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RESEARCH ARTICLE

Vascular Pathology: A Multifocal Cadaveric Analysis of Arterial Aneurysms

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Abstract

This case study aims to describe an extensive and rare case regarding the discovery of multisystemic aneurysms identified during cadaveric dissection spanning multiple vascular territories including the abdominal aorta, common and internal iliac, femoral, and popliteal arteries. This case serves to provide an understanding of anatomical diversity and the potential clinical implications for the development of widespread arterial aneurysms (AAs), including the prospective possibility regarding the influence of chronic obstructive pulmonary disease (COPD) and connective tissue disorders on aneurysm formation. Anatomic and pathologic observations were made of a phenol-fixed elderly male cadaveric donor with a known history of COPD during an eight-week anatomical dissection course. Biopsy tissue samples collected from the superior lobe of the right lung, left ventricle of the heart, left anterior descending coronary artery, lymph node, and right common iliac artery were histologically analyzed using hematoxylin and eosin (H&E) staining. Dissection revealed eleven atherosclerotic and non-ruptured aneurysms located in the abdominal aorta, both common iliac, both internal iliac, both proximal and distal femoral, and both popliteal arteries. No evidence of surgical intervention was found. This case illustrates widespread AAs and emphasizes the value of cadaveric studies in detecting rare vascular presentations that may have significant implications for diagnosis, treatment planning, and surgical intervention. Additionally, the potential correlation with COPD and connective tissue disorders invites further investigation into systemic contributions to aneurysm formation.

Key Words: *Donor dissection; Iliac arteries; Femoral artery; Popliteal artery; Chronic obstructive pulmonary disease; Abdominal aortic aneurysm*

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Introduction

An arterial aneurysm (AA) is defined as a dilation of an artery where the diameter of the affected section becomes at least 1.5 times larger than its natural diameter, frequently occurring in the aorta and cerebral arteries [1]. Aneurysms of the common iliac, internal iliac, femoral, and popliteal arteries are infrequent, particularly when occurring in conjunction with abdominal aortic aneurysms. Approximately 3% of patients with aortoiliac aneurysms also present with peripheral aneurysms [2]. Multiple aneurysms in the elderly population are generally ascribed to atherosclerosis, yet aneurysms of the magnitude seen in this case require consideration of other possible genetic conditions.

Sizing of vessels are adjusted in accordance with age, sex, and other factors. Considering these factors, the average diameter of the abdominal aorta is <3.0 cm in diameter and a diameter exceeding 3.0 cm is considered an abdominal aortic aneurysm (AAA) [3]. Within this classification, another exists: a giant AAA is defined as an AAA with a maximum transverse diameter >10.0 cm. While there are various definitions used to classify a giant AAA, an aneurysm exceeding 10.0 cm is uniquely rare with only 37 cases documented from 1988 to 2020 that detail the repair of giant AAA [4].

Peak prevalence generally occurs in the elderly population with the frequency of AAA varying from 0.5 to 3%. One study found that in 1.1 million AAA cases, the average prevalence is 1.4% in individuals in an age range from 50 to 84 [5]. Common risk factors for AAA include atherosclerosis, smoking, hypertension, advanced age, and being a White male. Other causes have been found to have a high degree of variation, including genetic conditions, human immunodeficiency virus, chronic inflammatory conditions, such as syphilis, along with connective tissue disorders [3].

Connective tissue disorders, specifically Marfan Syndrome, Ehler-Danlos Syndrome, and other familial forms of connective tissue disease, make up the major inherited disorders known to cause aortic diseases such as aneurysm formation [6]. Marfan Syndrome is highly associated with aneurysms of the aortic root and ascending aorta, while aneurysms of the descending thoracic aorta and abdominal aorta are less common [7]. While less common, adults with Marfan Syndrome are more at risk of developing AAA due to mutations of the fibrillin gene leading to weakening and distension of the aortic vessel walls [6].

COPD characterized by limited airflow progression and tissue destruction has become the third leading cause of death globally [8]. It is associated with structural changes due to chronic inflammation from overexposure to noxious particles and gases which can be commonly found in cigarette smoke [9]. COPD is a disease defined by its comorbidities specifically cardiovascular, metabolic, and musculoskeletal attributes. Patients with this disease present with a high risk of congestive heart failure and ischemic heart disease; these are risks that are also associated with AA formation. Upon further examination, in a study with 231 COPD participants, 33 were found to have had AA whether current or previously treated, indicating a growing need for increased research of these two life threatening conditions [10].

