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Case Report

A unique proposed etiology for inferior mesenteric artery aneurysm: A case report ☆

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ABSTRACT

The etiology of large artery aneurysms has long been established as secondary to atherosclerotic disease and degenerative changes in the vessel walls. Less common, are aneurysms of the visceral arteries; the splanchnic and renal arteries. Rarer yet, are inferior mesenteric artery aneurysms, accounting for approximately 1% of visceral artery aneurysms. While causes range from inflammatory to congenital disease, a proposed etiology of proximal, solitary inferior mesenteric artery aneurysms, is correlated to the "jet disorder phenomenon," first described in a 1990 case report by Sugrue, and Hederman. This paradigm states that aneurysm formation may occur secondary to celiac and superior mesenteric artery occlusion, causing increased, and turbulent arterial flow distally. We present a case that demonstrates a small inferior mesenteric artery aneurysm without findings of celiac or superior mesenteric artery stenosis or occlusion. This patient did, however, have a large thrombosed common hepatic artery aneurysm which may serve as an alternate cause of jet disorder phenomenon. The findings in this case offers support for focused screening of proximal arterial vasculature when an inferior mesenteric artery aneurysm is encountered.

Introduction

An aneurysm is a permanent, localized, dilatation of an artery to a diameter that is greater than 50% of its normal diameter [1]. Dilations can involve all 3 vessel wall sections; the intima, media, and adventitia. Focal dilatations that involve less that all 3 vessel wall sections are defined as pseudoaneurysms. Focal arterial dilatation can occur in any vessel with the most commonly studied, and encountered, being the abdominal aortic aneurysm (AAA). Secondary to atherosclerosis, AAA currently affects over 1 million Americans, with a 4-5:1 predilection for males over females aged 60-70 [1]. There has been a noted decrease in AAA related mortality with the added diagnostic tools such as Computed Tomography (CT), CT angiography, Magnetic Resonance (MR), MR angiography, and standard diagnostic and/or therapeutic angiography techniques. Strong evidence has been developed for implementing screening programs in efforts to reduce devastating

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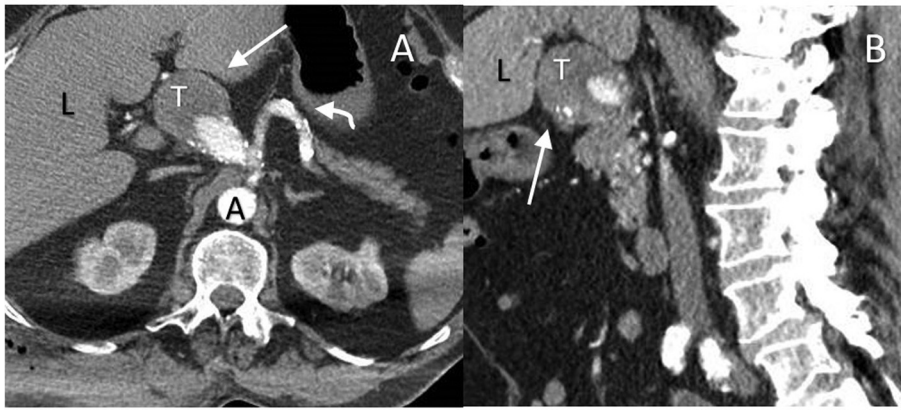

Fig. 1 – Axial (A) and sagittal (B) contrast-enhanced CT images of the abdomen. The hepatic artery aneurysm (arrow) is seen in the midline with thrombus (T) within it. The splenic artery (curved arrow) contains calcified atherosclerotic plaques, as did much of the other arterial vasculature, extending distally to the level of the iliac vessels. Liver (L) and Aorta (A) are labeled for orientation purposes.

consequences of AAA rupture. The Society of Vascular Surgery (SVS) and the United States Preventive Services Task Force (USPSTF) recommend screening of male smokers over the age of 65 with one-time abdominal ultrasound. The SVS additionally recommends screening in women of the same age with a smoking history or a family history of AAA. The combination of screening programs, improved diagnostic imaging techniques, and improved knowledge of clinical risk factors has led to an estimated 6.2%-6.7% decrease in mortality [1].

