 mm/hr to 29 mm/hr). A computed tomography angiography (CTA) of the chest, abdomen and pelvis showed a 50-mm aneurysm without any evidence of periaortic inflammatory changes (Figures 1a and 1b).

She underwent endovascular repair of the juxta-renal AAA with a patient-specific fenestrated aortic stent graft (Cook Medical, Bloomington, IN) with two custom made fenestrations for the both renal arteries and superior mesenteric artery scallop. A CT angiogram obtained on postoperative day 1 showed no evidence of an endoleak. The patient was dismissed home on postoperative day 3 after an unremarkable postoperative course. Serial surveillance CT angiograms demonstrated a progressive decrease in the size of the aneurysm sac from 50 mm to 33 mm over the span of 34 months.

Three years after the repair, the patient presented with a 4-week

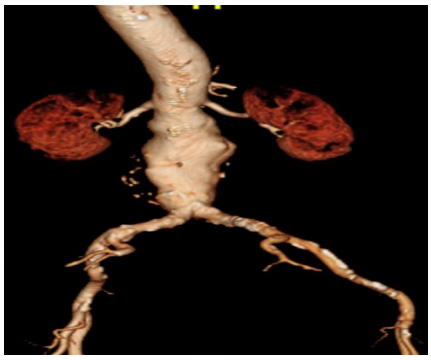

Figure 1a: CT angiogram demonstrating an asymptomatic juxta-renal abdominal aortic aneurysm.

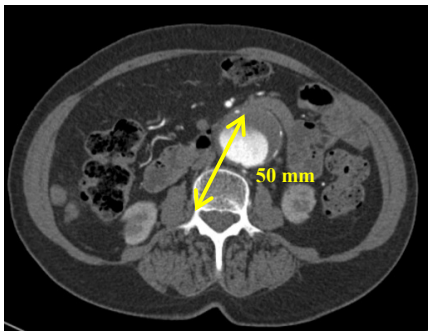

Figure 1b: CT angiogram demonstrating an abdominal aortic aneurysm without evidence of inflammatory changes.

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Received May 21, 2017; **Accepted** July 19, 2017; **Published** July 24, 2017

Citation: Salami A (2017) Comparative Effectiveness of a Regional Virtual Tumor Board vs. Standard Care for Patients with Hepatocellular Cancer in a Regional Health Network. J Clin Case Rep 7: 999. doi: 10.4172/2165-7920.1000999

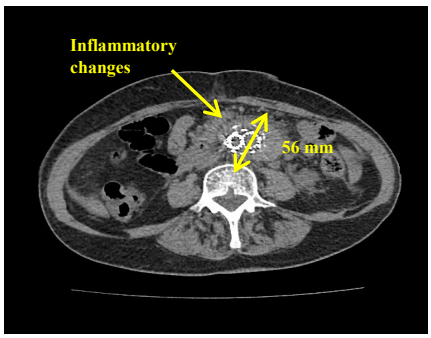

Figure 2: CT angiogram demonstrating inflammation around the excluded aneurysmal sac.

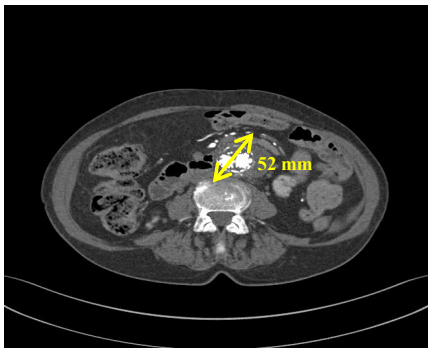

Figure 3: Interval CT angiogram demonstrating decreased inflammation around the excluded aneurysmal sac.

