CASE REPORT

Recurrent cardiac tamponade: an initial presentation of lung adenocarcinoma

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Accepted 28 April 2014

SUMMARY

A 63-year-old hypertensive woman presented initially to the surgical team with right upper quadrant pain, the patient was otherwise asymptomatic and clinically well. An abdominal CT scan excluded any surgical diagnoses but rather showed a pericardial effusion. When the cardiology team urgently reviewed her, they found her to be hypotensive and tachycardic with a raised jugular venous pressure. A diagnosis of cardiac tamponade was made and was transferred to the coronary care unit for an emergency pericardiocentesis. She developed tamponade on further occasions requiring pericardiocentesis. The underlying cause was investigated and following pericardial fluid analysis and subsequent imaging, metastatic lung adenocarcinoma was diagnosed.

BACKGROUND

Cardiac tamponade is an uncommon life-threatening medical emergency. Patients usually present in extremis with muffled heart sounds, hypotension, tachycardia and with prominent jugular venous pressure (JVP).¹ There are various underlying causes of tamponade with the commonest being iatrogenic, followed by malignancy, atherosclerotic disease of the heart and idiopathic causes.² Malignant cardiac tamponade accounts for up to 30% of cases seen in autopsy studies, but it is less common to identify tamponade during life.^{1 3} The most common cause of death in patients with metastases to the heart is from cardiac or respiratory failure.²

This case highlights the unusual presentation of lung adenocarcinoma and encourages clinicians to always look for an underlying diagnosis when encountering cardiac tamponade.

CASE PRESENTATION

A previously healthy 63-year-old woman presented to the surgical assessment unit with a 7-day history of right upper quadrant pain. She had a history of hypertension, was a non-smoker and only drank alcohol occasionally. The patient stated she had lost one stone in weight over 8 weeks but this was intentional. She did not have any red flag signs concerning malignancy. On examination, her pulse was regular at 80 bpm and the chest had equal bilateral air entry. The abdomen was tender in the right upper quadrant with the presence of guarding. An ECG showed non-specific ST changes with normal sized complexes, and a chest (figure 1) and abdominal X-ray did not reveal any abnormalities.

A provisional diagnosis of cholecystitis was suggested with an ultrasound booked for the morning. In the interim, she was given adequate analgesia and intravenous antibiotics.

The ultrasound scan showed no evidence of gallstones, oedema or increased wall thickness of the gallbladder; however, a right pleural effusion was noted. As the patient's pain was not being adequately controlled with analgesia, a CT of the abdomen was requested to exclude a ruptured gallbladder. The scan did not reveal any pathology in the gallbladder but rather noted bilateral pleural effusions with a significant pericardial effusion of 25 mm (figure 2). The patient was referred urgently for a cardiology review where the patient was found to be hypotensive and tachycardic with a pulsus paradoxus. A distended JVP could be seen from the end of the bed. A bedside echo illustrated a pericardial effusion of 2.5 cm with right ventricular collapse. A diagnosis of cardiac tamponade was made and the patient was immediately transferred to the coronary care unit where an emergency pericardiocentesis was performed.

The procedure was a difficult drain insertion and revealed a bloody effusion. A repeat echo postdrain insertion showed an improvement in fluid in the pericardial sac, which correlated with a clinical improvement in the patient. The echo also demonstrated fibrin deposits and a thrombus within the effusion could be seen over the right lateral ventricular wall.

Owing to this unusual presentation, a CT of the thorax was arranged which revealed evidence of a left upper lobe bronchial neoplasm measuring

Figure 1 Chest X-ray of the patient on initial presentation, no obvious malignant lesion or effusions are notable.

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To cite: Kumar A, Puttanna A. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-202553

Figure 2 CT of the abdomen revealing pericardial effusion of 25 mm with a large right-sided pleural effusion. A moderate left-sided pleural effusion is also present.

26×15.5 mm (figure 3) with mediastinal lymphadenopathy and possible liver metastases. A small volume bilateral pulmonary embolus was also present. The results of the pericardial fluid cytology returned the following day and confirmed the diagnosis of metastatic adenocarcinoma of the lung (T1b, N3, M1a).

INVESTIGATIONS

Admission blood results showed a white cell count of 11.2/L ($4-11 \times 10^9$) and a C reactive protein of 15 mg/L (<3 mg/L), which increased to 13.3 and 28 mg/L the next day. Liver function tests were raised with alanine transaminase 419 IU/L (3–35 IU/L), alkaline phosphatase 127 IU/L (30–35 IU/L), bilirubin 8 μ mol/L (3–17 μ mol/L) and gamma glutamyl transferase 228 U/L (0–45 U/L). Amylase was 61 Somogyi U/dL (0–180 Somogyi U/dL). The rest of her blood parameters were within normal limits. Blood and urine cultures were negative.

Before the CT of the thorax identified the lung mass, other differentials were excluded. A full autoimmune screen, including antinuclear, antineutrophil cytoplasmic and double-stranded antibodies, was carried out and found to be negative. Immunoglobulin levels were taken and found to be within

Figure 3 CT of the thorax revealing a left lung upper lobe mass present with a large right-sided pleural effusion.

normal limits, as was C3, C4 and CA 125. Electrophoresis was negative.

Pericardiocentesis revealed a clear fluid appearance, fluid total protein 31 g/L (60–80 g/L), albumin 13 g/L (35–50 g/L), serum albumin 33 g/L and lactate dehydrogenase 1669 IU/L (70–250 IU/L). Pericardial fluid cytology indicated that malignant cells were present from a metastatic adenocarcinoma. This was further confirmed by histology results, which showed positive tumour markers of BerEP4 and cytokeratin 7 with a few nuclei being positive for thyroid transcription factor-1.

