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ENDGAMES

PICTURE QUIZ

A 16 year old boy with chest pain

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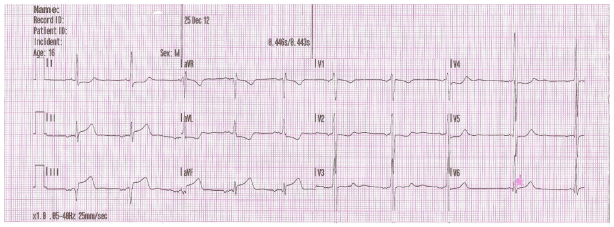
A 16 year old boy awoke with a crushing pain in the centre of his chest. He was clammy, nauseated, and vomited once. He had a history of antiphospholipid syndrome and had been prescribed low molecular weight heparin injections. He admitted to being poorly adherent to this treatment before presentation. He also had hypertension, which was thought to be secondary to poor blood supply to a shrunken, poorly functioning right kidney. He was a smoker and occasional user of cocaine and amphetamines, but he had not used either illicit drug for at least two weeks before the event. An ambulance was called, and the paramedics performed electrocardiography.



Questions

1. What does his electrocardiograph show?
2. What is the differential diagnosis for these electrocardiographic findings in a patient of this age?
3. What initial management would you institute for this patient before further investigation?
4. What is the priority for this patient after immediate management?

Answers

1. What does his electrocardiograph show?

Short answer

Sinus rhythm at 58 beats/min, normal axis, ST elevation in leads II, III, and aVF, with reciprocal ST depression in leads I and aVL. Appearances are consistent with an inferior ST elevation myocardial infarction (STEMI).

Long answer

Sinus rhythm at 58 beats/min, normal axis, ST elevation in leads II, III, and aVF, with reciprocal ST depression in leads I and aVL. There is also T wave inversion in leads V1, V2, aVR, and aVL (fig 2).

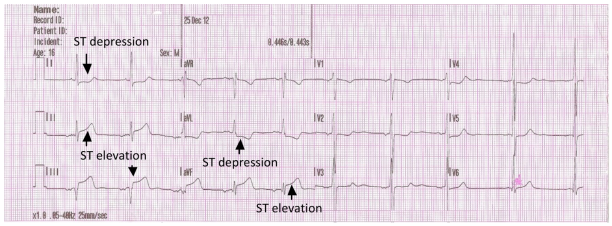


Fig 2 Electrocardiograph showing ST elevation in leads II, III, and aVF, with reciprocal ST depression in leads I and aVL

The electrocardiograph indicates an inferior STEMI. The right coronary artery supplies the inferior surface of the heart in around 85% of cases. The left circumflex artery supplies the inferior surface of the heart in the remaining 15% of patients.

The table⇓ gives details of the coronary artery territories and their corresponding electrocardiographic leads.

2. What is the differential diagnosis for these electrocardiographic findings in a patient of this age?

Short answer

Causes of ST elevation in young people include "high take off," myopericarditis, and acute STEMI. Causes of myocardial infarction in young people include coronary thrombosis or embolus, accelerated atherosclerotic disease (for example, in familial hypercholesterolaemia), coronary or aortic vasculitis, coronary spasm, congenital coronary abnormalities, and cardiac

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abnormalities such as a patent foramen ovale and aortic or coronary dissection.

Long answer

A common cause of ST elevation in younger patients is "high take off," where the ST segments appear to be elevated but simply represent benign early repolarisation rather than infarction. Myopericarditis is another cause of ST elevation in young people, but patients with this condition usually have well localised pain, sharp in character, which is relieved by lying forwards and exacerbated by deep inspiration. In addition, ST elevation is usually widespread and is not in keeping with a lesion in a single coronary artery territory. ST elevation in myopericarditis is usually concave or saddle shaped rather than convex, which is typical of STEMI (fig 3). The presence of reciprocal ST depression also makes the diagnosis of myopericarditis less likely.

