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CASE REPORT

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An extensive chronic aortic dissection presenting with acute embolic stroke

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ABSTRACT

Herein, we present a rare case of extensive chronic aortic dissection with extension to bilateral subclavian arteries, bilateral common carotid arteries, right internal carotid artery, bilateral proximal external iliac arteries and simultaneous presentation of acute embolic stroke and seizure. The rarity of this case presentation and the presence of neurological features necessitated a high index of clinical suspicion to reach the definitive diagnosis. This study also demonstrates a unique situation requiring correlation between chronic aortic dissection and multi-organ system dysfunction from chronic ischemia.

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KEYWORDS
Chronic aortic dissection; beta blockers; stroke; seizure; imaging

1. Introduction

Aortic dissection may present in acute or chronic form. The most common presentation of acute aortic dissection is sudden-onset, severe, tearing chest and/or back pain [1]. Aortic dissection also presents in a chronic form that is divided into type A and type B, which is the same classification used to describe the subdivisions of acute dissection [2]. A chronic dissection is arbitrarily defined as one that has been present for greater than 14 days [3,4]. Type A aortic dissection usually presents in an acute catastrophic event rather than a chronic ailment. By contrast, type B aortic dissection presents more commonly as a chronic condition, although acute type B is also present [5,6]. With regard to treatment modalities, type A dissection is more amenable to surgical correction, whereas type B dissection is initially controlled with antihypertensive medication and followed up with surgery should the pharmacological approach be unsuccessful [2,5,6]. All medically treated and many of the few surgically treated acute descending aortic dissections progress to a chronic phase that is characterized by a variable degree of aneurysmal dilatation, false lumen thrombosis, and dissection flap (septal) fenestrations [2].

2. Case report

A 45-year-old African-American female with past medical history of hypertension, tobacco abuse and DeBakey type 1 thoracic aortic dissection status post-repair three years ago was admitted to our hospital for the evaluation of new onset seizure. She did not experience upper extremity numbness or weakness or pain or any symptoms of amaurosis or Transient Ischemic Attack. She denied any leg pain or pain at rest. Her past medical history was significant for an 11-year history of essential hypertension treated with captopril and atenolol. She also underwent repair of an aortic dissection three years ago. Physical examination revealed a blood pressure of 137/78 mm of Hg, pulse of 64 bpm, temperature of 98.6°F, and oxygen saturation of 98% on room air. Her heart and lung examinations were completely within normal limits. Carotid auscultation revealed normal carotid upstrokes, normal radial pulses bilaterally, with bilaterally normal popliteal, posterior tibial, and dorsalis pedis artery pulses. Laboratory investigations showed normal complete blood count, electrolytes and cardiac enzymes. Lipid panel showed cholesterol of 133 mg/dl, triglyceride of 59 mg/dl, HDL of 56 mg/dl and LDL of 65 mg/dl. Chest X-ray revealed mildly enlarged cardiomediastinal silhouette. CT of the brain was obtained, which showed evidence of a new right parietal and left frontal cortical infarct. In addition to definitive stroke management, steps were taken to localize the source of emboli, characterize the extent of the pre-existing aortic dissection, and determine whether the stroke was attributable to the dissection. Carotid Doppler ultrasound revealed dissection of the right common and internal carotid arteries. Computerized tomography angiography (CTA) of the neck revealed extensive arterial dissection involving the aortic arch, great vessel origins, the entire length of the right common carotid artery and the proximal right internal carotid artery. CTA of the chest and back showed evidence of chronic dissection of the ascending and descending thoracic aorta extending into the abdominal aorta to the bilateral infrarenal aorta

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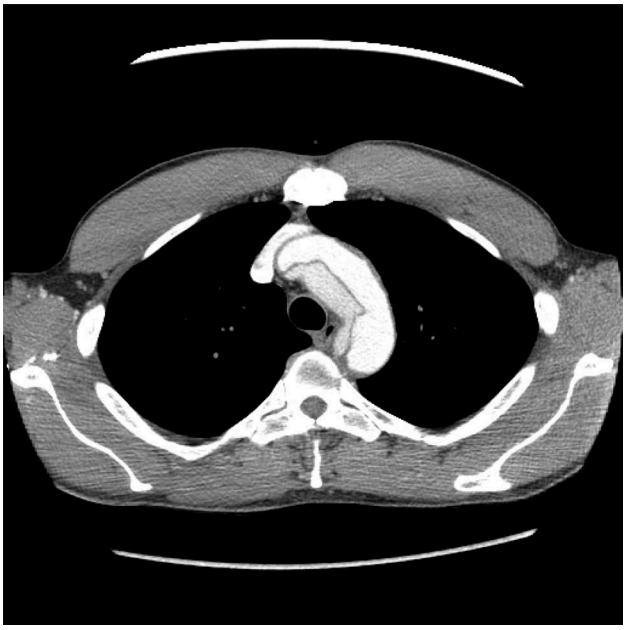

Figure 1. Aortic dissection at the level of the upper chest.

