

A pulled muscle," - Aortic Dissection

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Citation

A Bhagra, N Anand, M Fernanda Bellolio. *A pulled muscle," - Aortic Dissection*. The Internet Journal of Radiology. 2007 Volume 9 Number 1.

Abstract

We report a case of aortic dissection in a patient with hypertension who presented with chest pain. Chest pain has many causes, including myocardial infarction, pulmonary embolism, pericarditis, pneumothorax, pneumonia, pleurisy, acute pancreatitis, and penetrating duodenal ulcer, making diagnosis difficult. In the acute setting, management of aortic dissection is limited to control of hypertension, early recognition of the dissection, and activation of surgical services. To reduce morbidity and mortality, aortic dissection should always be considered in patients who present with severe chest pain of abrupt onset.

INTRODUCTION

Aortic dissection is a potentially fatal process that occurs when a false lumen is created in the aorta either proximally or distally to the left subclavian artery, with extension distally to the abdominal aorta (1). Most patients who are not in the vicinity of medical care will die of free pleural rupture or renal or visceral complications, but the systemic symptoms may also include shock, dyspnea, stroke, paraplegia, anuria, abdominal pain, or extremity ischemia (2). Because aortic dissection can remain silent, with only subtle clues, the diagnosis can easily be missed. Late diagnosis or missed diagnosis can result in considerable mortality if rupture occurs.

We report the case of a patient with numerous cardiovascular risk factors who presented with chest pain and discuss the predisposing factors, diagnosis, and treatment options both acutely in the emergency department (ED) and long term.

CASE REPORT

A 72-year-old man with a 1-hour history of right-sided chest pain came to the ED by ambulance, accompanied by his wife. The patient stated that he experienced an abrupt pain that traveled from his chest to the tip of his scapula as he bent over his desk to reach for something in the wastebasket. The chest pain was relieved by sublingual nitroglycerin, which the patient took before coming to the ED. The pain was rated as 2 on a scale of 10 (2/10). He has had similar chest pain previously. He had past medical history of hypertension and gastroesophageal reflux disease. His past surgical history was notable for an L3 disectomy and a total

knee arthroplasty. He had no medication allergies, or relevant family history. The review of systems was noncontributory. Again and again, the patient stated that the pain felt as though he "had pulled a muscle."

The patient was alert and oriented. The vital signs were as follows: pulse 69 beats/min; respiratory rate 14 breaths/min; and blood pressure 184/123 mm Hg. Oxygen saturation was 97% with room air. Immediately on arrival, he was given oxygen 2 L/min, through a nasal cannula, and a normal saline intravenous catheter was placed. Aspirin 325 mg, and sublingual nitroglycerin 0.4 mg, were administered. Despite this treatment his pain remained at 2/10. Morphine 2 mg, and diazepam 5 mg, were given intravenously, but the pain persisted. The color and appearance of the skin were normal, without diaphoresis. The head was normal, with moist mucous membranes. Jugular venous pressure was estimated at 4 cm H₂O. On cardiovascular examination, the heart was in normal sinus rhythm, with no audible gallops or rubs. A 2/6 systolic ejection murmur was heard at the upper right sternal border. There was no change with a Valsalva maneuver. The lung fields were clear. Findings on abdominal examination were unremarkable. Examination of the extremities did not show edema, cyanosis, or erythema. Pulses were all within normal limits.

The results of a complete blood count, electrolyte panel, and cardiac enzyme tests were all within normal limits. Electrocardiography (ECG) revealed occasional premature ventricular contractions, but otherwise no changes were noted when compared with a previous ECG. Chest radiography showed a normal cardiac silhouette and clear

lung fields. Two sets of cardiac enzyme tests and ECGs were negative, and the patient underwent an exercise treadmill test, on which he achieved a functional aerobic capacity of 32% and a Duke score of 1.4. The latter placed him in an intermediate risk category; however, because he had good follow-up and was reluctant to be admitted for observation, it was thought safe to discharge him to home.

The differential diagnosis included myocardial infarction, gastroesophageal reflux disease, musculoskeletal pain, and angina. Aortic dissection was included in the initial differential diagnosis but was thought unlikely because of the absence of such objective findings as a difference in blood pressure between the arms or a widened mediastinum seen on chest radiography.

After the initial work-up in the ED, the patient was discharged and told to return if the pain persisted or worsened. He returned to the ED the next day because his symptoms persisted, and computed tomography of the abdomen, chest, and pelvis were performed. The scan showed a Stanford type B dissection extending from the distal end of the left subclavian artery to the iliac arteries (Figs. 1 and 2).

