The Wolf in Sheep's Clothing: An Illustrative Case Report Of Aortic Dissection And Review Of Diagnostic Clinical Features

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Citation

Abstract
A 54 year-old man with hypertension presented to the emergency department with chest pain, nausea and syncope. Initial arterial blood pressure was high and pulse examination was normal. Cardiac auscultation revealed a murmur of mild aortic sclerosis. 12-lead electrocardiogram showed normal sinus rhythm, 1 mm ST depression in the lateral leads and left ventricular hypertrophy and no dynamic ST or T wave changes were noted subsequently. Cardiac enzymes levels were negative. Chest pain subsided with initial treatment, but recurred after discontinuation of the medications. Chest X-ray (CXR) showed mild increase in cardiac silhouette size and slightly prominent ascending aorta. In view of recurrent chest pain, syncope and abnormal CXR, an emergent computerized tomogram (CT) of the chest was performed. A Stanford Type 'A' dissection of aorta with typical double barrel appearance (figure 1) was found. Emergent surgical repair of the dissecting aneurysm as well as pericardial effusion found intra-operatively was successfully performed and patient recovered.

INTRODUCTION
Chest pain is a common presenting symptom in the ED contributing to 5% of total ED visits. Yet, only 15% of these patients have chest pain from acute myocardial infarction related to coronary artery disease (CAD). Due to heightened awareness and malpractice litigation, ED physicians tend to consider CAD as differential diagnosis in every individual presenting with chest pain. However, other diagnoses that are potentially lethal could be overlooked. We present a teaching case of “chest pain, rule out myocardial infarction” with interesting findings and discuss the pitfall of this routine approach to chest pain in the ED.

CASE REPORT
A 54 yr-old male with uncontrolled hypertension, Parkinson's disease presented to the ED with acute chest pain, nausea, sweating and syncope. He described a severe pressure-like sensation in the left precordium with radiation to his left arm. Initial vitals were: BP 280/110 mmHg; Pulse Rate 80/min; Respiratory Rate 19/min. BP and pulse were equal in all four limbs. Cardiac exam revealed mild aortic sclerosis. There was no carotid bruit. Neurological examination was normal. 12-lead standard electrocardiogram (EKG) showed 1 mm ST segment depression in the lateral chest leads and left ventricular hypertrophy with no dynamic ST or T wave changes on subsequent recordings. CXR revealed mild increase in cardiac silhouette size and slightly prominent ascending aortic silhouette with normal mediastinal width. Initial cardiac enzyme set was negative. Lab data included hematocrit 39% and serum creatinine 1.3mg/dl. An initial diagnosis of “chest pain, rule out myocardial infarction” was entertained and a decision was made to admit the patient. He was initially administered aspirin, brief anticoagulation, parenteral morphine and nitroglycerin infusion. After initials vitals were stabilized, cardiac symptoms resolved momentarily. Soon after admission, severe chest pain recurred with discontinuation of nitroglycerin infusion. A repeat EKG was found to have similar ST segment changes compared to the initial EKG and had no other dynamic ischemic abnormalities. In view of recurrent chest pain disproportionate to the electrocardiographic and roentgenographic abnormalities, anticoagulation and anti-platelet therapy was withheld and a computerized tomogram (CT) of the chest was performed. A Stanford Type ‘A’ dissection of aorta extending from the aortic root to the bifurcation of aorta with typical double barrel appearance (Figure 1) was found.
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Figure 1
Figure 1: Contrast enhanced CT slice of the chest cavity showing double barrel appearance of the ascending and descending aorta.

The patient underwent emergent aortic repair and pericardial window for pericardial effusion noted intra-operatively. His post-operative course remained uneventful and the patient was discharged with primary care follow-up.

DISCUSSION

It is of paramount importance in the ED to carefully consider in patients with chest pain for all potentially lethal diagnoses such as aortic dissection or pulmonary embolism in addition to myocardial infarction. We compared our case to those reported in literature to understand and identify clinical clues that could be useful to clinicians in the diagnosis of aortic dissection.