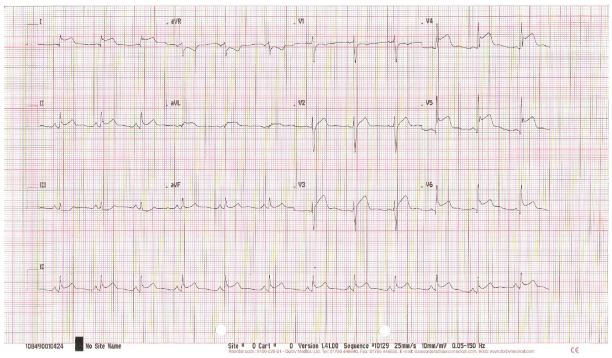

Fig 3 Electrocardiograph showing myopericarditis with widespread concave ST elevation and no reciprocal ST depression

Importantly, pulmonary embolism can mimic a myocardial infarction. Although pleuritic, rather than typical cardiac chest pain would be expected, the possibility of pulmonary embolism should still be considered. In such cases, electrocardiographic changes may be seen in the inferior leads, and large emboli can lead to the release of troponin and clinical deterioration with haemodynamic compromise. If pulmonary embolism is suspected, specific investigation such as computed tomography pulmonary angiography is required. If this is not immediately available, focused echocardiography may help discriminate between an inferior myocardial infarction and pulmonary embolism. Regional wall motion abnormalities in the right coronary territories might be apparent in myocardial infarction, whereas right ventricular and pulmonary arterial dilatation may be seen in pulmonary embolism, along with right ventricular hypokinesia sparing the apex (McConnell's sign).[1]

Myocardial infarction is extremely rare in young people. The estimated prevalence in the literature varies, but the box shows data from the United Kingdom Myocardial Ischaemia National Audit Project (MINAP) registry.

Unfortunately, MINAP does not collect data for patients under 20 years of age, and the data in the box come from those aged 20-30 years (personal communication, Lucia Gavilova, MINAP project manager, 2013).

The differential diagnosis for causes of myocardial infarction in young people is broad. Congenital coronary abnormalities can increase the likelihood of myocardial infarction, possibly because of compressive effects of the myocardium on arteries with an aberrant course.[2] The presence of a patent foramen ovale or an atrial septal aneurysm may lead to paradoxical embolism into the coronary circulation, although such cases are extremely rare.[3] Other rare embolic causes of myocardial infarction in young people include septic emboli from endocarditis (left sided) or clots formed around a left atrial myxoma.

Accelerated atherosclerotic disease can also cause myocardial infarction in very young people. This may occur in the setting of familial hypercholesterolaemia[4] or in the presence of multiple risk factors.[5] Aortic dissection, although rare in this age group, may be linked to inherited conditions such as Marfan's syndrome and may extend to the coronaries.[6] Coronary dissection in isolation is also possible, although again very rare. Vasculitis affecting the aorta or coronaries is another potential cause of myocardial infarction in young people.

Coronary spasm leading to vascular occlusion and myocardial infarction can occur spontaneously or may be provoked by drugs. Cigarette smoking seems to be a risk factor for such events. There have also been case reports of drugs such as cocaine, amphetamines, marijuana, khat, butane, and inhaled glue, as well as alcohol, causing coronary spasm.[7] Prescription drugs such as aspirin, β adrenergic blockers, pseudoephedrine, and atomoxetine, for treating attention-deficit/hyperactivity disorder, have also been implicated in this phenomenon.[7]

The coronary slow flow phenomenon is an uncommon, yet interesting, cause of acute coronary syndrome that can occur in young people. It describes the finding of delayed opacification of the distal coronary vessels with contrast during an angiogram, despite the absence of obstructive coronary disease. It is thought to be related to coronary microvascular dysfunction and is associated with ST elevation, ST depression, and T wave changes.[8] It may cause acute coronary syndromes, and patients with the condition are more likely to be male, hypertensive, hypercholesterolaemic, and active smokers.[8] Coronary angiography is needed to make a diagnosis.