Aneurysmal disease that affects the renal and splanchnic arteries is encompassed in the term visceral artery aneurysm (VAA). Visceral artery aneurysms are quite rare with cited incidence rates ranging from 0.01%-0.2% [2]. Although VAAs account for 5% of all abdominal aneurysms, frequency of splanchnic disease is highest in the splenic artery (60%) and lowest in the pancreaticoduodenal and gastroduodenal arteries, approximately 1.5% [3]. Proper inferior mesenteric artery (IMA) aneurysms occur with a frequency of 1% [4,5]. Atherosclerotic disease is also seen in instances of VAA, with histologic changes of medial layer degeneration, decreased vessel wall smooth muscle, and disruption of the elastic fibers [6]. Arteriosclerosis, although present, is commonly thought to represent a secondary cause of aneurysm formation. Additional etiologies include collagen connective tissue diseases such as fibromuscular dysplasia, collagen vascular diseases such as Marfan syndrome, inflammatory illnesses, and congenital diseases such as Ehler Danlos syndrome [6]. Pseudoaneurysms arise secondary to trauma, inflammation, infection, or iatrogenic injury, and are treated as soon as possible, as they have a high risk of rupture [2,6]. Visceral artery aneurysms remain an important clinical topic because of their indolent and often asymptomatic presentation. Efforts have been made to develop clinical consensus guidelines as to the management of this disease because nearly 22% of these aneurysms present as ruptures with 8.5% resulting in death [3].

Discussion of inferior mesenteric artery aneurysms may span the proximal and distal branches, affecting the jejunal, ileal, and colic arteries. For the purposes of this discussion, we specifically reference the aneurysm that forms at the proximal IMA, near the aorta. These aneurysms account for less than 1% of VAAs with similar incidence occurring in men and women in their sixth decade [4–7]. Literature review has revealed etiologies to include; medial degeneration, inflammatory disease such as pancreatitis, infectious etiology such as endocarditis, and even congenital disease such as Neurofibromatosis 1 (NF1). A compelling phenomenon theorizes a mechanical and physical etiology for solitary proximal IMA aneurysms. This has been coined “jet disorder phenomenon” and was first described by Sugrue and Hederman in a 1990 case report [8]. The phenomenon states that, secondary to celiac artery and superior mesenteric artery (SMA) occlusion in patients with severe atherosclerosis, there is increased and turbulent arterial flow distally, through the inferior mesenteric artery, causing progressive dilation, and aneurysm formation. Here, we present a case of an inferior mesenteric artery aneurysm in a patient with significant atherosclerosis, without stenosis or occlusion of the celiac or superior mesenteric arteries.

Case presentation

Seventy-nine-year-old male with past medical history of atrial fibrillation, hypertension, hyperlipidemia, and a known aneurysm of the hepatic artery was admitted after a fall from chair height. In managing his acute injuries, he was found to have right 9th through 11th rib fractures, a right flank hematoma, right hemopneumothorax, and bilateral pulmonary contusions. His abdominal CT demonstrated a 4.3 cm hepatic artery aneurysm with an occlusive intraluminal thrombus (Fig. 1) and distally, a 1.2 cm proper inferior mesenteric artery aneurysm (Fig. 2). The common hepatic artery demonstrated no flow distally, indicating complete thrombosis. The proper hepatic artery was reconstituted by collateral vasculature of the pancreaticoduodenal artery branches. Additionally, there was another aneurysm involving a branch of the IMA, shown in Figure 3. His age, history of hypertension, and hyperlipidemia corresponded with the