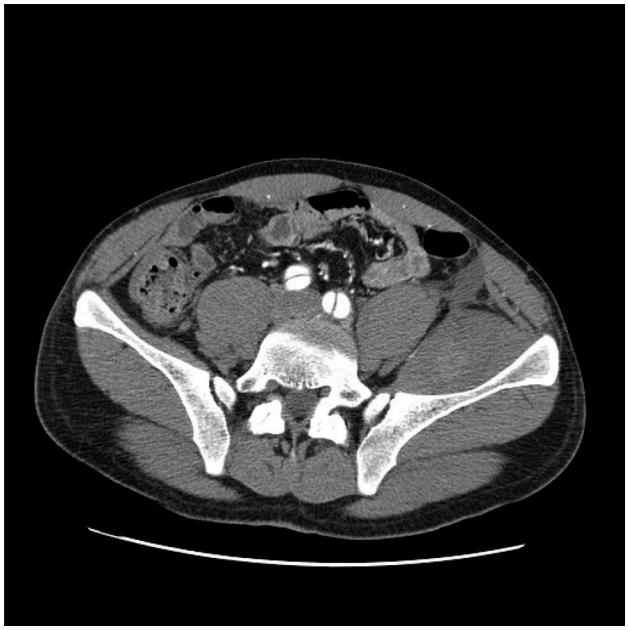

Figure 3. Aortic dissection at the level of the pelvis. Figure 1. Aortic dissection at the level of the upper chest.

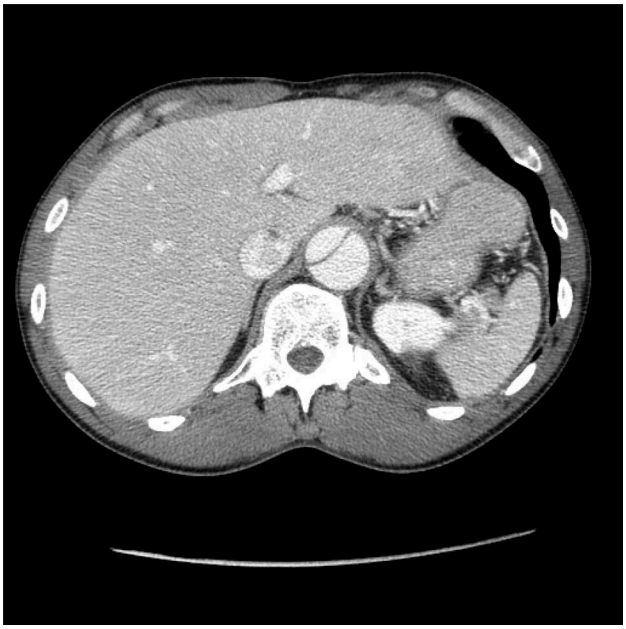

Figure 2. Aortic dissection at the level of the upper abdomen.

(Figures 1 & 2). Transthoracic echocardiography showed moderate aortic root enlargement without any evidence of clots or vegetation. Transesophageal echocardiogram (TEE) confirmed the absence of clots or vegetation. Magnetic resonance imaging (MRI) brain showed bilateral acute cortical infarcts involving the left parietal and right temporal occipital lobes. CT scan of the pelvis revealed aortic dissection extending into bilateral common iliac and proximal external iliac arteries (Figure 3). For her presumed embolic events, she was placed on heparin and Coumadin. Vascular surgery and cardiology consultation were obtained. Conservative medical management was recommended for her chronic aortic dissection.

3. Discussion

Any mechanism that initiates the weakening of the media layer of the aorta eventually leads to aortic dissection. The risk factors for aortic dissection include longstanding hypertension, connective tissue disorders, vascular malformations, vascular inflammation, trauma, and iatrogenic related to cardiac or aortic surgery and instrumentation [1,6]. In the consideration of associations with the development of aortic dissection, hypertension is the most common risk factor. The patient had hypertension as well as a previous history of vascular surgery [7]. Classically, aortic dissection is seen in a male with a history of hypertension in the seventh decade of life. Females constitute less than one-third of total aortic dissection cases, and the mean age of presentation in females is higher (67.9 years) compared to males (60.6 years); however, our patient is a relatively young female with the aforementioned risk factors [3]. Aortic dissection is typically an acute event characterized by sudden excruciating chest pain but may present with atypical or no symptoms [8–10]. Chronic dissection may rarely present with persistent fever, malaise and bilateral pleural effusions [9,11]. Chronic aortic dissection should be considered as a possible cause of fever of unknown origin in a patient with a past history of hypertension and a relatively recent episode of chest, back or abdominal pain [9].