Thrombophilia leading to coronary thrombus is an important cause of STEMI. This presentation has been described in the context of a patent foramen ovale leading to paradoxical embolism,[9] as well as isolated coronary thrombosis linked with genetic factors.[10]

3. What initial management would you institute for this patient before further investigation?

Short answer

Antiplatelet treatment with aspirin and an ADP receptor blocker such as ticagrelor, prasugrel, or clopidogrel should be started provided there are no contraindications. Opiate pain relief with antiemetic cover and nitrates for vasodilation should also be given. Any hypoxia should be corrected with oxygen therapy.

Long answer

In the context of probable STEMI, antiplatelet therapy with aspirin and an ADP receptor blocker should be given unless contraindicated. Although no data from randomised control trials are available to guide the optimal timing of administration of these drugs, international guidelines recommend that they are given as soon as possible in suspected STEMI.[11] Ticagrelor and prasugrel have a more rapid onset of action and greater potency than clopidogrel, so should be considered first line drugs in this situation.[12 13] Prasugrel is not recommended in people over 75 years of age and is contraindicated in those with a history of stroke or transient ischaemic attack.[11] Caution is needed when considering these drugs in patients with a high bleeding risk or anaemia. Opiate pain relief with antiemetic

Estimated prevalence of suspected myocardial infarction in people under 30 years of age

The figures below are based on discharge diagnoses from patients admitted in the 2010-11 and 2011-12 fiscal years.

- Myocardial infarction (ST elevation): 163
- Threatened myocardial infarction: 1
- Acute coronary syndrome (troponin positive): 92
- Acute coronary syndrome (troponin negative): 15
- Chest pain of uncertain cause: 66
- Myocardial infarction (unconfirmed): 4
- Other diagnosis: 175

cover and nitrates for vasodilation should also be given. Although there is a lack of evidence to suggest a definitive benefit for the routine administration of oxygen to those with an acute coronary syndrome,[14] European guidelines recommend that hypoxia be corrected with oxygen therapy.[11]

4. What is the priority for this patient after immediate management?

Short answer

Urgent transfer to a tertiary centre for coronary angiography. Subsequent treatment will depend on the confirmed diagnosis and the condition responsible.

Long answer

The priority for this patient after immediate management is urgent transfer to a tertiary centre for coronary angiography. Subsequent treatment will depend on the confirmed diagnosis and the disease process responsible.

In a suspected acute STEMI, rapid reperfusion is a priority and should be considered immediately. American registry data suggest that every 30 minute delay before the start of primary percutaneous coronary intervention increases in-hospital all cause mortality by around 10%.[15] As such, prompt intervention is key. If there is more than a 120 minute delay between the availability of primary percutaneous coronary intervention and the time that a fibrinolytic agent could have been administered, fibrinolysis should be used.[16] In the case of a delay in the availability of emergent angiography, investigative strategies such as serial troponin testing, focused echocardiography to assess for the presence of regional wall motion abnormalities or identify pericarditis, or coronary computed tomography scanning may be used to help elucidate the diagnosis. Such strategies should not delay definitive investigation or treatment, however.

Patient outcome

Paramedics started treatment with aspirin and intravenous opiates with antiemetic cover and transferred the patient to a tertiary centre for coronary angiography (fig 4).

