

Serious postoperative syncope

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In November, 2008, an arrest call was made for a 69-year-old woman after a syncopal episode in a rural hospital. The patient had had an elective right total hip replacement a day earlier. The syncope occurred when she mobilised with a physiotherapist. She regained full consciousness within seconds, but her blood pressure was unrecordable. She denied chest pain or discomfort, palpitation, dizziness, or dyspnoea. She had a history of treated hypercholesterolaemia. Physical examination was unremarkable. The initial 12-lead electrocardiograms (ECGs), done within the first 15 min, showed sinus rhythm at 89 beats per min with 1 mm ST depression in V5 compared with the preoperative ECG (figure A). Nursing staff noted that she had self-administered a 6 mg bolus of morphine-based patient-controlled analgesia (PCA) before mobilising. On the basis of the initial clinical assessment, her syncope was attributed to either hypovolaemia or morphine-induced hypotension, especially in view of her good response to fluid resuscitation.

However, a repeat ECG 30 min later showed widespread ischaemic changes (figure B), although the patient still remained asymptomatic and haemodynamically stable. Troponin I and creatine kinase concentrations were high (0.14 µg/L and 429 U/L, respectively; normal renal function). She was started on aspirin and a heparin infusion. Her PCA was stopped, and she started complaining of chest pain 4 h later, although there were no new ECG changes, and the pain was relieved by glyceryl trinitrate. The PCA was resumed, and the patient remained free of chest pain overnight (29 mg morphine used). The next morning, troponin I and creatine kinase concentrations were higher (6.14 µg/L and 1042 U/L, respectively). An echocardiogram done in the early afternoon showed many areas of cardiac akinesia. She subsequently developed cardiogenic shock requiring a dobutamine infusion while

awaiting transfer to a tertiary hospital. On arrival there, her systolic blood pressure was 60 mm Hg (on inotropes) and a coronary angiogram showed severe left main stenosis and triple-vessel disease. A balloon pump was inserted and the left main lesion stented. Subsequently, her condition stabilised and she underwent coronary bypass surgery 7 days later. She made an uneventful recovery and remained well when we last contacted her in January, 2009.

PCA has become a common standard for postoperative pain management since its introduction in 1971.¹ On the basis of a study of postoperative myocardial infarction after non-cardiac surgery,² we estimate the incidence of painless postoperative myocardial infarction to be 5%. The authors of that study suggested that such cases might be related to postoperative use of opioid analgesia.² Our case demonstrates the initial masking of chest pain until the removal of PCA. A change from self-administered to medically controlled morphine in this patient might have allowed symptomatic detection of sustained ischaemia with the postoperative pain still controlled. Of note, there is concern over intravenous morphine use in acute coronary syndromes³ and the recommendation for its use has been downgraded in the new ACC/AHA guideline.⁴ It has been reported that 56% of postoperative myocardial infarctions may manifest as hypotension, pulmonary oedema, or atrial fibrillation; chest pain may be absent.² These manifestations can be attributed to common postoperative complications and managed accordingly, especially in patients who have had major surgery without obvious initial ECG changes or chest pain. Therefore, care should be taken to exclude ischaemia in postoperative patients who have abnormal cardiovascular symptoms or signs, especially those using PCA or being given large amounts of opioid-based analgesia.

Contributors

CHK, SS, and BB managed the patient; CHK, DK, NMH, and BB wrote the report.

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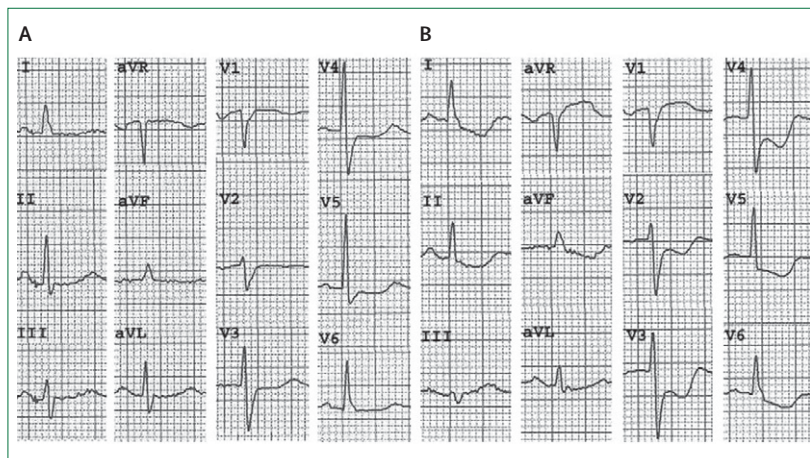


Figure: Examples of ECGs done immediately after syncopal episode (A) and 30 min later (B)