

CASE REPORT

Unique Presentation of an Inflammatory Abdominal Aortic Aneurysm With Rhabdomyolysis

Jeanette H. Man^{*}, Jun Xu

Department of Vascular Surgery, University of Iowa Hospitals and Clinics, Iowa City, IA, USA

Objective: This study reports the case of a 72 year old male who presented with rhabdomyolysis and a symptomatic juxtarenal inflammatory abdominal aortic aneurysm (IAAA). He underwent open repair of his IAAA with a polytetrafluoroethylene graft using the transperitoneal approach.

Results: The patient's aneurysm had significant inflammation with a thick rind of friable tissue overlying the native aorta. He had no history of autoimmune disease to serve as a potential trigger of his symptomatic IAAA. Prior to his presentation, however, he did experience three months of myalgia, with a concomitant creatine kinase elevation to 20,000 U/L and gross haematuria.

Conclusion: It is proposed that rhabdomyolysis and its accompanying inflammatory state may serve as a trigger for IAAA.

Published by Elsevier Ltd on behalf of European Society for Vascular Surgery. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Article history: Received 22 January 2019, Revised 3 March 2019, Accepted 7 March 2019,

Keywords: Inflammatory abdominal aortic aneurysm, Rhabdomyolysis

INTRODUCTION

Inflammatory abdominal aortic aneurysms (IAAA) are a rare variant of aortic aneurysms accounting for 5–10% of all abdominal aortic aneurysms (AAA). They are characterized by inflammatory thickening and fibrosis of the aneurysm wall. IAAA was first described in 1972 by Walker but its etiology remains unknown.¹ This study reports a case of symptomatic IAAA accompanying rhabdomyolysis. It is proposed that rhabdomyolysis may serve as a trigger for IAAA. Consent was obtained from the patient to discuss his medical history.

CASE REPORT

The patient was a 72 year old male with a medical history of previous tobacco use, hypertension, schizophrenia, and chronic obstructive pulmonary disease. He presented with myalgia. Specifically, he complained of having bilateral lower extremity weakness and body aches for several months. On the day of presentation, he complained of non-specific abdominal and flank pain. The patient also had gross haematuria, which he had not noticed. Laboratory evaluation was significant for creatine kinase elevation at 20,000 U/L. Computed tomography angiography showed a

juxtarenal AAA with stranding in the retroperitoneal fat planes (Fig. 1).

Procedure

Open repair was performed via a transperitoneal approach. The IAAA could not be treated by endovascular repair because of insufficient length, severe angulation of the neck, and heavily calcified distal landing zones. Intra-operatively, there was significant retroperitoneal fibrosis and the duodenum was densely adherent to the aneurysm, which was left undisturbed. The entire aneurysm wall also had significant inflammation with a thick rind of friable fibrotic tissue (Fig. 2). On opening the aortic sac, thrombus was removed and sent for gram stain and culture. Both were negative for mycotic processes. A 22 mm PTFE tube graft was placed. The rest of the surgery was uneventful. Post-operatively, the abdominal pain resolved, and renal function recovered.

Follow up

The patient returned to clinic at one month and a year. He continued to deny any abdominal pain and had full renal function. There were no post-operative complications.

DISCUSSION

The etiology of IAAA remains unknown. Hypotheses range from unknown antigens within the atherosclerotic plaque to a generalised autoimmune process from low level autoantibodies.^{2,3} It is unknown whether the present patient had an unknown antigen that contributed to his IAAA, but

^{*} Corresponding author. Department of Vascular Surgery, University of Iowa Hospitals and Clinics, 200 Hawkins Dr, Iowa City, IA 52242, USA.

E-mail address: jeanette-man@uiowa.edu (Jeanette H. Man).

2405-6553/Published by Elsevier Ltd on behalf of European Society for Vascular Surgery. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

<https://doi.org/10.1016/j.ejvssr.2019.03.001>

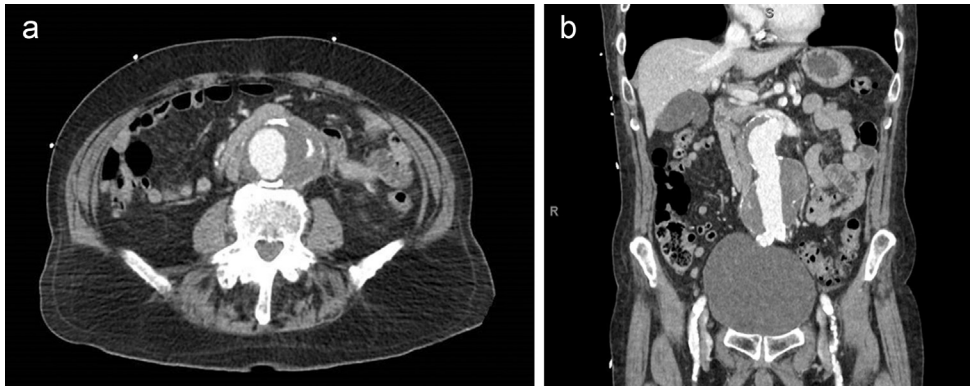


Figure 1. Diagnostic imaging of the inflammatory abdominal aortic aneurysm. Computed tomography angiography showed a juxtarenal abdominal aortic aneurysm without evidence of leakage. Note the inflammatory mass surrounding the aorta on the axial (A) and coronal view (B).

he had no history of autoimmune diseases. Because of his unique presentation of having months of myalgia prior to his eventual presentation with vague abdominal pain, it is proposed that rhabdomyolysis and its accompanying inflammatory state may have served as a trigger for the symptomatic IAAA. Although there are no case reports demonstrating a possible correlation between rhabdomyolysis and IAAA, prior basic science studies have postulated a causal mechanism between rhabdomyolysis and systemic inflammation. During the process, damaged muscle cells release cellular toxins into the systemic circulation that promote release of pro-inflammatory cytokines.⁴ The cause of rhabdomyolysis is unknown in the present patient, but it may have resulted from prolonged immobility.