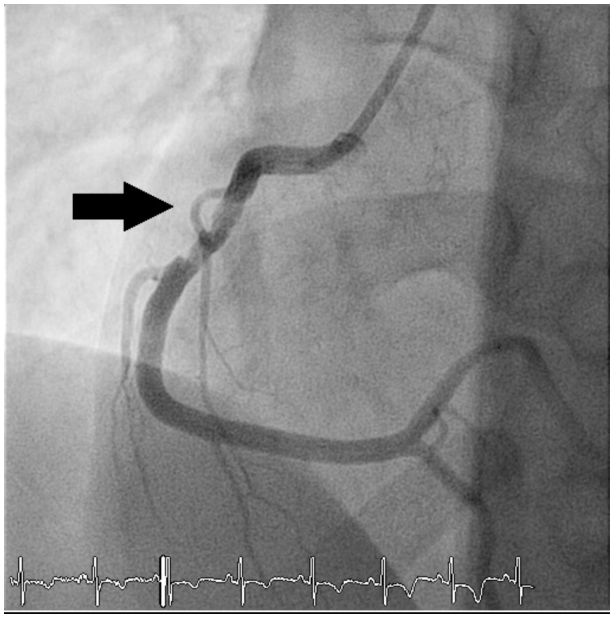

Fig 4 Right coronary angiogram showing proximal moderate narrowing and hazy appearance (arrow), consistent with non-occlusive intraluminal thrombus

Intravascular ultrasound was used to further elucidate the disease process within the right coronary artery (fig 5).

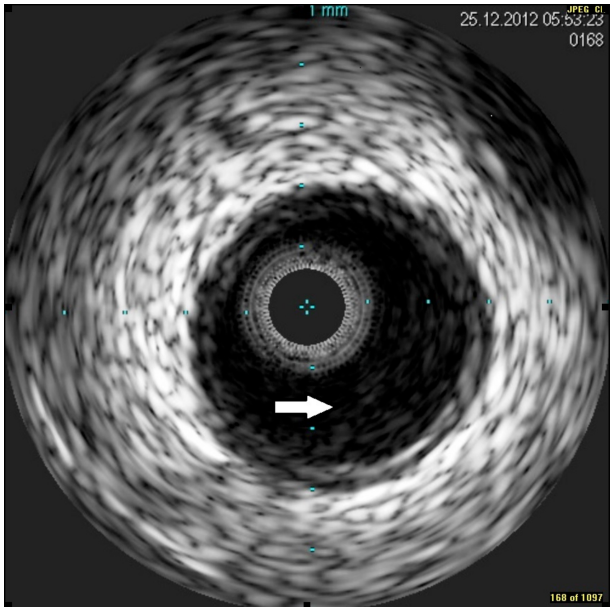

Fig 5 Intravascular ultrasound of proximal right coronary artery showing luminal thrombus (arrow) adherent to the artery wall, with no evidence of atheromatous plaque or dissection

Because coronary flow was normal, a stent was not inserted. The patient was treated with aspirin, prasugrel, and intravenous abciximab (bolus followed by a 12 hour infusion). High sensitivity troponin T was 944 ng/L (≥14 ng/L indicates myocardial damage) the morning after the procedure.

An echocardiogram before discharge showed good biventricular systolic function, with no regional wall motion abnormalities. There was no evidence of a patent foramen ovale or septal abnormality. The valves were normal.

The cardiac rehabilitation team provided advice on smoking cessation and abstinence from illicit drugs. The importance of adherence to anticoagulation was explained in detail.

The patient made a full recovery and his local centre modified his anticoagulation regimen to aspirin and warfarin because he had expressed a preference for oral, rather than subcutaneous, anticoagulation.

In this case, a careful history and the use of intravascular ultrasound to visualise the clot within the right coronary artery led to the diagnosis. The problem was adequately managed with antiplatelet and anticoagulant therapy, without the need for stenting.

Each case should be considered individually with a careful history to identify risk factors, particularly drug misuse and a history of thrombophilia. Poor adherence to prescribed drugs should always be considered in this age group, and patient education and a multidisciplinary team approach should be promoted.

Many thanks to Lucia Gavilova, MINAP project manager, for the data in answer 2.

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Patient consent obtained.

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Table

Table 1| Coronary artery territories and their associated electrocardiographic leads

Leads	Territory the lead represents
V1, V2, V3, V4	Right ventricle and septum (anterior surface of heart). Mostly supplied by the left anterior descending artery
V5, V6, aVL, I	Left ventricle (lateral surface of the heart). Mostly supplied by the left circumflex artery
II, III, aVF	Inferior surface of the heart. Mostly supplied by the right coronary artery
aVR	Right atrium