"Yet another chest pain to clerk? - Watch the murmur!"

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Abstract:

Most doctors in acute medical specialties are familiar with the management of patients presenting with chest pain. Whilst the range of differential diagnoses is wide, in practice the most common presentations are acute coronary syndrome (ACS), pulmonary embolus (PE) and musculoskeletal chest pain. Occasionally rarer (and potentially very serious) causes are seen, and it is then of utmost importance that clinicians recognize these patients appropriately. Often subtle clues in the history or abnormalities in the physical examination raise suspicion that the patient might not be the average "yet another chest pain".

Case report:

A 40 year old man presented to the emergency department (ED) with a few hours' history of sudden onset chest central chest pain whilst exercising on his home trainer. It was described as a sharp pain in the centre of his chest radiating to his back that changed with posture and respiration. His past medical history and family history were unremarkable. He did not take any medication and had no allergies. On examination he was comfortable, hemodynamically stable, and pain free after some ibuprofen. His chest was clear although a systolic-murmur was noted. His pulse was 76, blood pressure 130/80 bilaterally and oxygen saturations were 100% on room air. The ECG was normal, and chest X-ray is shown in figure 1. Blood results revealed elevated high sensitivity troponin of 40.00 (normal <40ng/l) and D-Dimer of 657 (normal <300). He was seen the next morning by the post-take consultant who noted a diastolic murmur. Urgent bedside transthoracic Echo with a handheld device (GE VScan™) revealed significant aortic regurgitation, a markedly dilated aortic root and a visible dissection flap (figure 2). The patient was transferred to CCU and underwent urgent contrast CT-Aortography that revealed a type A aortic dissection with marked 9cm enlargement of the ascending aorta (figure 3). Intravenous metoprolol was administered and the patient was started on a labetalol infusion to lower his blood pressure and heart rate. He was transferred urgently to a tertiary centre for operative repair and was discharged home a week later.

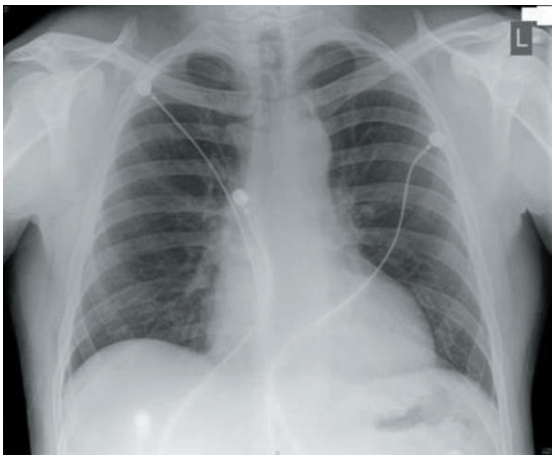

Figure 1. *Chest X-Ray:*

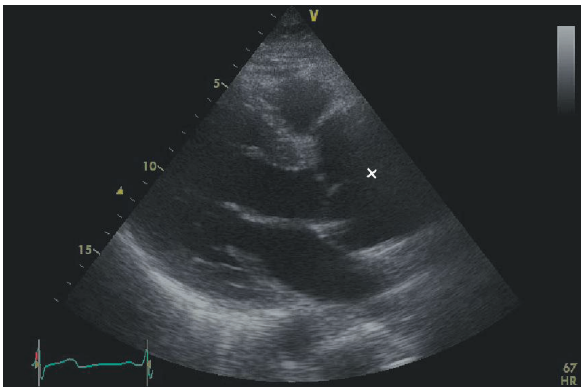

Figure 2. *Transthoracic Echo (parasternal long axis view): markedly dilated aortic root (marked with "x") with hint of dissection flap seen anteriorly. Also visible in this view are open mitral valve (with anterior and posterior leaflets pointing towards 10cm depth marker), and the thin aortic valve leaflets.*

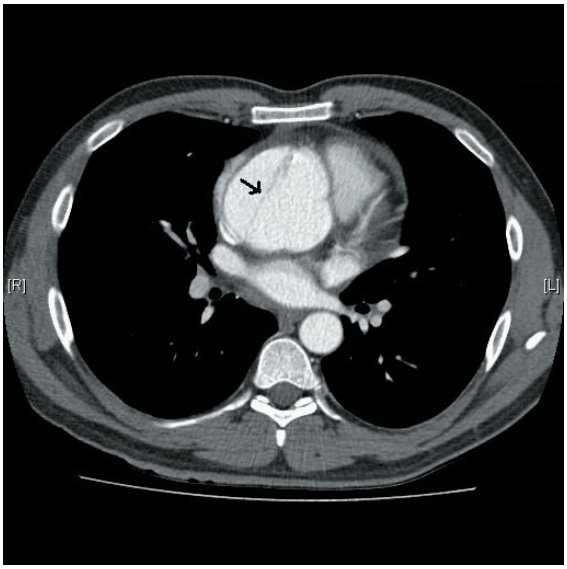

Figure 3. *CT-Aortogram: dilated ascending aorta with visible dissection flap (arrow)*

Discussion

Aortic dissection is a tear in the aortic intima resulting in blood separating the media from the adventitia. Patients usually present with a sudden onset of chest pain (70%) radiating to the back (50%), especially the interscapular area. The pain typically is described as "the worst pain ever" (90%), of immediate severity (in contrast to pain from myocardial infarction that increases over time), and often of a tearing or ripping nature (50%). Occasionally there might be radiation to the abdomen (30%), neck or jaw. About 10% of patient will have no pain. Sometimes ECG abnormalities will be present, usually ST-depression, T-wave inversions or signs of left ventricular hypertrophy. Rarely (1-2%) patients with aortic dissection may present with inferior ST-elevation, indicating involvement of the aortic root and the origin of the right coronary artery. Anterior STEMI (ST elevation myocardial infarction) in this context would be extremely unusual, as dissection of the left main coronary artery would likely be fatal immediately.

