Case Report

An Unusual Presentation of Right Common Iliac Artery Occlusion Secondary to Dissection of the Descending Aorta

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Received: 25 June 2020; **Accepted:** 03 July 2020; **Published:** 10 July 2020

Citation: Juna Musa, Loran Rakovica, Mina N.Z Mikel, Masum Rahman, Jason Cai, Ali Guy, Erisa Kola, Ina Kola, Nosehy Z.M. Yousef. An Unusual Presentation of Right Common Iliac Artery Occlusion Secondary to Dissection of the Descending Aorta. Journal of Surgery and Research 3 (2020): 212-215.

Abstract

Aortic dissection is a catastrophic condition which presents as an acute emergency with high morbidity in the vent of treatment delay or misdiagnosis. Aortic dissection may manifest in different forms depending on location and may mimic other disorders. 6.4 % to 17 % of dissections have been reported as painless. Uncommon presentations include syncope, lower extremity weakness and pain, mesenteric ischemia, peripheral ischemia, and congestive heart failure.

In this case report, a 60-year-old man with a history of hypertension and heavy smoking was admitted from the emergency department. He presented with a four-

hour history of pain and weakness in his right lower limb. The patient was found to have a distal aortic dissection complicated by right common iliac artery occlusion, hence surgical correction was performed. The condition was promptly recognized and treatment was initiated without delay.

Keywords: Aortic Dissection; Lower Limb Ischemia; Interventional Endovascular Management; Artery Coronary Syndrome (ACS)

Introduction

Aortic dissection remains one of the most common disastrous events affecting the great vessels. Patients usually present with sudden onset excruciating chest pain, which is often described as ripping or shearing and is frequently maximal at onset. Neurological manifestations, such as acute ischemic stroke, spinal cord ischemia or peripheral nerve ischemia may be additional presenting features. Rarely, the dissection may cause acute aortic regurgitation leading to myocardial infarction or cardiogenic shock [1]. However, a dissection may not be initially suspected when isolated lower extremity ischemia is the only presenting feature, leading to delayed diagnosis and treatment. Only 10 cases (0.6%) of aortic dissections were reported to have lower extremity ischemia with symptoms isolated to one or both lower limbs [2].

Nicholls first described rupture of the inner coat of the aorta in the absence of rupture of the outer coat in 1728. It was not until middle of 1990s that a clear understanding of the natural history, pathogenesis and etiology of aortic dissection evolved [3]. The term "dissecting aortic aneurysm" is often used to describe this acute catastrophic presentation. But strictly speaking, the term "aneurysm" is erroneous because entry of blood into the outer two-thirds of the aortic media often precedes dilatation of the vessel and dilatation may or may not occur subsequently. Therefore, a more appropriate term would be "dissection of the aorta" [4].

The peak incidence of aortic dissection occurs between the sixth and seventh decades of life, with men affected at least twice as often as women. In the United States, aortic dissection has an incidence of 5 to 10 cases per 100,000 per year and accounts for about 1.5 deaths per 100,000 males and 0.6 deaths per 100,000 females per year. About 30% of the patients with acute aortic dissection suffer from organ or limb ischemia [5]. Lower extremity symptoms accompanying dissecting hematomas of the aorta are well described. Pacifico and Spodick reviewed 1751 cases of aortic dissection and reported that 10% of these had lower extremity symptoms.

Case presentation

A 60 year-old male presented to the emergency department with sudden onset of severe pain and weakness of the right lower limb for the past four hours associated with profuse sweating. He was a heavy smoker. He also had a history of hypertension and had been on atenolol for one year.

Clinical examination revealed an anxious, restless, pale, profusely sweaty, and obese gentleman. His heart rate was 68 beats per minute and regular. All peripheral pulses were absent in the right lower limb. His blood pressures (BP) were 240/115 mmHg and 234/112 mmHg on the right and left upper limbs respectively, and it was 178/98 mmHg in the left lower limb. BP was unrecordable in the right lower limb. Examination of the right lower limb revealed 3/5

