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Case Report

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PERICARDIAL EFFUSION IN AN ANTICOAGULATED PATIENT: CASE REPORT FROM A REMOTE LOCALITY

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ABSTRACT

Pericardial effusion is a potentially life-threatening condition, often requiring urgent specialist intervention before progression to cardiac tamponade and subsequent cardiac arrest. One such case was seen in Lorne and the Islands emergency department one week post pulmonary endarterectomy for severe pulmonary hypertension. Imaging identified a 35ml effusion with significant chamber compression. This presented a challenging management and retrieval case which was complicated by the patient's concomitant use of apixaban and remote location. The case is discussed, and a literature review undertaken.

KEYWORDS: tamponade, pericardial effusion, rural medicine, emergency, apixaban.

INTRODUCTION

Pericardial tamponade is a serious, life-threatening condition characterised by the compression of all cardiac chambers due to increased intrapericardial pressure. This increase in pressure is usually the result of excessive fluid buildup within the pericardial cavity (a.k.a. pericardial effusion). There are a myriad of aetiologies that would precipitate a pericardial effusion, including chest trauma (2.1%), connective tissue disorder (4.1%) and neoplastic disease.[1] Depending on the aetiology, composition would vary from serous fluid to blood.

While pericardial effusion is a relatively uncommon condition, seen in up to 6.5% of the adult population based on the framingham cohort study, it poses significant risk to life if the effusion progresses to tamponade. Patients may develop early symptoms of cardiogenic shock (shortness of breath, chest pain and in more severe cases, syncope, lightheadedness and altered mental status). If left untreated, these symptoms are likely to worsen, eventually leading to cardiac arrest.[2] As such, prompt recognition and treatment of cardiac tamponade is crucial.

CASE REPORT

A 52 year old lady was seen in Lorn and the Islands emergency department, having been brought in by ambulance following a brief loss of consciousness at home. The patient had been seen in primary care the day prior, and diagnosed with urinary tract infection. Other relevant medical history was of chronic thromboembolic pulmonary hypertension, lupus related antiphospholipid syndrome, and recent pulmonary endarterectomy 2 weeks prior. Following this procedure, the patient had been started on the anticoagulant apixaban.

Physical examination rapidly identified haemodynamic instability, with no recordable NIBP and no peripheral pulses, though femoral pulses were felt. Given the patients recent medical history, bedside transthoracic echocardiogram was conducted; this showed a large pericardial effusion of approximately 35mm in diastole with tamponade. Subsequent CT thorax reported 4cm pericardial effusion with compression of all four chambers of the heart (see **Figure 1**).

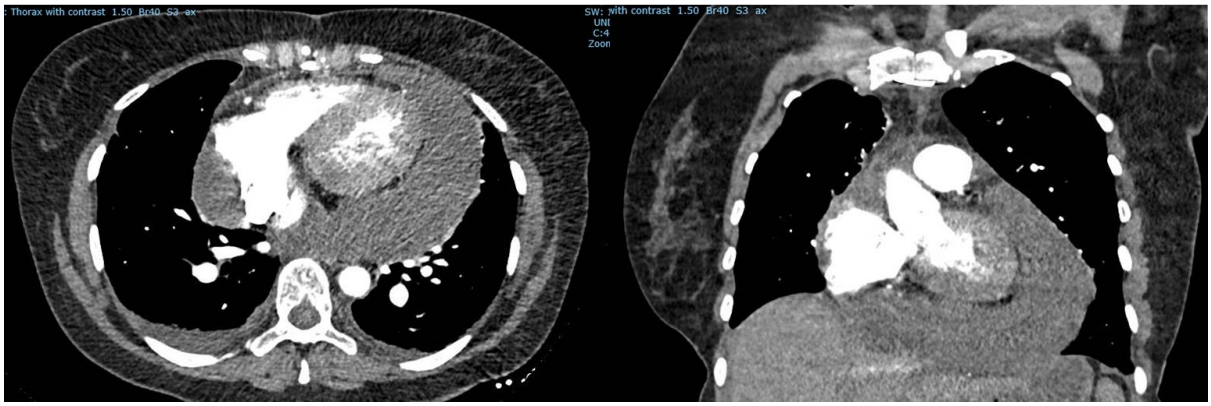

Figure 1: CT Thorax (Axial and Coronal view) of pericardial effusion with tamponade.

Initial biochemical results were: Hb 106 g/l, APTT 31, APTTr 1.0, PT 17, Fibrinogen 4.8, and INR 1.5. Given the peri-arrest status of the patient, serious consideration was given to immediate pericardiocentesis. After consultation between on-site surgical, anaesthetic, and medical specialists with a tertiary centre cardiothoracic unit, and with consideration to the patients' anticoagulated state, the decision was made to delay pericardiocentesis and attempt reversal of anticoagulant with prothrombin complex before urgent aeromedical evacuation to a cardiothoracic unit inland.

The patient's condition did not deteriorate further, and they were retrieved to the tertiary centre within 3 hours of arrival at the emergency department.

