Case Report

Temporary Cardiac Tamponade Secondary to Chest Tube Placement for Pneumothorax

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A 87-year-old woman was hospitalised because of a third-degree atrioventricular block. After the insertion of a temporary pacemaker lead through the left subclavian vein, she developed an ipsilateral pneumothorax. Although there were clinical and echocardiographic signs of cardiac tamponade after chest tube placement for pneumothorax, a second echocardiogram performed after transportation for surgical drainage failed to demonstrate the presence of any pericardial fluid, while the patient showed an unexpected clinical improvement. A new X-ray showed a collection of left pleural fluid. Over the following days a limited amount of blood was drained through the tube with disappearance of the pleural effusion and no further signs of major bleeding. A permanent DDD pacemaker was subsequently implanted and the patient was discharged in a good condition.

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Cardiac tamponade following chest tube placement for pneumothorax is an extremely rare and serious complication.[1,2] We report a case of cardiac tamponade during left tube thoracostomy for pneumothorax with an unexpected clinical improvement and disappearance of haemopericardium, without a pericardiocentesis or any surgical intervention.

Case description

An 87-year-old woman with a history of hypertension was admitted to our department because of symptomatic third-degree atrioventricular block. Laboratory examination was unremarkable. After initial evaluation, a temporary pacing lead was inserted through the left subclavian vein.

On the next day she started experiencing dyspnoea with tachypnoea, high percussion tones and diminished breath sounds over the left lung. Vital signs were normal. Arterial blood gas analysis showed pH 7.49, PCO_2 30 mmHg, PO_2 49 mmHg, bicarbonate 23 mmol/L and oxygen saturation 88%. Chest X-ray showed a subtotal left-side pneumothorax (Figure 1A).

Accordingly, a chest tube was inserted between the anterior axillary and the midclavicular line, in the 5th intercostal space, with an immediate return of blood. The patient became pale, diaphoretic and drowsy, while the monitoring electrocardiogram showed sinus tachycardia. On physical examination blood pressure was impalpable and the jugular veins were distended. Continuous radial artery pressure recording showed a pulsus paradoxus waveform. A new X-ray showed that the tip of the catheter was close to the cardiac silhouette and indicated partial regression of the pneumothorax (Figure 1B). Transthoracic echocardiography (TTE) revealed a moderate pericardial effusion with signs of cardiac tamponade (Figure 2, views A 1-3 and C1).

Since the patient's clinical condition started to improve after fluid administration and the amount of pericardial fluid was not considered sufficient for a safe pericar-

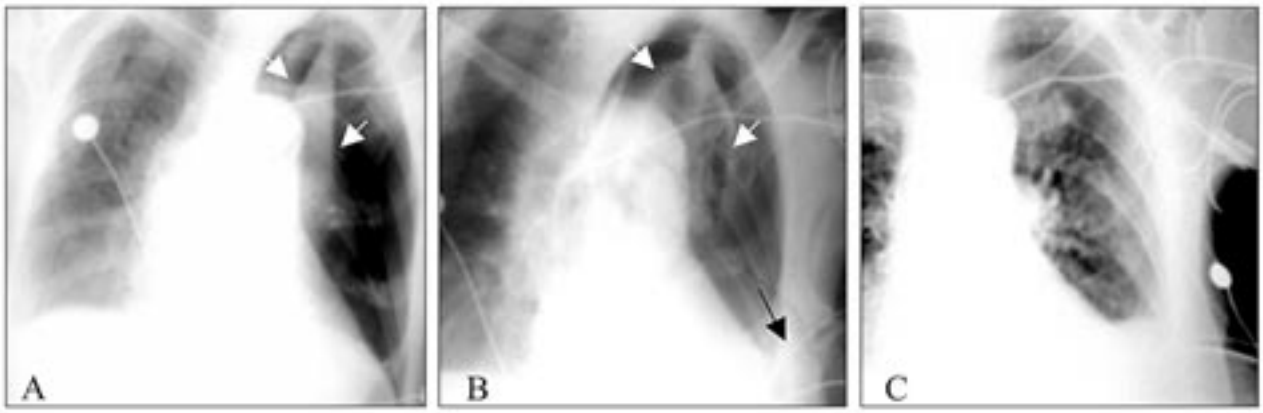

Figure 1. Chest X-rays of the patient. **A**: Subtotal lung collapse (white arrows). **B:** After tube placement. The left lung is partially deployed. The tip of the tube is close to the cardiac silhouette (black arrow). **C:** 12 hours later. Complete left lung deployment and left pleural fluid effusion. The tip of the tube is within the fluid.