3.1. Presentation

Symptoms of chronic dissection are often vague, non-specific, and related to the mass effect of the aneurysm [2]. Classic type A aortic dissection predisposes to ischemic stroke as the most common cause of focal neurological deficits in approximately one-third of cases [3,12]. Chronic aortic dissection may present with varied and atypical features that require a high index of clinical suspicion with close, long-term follow-up and treatment once diagnosed. The features of chronic aortic dissections depend on the extent and the type of the arteries involved. Chronic type A dissection may present with blood pressure discrepancy in the upper extremities, Horner syndrome and hoarseness. If the dissection extends to the carotid arteries, neurologic symptoms such as stroke or altered mental status may occur. With regard to chronic type B dissection, there may be sustained loss of peripheral pulses, impaired renal perfusion, compromised visceral perfusion and other signs [4]. Occasionally, there may be no suggestive or pathognomonic signs of aortic dissection [13].

3.2. Diagnosis

Any clinical suspicion of acute aortic syndrome should prompt confirmatory non-invasive imaging. With respect to sensitivity and specificity for acute aortic pathology, contrast-enhanced spiral CT scanning, Transthoracic or Transesophageal Echocardiography (TTE or TEE), and MRI all have comparable accuracy of near 100% [1,14]. The selection of test depends upon a number of factors such as availability, urgency, confirmation of the tear, localization, extent, classification and determination of emergent intervention [1]. In acute phase, CT is the most used imaging technique owing to its wider availability and accuracy [3,15]. Transthoracic echocardiography, although less accurate than CT, is useful in hemodynamically unstable patients as it is portable [15]. Other supportive workup modalities include electrocardiogram, Chest X-ray, biomarkers (D-dimer, Troponin T, Troponin I) [16]. It is suggested that left ventricular diastolic function is severely reduced in patients having aortic dissection with a double-barrel and narrowed true lumen [17]. The gold standard for diagnosis of aortic dissection is spiral CT angiogram as its sensitivity of the chest, abdomen and pelvis for detection of aortic dissection is 97% and the specificity is 100% [18]. Magnetic resonance angiography is equally precise and used especially in patients with renal insufficiency. TEE is accurate in the diagnosis but does not provide anatomical details for the monitoring of type B chronic aortic dissection. Occasionally, the patient may never have symptoms, and the diagnosis is made from imaging studies obtained during the workup of unrelated problems [14].

3.3. Management

Classical treatment recommendations are direct surgical intervention for type A and medical treatment for type B aortic dissection in the absence of complications. Continuous medical treatment is a strong recommendation for long-term management irrespective of the type of aortic dissection and initial treatment [19]. With chronicity of dissection, aortic remodelling continues, but there is a decline in morbidity, mortality and need for intervention. Therefore, the distinction between acute and chronic dissection is important to make [20]. Chronic aortic dissections are treated with anti-hypertensive drugs while keeping the patient on strict periodic follow-up with imaging surveillance and careful periodic clinical follow-up. The goal for blood pressure is less than 140/90, maintained with beta-blockers, calcium channel blockers or angiotensin receptor blockers [15]. Various surgical approaches have been recommended, which should be tailored as per the type, presentation and desired outcome. Early outcomes are favourable and late outcomes are less than desirable after open repair of chronic distal aortic dissection, regardless of the extent of repair. High-risk and late-stage patients with larger and more extensive aneurysmal degeneration warrant further investigation, including the use of newer, less-invasive techniques. Select patients at risk for aneurysmal degeneration should undergo a more aggressive initial approach with aortic dissection repair [21]. Less commonly, patients may present with frank rupture and shock.

A prospective multi-centre comparative study on the treatment of type B aortic dissection showed that thoracic endovascular aorta repair had a significantly lower aorta-related mortality compared with optimal medical therapy but failed to improve overall survival rate or lower the aorta-related adverse event rate [22]. For ongoing surveillance, patients who undergo thoracic endovascular aortic repair for aortic dissection should undergo a CT or MRI at 6 months and then an annual CT or MRI. They should also continue on long-term anti-hypertensive medication, preferably a beta-blocker or angiotension-converting-enzyme inhibitor, with a goal blood pressure of <140/90 mmHg [23].

4. Conclusion

Chronic aortic dissection may remain asymptomatic despite widespread extension. If there is no alternative explanation, aortic dissection should be suspected in a hypertensive patient who presents with typical or atypical symptoms in order to avoid missing this potentially life-threatening condition. Medical management

is recommended, unless there is occlusion of a major aortic branch leading to end-organ ischemia, persistent severe hypertension or pain, progressive dissection, aneurysmal degeneration and rupture.