DISCUSSION

Early detection and management is pivotal in preventing the progression of a pericardial effusion to tamponade. At present, there are no validated criteria for risk stratification of patients presenting with an effusion. As such, the aim of this case discussion would be to provide evidence-based recommendations for clinicians in various clinical settings, in dealing with patients with a tamponade.

Generally, patients presenting to the emergency department lie somewhere on the haemodynamic spectrum from preclinical tamponade to severe haemodynamic shock. Therefore, in any patient presenting with hypotension, tachycardia, tachypnoea, severe dyspnoea and/or *pulsus paradoxus*, cardiac tamponade should always be suspected. An electrocardiogram (ECG) is a fast and useful investigation that may show low QRS voltage gains or electrical alternans – indicative of tamponade.[3] If cardiac tamponade is suspected, the diagnostic investigation of choice is an echocardiogram. This allows direct visualisation of a large pericardial effusion, diastolic collapse of the right atrium and ventricle, and inferior vena cava plethora (dilation). Computed tomography (CT) and cardiac magnetic resonance imaging (MRI) are only indicated in complex cases (i.e. loculated effusions).[4]

Pericardiocentesis is a potentially dangerous procedure, and the risk-benefit profile for its immediate, urgent, or elective undertaking is based on the clinical presentation and echocardiogram, in addition to the patient's current and trending haemodynamic status. In a patient with an established diagnosis of cardiac tamponade who is haemodynamically compromised, emergency pericardiocentesis is mandatory. It is only by draining the fluid within the pericardial space that the external pressure exerted on cardiac chambers will be reduced; draining this space will improve ventricular filling, and by extension cardiac output as well. As such, there are no absolute contraindications to emergency pericardiocentesis in the event of a tamponade or haemodynamic shock.[4] Given that the signs and symptoms for cardiac tamponade are neither sensitive nor specific, a recent paper by *Halpern et al* introduced a scoring system to aid in the decision making process for pericardial drainage, based on clinical features, effusion size and echocardiographic findings.[5]

The patient described in this case was taking Apixaban, a factor Xa inhibitor. With a half-life of 12 hours, this class of drug provides adequate daily anticoagulation for patients without the need for regular monitoring. Their notable drawback is the lack of a reliable reversal agent.

To date, there is only one FDA-approved reversal agent for factor Xa inhibitors - andexanet alfa. Andexanet alfa is a recombinant human factor Xa that has a high affinity to factor Xa inhibitors. At present it is only indicated for anticoagulation reversal in life-threatening or uncontrolled bleeding in patients on apixaban or rivaroxaban. Additionally, it is only licensed for specialist use and is not widely available at present.[6] With such limited provision for reversal of factor Xa inhibitors, there is ongoing research into utilization of reversal agents used for other common anticoagulants. In a three-period crossover study, 4-factor prothrombin complexes (i.e. Beriplex and Cofact) have shown to reverse the effects of apixaban on endogenous thrombin potential (ETP), returning the ETP levels to pre-anticoagulation baseline 4 hours after prothrombin complex infusion. Additionally, it was shown to have a reversal effects on prothrombin time and INR. These

results suggest that 4- factor prothrombin complexes could prove useful in the management of bleeding attributed to factor Xa inhibitors. However, further evaluation is required to identify the adequate dosing requirements and any potential safety issues.[7]

CONCLUSION

In this particular case, the decision to delay pericardiocentesis was in line with the scoring system proposed by *Halpern et al*. Delaying this intervention also enabled the patient to be transferred to a specialised tertiary institution. In the event of any further deterioration, emergency percutaneous pericardiocentesis with echo guidance would have been conducted immediately. This was considered the best option, given the patient's anticoagulated state and the limited resources available.

Transfer to a tertiary centre was also in accordance with criteria listed by the European Society of Cardiology. They state that in a clinical setting with limited experience with pericardiocenthesis or in the presence of any contraindications such as ongoing anticoagulation therapy with INR above 1.5, thrombocytopaenia, or posterior/loculated effusions, prompt transfer to tertiary centre is recommended provided the patient is haemodynamically stable.

As there was no andexanet-alfa available in hospital for apixaban, reversal was attempted with beriplex. Although it was uncertain if this would help in reducing the bleeding risk, the patient did not develop any bleeding complications after her pericardiocenthesis (conducted in tertiary hospital after transfer). It is worth noting that the patient should have ideally been anticoagulated with warfarin rather than apixaban, given her background of lupus related antiphospholipid syndrome - this was subsequently rectified during her admission.

In summary, cardiac tamponade is a life-threatening condition rapid recognition, stratification and management. The urgency for invasive drainage and suitability for inter-hospital transfer should be based on the physiological trend and haemodynamic status. The use of an appropriate scoring system as a reference would greatly benefit clinicians in the decision making process when dealing with pericardial effusion at risk of progression to tamponade.

Grant
None.

Conflicts of interest
We the authors of this case report have no conflicts of interest to declare.

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