history of progressively worsening lower abdominal and back pain. She had an associated history of weight loss, anorexia, lethargy, and drenching night sweats, but denied having fevers or chills. Following admission, a CT angiogram revealed an interval enlargement of the aneurysm sac to 56 mm with significant periaortic inflammation. There was no evidence of an endoleak (Figure 2). Complete blood cell count was normal. However, CRP and ESR were elevated at 111.9 mg/L and 71 mm/hr, respectively. Kidney function tests were within normal limits (Creatinine: 0.8 mg/dl and GFR: >60 ml/min/1.73 m^2). Urine and blood cultures were not suggestive of an ongoing infection. Transthoracic echocardiogram was negative for valvular vegetations. A tagged white blood cell scan was obtained for a presumed stent graft infection, and showed slightly increased white blood cell localization around the aortic endograft. High dose steroid therapy with prednisone was initiated at a dose of 1 mg/kg. The patient demonstrated rapid response with improvement in symptoms within 48 hours of therapy. CRP also decreased to 30 mg/L within a week of therapy. The patient was discharged home in significantly improved clinical condition on hospital day 5.

Her symptoms were completely resolved at 1 month follow up, with improved appetite and interval weight gain. Her CRP and ESR were both within normal limits, <3 mg/L and 13 mm/hr respectively. CTA abdomen and pelvis obtained during follow up showed decrease in the periaortic inflammation and interval decrease in the diameter of the excluded aneurysm sac to 52 mm from 56 mm in the previous imaging (Figure 3).

Discussion

IAAA is a rare complication of EVAR. We have described an atypical and remote presentation of *de novo* IAAA that presented 3 years following endovascular repair of a juxta-renal AAA with a fenestrated stent graft. Available case reports/series on the subject have typically described postoperative IAAA within 3 months to 17 months of surgery, usually in association with retroperitoneal fibrosis [6-11]. To our knowledge, an IAAA in a setting of a fenestrated aortic stent graft has not been previously been reported.

At its core, aneurysm development is thought to be multifactorial in etiology, with both genetic and environmental factors identified as contributing influences. However, the exact pathophysiology of IAAA remains unknown. A previous review suggested that IAAA are an inflammatory extension of a common atherosclerotic process that results in non-inflammatory aneurysms [12]. Another study corroborated this concept by describing the presence of identical HLA alleles in both IAAA and degenerative AAA [13].

Some authors have hypothesized that IAAA occur as a reaction to an infectious process. A recent review reported an increased prevalence of Chlamydia, Herpesvirus and Cytomegalovirus in IAAA [14]. Along similar lines, an experimental study demonstrated significant macrophage infiltration in aortic aneurysms following exposure of rabbits to Chlamydia antigens, a finding that was prevented by Azithromycin [15].

Post-implantation syndrome (PIS) is an acute-phase inflammatory response that frequently occurs after EVAR with an estimated incidence of 34% [16]. PIS usually presents within 24 hours to 48 hours of stent graft implantation and manifests as a systemic inflammatory response syndrome. The exact etiology of PIS is unclear, but it is thought that the severity of PIS may be related to the composition of the stent graft material [17,18]. Voute et al. recently reported that woven polyester stent grafts were associated with a higher risk of PIS following EVAR, compared to expanded polytetrafluoroethylene [17]. Some authors have suggested that IAAA following EVAR may be an insidious extension of reactions to graft material or PIS [7,8].

A woven polyester-based stent graft was used for our patient's AAA repair, raising suspicion that the patient's periaortitis may be related to PIS or an allergic reaction to the graft material. While not improbable, we believe this is unlikely since PIS usually presents acutely in the early postoperative period. The patient presented had no symptoms of PIS in the early postoperative period, and remained asymptomatic on successive surveillance visits until presentation 3 years postoperatively.

Conclusion

A study recently suggested that the autoimmune condition, IgG4-related sclerosing disease, may be associated with an increased risk for the development of IAAA [19]. However, there is limited evidence of an association between Sjögren's syndrome and IAAA in the literature. Although the patient had a history of Sjögren's syndrome with a mildly elevated ESR prior surgery, her CRP, a more specific marker for inflammation, was within normal limits preoperatively. She also had multiple comorbidities, including hypergammaglobulinemia that could have accounted for the elevation in ESR that was observed.

IAAAs have been managed with good success medically or surgically. We opted for a trial of medical, non-invasive treatment of the patient's IAAA using high dose corticosteroid therapy, which she responded well to as evidenced by a progressive decline in her presenting symptoms

and inflammatory markers. A similar approach, with or without Tamoxifen, has been used with success in previous reports.

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