Improving the early diagnosis of acute myocardial infarction

Ashis Banerjee

A large proportion of patients with acute myocardial infarction are initially seen in accident and emergency departments. Constraints on time and space require the examining doctor to reach rapid decisions about the effective disposal of the patient with suspected or proven myocardial infarction.

The failure to recognise acute myocardial infarction, particularly in the early stages when diagnosis may be difficult, can lead to disastrous consequences. These include the risk of sudden cardiac death from ventricular fibrillation in a patient inadvertently sent home, and of failure to administer thrombolytic therapy at the time of maximum benefit. The latter may occur even with patients hospitalised where the diagnosis is initially not made. Increasing trends towards litigation in the UK may lead to missed myocardial infarction being a significant financial burden on a hospital’s resources. In the US about 20% of the financial burden from litigation against emergency departments is believed to arise from this source. Effective risk management strategies need to develop mechanisms so that the likelihood of missed myocardial infarction is continually reduced. It would appear that no single investigative procedure is failproof in this respect. This review analyses the available strategies and suggests ways in which the present situation can be improved on.

History

As with any branch of clinical practice, the importance of good history taking must not be underestimated. The classical descriptions of angina by Heberden and acute myocardial infarction by Herrick have stood the test of time.

A typical history of central (or retrosternal) crushing, pressing or constricting chest pain, often with radiation into the neck and upper limbs, persisting beyond 20 minutes, is characteristic but not pathognomonic of myocardial infarction. In the absence of information to the contrary it is wise to suspect myocardial infarction until proven otherwise in these circumstances. This is particularly important as the electrocardiogram (ECG) may be nondiagnostic in the early stages of evolving infarcts. As a result, however, a proportion of patients with what will eventually turn out to be noncardiac chest pain may be admitted to coronary care units. This leads to a misdirected use of resources as well as possibly denying the benefits of intensive monitoring to more suitable candidates.

Specifically, pain of oesophageal origin may closely mimic that of cardiac origin. Retrosternal crushing pain, precipitation by exertion, and relief by rest and sublingual glyceryl trinitrate can occur with oesophageal angina. Swallowing-induced discomfort, persistence of a dull ache for several hours after an acute episode, and dysphagia may favour an oesophageal aetiology. In practice, relief of pain by antacid therapy can often be misleading. However, with the present state of knowledge, it is safer to assume cardiac origin until proven otherwise.

Further problems arise from the fact that atypical chest pain may also occur with an evolving infarct as the underlying cause. The use of structured history sheets which draw attention to cardiovascular risk factors may be helpful, as may interactive computer programs and computer-derived protocols. American experience suggests that about 5% of patients with chest pain and acute myocardial infarction can be inadvertently sent home. Setting up a mechanism for follow-up of discharged patients, as suggested in these studies, may have merit but is difficult to implement in practice.

Noncardiac presentations of myocardial infarction have been enumerated, in particular in the elderly (box 2), and in these specific situations the likelihood of myocardial infarction needs to be borne in mind. Certain conditions, in particular dissecting aneurysms of the thoracic aorta, may mimic closely cardiac pain, resulting in administration of thrombolytic therapy with disastrous consequences. In practice, pain that is maximal at the onset,
unrelieved by nitrates and radiating to the back, tends to favour dissecting aneurysm as the source of pain.

**Clinical examination**

Clinical examination is of limited value in the diagnosis of myocardial infarction. Signs of sympathetic activation such as pallor, sweating and tachycardia are often present. Presentation with acute left ventricular failure, or with cardiac arrhythmias, including ventricular tachycardia and atrioventricular block is not unusual. Third and fourth heart sounds may be audible. New murmurs of aortic or mitral incompetence may occur, as may pan systolic murmurs of ventricular septal rupture but usually only on the second or third day after infarction. Chest wall tenderness can be misleading and should only be evoked if the pressure on the tender area precisely reproduces the patient's discomfort. Chest wall tenderness can coexist with acute myocardial infarction and, in the author's experience, undue reliance must not be placed on it in isolation.

