CASE REPORT

Do we intervene inappropriately for ST elevation?

A Sanders, A Froude, F Probst

Emerg Med J 2006;23:e10 (<http://www.emjonline.com/cgi/content/full/23/2/e10>). doi: 10.1136/emj.2004.020537

ST elevation on a 12 lead ECG is one of the cardinal features of acute myocardial infarction (AMI), yet it also occurs with other clinical conditions such as spontaneous pneumothorax. Three cases are presented, all of whom had chest pain and ST elevation. All had pneumothoraces yet only one had an AMI. Thrombolysis was administered to one patient. With the current pressure on "door-to-needle" times, emergency physicians should take care to differentiate between these entities.

Following recent advances in AMI management, the 12 lead ECG has become the first part of the assessment of a patient with chest pain. With an ideal "door-to-needle" time of 20 minutes, ST elevation on ECG can easily determine a path of action for doctors. However, several clinical conditions produce these changes, one of which is spontaneous pneumothorax, for which thrombolysis is not standard therapy.

CASE REPORTS

Patient 1

A 74 year old man presented complaining of chest pain for 1 hour that improved after sublingual glyceryl trinitrate. He also complained of numbness of his left arm, nausea, shortness of breath, and sweating. He had a history of hypertension. Pulse was 120 beats/min, blood pressure 190/110 mm Hg, respiratory rate 30 breaths/min. A 12-lead ECG (fig 1A) showed ST elevation of 2 mm in leads V1–V3 and a diagnosis of AMI was made. The patient was thrombolysed with reteplase. A chest radiograph (fig 1B) showing a left sided pneumothorax was then obtained. A chest drain was inserted and the ST elevation resolved. Subsequently, troponin I was 7.6 ng/ml.

Patient 2

A 25 year old man presented complaining of epigastric tightness for 9 hours, with some shortness of breath. He had no known risk factors for ischaemic heart disease and had never used cocaine. ECG showed 1 mm ST elevation in leads V2–V3 (fig 2A). He appeared well with normal vital signs, normal heart sounds, central trachea, and bilateral air entry. AMI was considered, but as the patient seemed so well, the cardiologist decided that neither percutaneous transluminal coronary angioplasty nor thrombolysis were appropriate. Subsequent chest radiograph (fig 2B) showed a large left sided pneumothorax. Following aspiration, the ST elevation resolved. Serum troponin was never elevated.

Patient 3

A 52 year old man presented as a priority call with severe difficulty in breathing. On arrival he was distressed, unable to speak, and using accessory muscles. His respiratory rate was 25 breaths/min, oxygen saturation 67% on air, and pulse 123 beats/min. Initial examination revealed bilateral

resonant percussion and quiet breath sounds. On arrival, an ECG was performed (fig 3A) which showed anterior ST elevation, right bundle branch block (RBBB), and right axis deviation. AMI was considered as a diagnosis but as the patient was so unwell, the senior house officer sought assistance. The consultant requested an immediate chest radiograph, which revealed a right sided pneumothorax (fig 3B). A chest drain was immediately inserted, his respiratory rate and oxygen saturations improved, and subsequent ECGs showed sinus tachycardia with resolution of the RBBB. Serum troponin was not elevated. He reported later that he had had chest pain during his presentation, which had improved after insertion of a chest drain.

Figure 1 (A) ECG and (B) chest radiograph of patient 1.

Abbreviations: AMI, acute myocardial infarction; RBBB, right bundle branch block

DISCUSSION

ECG changes with pneumothoraces have been previously reported.¹⁻⁴ In this series, there was anterior ST elevation with a left sided pneumothorax (cases 1 and 2) and ST elevation, right axis deviation, and RBBB with a right sided pneumothorax (case 3). It has been suggested that these changes result from myocardial ischaemia secondary to hypotension and reduced coronary blood flow.³ In previous case reports, signs of circulatory collapse and pneumothorax under tension have been evident;^{2,3} however, our patients maintained adequate blood pressures throughout with little clinical indication of pneumothorax.