This case study explores the unique presentation of eleven multisystemic AAs in a cadaveric donor. The study aims to further understanding of the rarity of these aneurysms and the potential clinical implications they may pose, including the comorbidities with COPD and connective tissue disorders. Our university's whole body donation program aims to protect the anonymity of all donors. As a result, we are unable to confirm possible underlying causes leading to the presence of these aneurysms outside of the scope of post-mortem embalmed tissue pathology and gross observation.

Materials and Methods

A phenol-fixed cadaveric donor was dissected over eight weeks by five first-year medical students. The donor was a White male who died in his eighties with a cause of death reported to be due to complications of COPD. A full-body dissection was completed following instructions from Grant's Dissector, 16th Edition [11]. The dissection occurred in seven stages as follows:

1. Back, Spinal Cord, and Shoulder
2. Upper Limb
3. Thorax
4. Abdomen and Retroperitoneum
5. Pelvis and Perineum
6. Lower Limb
7. Head and Neck

The abdomen was dissected following the guidelines in Chapter 4 [11]. This began with a midline skin incision from the xiphoid process to the pubic symphysis. Transverse skin incisions were then made from the umbilicus to the midaxillary line on both sides of the donor to divide the abdomen into four quadrants. After removal of the fascia and reflection of the underlying muscles and abdominal wall, students were instructed to examine the organs within the abdominal cavity. At this point, an AAA as well as common iliac artery aneurysms were observed. The dissection proceeded into the pelvis and lower limbs using Chapters 5 and 6, leading to the discovery of additional AAs of the femoral and popliteal arteries [11]. Throughout the dissection, biopsy samples were collected from the superior lobe of the right lung, a lymph node associated with the left lung posterior to the pulmonary trunk, the left ventricle of the heart, the left anterior descending coronary artery, and the right common iliac artery aneurysm. The samples were processed and analyzed by the Department of Pathology at the UAMS College of Medicine using H&E staining and microscopic examination.

Results

During dissection of the thorax, the ascending and descending aorta were found to be visibly distended, yet cross-sectional evaluation of these arteries did not reveal any evidence of aneurysms. Table 1 shows a comparison of the average diameters of these arteries to the diameters recorded from the body donor [6].

TABLE 1
Diameters of the Distended Ascending and Descending Aorta

Artery	Donor Diameter (cm)	Average Diameter (cm)	Percent Difference (%)
Ascending Aorta	5.00	<2.1	>138%
Descending Aorta	6.20	<1.6	>288%

Upon completion of dissection, the donor presented a total of eleven non-ruptured, atherosclerotic AAs with no evidence of surgical intervention. These aneurysms were located in the abdominal aorta, both common iliac arteries, both internal iliac arteries, both femoral arteries at each of their proximal and distal ends, and in both popliteal arteries. Selected images of these aneurysms can be seen in Figure 1.

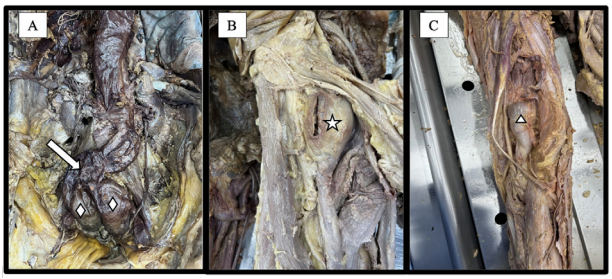

Figure 1) *(A) Image depiction of the arterial aneurysm system beginning in the abdominal aorta (white arrow) and descending into the bilateral common iliac arteries (white diamonds). (B) Aneurysm of the right proximal femoral artery labeled with white star. (C) Aneurysm of the left popliteal artery labeled with white triangle.*

Cross-sectional analysis of these aneurysms revealed that they are true aneurysms involving all three layers of the arterial wall – the intima, media, and adventitia, as confirmed grossly by a pathologist. No evidence of surgical intervention was observed in the arterial system in the regions of the aneurysms.

To classify these findings as true aneurysms, diameter measurements were taken and compared to average diameters reported in the literature [9,12]. A complete list of aneurysm measurements and their mathematical analysis can be found in Table 2. Visualization of the arterial diameters can be seen in Figure 2.

Gross findings in the thoracic cavity consisted of an enlarged heart weighing 683g in conjunction with distinct blackened discoloration of the lung tissue and surrounding lymph nodes. The pulmonary tissue presented with palpable crepitus and potential calcification of the bilateral lower lobes of the lungs. The findings in the lungs are consistent with COPD.

