EMERGENCY CASEBOOK

Pseudo myocardial infarction

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The case is presented of a 66 year old woman who attended the emergency department with severe abdominal pain subsequent to a bout of coughing, following a week’s history of productive cough. She was known to have chronic obstructive pulmonary disease and was also on warfarin for recurrent deep vein thromboses. She had no history of ischaemic heart disease. She was found to have a rectus sheath haematoma and an international normalised ratio of 7.7, and admission was arranged for coagulation control and analgesia. However, a routine electrocardiograph (ECG) demonstrated an ST elevation pattern consistent with an acute inferior infarction. Subsequent ECGs showed no ST elevation, although the axis and chest lead QRS morphology remained the same throughout the first 12 hours. Over the next three days, R wave progression decreased in the chest leads. Troponin I at admission and 24 hours later were both <0.2 ng/ml. ECG changes compatible with acute myocardial infarction have been reported in association with a number of non-cardiac presentations; however, to our knowledge, it has never been reported in relation to a rectus sheath haematoma. We speculated on the possible mechanism of such “pseudo myocardial infarction” and the importance of treating the patient, not the ECG.

A 66 year old woman attended the emergency department complaining of severe pain of sudden onset in the left abdomen, which had occurred a few hours earlier during a spasm of coughing. Shortly after the pain started, she noticed a large lump to the left of her umbilicus. She had been coughing for the past week, which was productive of yellow sputum. Her medical history of note included recurrent deep vein thromboses, for which she was taking warfarin, and chronic obstructive pulmonary disease. She had no history of ischaemic heart disease, myocardial infarction (MI) or diabetes.

Examination revealed a large, firm, tender mass, which appeared to be in the left rectus sheath. Apart from widespread crackles and a slight expiratory wheeze on chest examination, her general examination was otherwise unremarkable.

Chest and abdominal films were normal. International normalised ratio was 7.7, and admission was arranged for analgesia and control of her anticoagulation, with a diagnosis of left rectus sheath haematoma caused by a combination of over-anticoagulation and vigorous coughing due to an exacerbation of COPD. While awaiting transfer to the ward a routine electrocardiograph (ECG) was performed (fig 1).

The ECG was repeated, and showed an identical pattern of an inferior MI, with some lateral territory extension. She had no other symptoms beyond the cough and the abdominal pain and lump, and she was transferred to the coronary care unit for monitoring and serial troponin levels. Urea and electrolytes were normal. A repeat ECG 2 hours showed no ST elevation, although the axis and chest lead QRS morphology remained the same throughout the first 12 hours. Over the next 3 days, R wave progression decreased in the chest leads. Troponin I at admission and 24 hours later were both <0.2 ng/ml.

The patient’s stay was otherwise uneventful, her coagulation was controlled, and she was managed conservatively and discharged 4 days after admission.

DISCUSSION

All emergency physicians will be aware of the ECG changes associated with acute MI. However, the increasingly documented phenomenon of pseudo MI needs to be considered, especially in those cases in which thrombolytic therapy could have disastrous consequences.

ECG changes compatible with acute MI have been reported in association with acute surgical abdomen (pancreatitis, gangrenous appendix, perforated duodenal ulcers), and also with shock, severe metabolic stress, herpes zoster, phaeochromocytomas, hyperkalaemia secondary to diabetic ketoacidosis, and Wolf-Parkinson-White syndrome, but have not, to our knowledge, been reported in relation to a rectus sheath haematoma.

The exact mechanism by which this occurs is unclear but several ideas have been postulated. A “stress response” secondary to circulating catecholamines has been linked to pseudo MI, either secondary to the pain of the initial presentation (such as pancreatitis) or in relation to a phaeochromocytoma. This appears to a likely cause in this patient, in the absence of any other abnormalities.

Vagal stimulation causing coronary artery spasm or indirectly altering coronary blood flow has been considered, as has the effect of circulating proteolytic enzymes or electrolyte abnormalities. Myocarditis has also been linked to the ECG changes, but would have been unlikely with

Figure 1 ECG of the patient.

Abbreviations: ECG, electrocardiograph; MI, myocardial infarction
normal cardiac enzymes. Pseudo MI patterns of ECG changes have also been reported in left ventricular hypertrophy and Wolf-Parkinson-White syndrome. However, there was no evidence of these changes on this patient’s subsequent ECGs.

We hope that this case will emphasise the relationship between pseudo MI and an expanding list of presenting complaints, the main educational point being: treat the patient and not the ECG.

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