Targets for reperfusion have drastically altered the assessment of a patient with chest pain. In our emergency department, the standard is 10 minutes from presentation to ECG review for these patients. In these three cases, the ECG changes seen early in the assessment process prompted the doctors to consider thrombolysis.

In case 1 (before the availability of primary PTCA in our hospital) the ECG changes and chest pain led to a decision to administer thrombolysis. At 12 hours, troponin I was 7.6 ng/ml, showing myocardial damage. Thrombolysis was not delayed by the process of obtaining a chest radiograph and therefore the pneumothorax had not been diagnosed.

In case 2, the availability of primary PTCA meant early involvement of senior staff. This patient fitted the criteria for thrombolysis. It seems reasonable to speculate that he would have been administered thrombolytic therapy if a PTCA had not been available and if he had been seen by a less experienced doctor.

In case 3, the senior house officer managing the patient was considering AMI as the diagnosis based on the ECG changes, but sought assistance from a more senior staff

Figure 2 (A) ECG and (B) chest radiograph of patient 2.

Figure 3 (A) ECG and (B) chest radiograph of patient 3.

member as the patient was so unwell. This resulted in early diagnosis of the pneumothorax following prompt radiography. This patient did not strictly match the criteria for thrombolysis as he was unable to communicate about his chest pain.

There are other causes of ST elevation in addition to AMI (table 1). Intracerebral bleed, aortic dissection, tension pneumothorax, Brugada syndrome (abnormality of the sodium channels resulting in ECG changes of RBBB, J point

Table 1 Causes of ST elevation on 12 lead ECG

Acute MI
Coronary vasospasm
Ventricular aneurysm
Benign early repolarisation
Pericarditis
Brugada syndrome
Intracerebral bleed
Aortic dissection
Tension pneumothorax
Left ventricular hypertrophy
Left bundle branch block

elevation, and concave ST elevation), and pericarditis have been reported to cause these changes.⁵⁻⁶ It is possible that these could be confused in an acute setting with narrow time constraints. In addition, correct interpretation of ECGs by junior doctors has been shown to be variable.⁷

CONCLUSION

There is no doubt that early coronary reperfusion is beneficial for cases of AMI. Chest pain with ST elevation is not always synonymous with an AMI. In the rush to meet government targets, clinicians must avoid making a hasty but wrong diagnosis.

Authors' affiliations

A Sanders, A Froude, F Probst, Emergency Department, Charing Cross Hospital, UK

Competing interests: none declared

Correspondence to: Dr A Sanders, Specialist Registrar, Emergency Department, Charing Cross Hospital, Fulham Palace Rd, London W6 8RF, UK; alisanders@btinternet.com

Accepted for publication 5 January 2005

REFERENCES

- 1 **Strizik B**, Forman R. New ECG changes associated with a tension pneumothorax: a case report. *Chest* 1999;**115**:1742-4.
- 2 **Monterrubio VJ**, Fernandez BD, Alzugaray Fraga RJ, *et al*. [ST elevation and tension pneumothorax]. (Spanish). *Revista Espanola de Cardiologia* 2000;**53**:467-70.
- 3 **Slay RD**, Slay LE, Luehrs JG. Transient ST elevation associated with tension pneumothorax. *JACEP* 1979;**8**:16-18.
- 4 **Raev D**. A case of spontaneous left-sided pneumothorax with ECG changes resembling acute myocardial infarction. *Int J Cardiol* 1996;**56**:197-9.
- 5 **Atarashi H**, Ogawa S. Idiopathic VFI. New ECG criteria for high-risk Brugada syndrome. *Circ J* 2003;**67**:8-10.
- 6 **Terradellas JB**, Bellot JF, Saris AB, *et al*. Acute and transient ST segment elevation during bacterial shock in seven patients without apparent heart disease. *Chest* 1982;**81**:444-8.
- 7 **Gillespie ND**, Brett CT, Morrison WG, *et al*. Interpretation of the emergency electrocardiogram by junior hospital doctors. *J Accid Emerg Med* 1996;**13**:395-7.