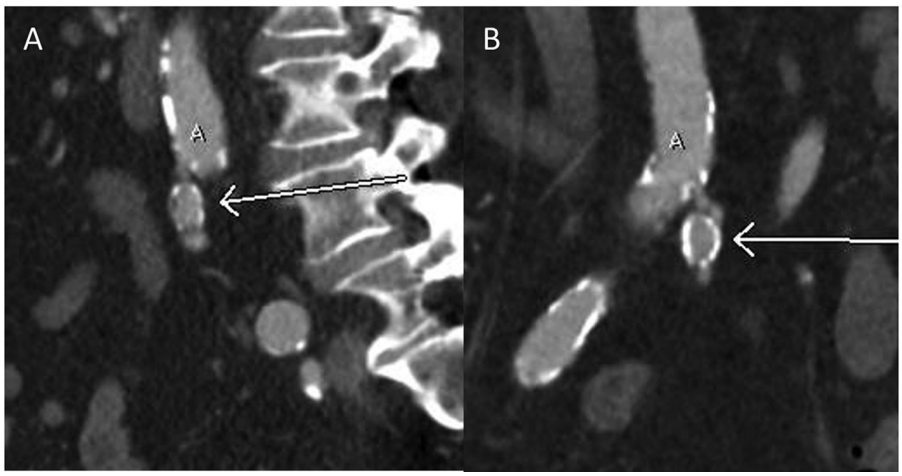

Fig. 2 – Sagittal (A) and coronal (B) CECT images of an inferior mesenteric artery aneurysm (arrow). Scattered atherosclerotic calcification is seen throughout the abdominal aorta, *A*.

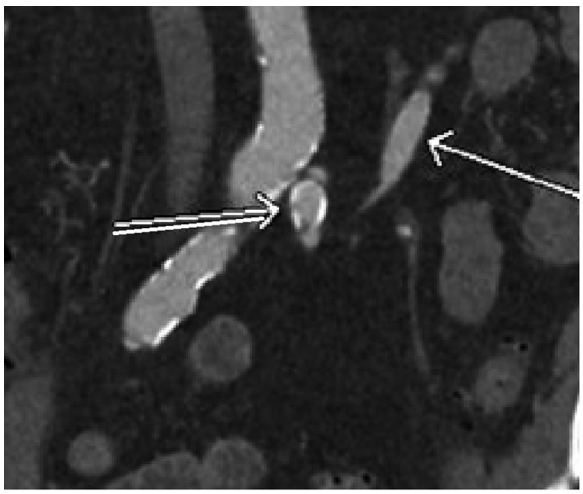

Fig. 3 – Coronal CECT demonstrating the proximal IMA aneurysm *(double arrow)* and another distal aneurysmal vascular dilation *(single arrow)*.

diffuse severe atherosclerotic disease that was demonstrated. Although scattered calcifications were present, the celiac, and superior mesenteric arteries were patent (Fig. 4). Our patient had no history of vasculitis, systemic lupus erythematosus, or NF1, suggesting atherosclerosis as the cause for his aneurysmal disease. He had leukocytosis on this admission, however, that was in the immediate traumatic period. There were no historical lab results to indicate an underlying inflammatory process. He was asymptomatic regarding his visceral artery aneurysms. As an outpatient, the hepatic artery aneurysm was being followed by his PCP with annual screening. As a patient with significant comorbidities, current recommendations are to treat the hepatic artery aneurysm once larger than 5 cm. Current recommendations for elective treatment of the IMA aneurysm is when size exceeds 2 cm [6]. He was admitted for care of his acute injuries and a visceral angiogram was recommended on an outpatient basis.