power with diminished tone, normal deep tendon reflexes, equivocal plantar response and impaired superficial and deep sensation. Examination of fundus revealed grade II hypertensive retinopathy. Heart, chest and abdominal examinations were unremarkable. ECG showed left ventricular hypertrophy by voltage criteria. Chest X-ray revealed mediastinal widening with clear lungs. Cardiac enzyme and D-dimer levels were normal. A complete blood count showed a hemoglobin level of 14.7 g/dl and a total WBC count of 16.1 x 10^{12}/L with predominantly neutrophils. Intravenous morphine and nitroglycerine were started and the patient was admitted to the intensive care unit. Atenolol 100 mg was administered orally. BP was brought down to 136/65 mmHg. Echocardiography showed concentric hypertrophy of the left ventricle with diastolic dysfunction. Abdominal ultrasound revealed a dissection of the descending aorta. Digital subtraction angiography, undertaken after vital signs were stabilized, revealed dissection of the aorta from an area distal to the origin of the left subclavian artery extending inferiorly to right common iliac artery.

The patient underwent an emergency operation. On exploration, a large hematoma was found around the aorta. On opening the aorta, a tear measuring 1.5 cm, beginning 2 cm below the subclavian artery, and dissecting distally to right common iliac artery was seen. A 2 cm segment of dissected aorta from the proximal end was excised and the layers were sutured together. The excised segment was replaced with a 22-mm size Dacron tube graft (Uni-graft, B Braun, Melsungen, Germany). No complications occurred during the surgery. The patient made an excellent recovery without any residual vascular or neurological deficits and was discharged on atenolol 50 mg daily and captopril 25 mg twice daily. During regular follow-up after discharge, he remained clinically stable.

Discussion

An aortic dissection entails a cleavage of the intima from the media and adventitia. It is believed that the dissection begins with formation of an intimal tear, exposing the underlying media to the driving force (pulse pressure) of intraluminal blood. When the intimo-medial tear allows the blood flow to enter the aortic wall, creating a new secondary channel, the false lumen. This channel can propagate proximally to the aortic valve or can propagate distally in a spiraled or straight manner. It is a relatively rare but highly fatal disease [6]. Determining incidence of aortic dissection is rather difficult due to most cases being diagnosed post mortem and often initially misdiagnosed. Estimated incidence is 2.0-3.5 people per 100.000 individuals every year [7]. A known history of high blood pressure, male sex, age in 60s and 70s, connective tissue disease that affects blood vessel walls, structural valve disease or previous heart surgery predisposes a risk for development of aortic dissection [8]. Less than 10% of all cases, aortic dissection occurs in individuals younger than age 40 – often normotensive, however a known history of cardiac surgery or bicuspid valve, Marfan syndrome, Ehler-Danlos syndrome or similar connective tissue diseases.

Classification varies by localization, duration, initial presentation. Thoracic aortic dissections are classified based on anatomic localization and extent of emergency of treatment. Two most frequently used system for classification, the Stanford system and DeBakey system, describe the anatomic range of dissection as opposed to the initial tear localization. Another classification, the European Society of

Cardiologists system for aortic dissection, classifies the aortic dissection in five groups and may be helpful in deciding which type of dissection are candidate for endovascular treatment (Table 1) [9].

DeBakey Classification	Type I	Originates in the ascending aorta, propagates to the aortic arch.
	Type II	Originates in and is confined to the ascending aorta.
	Type III	Originates in the descending aorta and extends distally down the aorta or, rarely, retrograde into the aortic arch and ascending aorta
Stanford Classification	Type A	All dissections involving the ascending aorta, regardless of the site of origin
	Type B	All dissections not involving the ascending aorta
European Society of Cardiology (ESC)	Class 1	Classic aortic dissection with intimal flap
	Class 2	Intramural Hematoma
	Class 3	Discrete dissection – no hematoma
	Class 4	Penetrating atherosclerotic ulcer
	Class 5	Iatrogenic/traumatic dissection

Table1: Classification schemes of aortic dissection

The DeBakey system categorizes the dissection based on where the initmal tear is localized and the extent of dissection – based on ascending aorta or descending aorta [10], whereas the Stanford classification is divided in two groups: group A, involves the ascending aorta and aortic arch, possibly descending aorta, and group B which involves descending aorta or the arch with no involvement of ascending aorta [11].