diocentesis, the patient was transferred to the cardiac surgery department for surgical drainage. TTE performed immediately after transportation, 6-8 hours after tube placement, surprisingly failed to demonstrate the presence of any pericardial fluid (Figure 2, views B1-3), while a new X-ray showed no signs of pneumothorax. The patient was transferred back to our department 12 hours later in a good condition, with the chest tube left in place. A new TTE again confirmed the absence of pericardial fluid, while a new X-ray showed complete left lung deployment and collection of left pleural fluid (Figure 1C). The ECG showed signs of acute pericarditis (ST elevation in all leads except aVR and V_{1-2}) and troponin I was 14.4 ng/ml.

Over the next three days haemoglobin concentration decreased from 12.2 to 9.2 g/dl while a total of 300 ml of haemorrhagic fluid was drained through the tube with disappearance of pleural effusion. Since the laboratory tests subsequently started to normalise and the clinical course was uneventful, a permanent DDD pacemaker was implanted and the patient was discharged in a good condition. Follow up TTE several weeks later showed no recurrence of a pericardial effusion.

Discussion

Placement of a chest tube for pneumothorax, when performed by an experienced physician, is generally safe.[1] Insertion sites are the 5th intercostal space at the anterior or middle axillary line and the 2nd or 3rd intercostal space at the midclavicular line.

Cardiac tamponade as a complication of chest tube or needle decompression of pneumothorax is extremely rare,[2-4] while other iatrogenic causes are mainly attributed to intracardiac instrumentation, such as needles or catheters.[5-9] Despite the ominous prognosis in case of external trauma, both cardiac tamponade and pneumothorax can control major cardiac bleeding and promote haemostasis.[10-12] In contrast, untimely release of tamponade with pericardiocentesis can cause rapid decompensation because clotting of the pericardial haematoma can be haemostatic for cardiac wounds.[12]

In our case, the trocar of the chest tube, a sharp, pointed rod 8 mm thick, was inserted close to the midclavicular line at the 5th intercostal space (Figure 1B) and resulted in cardiac tamponade, either by direct damage to the myocardium or by laceration of a pericardial vessel. This was suggested by the presence of clinical and echocardiographic signs of cardiac tamponade, diagnostic blood pressure recordings, ECG changes and the drainage of blood, all of them having occurred immediately after tube placement. Since there was elevation of cardiac enzymes, damage to the myocardium was our final explanation for haemopericardium.

Other possible causes of this complication were ruled out by the patient's history, physical examination, laboratory and microbiologic investigations. Left subclavian vein cannulation is a more common cause,[7] but delayed cardiac tamponade following pacing lead insertion is rare and is attributed to delayed perforation of the right ventricle.[13-15] To control this frequently fatal complication, as opposed to the case of external cardiac