TABLE 2
Mathematical Analysis of Arterial Aneurysms

Artery	Donor Diameter (cm)	Average Diameter (cm)	Percent Difference (%)
Abdominal Aorta	10.30	<3.00	>243%
Common Iliac Artery	L 7.00; R 6.30	1.18	L 493%; R 434%
Internal Iliac Artery	L 6.00; R 5.30	0.75	L 700%; R 606%
Femoral Artery (proximal)	L 5.00; R 5.00	1.02	L 390%; R 390%
Femoral Artery (distal)	L 3.60; R 3.80	0.77	L 368%; R 394%
Popliteal Artery	L 3.00; R 2.80	0.69	L 335%; R 306%

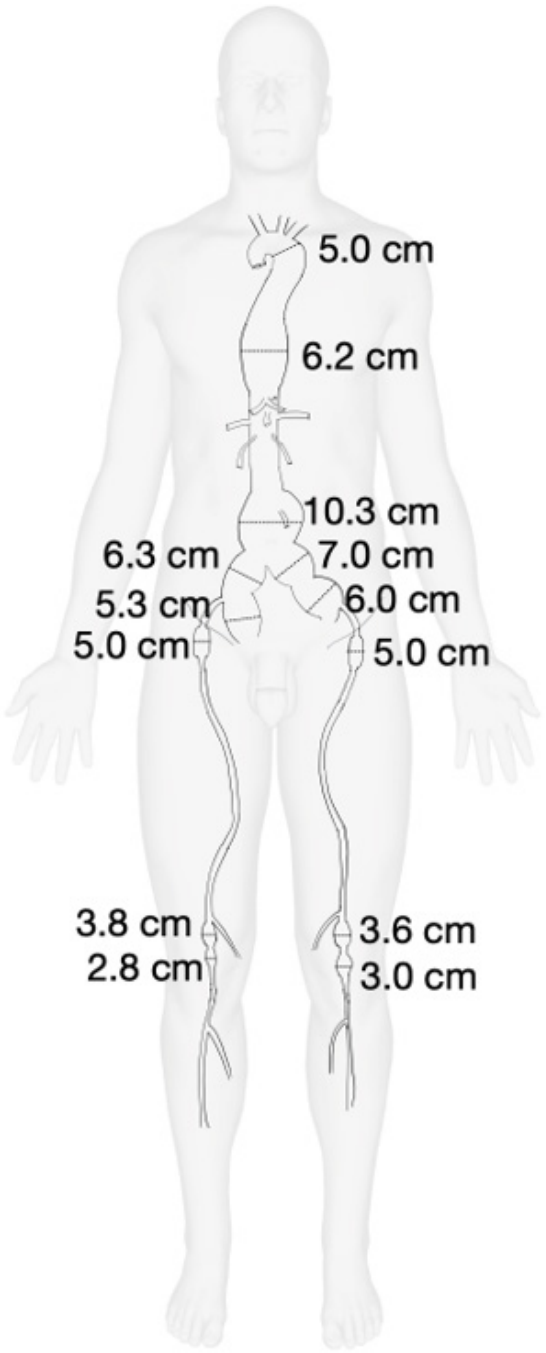

Figure 2) *Anatomical illustration of arterial diameters as measured on the donor. Illustration done by author Jade Michael Matthews.*

Significant histologic finding of the cardiovascular system includes marked calcific atherosclerotic disease of the left anterior descending coronary artery (Figure 3A), evidence of a remote myocardial infarction of the left ventricle (Figure 3B) with the uninvolved heart showing significant myocyte hypertrophy (Figure 3C). A representative section of the right iliac aneurysm showed calcific atherosclerosis with thinning of the arterial wall.

