Cardiac tamponade secondary to haemopericardium in a patient on warfarin

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Acute cardiac tamponade requires urgent diagnosis and treatment. We report a case involving a 70-year-old man who was receiving warfarin treatment for 12 years following mitral valve replacement. The international normalised ratio (INR) was checked and echocardiography performed regularly in the clinic. The last INR was 2.1, checked 2 weeks before admission to the emergency department. The last echocardiography performed 3 months previously revealed no pericardial effusion. The patient suffered from progressive dyspnoea and orthopnoea for several days. Cardiac tamponade was diagnosed, and the INR at that time was 7.52. Urgent pericardiocentesis and pericardiotomy were undertaken and 1300 ml of pericardial blood was drained. Following surgery the patient's recovery was uneventful. An intravenous vitamin K injection and fresh frozen plasma transfusion were administered to reverse the patient's over-anticoagulated state. The final pathology revealed chronic inflammation and there was no malignancy, and no bacteria or mycobacterium were seen. Emergency physicians should remember that over-anticoagulation with warfarin may contribute to certain complications, including haemopericardium, and that strict control of target INR should be the goal for patients who require continuous warfarin treatment.

Acute cardiac tamponade is a potentially life-threatening condition resulting from fluid accumulation in the intrapericardial space. The fluid may arise from traumatic causes, either from an accident or it may be iatrogenic origin, or from atraumatic conditions such as malignancy, infection, uraemia or coagulopathy. Anticoagulation agents are required to treat a large number of patients following open heart surgery, particularly those with valve replacement. The potential role of anticoagulation treatment in the formation of haemopericardium has been suspected for a long time but not established or excluded.¹

We report the case of a patient admitted to the emergency department with cardiac tamponade secondary to haemopericardium without trauma, who had been treated with warfarin for 12 years following mitral valve replacement.

CASE REPORT

A 70-year-old man presented to the emergency department with a 10 day history of progressive dyspnoea and orthopnoea. He had undergone mitral valve replacement for severe mitral regurgitation 12 years previously. He had been taking warfarin (5 mg daily) since then and attending regular follow ups in the cardiology clinic. No more open heart surgery had been conducted following the mitral valve replacement.

In addition to warfarin, he also took furosemide, carvedilol, methyldopa and losartan for heart failure and hypertension control. The international normalised ratio (INR) was checked and echocardiography was performed regularly in the clinic. The last INR was 2.1, which was checked 2 weeks before admission to the emergency department. The last echocardiography undertaken 3 months previously revealed no pericardial effusion.

The patient denied fever, body weight loss, trauma or cough. A dull chest pain over the anterior chest wall without radiation was noted. At our emergency department, the initial chest radiograph revealed an enlarged heart shadow (fig 1) and pericardial tamponade was revealed by echocardiography. Pericardiocentesis was performed under echo guidance and 400 ml of blood was drained. The patient's INR in the emergency department reached 7.52. He denied taking any medications other than those prescribed in the clinic. All blood investigations, including white cell count, platelet, electrolytes, glucose, creatinine, liver enzyme, amylase, partial thromboplastin time and troponin-I, were within normal limits. The electrocardiogram (ECG) showed sinus tachycardia with normal QRS amplitude and no signs of right heart strain or ischaemia. The contrast computed tomography (CT) scan of the chest demonstrated cardiac tamponade, bilateral pleural effusion and haemopericardium. No structural cause for the

pericardial fluid was appreciated. The great vessels and thoracic lymph nodes appeared normal on the CT scan.

The patient was treated for a presumed haemopericardium with intravenous vitamin K and fresh frozen plasma in order to reverse the anticoagulated state. He underwent an emergency pericardiotomy and 1300 ml of dark blood pericardial effusion was drained. The patient eventually recovered successfully. The final pathology revealed chronic inflammation and there was no malignancy, and no bacteria or mycobacterium were detected.

The patient was discharged and followed closely as an outpatient in the clinic. He continues to do well and has not had a recurrence of dyspnoea or orthopnoea.

DISCUSSION

Late cardiac tamponade was first reported in 1968. It is defined as cardiac tamponade occurring 7 days or more after an operation. The published literature to date reports cases presenting at 1–4 weeks post-cardiac surgery.^{1–4} Most patients had undergone valvular surgery and nearly all were on anticoagulation treatment.

Many investigators have asserted that excessive anticoagulation in the postoperative period is responsible for late postoperative tamponade. Some authors have suggested that late cardiac tamponade may due to bleeding or formation of pericardial effusion. Despite the existence of strong evidence favouring a causative role of anticoagulation in the genesis of late tamponade, some studies have challenged this result.⁴ However, there are still few case reports directly pointing to the

Figure 1 Chest x ray taken at admission, showing cardiomegaly, pleural effusion and tortuous aorta.

association between warfarin and haemopericardiac tamponade.

Our case involves a man who developed late cardiac tamponade after 12 years of warfarin treatment for mitral valve replacement, with an INR of 7.52. Tracing back his history, there were no echocardiographic findings of pericardial effusion and all INRs were within normal therapeutic limits during these 12 years. Furthermore, he did not take any antithrombotic drugs (aspirin, dipyridamole) or antibiotics (cephalosporin, vancomycin) that are known to augment prothrombin times.

Thus, we believe that the cardiac tamponade in our patient, secondary to haemopericardium, was associated with his over-anticoagulated status caused by the warfarin. This is the first case to report the development of haemopericardium as a result of the patient's over-anticoagulated status, 12 years after cardiac surgery.

Poor anticoagulation control may precipitate the development of haemopericardium. Emergency physicians should remember that over-anticoagulation with warfarin may contribute to certain complications, including haemopericardium, and that strict control of target INR should be the goal for patients who require continuous warfarin treatment.

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