Disclosure statement

No potential conflict of interest was reported by the authors.

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References

[1] Tsai TT, Nienaber CA, Eagle KA. Acute aortic syndromes. Circulation. 2005;112(24):3802–3813.
[2] McGee ECJ, Pham DT, Gleason TG. Chronic descending aortic dissections. Semin Thorac Cardiovasc Surg. 2005;17(3):262–267.
[3] Hagan PG, Nienaber CA, Isselbacher EM, et al. The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease. JAMA J Am Med Assoc. 2000;283(7):897–903.
[4] Fann JI, Sarris GE, Mitchell RS, et al. Treatment of patients with aortic dissection presenting with peripheral vascular complications. Ann Surg. 1990;212(6):705–713.
[5] Genoni M, Paul M, Jenni R, et al. Chronic beta-blocker therapy improves outcome and reduces treatment costs in chronic type B aortic dissection. Eur J Cardio-Thorac Surg Off J Eur Assoc Cardio-Thorac Surg. 2001;19(5):606–610.
[6] Stanger O, Oberwalder P, Dacar D, et al. Late dissection of the ascending aorta after previous cardiac surgery: risk, presentation and outcome. Eur J Cardiothorac Surg. 2002;21(3):453–458.
[7] Nienaber CA, Eagle KA. Aortic dissection: new frontiers in diagnosis and management part I: from etiology to diagnostic strategies. Circulation. 2003;108(5):628–635.
[8] Smith MA, Singer C. Fever of unknown origin: unusual presentation of dissecting aortic aneurysm. Am J Med. 1988;85(1):126–127.
[9] Gorospe L, Sendino A, Pacheco R, et al. Chronic aortic dissection as a cause of fever of unknown origin. South Med J. 2002;95(9):1067–1070.
[10] Schattner A, Klepfish A, Caspi A. Chronic aortic dissection presenting as a prolonged febrile disease and arterial embolization. Chest. 1996;110(4):1111–1114.
[11] Magri J, Corso R, Manche A, et al. Aortic dissection: an unusual presentation. Br J Hosp Med Lond Engl 2005. 2013;74(5):290–291.
[12] Lee S-J, Kim J-H, Na C-Y, et al. Eleven years of experience with the neurologic complications in Korean patients with acute aortic dissection: a retrospective study. BMC Neurol. 2013;13(1):46.
[13] Wong T, Kilner PJ, Gatzoulis MA. Asymptomatic aortic dissection in Marfan syndrome. Heart. 2002;87(1):66.
[14] Nienaber CA. The role of imaging in acute aortic syndromes. Eur Heart J Cardiovasc Imaging. 2013;14(1):15–23.
[15] Nauta FJ, Trimarchi S, Kamman AV, et al. Update in the management of type B aortic dissection. Vasc Med Lond Engl. 2016;21(3):251–263.
[16] Nienaber CA, Powell JT. Management of acute aortic syndromes. Eur Heart J. 2012;33(1):26–35.
[17] Shingu Y, Shiiya N, Mikami T, et al. Left ventricular diastolic dysfunction in chronic aortic dissection. Ann Thorac Surg. 2007;83(4):1356–1360.
[18] Hansmann HJ, Doebert N, Richter GM, et al. Different spiral CT protocol types and their significance in the diagnosis of aortic dissections: results of a prospective study. RoeFo - Fortschritte Auf Dem Geb Roentgenstrahlen Neuen Bildgeb Verfahr. 2000;172(11):879–887.
[19] Tolenaar JL, Van Bogerijen GHW, Eagle KA, et al. Update in the management of aortic dissection. Curr Treat Options Cardiovasc Med. 2013;15(2):200–213.
[20] Nienaber CA, Rousseau H, Eggebrecht H, et al. Randomized comparison of strategies for type B aortic dissection: the INvestigation of STEnt Grafts in Aortic Dissection (INSTEAD) trial. Circulation. 2009;120(25):2519–2528.
[21] Pujara AC, Roselli EE, Hernandez AV, et al. Open repair of chronic distal aortic dissection in the endovascular era: implications for disease management. J Thorac Cardiovasc Surg. 2012;144(4):866–873.
[22] Jia X, Guo W, Li T-X, et al. The results of stent graft versus medication therapy for chronic type B dissection. J Vasc Surg. 2013;57(2):406–414.
[23] Erbel R, Aboyans V, Boileau C, et al. 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The task force for the diagnosis and treatment of aortic diseases of the european society of cardiology (ESC). Eur Heart J. 2014;35(41):2873–2926.