**Electrocardiography**

The typical ECG changes of acute myocardial infarction were first described by Pardee. 20 A diagnostic ECG can be regarded as the gold standard in the current state of knowledge. Regional ST segment elevation predicts acute myocardial infarction with nearly 100% specificity. 21 Some patients with pericarditis and old Q wave myocardial infarction can cause diagnostic problems. The problems with ECG diagnosis arise from several sources. The initial ECG may be normal or non-diagnostic. The earliest ECG changes may not involve ST segment elevation (eg, hyperacute T waves). 22 Pre-existing ECG changes may interfere with assessment of the latest tracing, eg, left bundle branch block. Criteria for the diagnosis of myocardial infarction in the presence of left bundle branch block are now available. 23 Pseudo-infarction patterns may occur with a variety of cardiac disorders (box 3). Certain forms of myocardial infarction (eg, right ventricular) produce changes mimicking cardiac chamber hypertrophy. Sub-endocardial infarcts may be difficult to recognise, especially by the inexperienced. The ECG may remain normal and myocardial infarction only diagnosed by positive radionuclide scan. 24

Ways of improving the diagnostic yield from ECGs include review of previous ECGs, where available, and serial ECGs to assess evolving infarcts. 25 Audit of all emergency department ECGs by cardiologists or accident and emergency physicians to pick up less obvious missed myocardial infarctions may be useful but does not produce a diagnosis at the time of presentation. 26

**Biochemical markers**

Myocardial necrosis and increased cell membrane permeability may allow the release into the circulation of a variety of myocardial enzymes and other proteins. These biochemical markers may aid diagnosis and quantification of the extent of myocardial necrosis. Elevation of aspartate transaminase and lactate dehydrogenase (LDH) in serum of patients with acute myocardial infarction was documented by Karmen et al in 1955. 27 These enzyme elevations are nonspecific and can be produced by muscle damage elsewhere (including intramuscular injections) as well as by hepatocellular necrosis. Characteristic temporal patterns of enzyme elevation mean that timed sequential measurements may be helpful in the acute phase. Serum LDH begins to rise at 12 hours, peaks at 48–72 hours and does not return to normal for 7–10 days. LDH levels rise and, more specifically, LDH1 to LDH2 ratios (heart muscle contains primarily LDH1 isoenzyme), may be useful in late presentations but by then ECG changes would be more likely in the vast majority of instances. 28,29

The use of creatine kinase (CK) measurement has proved more useful. 30 CK is a dimeric molecule of two subunits, M and B. Combinations of subunits form the isoenzymes CK-MM, CK-MB and CK-BB. These isoenzymes may be separated electrophoretically. Serum of healthy individuals contains predominantly the CK-MM isoenzyme. The major source of CK-MB is the myocardium, but it is also found in skeletal muscle. Following the onset of symptoms of myocardial infarction, total CK and CK-MB levels rise in the serum within three to six hours, with peak levels being attained between 16–38 hours. Levels return to normal by 34–36 hours. Elevated total CK levels may be detected for up to 36–60 hours. CK-MB estimations are useful as an early screening test (starting at four hours after presentation) and when levels rise on serial estimations. They are also a useful tool to aid the decision to discharge from the accident and emergency department. 31–34

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**Box 3**

**Pseudo-infarction patterns on ECG**

- hypertrophic obstructive cardiomyopathy
- acute myocarditis
- acute pulmonary embolism
- cardiac tumours
- cardiac amyloidosis

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**Case report**

A 37-year-old previously fit AfroCaribbean man attended the accident and emergency department following a syncopal episode. He had no chest pain at any stage. When seen he had recovered. Apart from being an occasional smoker no other cardiovascular risk factors were present. A thorough physical examination revealed bradycardia but no other abnormalities. A 12-lead ECG showed sinus bradycardia. He was discharged home but re-attended three hours later with signs of shock. Refractory ventricular tachycardia ensued which degenerated into ventricular fibrillation and he was declared dead after all resuscitation attempts failed. A post mortem revealed aneurysma of the origin of the left coronary artery with occlusive thrombus.

