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Case Report

Analysis of the Architectural Characteristics of the Abdominal Aortic Aneurysm in Dizygotic Twins

Maged Metias[1,2*], Anand Patel[2], Vikram Iyer[1,2], Theodore Rapanos[1,2]

[1]Department of Surgery, Division of Vascular Surgery, McMaster University, Hamilton Ontario, Canada; [2]Michael G. DeGroote School of Medicine, McMaster University, Hamilton Ontario, Canada

ABSTRACT

We report a case of dizygotic twins who were diagnosed with abdominal aortic aneurysms (AAAs) and analyze the differences in architectural characteristics of the abdominal aorta and visceral vessels. Both twins underwent an endovascular repair of their AAAs at the same age. This paper adds to the growing body of evidence of the genetic nature of aneurysmal degeneration.

Keywords: Abdominal aortic aneurysm; Dizygotic; Architectural characteristics; Dizygotic twins

INTRODUCTION

Negating collagen vascular diseases, family history of Abdominal Aortic Aneurysms (AAAs) along with age, smoking, atherosclerosis areeach independent risk factors for the development of AAAs [1]. Some estimate that 15 percent of patients with AAAs have a positive family history of the pathology [2]. The proportion of individuals with a familial AAA is approximately 13 percent, in which 17% of men with a relative with an AAA were also found to have an AAA on ultrasound surveillance [3]. One study found that familial AAAs were most common between brothers [4]. Here we discuss anatomic variations in the architecture of the AAA in dizygotic twin brothers who underwentan endovascular intervention at the same age. Consent was obtained from both patients for publication.

CASE 1

A 71-year-old male presented to the vascular surgery service after an incidental finding of an asymptomatic 4.3 cm Abdominal Aortic Aneurysm (AAA) found during ultrasound of his bladder.

His past medical history was significant for hypertension, dyslipidemia, type II diabetes, and obstructive sleep apnea with CPAP non-compliance. He was medically managed for these co-morbidities while placed on Aspirin 81 mg, Rosuvastatin 10 mg, telmisartan 20 mg, and metformin 500 mg daily. His blood group was O positive and had a positive family history of an abdominal aortic aneurysm in his father. He was an ex-smoker of 50 pack years who quit 6 months prior to his presentation. His height and weight were 182 cm and 103.6 kg, respectively yielding a BMI of 31.3 kg/m2. His computed tomography (CT) findings are summarized in Table 1 and Figures 1-3. Of note, his presenting aneurysm measured 4.1 × 4.6 cm. His six-month follow-up measurements discovered a rapidly growing AAA measuring 5.9 × 4.3 × 54 cm with extension of the aneurysm into the common iliac arteries (CIAs) measuring 4.2 cm and 2.0 cm on the right and left CIA, respectively. He also had two accessory renal arteries on the left and another on the right as well as a patent inferior mesenteric artery (IMA). He was consented for an endovascular approach to repair the AAA.

Intraoperative, the patient had a main body installed (Cook Medical, ZIMB-30-98) with limb extensions into the left (ZSL-24-59) and right (ZISL-24-59) CIAs.

CASE 2

71-year-old twin male underwent an ultrasound in light of his twin brother's AAA diagnosis. He was discovered to have an 8.2 × 8.3 cm AAA on ultrasound. He was asymptomatic.

His past medical history was significant for hypertension, dyslipidemia, mixed ischemic/nonischemic cardiomyopathy with an ejection fraction of 26%. Myocardial infarction with a stent insertioninto the right coronary artery with subsequent occlusion. Furthermore, the patient was positive for ventricular tachycardia (VT) with a bi-ventricular ICD and subsequent VT ablation, COPD, asthma, depression, gastric-esophageal reflux disease, tonsillectomy, inguinal hernia and resection of a basal cell carcinoma of the skin.