Chest or back pain, often described as “tearing,” is the classic presentation of aortic dissection. However this type of pain is rarely seen. In a large series of aortic dissection cases described by Armstrong et al, a combination of chest pain and back pain were common symptoms present in 81% of patients, while chest pain was alone was present only among 41% of patients. In about 1/3 rd of the cases, the chest pain was dull and myocardial ischemia was suspected as cause of pain. The character and location of chest pain did not have good predictive value in the diagnosis of aortic dissection. In our case also, chest pain was reported as pressure like sensation radiating to left arm which is typical of CAD and not a characteristic feature of aortic dissection.

The presence of pulse deficits or focal neurological symptoms and deficits on clinical examination raises the likelihood of an acute thoracic aortic dissection. In our patient, there were no pulse deficits, but he presented with syncope which by itself occurs rarely in aortic dissection and therefore is very unlikely to prompt a search for aortic dissection by most clinicians. Aortic regurgitation can occur in 1/3rd of these patients, which was absent in our case.

Plain chest radiography is of limited value in screening for aortic dissection. Firstly, a widened mediastinal shadow observed in patients with aortic dissection can occur due to non-dissecting aortic abnormalities. Further, inter-observer variability in interpretation of chest roentgenograms for aortic pathology can be significant. Subtle abnormalities on chest roentgenograms may increase sensitivity of diagnosing aortic pathology but may be reliably detected by clinicians. A normal chest roentgenogram lowers the likelihood of aortic dissection. In our patient, CXR showed a subtle aortic abnormality that served as an additional clue for investigating aortic dissection.

While normal 12 lead EKGs are noted in aortic dissection cases, nonspecific EKG abnormalities have been reported in many cases. As in our case, the presence of abnormalities suggesting myocardial ischemia such as ST segment or T wave changes can occur in aortic dissection and are especially problematic given the potential for misinterpretation of the presentation as acute coronary syndrome.

In our patient, nitroglycerin administration resulted in improvement in chest pain, which is not a feature of aortic dissection. However, response to nitroglycerin must not be considered as suggestive of myocardial ischemia. Blood pressure and pulses were equal in all four limbs without any new bruit in the major vessels. CXR did not show a widened mediastinum. On one hand, this patient appears to have been treated appropriately for acute coronary syndrome initially, considering the presence of pressure-type chest pain, ST-segment abnormalities, and normal mediastinal width on chest radiograph. However, neurological symptoms such as dizziness, confusion and syncope may be associated with aortic dissection as in our case, who had syncope. More importantly, significant recurrent chest pain with minimal electrocardiographic and radiological abnormalities and relatively normal physical examination findings in our patient served as the main clues in the diagnosis of aortic dissection.
Patients who have atypical clinical symptoms or subtle abnormalities on EKG and chest roentgenograms are at higher risk of receiving anticoagulation for acute coronary syndrome or pulmonary embolism diagnoses when actually they were noted to have aortic dissection later. Our case certainly fits the description of those found in other studies to be at higher risk for anticoagulation. In our patient anticoagulation was withheld as soon as diagnosis of aortic dissection was suspected and hence did not adversely impact patient outcome.

CONCLUSION

Our case underscores the importance of considering aortic dissection as differential diagnosis of chest pain in the ED. “Chest pain, rule out myocardial infarction” approach must not apply for all patients with chest pain in the ED. This is especially important as anticoagulation administration necessary for CAD or acute pulmonary embolism may be detrimental for certain patients presenting with chest pain due to other potentially lethal conditions such as aortic dissection. The presentation of aortic dissection consists of more often atypical rather than classic symptoms or signs and imaging results, and this hidden danger metaphorically could be “the wolf in sheep's clothing” and therefore needs to be recognized early. A high index of suspicion, features such as neurological symptoms, lack of response to initial treatment of suspected CAD, chest pain that is disproportionately severe with subtle electrocardiographic, radiographic and physical findings as in our case may be important clues for this potentially lethal diagnosis.

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