Postictal electrocardiographic changes mimicking acute myocardial infarction

AC Hodson, A Poullis

CASE REPORT

A 63-year-old male presented to the accident and emergency department having collapsed. On arrival in the department a generalized tonic–clonic fit was witnessed which lasted less than a minute and resolved spontaneously. The patient was asymptomatic following this episode.

His past medical history included a cerebrovascular accident 3 years previously resulting in a mild residual left-sided hemiplegia for which he was taking warfarin. He had suffered from what he described as two 'minor fits' over the previous 10 months for which he had not sought medical attention. Further history was unremarkable.

On examination the patient had a Glasgow coma scale of 15/15. Cardiovascular examination revealed a dyskinetic apex beat displaced to the sixth intercostal space in the anterior axillary line. Respiratory and abdominal examination were unremarkable. Neurological examination revealed a mild left-sided hemiplegia and left seventh cranial nerve upper motor neurone palsy.

Routine haematology and biochemistry were normal and a chest X-ray revealed cardiomegaly. His electrocardiogram (ECG) initially showed anterior ST segment elevation and anterior Q waves (*Figure 1*). A provisional diagnosis of hypoxic fits secondary to an arrhythmia following acute anterior myocardial infarction was made and the patient was admitted to the coronary care unit for serial ECGs, cardiac enzymes and observation.

A repeat ECG after 6 hours (*Figure 2*) showed changes suggestive of an evolving acute anterior myocardial infarct with development of lateral T wave inversion. In view of the absence of any history of chest pain, ongoing anticoagulation with warfarin and normal cardiac enzymes, a decision was made not to thrombolyse this patient.

Serial cardiac enzymes (creatinine kinase and hydroxybutyric dehydrogenase) and a troponin T (performed 12 hours after admission) were normal. An echocardiogram revealed an antero-apical left ventricular aneurysm with no associated thrombus visualized. Repeat ECGs revealed no further changes.

With the above information the diagnosis of epilepsy secondary to a previous cerebrovascular accident and a left ventricular aneurysm was made. Treatment with phenytoin was commenced with cautious monitoring of the international normalized ratio (phenytoin can reduce or enhance the anticoagulant effect of warfarin). The patient had no further fits. The warfarin was continued in view of his left ventricular aneurysm.

INTRODUCTION

The electrocardiogram (ECG) is of critical importance in the diagnosis of acute myocardial infarction. This article describes a case in which the ECGs in isolation were highly suggestive of an evolving acute myocardial infarction, although the true pathology was of central nervous system origin.

DISCUSSION

ST segment elevation with distinctive pattern progression is one of the diagnostic criteria of acute myocardial infarction. Left ventricular aneurysms cause ST segment elevation but this does not change over the time scale described in this case.

Some of the changes in the QRS complex may be accounted for by the change in heart rate as this alters the terminal deflection of the QRS complex; however, this would not account for the new lateral T wave changes.

Many non-cardiac conditions can cause ECG changes. Various ECG abnormalities have been described but in this case the search focussed on non-cardiac conditions causing ST segment changes. The commonest non-cardiac cause of ST elevation described is subarachnoid haemorrhage. Other reports include ST elevation in oesophageal rupture (Mosseri et al, 1986), pancreatitis (Patel et al, 1994), perforated duodenal ulcer (Thomas et al, 1987), pneumothorax (Diamond and Estes, 1982) and following elective DC cardioversion (Chun et al, 1981).

A number of small studies and case reports have shown ECG changes occurring in the postictal state (Mohr et al, 1974). Of all the postictal changes documented in the literature none have shown the changes of an evolving acute myocardial infarct. The commonest ECG changes to occur postictally are rate disturbances and these have been suggested as a possible cause of sudden unexplained death among patients with chronic epilepsy. Changes in rate, rhythm and non-specific ischaemic patterns have been suggested as a cause of death in status epilepticus. Reports of postictal ST segment change are limited to anterolateral ST depression with T wave inversion and no Q wave formation (Mohr et al, 1974), and to anterolateral ST elevation with no evolution of the ECG changes (Miyagawa, 1993).

The mechanism behind postictal ECG changes are unclear but there are several possibilities. Catecholamine activity secondary to autonomic arousal has been widely described as the cause of ST segment elevation secondary to subarachnoid haemorrhage. Direct activation of brainstem cardioarrhythmogenic centres have been studied in animals as part of research into the cause of sudden epileptic death (Mameli et al, 1993); however,

Dr AC Hodson is House Officer and **Dr A Poullis** is Senior House Officer in the Department of General Medicine, Chelsea and Westminster Hospital, London

Correspondence to: Dr A Poullis, Department of Gastroenterology, St George's Hospital, London SW17 0RE