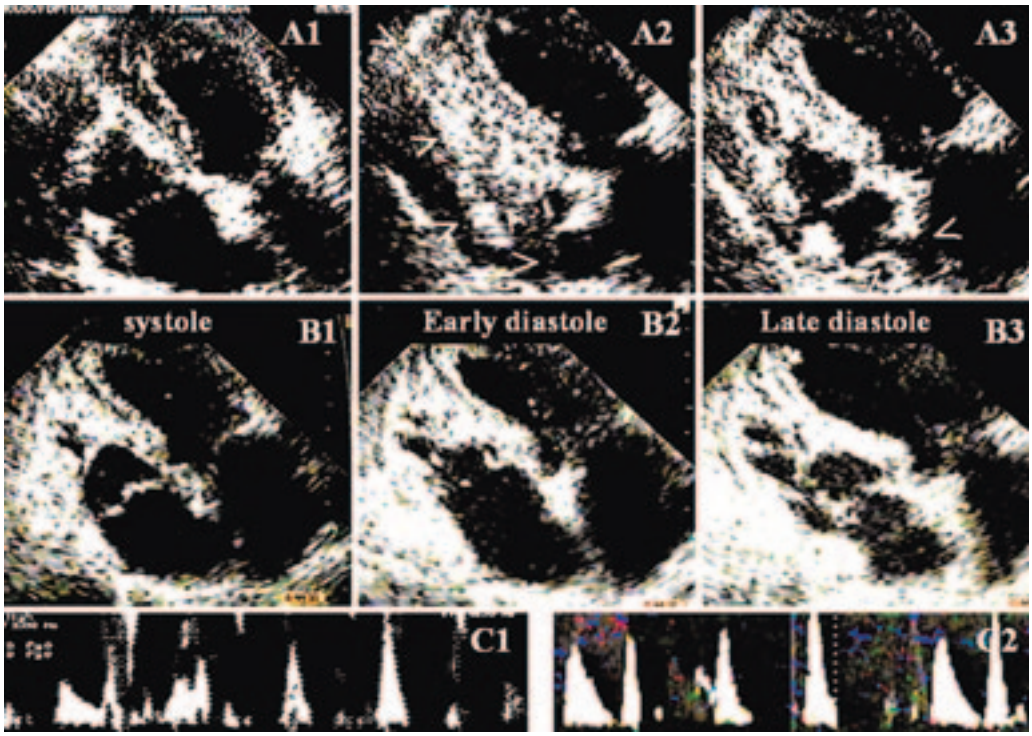

Figure 2. Transthoracic echocardiograms, 4-chamber view, in systole, early and late diastole. **A1-3**: Pericardial effusion after tube placement. Note the diastolic collapse of the right chambers in early diastole and of the right atrium in late diastole. (white arrows). **B1-3:** Same views 6-8 hours later. Disappearance of pericardial fluid. There are no signs of cardiac tamponade. **C1**: Exaggerated respiratory variation of mitral inflow during the 1st echo study. **C2:** Normal variation of mitral inflow during follow up.

trauma, immediate drainage of the haemopericardium and surgical intervention must be carried out.[16-18] Although this hypothesis cannot be excluded, it is difficult to explain the abrupt appearance of signs of cardiac trauma and tamponade immediately after chest tube placement, as well as the subsequent stable clinical course without prompt intervention. The only complication that can easily be attributed to this procedure is delayed pneumothorax, recognized before the event. This diagnosis can be made 8 to 96 hours postoperatively, it rarely occurs compared with immediate pneumothorax and challenges the effectiveness of pneumothorax diagnosis by chest films within 1-2 hours of subclavian venipuncture.[19]

To our knowledge, there are no previous reports of regression of cardiac tamponade resulting from haemopericardium without any intervention. However there are some cases of concomitant resolution of pneumopericardium together with post-traumatic pneumothorax after tube decompression.[20-23] In our case, pneumopericardium was ruled out by the absence of radiographic findings along with the presence of pericardial fluid accumulation on echocardiographic imaging, without any air-fluid interface.[23] We hypothesise that pericardial damage caused by the trocar of the tube created a pericardial fenestration that allowed communication between pericardial and pleural space. Myocardial haemorrhage was controlled and constrained to the pericardial space in the early period after the procedure, possibly because of the positive intrathoracic pressure and/or clotting of the pericardial fenestration. Normalisation of intrathoracic pressure after tube decompression of the pneumothorax created a leak of the accumulated blood through the fenestration to the pleural cavity and regression of cardiac tamponade without any further signs of major bleeding. To support this hypothesis, we rely on the following observations: 1) the appearance of left pleural fluid after the regression of pneumothorax and the disappearance of the pericardial fluid (Figures 1B-1C and Figure 2); 2) the gradual and limited drainage of blood by the tube and the fall in haematocrit over the following days.

This case firstly illustrates the importance of the appropriate insertion site of the chest tube for left-sided pneumothorax, depending on the position of the heart. Secondly, it emphasises the haemostatic role of cardiac tamponade and positive intrathoracic pressure, created by pneumothorax, in the acute phase of cardiac trauma. If there is clinical improvement with conservative measures, the haemopericardium can be drained later through a pericardial window with decreased risk for major bleeding.

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