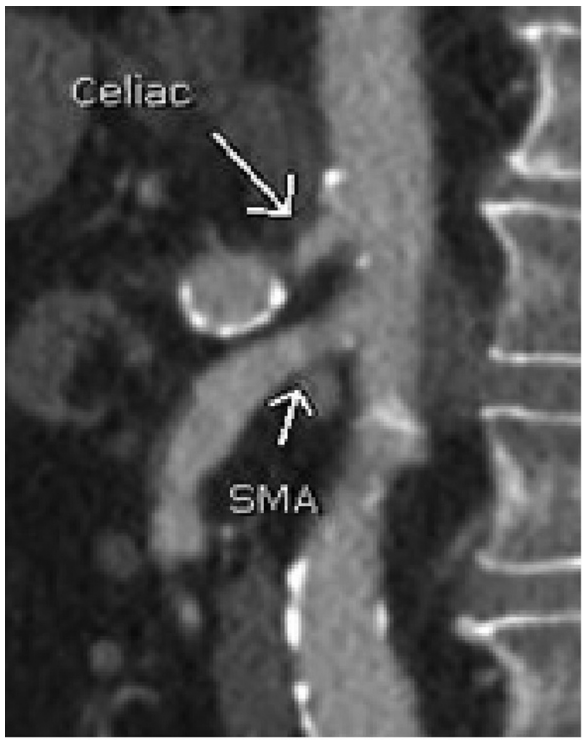

Fig. 4 – Sagittal CECT image demonstrating scattered atherosclerotic disease. Note the patency of the celiac and superior mesenteric arteries.

Our patient unfortunately succumbed to his acute injures and did not survive this admission.

Discussion

Of the visceral artery aneurysms that arise, IMA aneurysms account for less than 1%. While rare and commonly asymptomatic, IMA aneurysms, and visceral artery aneurysms in

general, remain of clinical concern because up to one-fourth of visceral artery aneurysms present with rupture and mortality rates of 10% and higher [6,9]. We strive to understand more about these diseases to develop methods and guidelines to provide excellent patient care. Further insight into the etiology of this insidious disease process yields superior clinical understanding and decision making.

Jet disorder phenomenon is a noted theory that accounts for the etiology of IMA aneurysms. It states that IMA aneurysms form due to increased and turbulent arterial flow causing progressive IMA dilatation secondary to celiac and SMA occlusion in patients with severe atherosclerosis. Since 1990, jet disorder phenomenon has been described in multiple case reports and the phenomenon has caused interventionists to additionally consider techniques to revascularize the celiac and SMA during intervention [10–14]. We have demonstrated a case of IMA aneurysm with patent celiac and SMA arteries. Uniquely, our patient had a hepatic artery aneurysm that was thrombosed, and occluded.

The implications of this are 2-fold. Firstly, we propose that the thrombosed hepatic artery aneurysm may impose the same mechanism of increased and turbulent flow leading to IMA aneurysm formation in the absence of celiac or SMA occlusion. The thrombus completely occludes the common hepatic artery and a significant amount of blood flow is diverted away from its natural hepatic target, causing increased distal flow, and thus, aneurysm formation in the IMA. If an IMA aneurysm is incidentally discovered, the mechanism of increased and turbulent distal arterial flow should be considered and a search for either occlusive disease or a significant upstream aneurysmal abnormality should ensue. Secondly, during the course of recommended screening for large proximal VAAs, care should be taken to evaluate distal arteries such as the IMA to aid in early detection of indolent aneurysmal disease.

Conclusion

Over the years, our increased understanding of the risk factors of AAA development such as smoking, hypertension, and cardiovascular disease has led to the implementation of screening programs, outlined by the SVS and the USPSTF, that have decreased mortality due to this disease by about 6.5% [1]. We propose that a disease process such as a thrombosed, proximal visceral artery aneurysm can cause a cascade mechanism akin to the jet disorder phenomenon, resulting in increased, and turbulent blood flow to the IMA that serves as the etiology of aneurysm formation. This knowledge may support selected screening of patients with longstanding proximal visceral artery aneurysms, leading to earlier detection, and treatment of distal IMA aneurysmal disease. Additionally, the differential of proximal disease that may be encountered when an incidental IMA aneurysm is found, is now broadened.

Authors' contribution

All authors contributed to writing the manuscript. All authors read and approved the final manuscript.

Patient consent

Written informed consent was obtained from the patient's family for publication of this case report, including accompanying images.

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Ethics approval

This is a retrospective case report not requiring ethics approval.

Consent to participate

All patient data has been removed and no informed consent is required to participate.

Consent for publication

All patient data has been removed and no informed consent is required to publish.

Availability of data and materials

Not Applicable.

Code availability

Not Applicable.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.radcr.2022.03.057.

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