Correspondence to: Maged Metias, Department of Surgery, Division of Vascular Surgery, McMaster University, Hamilton Ontario, Canada, Tel: +6472912071; E-mail: mnm_metias@hotmail.com

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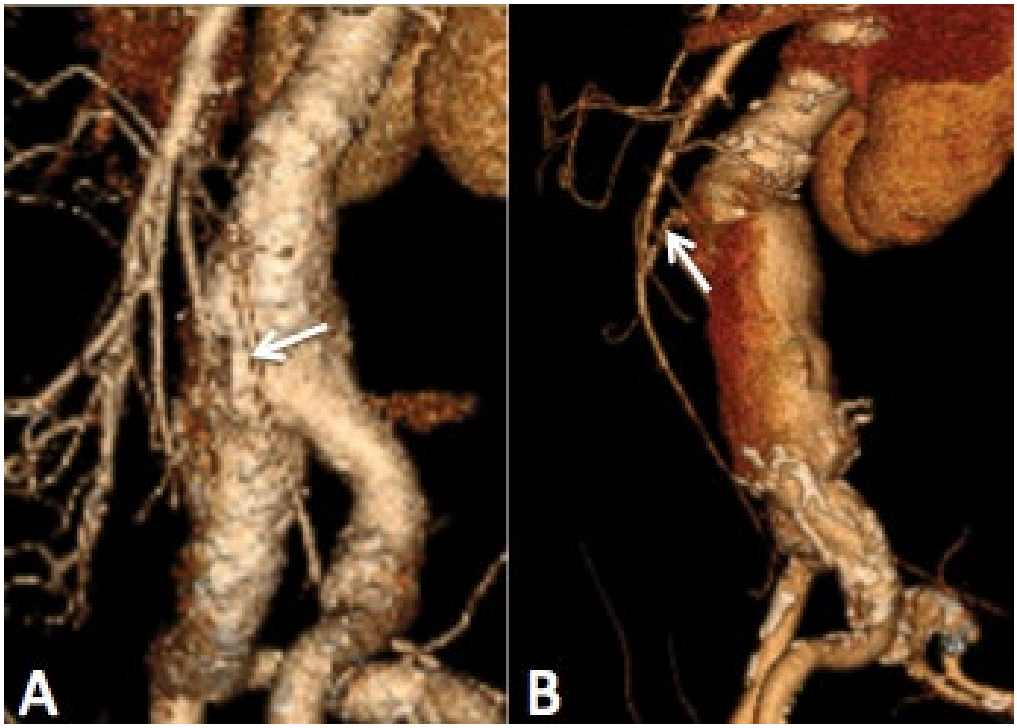

Figure 1: Three-dimensional renditions of the AAAs and associated visceral vessels. White arrows show patent IMA for both Case 1 (A) and Case 2 (B). Also noted here is the aneurysmal degeneration of the CIAs.