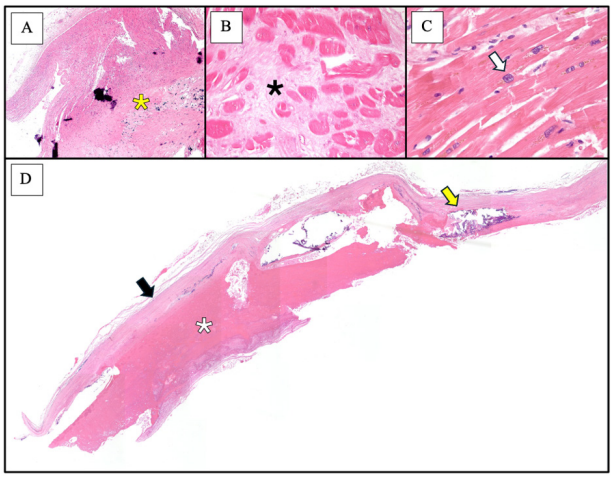

Figure 3) *(A) Tissue sample of the left anterior descending coronary artery with marked calcific atherosclerosis; plaque labeled with yellow asterisk (H&E, 40x magnification). (B) Left ventricle of the heart with evidence of a remote myocardial infarction; fibrosis labeled with black asterisk (H&E stain 200x magnification). (C) Left ventricle of the heart with myocyte hypertrophy; classic "boxcar" nucleus labeled with white arrow (H&E stain at 200x magnification). (D) Composite photograph from the right iliac artery aneurysm; black arrow thinned arterial wall, yellow arrow calcific atherosclerotic plaque, white asterisk attached blood clot (H&E, 40x magnification).*

Histologic findings of the lungs demonstrated dilated airspaces and thickened alveolar septa compatible with the reported diagnosis of COPD (Figure 4A). Lymph nodes with significant anthracosis were also found (Figure 4B-C). The donor's smoking history is unknown.

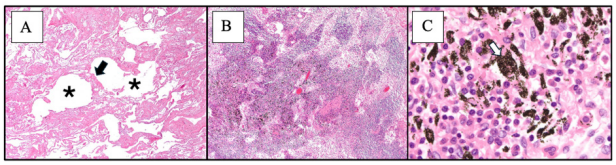

Figure 4) *(A) Tissue from the superior lobe of the right lung with dilated airspaces, indicated by black asterisks, and thickened alveolar septa (black arrow) (H&E 40x magnification). (B, C) Lymph node from the left lung posterior to the pulmonary trunk with significant anthracosis with representative anthracotic pigment-laden macrophage labeled with white arrow (H&E stain with 40x and 500x magnification for parts B and C, respectively).*

Discussion

The anomalies observed within the arterial system in this case provide a rare and extensive example of aneurysms spanning the abdominal aorta, both common and internal iliac arteries, both femoral arteries (each with aneurysms proximally and distally), and in both popliteal arteries. Isolated aneurysms are more likely to occur within the general population, especially in the elderly population. The presence of eleven non-ruptured atherosclerotic aneurysms with no evidence of surgical intervention is distinct and remarkable. These findings illustrate the necessity for comprehensive anatomical surveillance in both the clinical and cadaveric aspect.

Anatomical variations and degree of vessel dilation greatly exceed existing standard literature values. The AAA found in the cadaveric donor had a transverse vessel diameter of 10.3 cm resulting in its categorization being a giant AAA. This classification is associated with an increased risk of rupture and limited documentation in literature with only 37 documented cases of surgical repair between 1988 and 2020 [4].

The internal iliac arteries were particularly enlarged resulting in a 600%-700% increase in vessel diameter based on average values found in the literature (Table 2). This highlights the extensive nature of this specific pathological presentation. The enlargement found in the remaining vessels found in conjunction with one another raises the suspicion for potential systemic and genetic conditions beyond the stereotypical finding of atherosclerosis.

Histological findings demonstrate and further contextualize the vascular degeneration observed. The atherosclerotic buildup of calcium and plaque within the arterial walls of the left anterior descending coronary artery and right common iliac artery likely restricted blood flow through these vessels, contributing to arterial wall weakness and aneurysm development [13,14].

The histologic sections from the left ventricle of the heart shows evidence of a remote myocardial infarction, in the setting of marked atherosclerotic disease of the left anterior descending coronary artery. The uninvolved myocardium showed evidence of

significant myocyte hypertrophy. The finding of myocyte hypertrophy could be attributed to excess pressure or volume stress and often accompanies various heart diseases when used to accommodate for the decreased efficiency of the vessels [10]. This could have manifested through the presentation of hypertension or impaired vascular compliance [13-15].

In the lungs, histological evidence showed dilated airspaces and thickened alveolar septa; these findings are consistent with the reported history of COPD. An associated lymph node had prominent anthracosis, which could be attributed to smoking, general air pollution, or inhalation of noxious gases. Information about the patient's smoking history or environmental exposure was not available. Some studies indicate that there is increased risk of aortic aneurysm in patients with COPD [10]. It is unclear if there is correlation between COPD and the aneurysms seen in this case.