The most feared complication of aortic dissection is malperfusion syndrome [12]. Diminished blood flow results in ischemia followed by impairment of function. Manifested clinically based on specific system, extremity pain due to limb ischemia or abdominal pain due to visceral ischemia, or manifested

with laboratory abnormalities such as rising creatinine due to renal failure and physiologically with neurologic abnormalities related to stroke. The malperfusion syndrome is postulated to contribute to symptoms with two mechanisms: either with dynamic compression of true lumen, or a static mechanism owning to extension of the initmal flap into the branch vessels and subsequent thrombosis of false lumen and occlusion of true lumen [13]. 30% of patients presenting with type A aortic dissection (Stanford classification) also present with malperfusion syndrome versus type B dissection in which the malperfusion syndrome is approximately 20% [14,15]. Malperfusion is associated with significant rise in complications.

Smoking, hypertension, male Blood in the false lumen may occlude any branch of the involved aortic segment at its origin, leading to acute ischemia in the regions supplied by the artery gender, hypercholesterolemia, and a recent history of coronary artery bypass grafting are common predisposing risk factors for the rare presentation of isolated lower extremity ischemia due to aortic dissection [5]. In this case, our patient was typically hypertensive male with acute ischemia of the right lower limb due to occlusion of the right common iliac artery from the dissecting process.

Painless aortic dissection is not unlikely. It has been reported in previous studies with an incidence of 6.4 % and associated with other symptoms like syncope or lower extremity weakness or peripheral ischemia of lower extremity.

Initial treatment is aimed at the reduction of the systolic BP and left ventricular ejection fraction. The presence of any hemodynamic instability should be addressed and corrected immediately. Pain relief can be achieved by using IV morphine. Reduction of systolic BP should be achieved using beta-blockers such as IV propranolol, metoprolol, or esmolol due to the dual targets beta-blockers to left ventricular ejection fraction and heart rate. Calcium channels blockers like diltiazem is an alternative choice where beta-blockers are contraindicated (e.g., with comorbid chronic obstructive pulmonary disease). A majority of cases with very severe hypertension which may require a concurrent sodium nitroprusside infusion for appropriate BP control. Ideally, systolic BP should be reduced to 100–120 mmHg, mean BP should be reduced to 60–75 mmHg, and heart rate should be controlled at around 60 beats per minute. After these aims are achieved, the subsequent management of aortic dissection differs depending on the type of dissection.

Trans-esophageal echocardiography, besides trans-thoracic echocardiography, can be used for decision making in the emergency room with high accuracy. When a more spatial resolution is necessary, CT or MRI is used in addition. CT is the technique used most often in patients with suspected aortic dissection. The sensitivity is greater than 90%, specificity being 85%. CT scan with the contrast is used most often in patients with suspected aortic dissection. MRI has the highest accuracy for detecting any type of aortic dissection, as its sensitivity and specificity are almost 100% [16]. Aortography is highly valuable to diagnose classical aortic dissection. It is the standard technique for guiding interventions in aortic dissection.

Close follow-up of patients, after medically or surgically treated patients with aortic dissection, is to be carried out by a specialized team. It must include the assessment of signs of aortic expansion, aneurysm

formation, signs of leakages at anastomoses/stent sites recurrence of the dissection, aortic regurgitation and peripheral vascular compromise. The single most important goal is excellent blood pressure control (<135/80 mmHg). After hospital discharge, a regular outpatient visits at 1, 3, 6 and 12 months and thereafter yearly is recommended with careful repeated examinations, periodic chest X-rays and serial aortic imaging. If complications are suspected, the first choice is MRI, 2nd and 3rd choices being CT and TEE respectively [17,18].

Conclusion

Aortic dissection is an acute medical emergency, which has a wide range of clinical presentations. Isolated lower extremity ischemia, as the presenting feature of aortic dissection is rare. The high degree of suspicion and awareness of its characteristics are needed to diagnose the true origin of this condition, especially in the presence of known risk factors. The early diagnosis and prompt initial medical treatment are the keystones of management of aortic dissection. The combination of surgical and interventional endovascular approaches has improved clinical outcomes in certain subgroups of aortic dissection.

Acknowledgements

The authors are deeply indebted to Dr. P.S. Jairaj, Dr. V. John and Dr. Taha Delemi from the cardiothoracic department of Royal Hospital, Muscat for their kind cooperation and help.

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