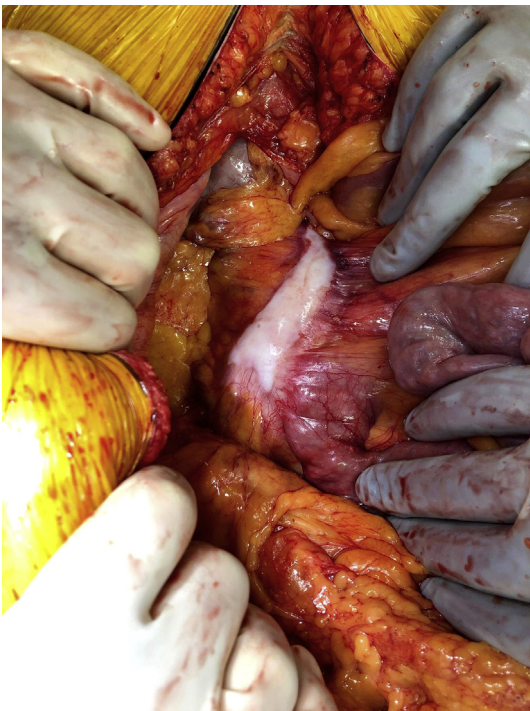


Figure 2. Intra-operative photo of the inflammatory abdominal aortic aneurysm. There was significant pearly white, peri-aortic inflammation with a thick rind of retroperitoneum overlying the native aorta.

Interestingly, the patient also had schizophrenia as a comorbidity, which has been increasingly associated with chronic inflammation and an increased risk of vascular disease.^{5,6} Studies have described high levels of pro-inflammatory cytokines in schizophrenia patients resulting in immune dysbalance.⁷ This is further reinforced by research demonstrating the therapeutic benefit of anti-inflammatory medication in treating schizophrenia.^{8,9} The present patient's diagnosis of schizophrenia may have served as an additional source of underlying chronic inflammation. Further studies are needed to elucidate whether chronic inflammation and genetic predisposition to vascular disease in schizophrenia patients increase the risk of IAAA.

Meanwhile, IAAA can be treated successfully by either an open or endovascular approach. Open repair was performed before endovascular treatment was possible. With an open approach, the difficulties encountered are largely technical because of the hostile nature of the retroperitoneum from the peri-aortic adhesions involving the duodenum, inferior vena cava, left renal vein, and ureter. In contrast, endovascular repair is an attractive option because it eliminates difficult dissection. Endovascular repair is only feasible, however, when the anatomy of the aorta is amenable. Treatment should be individualized for each patient and according to the surgeon's expertise.

Conclusion

This is the first reported case in which a patient presented with rhabdomyolysis and a symptomatic IAAA that was treated successfully by open repair. Although the etiology of IAAA remains unknown, it is proposed that rhabdomyolysis may be a possible trigger for IAAA.

CONFLICT OF INTEREST

None.

REFERENCES

- 1 Walker DJ, Bloor K, Williams G, Gillie I. Inflammatory aneurysms of the abdominal aorta. *Br J Surg* 1972;59:609–14.

- 2 Hellmann DB, Grand DJ, Freischlag JA. Inflammatory abdominal aortic aneurysm. *JAMA* 2007;**297**:395–400.
- 3 Vaglio A, Greco P, Corradi D, Palmisano A, Martorana D, Ronda N, et al. Autoimmune aspects of chronic periaortitis. *Autoimmune Rev* 2006;**5**:458–64.
- 4 Hamel Y, Mamoune A, Mauvaise FX, Habarou F, Lallement L, Romero NB, et al. Acute rhabdomyolysis and inflammation. *J Inherit Metab Dis* 2015;**38**:621–8.
- 5 Moises HW, Wollschläger D, Binder H. Functional genomics indicate that schizophrenia may be an adult vascular-ischemic disorder. *Transl Psychiatry* 2015;**5**:e616.
- 6 Kritharides L, Chow V, Lambert TJ. Cardiovascular disease in patients with schizophrenia. *Med J Aust* 2017;**206**:91–5.
- 7 Müller N, Weidinger E, Leitner B, Schwarz MJ. The role of inflammation in schizophrenia. *Front Neurosci* 2015;**9**:372.
- 8 Müller N, Riedel M, Schwarz MJ, Engel RR. Clinical effects of COX-2 inhibitors on cognition in schizophrenia. *Eur Arch Psychiatr Clin Neurosci* 2005;**255**:149–51.
- 9 Sommer IE, de Witte L, Begemann M, Kahn RS. Nonsteroidal anti-inflammatory drugs in schizophrenia: ready for practice or a good start? A meta-analysis. *J Clin Psychiatry* 2012;**73**:414–9.