The nature of the aneurysms found in this case study invites the consideration of an underlying connective tissue disorder due to the high degree of dilation found in multiple arterial territories. Although a history of a connective tissue disorder is unknown in this case as a result of the anonymous nature of the anatomical donation program, Marfan and Ehler-Danlos syndromes as well as other connective tissue disorders carry an increased risk of aneurysm formation. This is due to the formation of abnormal fibrillin or elastin structures resulting in increased stress to the vascular system [16,17].

This case emphasizes the importance of cadaveric dissection in the identification and study of rare anatomical findings, which may not be present clinically within a patient's lifetime. Educationally, the case reinforces the need for correlated analysis between gross anatomy, histology, and pathology. Clinically, it illustrates the heightened need for awareness of vascular anomalies. This report contributes to the ever-growing body of literature studying rare anatomical variations while supporting the need for further interdisciplinary research regarding aneurysm formation, clinical comorbidities, and risk factors.

Conclusion

This cadaveric donor case report documents the uncommon and distinctive presentation of eleven atherosclerotic and non-ruptured AAs spanning multiple vascular territories. The aneurysm locations are as follows: abdominal aorta, both common and internal iliac arteries, both femoral arteries (each with aneurysms proximally and distally), and both popliteal arteries. The combination of gross anatomical findings, histological evidence, and the donor's known history of COPD suggests a complex multifactorial etiology. This includes the involvement of atherosclerotic degradation and possible systemic contributions of connective tissue diseases. While a definitive cause and correlation cannot be established, the severity of this case provides insight into vascular pathology and demonstrates the need for the integration of anatomical education with clinical practice. These findings support the demand for increased knowledge regarding anatomical variations in both a surgical and educational setting. This case report highlights the potential for cadaveric study to inform future research into systemic and chronic risk factors of widespread AA development.

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References

1. Norman PE, Powell JT. Site specificity of aneurysmal disease. Circ. 2010;121:560-8.
2. Takagi H, Mori Y, Manabe H, et al. Multiple arterial aneurysms. J Vasc Surg. 2003:6.
3. Shaw PM, Loree J, Oropallo A. Abdominal Aortic Aneurysm. StatPearls, Treasure Island, Florida, United States. 2025.
4. Ng JI, Nguyen T, Tarpara A, et al. Giant abdominal aortic aneurysms. J Vasc Surg Cases Innov Tech. 2021;7:659-64.
5. Kent KC, Zwolak RM, Egorova NN, et al. Analysis of risk factors for abdominal aortic aneurysm in a cohort of more than 3 million individuals. J Vasc Surg. 2010;52:539-48.
6. Erbel R, Eggebrecht H. Aortic dimensions and the risk of dissection. Heart. 2006;92:137-42.
7. Hagerty T, Geraghty P, Braverman AC. Abdominal aortic aneurysm in Marfan syndrome. Ann Vasc Surg. 2017;40:294-e6.
8. Negewo NA, Gibson PG, McDonald VM. COPD and its comorbidities: impact, measurement and mechanisms. Respirol. 2015;20:1160-71.
9. Agarwal AK, Raja A, Brown BD. Chronic Obstructive Pulmonary Disease. StatPearls, Treasure Island, Florida, United States. 2023.
10. Ando K, Kaneko N, Doi T, et al. Prevalence and risk factors of aortic aneurysm in patients with chronic obstructive pulmonary disease. J Thorac Dis. 2014;6:1388.
11. Detton AJ. Grant's Dissector. (16thedn), Wolters Kluwer, Singapore. 2024.
12. Lorbeer R, Grotz A, Dorr M, et al. Reference values of vessel diameters, stenosis prevalence, and arterial variations of the lower limb arteries in a male population sample using contrast-enhanced MR angiography. PloS One. 2018;13:e0197559.
13. Frey N, Katus HA, Olson EN, et al. Hypertrophy of the heart: a new therapeutic target?. Circ. 2004;109:1580-9.
14. Rafieian-Kopaei M, Setorki M, Doudi M, et al. Atherosclerosis: process, indicators, risk factors and new hopes. Int J Prev Med. 2014;5:927-46.
15. Poznyak AV, Sadykhov NK, Kartuesov AG, et al. Hypertension as a risk factor for atherosclerosis: cardiovascular risk assessment. Front cardiovasc med. 2022;9:959285.
16. Milewicz DM, Braverman AC, De Backer J, et al. Marfan syndrome. Nat Rev Dis Primers. 2021;7:64.
17. Malfait F. Vascular aspects of the ehlers-danlos syndromes. Matrix Biol. 2018;71:380-95.