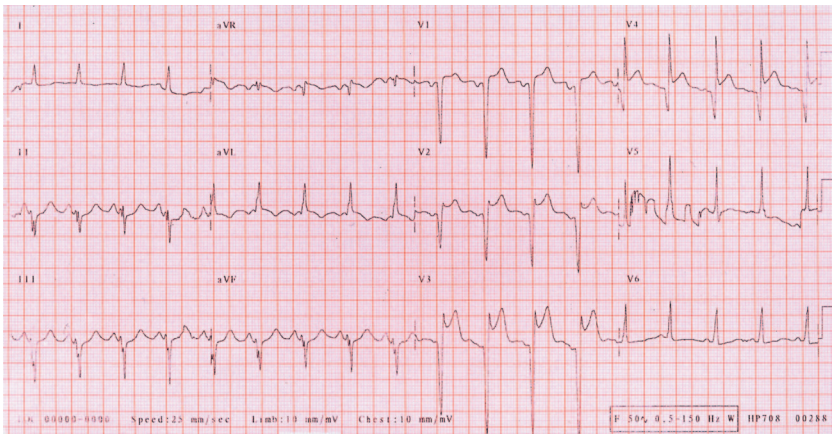

Figure 1. Initial electrocardiogram.

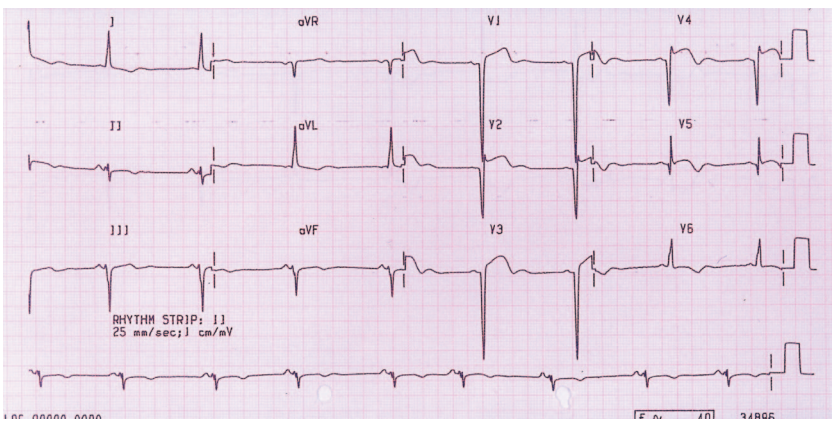

Figure 2. Repeat electrocardiogram after 6 hours.

ECG changes mimicking ischaemic change have not been seen. Coronary artery spasm is well documented to occur with externally administered pharmacological agents, but in this case the patient was not taking any vasoactive drugs.

In this case a patient with pre-existing infarction and left ventricular aneurysm has been described, in whom postictal ECGs showed a sequence of further ST elevation and T inversion suggesting an evolving infarct. These changes may have been the result of a combination of autonomic arousal secondary to the epileptic fit causing a catecholamine surge and neuronal changes in regulatory centres on a background of a left ventricular aneurysm. It is well known that left ventricular aneurysms cause persistent ST elevation but there are no reports of changes evolving over the time sequence observed in this case.

With thrombolysis and primary angioplasty available as treatments for acute myocardial infarcts early diagnosis is essential. ECGs are a vital confirmatory test but must be interpreted in the context of the history, examination and supporting investigations.

CONCLUSIONS

A number of non-cardiac conditions have been described as causing ST segment changes. A neurological case is reported in which the ECG changes evolved and mimicked an acute myocardial infarction. HM

Chun PKC, Davia JE, Donahue DJ (1981) ST segment elevation with elective DC cardioversion. *Circulation* **63:** 220–4

Diamond JR, Estes NM (1982) Electrocardiographic changes associated with iatrogenic left pneumothorax simulating anterior myocardial infarction. *Am Heart J* **103**: 303–5

Mameli O, Melis F, Giraudi D, Cualbu M, Mameli S, De Riu PL, Mameli P (1993) The brainstem cardioarrhythmogenic triggers and their possible role in sudden epileptic death. *Epilepsy Res* **15(3):** 171–8

Miyagawa K (1993) Prominent elevation of the ST segment by convulsion. *Chest* **104(2):** 653–4

Mohr HJ, Fricke G, Simon H (1974) Genuine epilepsy simulating myocardial infarction. *Medizinische Klinik* **69(25):** 1112–6

Mosseri M, Eliakim R, Mogle P (1986) Perforation of the oesophagus electrocardiographically mimicking myocardial infarction. *Israeli J Med Sci* **22(6):** 451–4

Patel J, Movahed A, Reeves WC (1994) Electrocardiographic and segmental wall motion abnormalities in pancreatitis mimicking myocardial infarction. *Clin Cardiol* **17(9):** 505–9

Thomas I, Mathew J, Kumar VP et al (1987) Electrocardiographic changes in catastrophic abdominal illness mimicking acute myocardial infarction. *Am J Cardiol* **59(12):** 1224–5