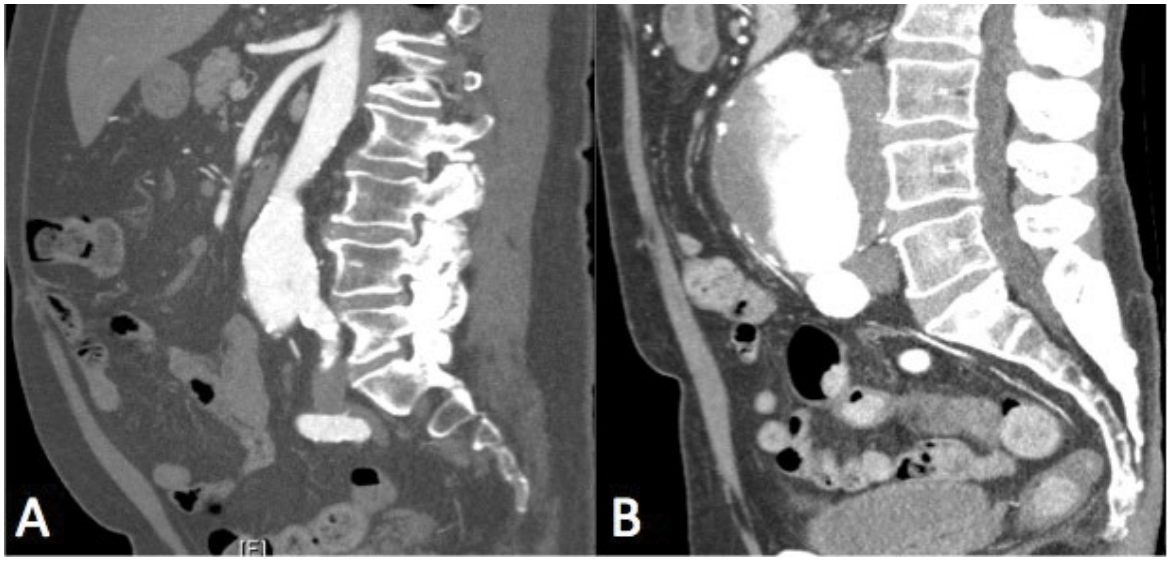

Figure 2:Sagittal view of the AAA of the patient in Case 1 (A) ad Case 2(B) at the age of 71 years. Image B demonstrates a larger aneurysm of 7.8 * 7.5 cm with higher thrombus burden.

Table 1: Dimensions and anatomical location of the AAAs and visceral vessels.

	Case 1	Case 2
Diameter of supra-renal abdominal aorta	26.6 mm	24.4 mm
Neck length	49 mm	3.7 cm
Neck diameter	20.2 mm	24.0 mm
Aneurysm Diameter	4.1 × 4.6 cm	7.8 × 7.5 cm
Length or AAA	69.7 mm	99.56
Distance from lowest renal to bifurcation	107.34 mm	143.27 mm
Right CIA maximal diameter	4.2 cm	2.2 cm
Left CIA maximal diameter	2.0 cm	2.0 cm
Accessory renal	Left	None
Clock position of right renal artery	10:00	9:00
Clock position of Left renal artery	3:00	2:30

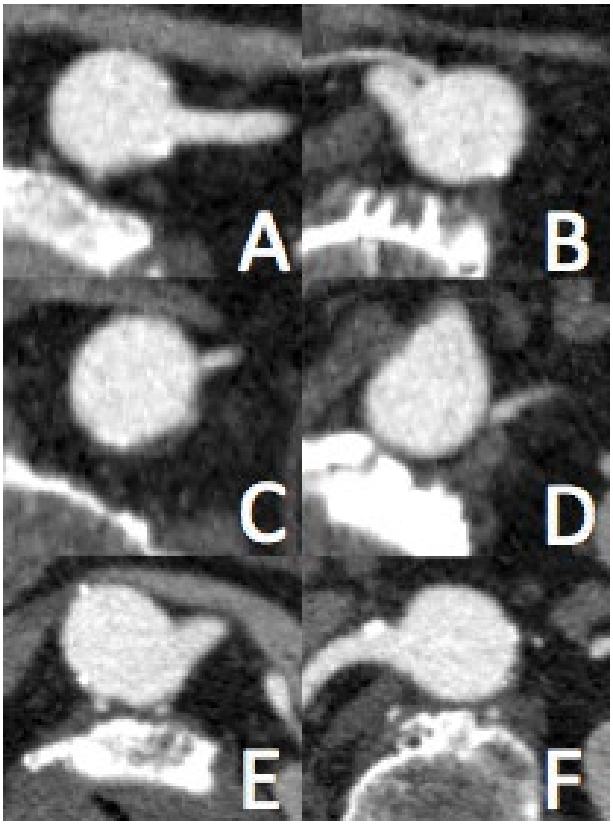

Figure 3: Renal and accessory renal vessels of both Case 1 and Case 2. Case 1: (A) Left renal artery; (B) Right renal artery and an accessory renal artery; (C) Left proximal accessory renal artery; and (D) Left distal renal artery. Case 2: (E) Left renal artery; and (F) Right renal artery.