Figure 1

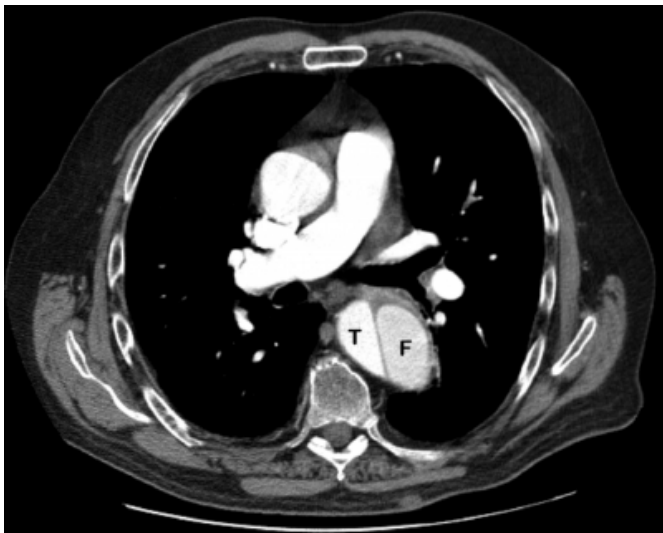
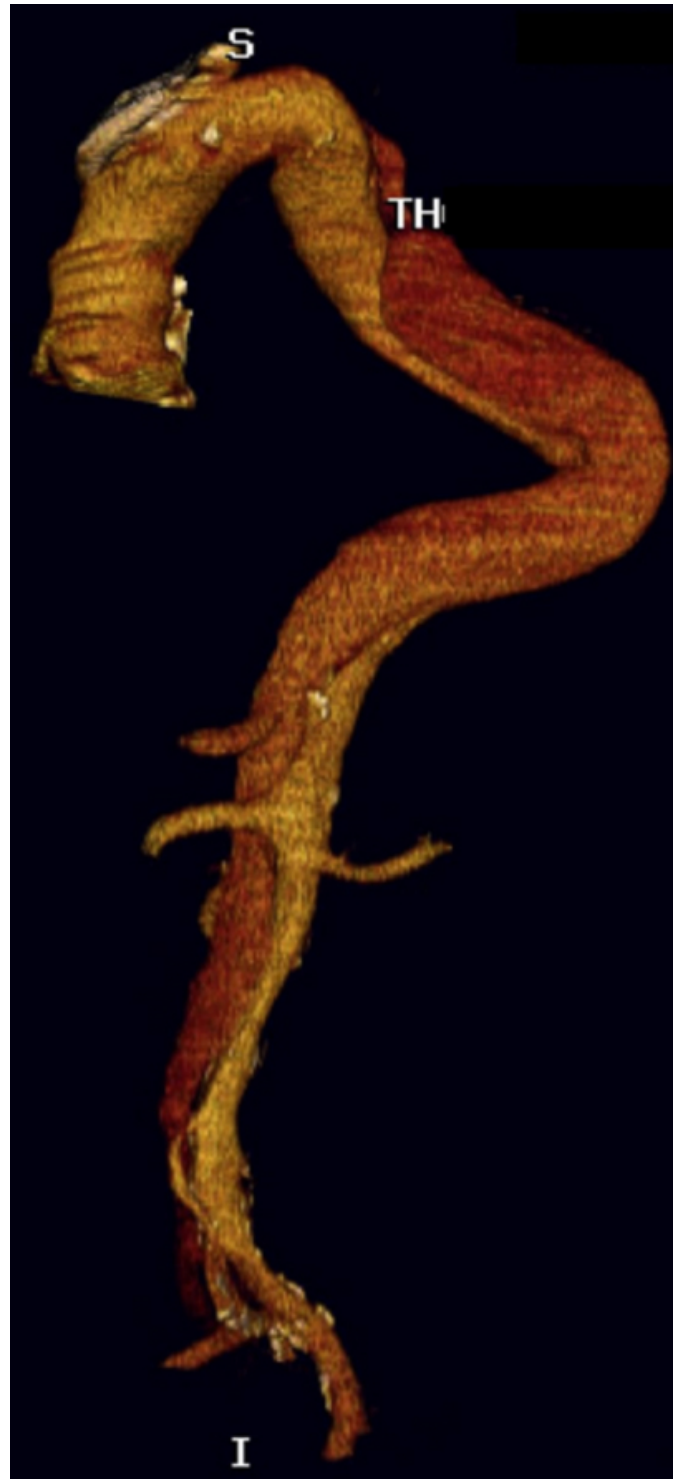


Figure 2



DISCUSSION

Chest pain is a vague complaint that encompasses numerous conditions, including myocardial infarction, pulmonary embolism, pericarditis, pneumothorax, pneumonia, pleurisy, acute pancreatitis, and penetrating duodenal ulcer. Although the exact cause of aortic dissection is uncertain, the reported incidence is 5 to 30 cases per million population annually (3).

