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Spontaneous haemothorax as a complication of anti-coagulation following coronary angioplasty

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Haemorrhagic complications are well recognized when heparin is used during percutaneous transluminal coronary angioplasty (PTCA). We present a 74-year-old female who developed a large acute spontaneous haemothorax 20 h after coronary angioplasty. Spontaneous haemothorax has rarely been described and is predominantly associated with pulmonary embolism. In the absence of a pulmonary embolus, a spontaneous haemothorax during anti-coagulation has only been described twice in the world literature since 1862 (1). This is the first description of this complication following the use of heparin during PTCA.

Introduction

A spontaneous haemothorax is defined as the presence of a bloody pleural effusion in which the pleural fluid haematocrit is 50% or more of the peripheral blood haematocrit (2). A spontaneous haemothorax occurs with no obvious trauma. Anti-coagulation therapy has been reported as the cause of spontaneous haemothorax in 14 cases, 12 of which were associated with a primary diagnosis of pulmonary embolus (2).

Case Report

A 74-year-old female was transferred to our department with unstable angina. She had a 3-yr history of exertional angina with a deterioration over the previous 3 weeks. There was a past history of rheumatoid arthritis alone, controlled by 5 mg prednisolone daily. Other medication included propranolol 10 mg t.d.s., diltiazem 60 mg t.d.s. and isosorbide mononitrate 10 mg b.d. Cardiovascular and respiratory examination were unremarkable, blood pressure was recorded at 140/80 mmHg. Chest X-ray revealed clear lung fields with evidence of old apical tuberculosis affecting predominantly the right side. Full blood count revealed haemoglobin $13{\cdot}6\ g\ dl^{-1}$, white cell count $10{\cdot}1 \times 10^9\ l^{-1}$ and platelet count $315 \times 10^9\ l^{-1}$.

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Coronary angiography was performed using the right femoral artery on the first day after admission. This showed hypokinesia of the anterior left ventricular wall, a 95% stenosis in the first diagonal branch, and occluded left anterior descending artery beyond the first diagonal branch and atheromatous circumflex and right coronary arteries. Due to continuing unstable angina, the patient proceeded to percutaneous transluminal coronary angioplasty (PTCA) to the left anterior descending and first diagonal arteries. This was performed successfully, without complication and with good angiographic result. During the angioplasty a 20 000 unit bolus of heparin was routinely administered and an infusion of 1000 units h^{-1} of heparin was commenced following the procedure. Nursing observation of pulse rate, blood pressure, femoral puncture site, pedal pulses and patient well-being were taken every 30 mins until midnight and then every 2 h through the night.

The following morning, 20 h after the procedure, the patient became acutely hypotensive. The blood pressure was recorded as 70/40 mmHg with pulse rate 130 beats min^{-1}. The electrocardiogram revealed no acute ischaemic changes and beside echocardiography showed no evidence of a pericardial effusion. Clinically she was pale and drowsy, with tenderness over the right femoral artery and dullness to percussion at the right lung base. The haemoglobin checked immediately was $8{\cdot}1\ g\ dl^{-1}$ and activated partial thromboplastin time (APTT) was 100 s (normal laboratory range 35–45 s). The heparin infusion was stopped in view of the accumulating evidence of serious haemorrhage. Intravenous hydrocortisone was given to counteract possible adrenal suppression

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secondary to the maintenance steroid therapy. Computerized axial tomography (CAT) was performed to exclude any retroperitoneal haemorrhage which was felt to be the likely diagnosis. This confirmed the femoral artery, iliac vessels and abdominal aorta to be intact but revealed a large right-sided collection of pleural fluid. This was confirmed on chest X-ray. No attempt at subclavian or jugular venous catheterization had been made.

The heparin infusion was stopped and the patient was transfused with four units of fresh blood. A large pleural drain was inserted and immediately drained 1·2 l of fresh blood. The drain was left in place for 8 days as it continued to drain a further 1·4 l of blood and then blood-stained fluid. After its removal, the patient was well with haemoglobin 15·6 g dl^{-1}, APTT 37 s and chest X-ray showing resolution of the haemothorax.

Discussion

The world literature on spontaneous haemothorax has recently been comprehensively reviewed (1). The differential diagnosis of causes includes anti-coagulation treatment, bleeding from systemic vessels (aortic dissection, ruptured patent ductus arteriosus, leaking internal mammary artery aneurysm), bleeding from pulmonary vessels (arteriovenous malformations), active tuberculosis, costal exostoses and sub-diaphragmatic causes (endometriosis, splenic artery aneurysm). Anti-coagulation treatment has been described as one of the most frequent causes (2). Twelve of the 14 cases described in the world literature have been related to heparin therapy for a primary diagnosis of pulmonary embolism. Bleeding from a pulmonary infarct may cause the development of a haemothorax. The other two reported cases include one patient treated for a deep venous thrombosis in whom a pulmonary embolus was not excluded, and one patient with an artificial heart valve (1).

Anti-coagulation may unmask a separate risk factor for haemothorax, such as a pulmonary arteriovenous malformation (3) or a costal exostosis (4). In our patient, the original chest X-ray revealed evidence of old, healed apical tuberculosis. The history had not suggested any active tuberculosis. Previous reports of spontaneous haemothorax in tuberculosis have all been in active advanced disease (5). This patient had been on treatment with maintenance steroids which may have increased the bleeding tendency.

The source of the haemorrhage in our case was recognized by CAT scanning. Clinical examination had focused on the femoral puncture site and did not detect the haemothorax. Earlier clinical detection of the site of haemorrhage would have led to earlier intervention.

Heparin is commonly used following coronary angioplasty. With increasing use of intracoronary stents, more aggressive anti-coagulation regimens are being introduced and haemorrhagic complications are frequently reported (6). In our case, a 20 000 unit bolus of heparin was given during the angioplasty. There is some evidence that bolus administration of heparin may be associated with a greater bleeding tendency (6).

We know of no other reports of haemothorax complicating anti-coagulation therapy during coronary angioplasty. This complication should be considered as the differential diagnosis to retroperitoneal bleeding when hypovolaemic collapse occurs following PTCA in the absence of any other obvious site of blood loss. Patients are frequently on treatment with β-blockers following PTCA. This treatment may mask an early tachycardia associated with bleeding. Observation with cardiac monitoring of pulse rate needs to be supplemented with assessment of blood pressure. The diagnosis is important to make as the presence of a haemothorax mandates discontinuation of anti-coagulation and drainage of the pleural space by chest drain (1).

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