This case illustrates some of the potential pitfalls in myocardial infarction: young age, no chest pain and no significant cardiovascular risk factors in a previously fit individual. Any risk reduction strategy would have to include assessment of this type of patient.
Specific muscle proteins found to be of some use in improving the early diagnosis of myocardial infarction include myoglobin and troponin T. Myoglobin and light chains of myosin are released into serum within two to six hours, peak at four to six hours and return to normal within 12–24 hours. These proteins are also found in skeletal muscle and elevation is not specific for cardiac muscle damage.

The problem with use of biochemical markers is that they have temporal limitations and are not tissue-specific. It is also not possible to diagnose perioperative myocardial infarction, especially after cardiac surgery, with any confidence.

**Echocardiography**

The use of ultrasound to delineate focal areas of poor myocardial contractility (akinisia or dyskinesia) secondary to infarction has been employed with variable success. In general, the procedure is operator dependent and yields inconsistent results. It does not therefore appear to be a reliable tool to aid the early diagnosis of acute myocardial infarction.

**Radionuclide scans**

Occasionally myocardial infarction may be visualised by radionuclide agents, either as a hot spot of increased uptake or a cold spot of reduced uptake. Specificity may be improved by the use of indium-III labelled monoclonal antimyosin antibodies. Access to radionuclide scanning is not generally available to accident and emergency departments in the UK. Scanning is best performed under cardiological supervision. It is particularly useful in patients with acute myocardial infarction and presentations with normal ECG and

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**Suggested strategy for suspected myocardial infarction (MI) in accident and emergency (A&E)**

<table>
<thead>
<tr>
<th>MI suspected by general practitioner (GP) or triage nurse</th>
<th>Accident &amp; Emergency Department</th>
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<tbody>
<tr>
<td>Vital signs on arrival</td>
<td>Resuscitation room/cubicle with monitoring facilities</td>
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</table>

**Diagnostic**

- Immediate 12-lead ECG

**Nondiagnostic**

- Normal ECG

**High probability of MI**

- (cardiac chest pain, other possible presentation of MI, two or more cardiovascular risk factors)

- Low probability of MI (noncardiac pain, no or one cardiovascular risk factor)

**High probability of MI**

- Admit

**Low probability of MI**

- Observe in A&E

- Serum CK-MB rapid assay

- Repeat 12-lead ECG hourly for 4 hours

**Serum CK-MB rapid assay**

- Discharge & GP follow-up

**Admit**

- All discharged patients should be followed up within 24 hours and ECG formally reported on.

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**Box 4**

**Strategies for improving early diagnosis of myocardial infarction in accident and emergency**

- structured history sheets, as aide memoires
- improved awareness of noncardiac presentations, particularly in the elderly
- ECG on arrival in cases of strong suspicion
- observation of high-risk patients with serial ECG monitoring, 12-lead ECGs and serial cardiac enzyme estimations if a holding space is available (eg, short stay ward)
- use of serum CK-MB estimations in atypical cases or typical cases with nondiagnostic ECG, where rapid assay is available
- referral on clinical suspicion of doubtful cases for formal admission
- routine reporting and audit of ECGs
- introduction of artificial intelligence systems able to analyse data from multiple sources

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**Box 5**
Learning points
- acute myocardial infarction can be difficult to diagnose
- high-risk patients should be admitted to the intensive care unit
- the use of short stay observation units in accident and emergency departments may be cost effective as the highest risk of sudden death is in the first six hours after onset of symptoms
- repeating the 12-lead ECG at hourly intervals while under observation is recommended
- rapid assay of serum CK-MB isoenzyme is a useful adjunct to aid diagnosis in doubtful cases

Box 6


- Olson HG, Lyons KP, Butnir S, Pitts KM. Validation of Tc-99m stannous phytrophosphate myocardial scintigraphy for diagnosing acute myocardial infarction more than 48 hours after serum creatine kinase - MB has returned to normal. Am J Cardiol 1983; 52: 245 – 51.

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