Two classification schemes are used to describe aortic dissection: the Stanford and the DeBakey classifications. Stanford type A dissection involves the ascending or distal aorta (or both), and a type B dissection involves the descending aorta distal to the left subclavian artery. DeBakey type I dissection involves both the ascending and descending aorta, type II involves only the ascending aorta, and type III involves only the descending aorta.

Risk factors for aortic dissection include male sex, age 50 to 55 years for proximal dissection and 60 to 70 years for distal dissection, chronic systemic hypertension (present in 62% to 78% of patients with aortic dissection), aortic aneurysm, cocaine use, pregnancy, Turner syndrome, Noonan syndrome, Marfan syndrome, and Ehlers-Danlos syndrome (3).

Several theories have been proposed to explain the pathophysiologic mechanism of aortic dissection, and their common feature is the creation and propagation of an intimal flap. The propagation continues until a rupture occurs either into the lumen or through the adventitia. According to one theory, this may be due to cystic medial necrosis (4,5). Degeneration of the media of the aorta weakens the cohesion between the aortic layers, allowing the creation of an intimal flap. Cystic medial degeneration generally is more extensive in older persons and in those with hypertension or Marfan syndrome (3). According to other studies, intimal flaps tend to form in areas of the aorta subjected to great pressure changes and flexion stress. These areas include the ascending aorta and the first part of the descending aorta. Another theory proposes that penetrating atherosclerotic aortic ulcers weaken the aortic wall and predispose to aortic vascular pathology. This tends to occur in the descending thoracic aorta of elderly persons (3).

Aortic dissection classically presents with pain described as "sharp," "ripping," "tearing," and "abrupt." The location of the pain depends on the site of the dissection. According to a report from the International Registry of Acute Aortic Dissection, the most common pain was chest pain (73% of patients), followed by back pain (53% of patients) and abdominal pain (30% of patients) (6). The pain is differentiated from the pain of myocardial infarction by the absence of radiation into the neck, shoulder, or arm unless there is associated coronary artery extension. Furthermore, more than one-third of patients may have symptoms due to organ system involvement such as aortic regurgitation, stroke, pleural effusion, and acute gastrointestinal

hemorrhage. Our patient had abrupt onset of chest pain, but he did not have any signs of organ system involvement.

The diagnosis of aortic dissection requires a high degree of clinical suspicion because the symptoms and clues can be subtle. A missed diagnosis can be disastrous. The diagnostic work-up should begin with an ECG. However, the work-up must not stop with this test because the presence of a myocardial infarction does not rule out dissection. Multiple imaging modalities can be used if aortic dissection is suspected. These include CT, magnetic resonance imaging, and transesophageal echocardiography, which have the highest specificity, followed by transthoracic echocardiography and chest radiography, which are less specific (3). If a dissection is found, it is important to differentiate a proximal dissection from a distal one for purposes of treatment.

Treatment depends on the status of the patient, the degree of dissection, and the progression status of the dissection. In the ED, the mainstay of treatment is control of hypertension. Many treatment options are available for lowering blood pressure; currently, a β -blocker and a vasodilator are initially administered (3). Patients whose condition is stable, who have a distal dissection, or for whom surgery is contraindicated may be managed medically. The goal is to limit the extension of the dissection, to stabilize the dissection, and to prevent complications (7). If signs and symptoms of extension or worsening of the clinical condition becomes evident, surgery should be considered. Proximal dissections should be treated surgically. Numerous techniques are used, depending on the expertise of the surgeon and circumstances of the patient. Recently, endovascular stenting has been gaining popularity and is being used to manage distal dissections, especially when symptoms of organ or lower extremity malperfusion develop.

CONCLUSION

Aortic dissection is a potentially disastrous process that can be missed when a patient presents with a chief complaint of chest pain. This diagnosis should be considered for patients with hypertension and other cardiovascular risk factors who present with chest pain that is maximal at onset and described as "ripping" or "tearing," especially if the pain radiates to and down the back. An incorrect or missed diagnosis can result in rupture and a likely fatal outcome.

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