He was medically managed on the following therapies: ramipril 2.5 mg, crestor 20 mg. metoprolol 50 mg BID, amiodarone 200 mg daily, ASA 81 mg, escitalopram 10 mg, fluticasoen 250 mcg, ipratroprium 20 mcg, and omeprazoel 20 mg BID. Like his brother, his blood group was O positive. He was an ex-smoker of 50 pack years who quit 1 year prior to his presentation. His height and weight were 182 cm and 77.2 kg, respectively yielding a BMI of 23.3 kg/m. His CT findings are summarized in Table 1 and Figures 1-3. His imaging revealed an AAA measuring 7.8 × 7.5 cm. His CIAs were dilated bilaterally at 2.2 cm on the right and 2.0 cm on the left. Unlike his brother, he did not have any accessory renal arteries, but similarly had a patent IMA. He requested and was consented for an endovascular repair, which he underwent in that same year.

Intraoperative, the patient had a main body deployed (Cook Medical, Zenith® LP-30-108), along with limb extensions into the left (Alpha 24-93) and right (Alpha 24-77) CIA.

DISCUSSION

Collagen vascular diseases such as Marfans and Loeys-Dietz syndrome affect the growth factor-beta pathway and are known to be associated with genetic risk factors that predispose patients to developing aortic aneurysms [5]. However, familial AAAsare genetically different and therefore pathologically different in that they are associated with late onset formation. Some studies have shown a link between 19q13 and 4q31 areas of the chromosome associated with aneurysm formation [6,7]. In one case report, Kitagawa et al showed anatomical similarities between monozygotic twins [8]; however, the pair of twins discussed here is dizygotic and had some anatomical differences as seen in Table 1. The variation may be due to the mode of inheritance of AAAs in which some families of AAA reveal a difference in penetrance patterns. Physiological effects such as hypertension, dyslipidemia and smoking may influence the architectural formation of the aneurysm and thus its variability between relatives. Furthermore, Darling et al. compared the architecture of AAAs and found no significant difference between familial and non-familial aneurysms anatomically or in occlusive burden [9].

When first degree relatives, defined as a parent, sibling or child develops aneurysmal degeneration, it is classified as a familial AAA [10]. This is an important classification from a screening perspective and can be established by history or physical exam of first-degree relatives.First-degree relatives are 2-3 times as likely to develop AAAs compared to the general population [3,11]. The current recommendations for the repair of AAAs are 5.5 cm for males and 5.0 cm for females [12]. However, familial AAAs has been documented to grow at an accelerated rate [13], with a higher rate of rupture [14].

In a Swedish study, a significant proportion of siblings of those with an AAA were found to have aneurysmal degeneration below the age of 65 years [15]. As such, the Canadian Society for Vascular Surgery recommends ultrasound surveillance on first-degree relatives after the age of 55 years [16]. As per the European Society for Vascular Surgery, those with a family history of AAAs are recommended to undergo ultrasound surveillance after 50 yearsof age, regardless of gender.

There is limited data on the efficacy of open versus endovascular repair of familial AAAs. Familial AAAs has an earlier onset, and as such, may be more amenable to open repair. However, Coscas et al. found that open repair was associated with late failure in patients with a positive family history of AAAs [17]. Conversely, patients undergoing endovascular repair experience higher rates of secondary sac rupture and higher re-intervention rates as compared to open repair [18].

CONCLUSION

Our case report demonstrates dizygotic twin brothers with architectural differences in the aneurysm diameter and length as well as thrombus burden. The presence of accessory renal arteries was also dissimilar; case 1 contained two accessory renal arteries on the left side and one on the right while the twin brother was found to have none. However, there were some similarities particularly in the circumferential position of the main renal arteries between cases. The overall dissimilarity in architecture may be due to genetic, developmental as well as life style factors between dizygotic twins.

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