Physical examination may reveal pulse and blood pressure differences between left and right arm, low diastolic blood pressure, a diastolic murmur due to involvement of the aortic valve producing incompetence, focal neurological deficits (due to involvement of the carotid vessels in 15-20%) or raised JVP with soft heart sounds (indicating extension in the pericardial space causing an effusion with tamponade). Sometimes tamponade causes pulsus paradoxus - the disappearance of the radial pulse with deep inspiration - due to the increased RV-filling compressing the LV and decreasing cardiac output. The murmur of aortic regurgitation (AR) is often short and difficult to hear due to tachycardia, but often crucial in diagnosing or suspecting aortic dissection. The inexperienced clinician is advised to listen carefully for the second heart sound, after which diastole starts. Any noise heard immediately after this second heart sound is suspicious for AR.

Chest auscultation may reveal pulmonary oedema, as acute AR is poorly tolerated causing increased LV end diastolic pressure followed by left ventricular failure. Involvement of the descending and abdominal aorta can lead to acute renal failure due to involvement of the renal arteries, bowel ischaemia (due to superior mesenteric artery compromise) or claudication (involvement of the femoral arteries). Increased height, high arched palate, joint hypermobility, increased arm span or arachnodactyly can point to Marfan's syndrome as a cause of dissection.

In most cases aortic dissection is associated with hypertension. Other causes include bicuspid aortic valve (as in this case), Ehlers-Danlos syndrome, pregnancy (usually third trimester and post-partum), trauma or coarctation.

In about 90% of cases the CXR will show a widened mediastinum, irregularities of the aortic contour (separation of intimal calcification in the aortic arch, called "calcium sign") or a left sided pleural effusion. Blood tests often reveal

elevated troponin and D dimer levels, confusing the diagnosis.

The most common imaging modality used to confirm the diagnosis, assess the origin, extent and complications of the dissection, is CT aortography, which is usually readily available even out of hours. Transthoracic echo can mainly identify dissections involving the aortic valve and proximal root, and can assess for AR and pericardial effusion. The latter two are relatively easy to spot, and in fact in most European countries emergency physicians are trained in bedside ultrasound and focussed Echo to do exactly this.

Aortic dissections are classified as per Debakey (who ironically nearly died of an aortic dissection himself, requiring the very operation he invented) or by the Stanford scheme. The latter is most commonly used, and categorizes all dissections involving the ascending aorta and aortic arch (irrespective of how far distal they extend) as type-A, and all that arise after the origin of the left subclavian artery as type-B.

The mainstay of the immediate management[1] involves analgesia, large-bore intravenous access, crossmatch, arterial line and transfer to CCU or ITU. Aggressive blood-pressure control is of the utmost importance, as it reduces pulse pressure and so aortic wall stress, thereby possibly reducing proximal or distal propagation of the dissection. Intravenous beta-blockade with labetalol or metoprolol, GTN or sodium nitroprusside infusions are commonly used.

All Type A dissections (i.e. the ones involving the ascending aorta) need to be managed surgically, to prevent retrograde extension into the aortic valve or coronary arteries, or causing rupture into the pericardial sac with often lethal pericardial tamponade[2]. Without surgery 25% of patients die within the first 24h (i.e. a mortality rate of around 1%/hour), 75% within the first month. Usually operative interventions involve interposition grafts, root and or valve replacements (occasionally valve repair), with or without reimplantation of coronary arteries and head and neck vessels. Type B dissections are managed medically, unless complications like proximal extension, aortic enlargement or ischaemic complications develop. Occasionally endovascular stenting can be performed, especially with penetrating arteriosclerotic ulcers, causing false aneurysms.

After surgical repair patients should be followed up closely for the first 12 months and yearly thereafter, as further aortic aneurysmal enlargement is possible. Patients should be alerted to signs of subacute rupture or dilatation: back pain, dysphagia and dyspnoea. Stringent long term blood pressure control is required.

In summary we present a case of a patient with an aortic dissection that had few clinical symptoms. This can happen in about 10% of cases. Luckily the patient was not treated with anticoagulation and antiplatelets as might have happened with the elevated D dimer and troponin suggesting PE. Clinicians should be confident in diagnosing diastolic murmurs, and in the context of chest pain should consider aortic dissection as a cause.

References

1. Erbel R, Alfonso F, Boileau C, Dirsch O, Eber B, Haverich A, Rakowski H, Struyven J, Radegran K, Sechrem U, Taylor J, Zollikofer C, Klein VW, Mulder B, Providencia LA,: Task Force on Aortic Dissection, European Society of Cardiology. Diagnosis and management of aortic dissection. Eur Heart J. 2001 Sep;22(18):1642-81.

2. Golledge J, Eagle KA.(2008). Acute aortic dissection. Lancet;372:55-66