DIFFERENTIAL DIAGNOSIS

The initial presentation of right upper quadrant discomfort together with raised inflammatory markers indicated the possibility of infection (intra-abdominal or right lung base); however, the patient had no fever. It is likelier that cardiac tamponade manifested as hepatic congestion, hence the elevated liver enzymes.

The chest X-ray did not note any nodule in the lung which could be due to the difficulty in identifying nodules on plain films, hence the need for CT of the thorax.

The underlying cause of the tamponade had a variety of aetiologies, although results from the cytology and imaging suggested malignancy.

TREATMENT

Over the subsequent days, the patient was found to be in tamponade on two further occasions with significant effusions. As the drain kept blocking, repeat pericardiocentesis had to be performed, each time draining heavily blood-stained fluid. An ultrasound of her chest also showed gross fluid collection in the left base and a 4 cm pericardial effusion but good left ventricular motion. A chest drain was inserted, which drained 1.5 L of blood.

OUTCOME AND FOLLOW-UP

A consensus decision with cardiologists, respiratory physicians and oncologists in a multidisciplinary team meeting concluded that she had a poor prognosis of less than 6 months and was not suitable for surgery. It was ultimately decided to proceed with chemotherapy. She had finished her fourth cycle of chemotherapy (pemetrexed and cisplatin) and her latest CT scan showed the mass to have reduced to 21×9 mm and staging of T4, N3, M1a. The patient remains well at 7 months postdiagnosis and 5 months since start of chemotherapy with no further episodes of tamponade.

DISCUSSION

Cardiac tamponade occurs when blood, fluid or pus builds up in the space between the myocardium and the pericardium.¹ This prevents the cardiac ventricles from fully expanding during diastole and the excess pressure from the fluid prevents adequate systolic motion. As a result, stroke volume and cardiac output become significantly reduced.² The progression of cardiac tamponade is dependent on the fluid volume and rate of accumulation.¹ Unless diagnosis and management are initiated immediately, it can develop rapidly with potentially fatal consequences.^{3 4}

The classical feature of cardiac tamponade is Beck's triad, which includes hypotension secondary to reduced stroke volume, distended JVP due to impaired venous return to the heart and muffled heart sounds as a result of fluid within the pericardium.⁵ Systemic venous congestion may also present with hepatomegaly and peripheral oedema. Other signs include

pulsus paradoxus, where, the arterial blood pressure drops to 10 mm Hg on inspiration, as well as septic shock. Tachycardia and an elevated JVP are the two commonest signs seen in patients with tamponade.¹

Clinical features of malignant tamponade tend to be more non-specific and develop as an acute-on-chronic event of a large, slowly developing pericardial effusion.^{6,7} Thus, the classic features may not initially be seen⁵ and symptoms are usually respiratory in nature.³ Physical signs, ECG and chest radiographs can be non-specific.³ The diagnostic test of choice is echocardiography, which will show an enlarged pericardium with evidence of right ventricular diastolic collapse.⁷

Recent studies have indicated that the primary cause of tamponade is iatrogenic followed by malignancy, complications of acute myocardial infarction and idiopathic disease.⁸ Of the malignancies, more than half of all cardiac metastases are due to lung and breast cancer.⁹ The remainders are accounted for by haematological malignancies, melanoma, mesothelioma, sarcoma, digestive, thyroid and renal tumours.²⁻⁷

The pathogenesis behind malignant tamponade is Fraser's hypothesis of retrograde lymphatic dissemination from malignant mediastinal lymph nodes.⁴ The obstruction of lymphatic and venous outflow from mediastinal lymph nodes to the epicardial lymphatic plexus by the tumour will then result in an accumulation of fluid.^{3,7}

The incidence of primary tumours arising from the pericardium is below 0.1%, with pericardial disease being rarely the first presentation of a tumour. The likelihood of finding a neoplastic disease with the initial presentation of pericarditis or a small pericardial effusion is only 4–7%, although this increases to 23–33% for large pericardial effusions.¹⁰

In a case series of 90 patients, the underlying neoplasm giving rise to malignant pericardial effusion was most commonly from the lung (61%), followed by the gastrointestinal tract, unknown origin, breast, kidney, lymphoma and leukaemia.¹¹

The most frequent site of metastatic involvement of the heart in primary lung cancer is the pericardium.² Pericardial lesions were either the direct or contributory cause of death in between 36% and 85% of patients with symptomatic pericardial effusions.¹² It is sometimes difficult to identify effusions during life with only 30% of cases being correctly diagnosed before death.⁷ Unlike the patient in our case, the diagnosis of malignant pericardial effusion is usually made after the diagnosis of malignancy has been established.³

Effusions associated with neoplastic infiltration of the pericardium tend to be bloody, rapidly accumulating and large with the average aspirate ranging from 840 to 1200 mL.^{2,7} Our patient drained 250 mL in her first presentation; however, in her second presentation, 850 mL was drained. Cardiac tamponade rarely manifests as the initial presentation of malignancy, and once diagnosed, prognosis is poor with limited life expectancy.³ The 30-day mortality in patients with malignant cardiac tamponade is 32.9% with mean survival quoted to be 144 days.⁷

Management options include pericardiocentesis, pericardiectomy, creation of a pericardial window and chemotherapy or radiotherapy.²

Pericardiocentesis may be life-saving and brings about immediate symptomatic relief in 90% of patients as well as provide fluid for diagnosis.¹³ There is only 1% risk of major complications,¹³ which include puncturing of the coronary artery or myocardium, arrhythmias or pneumothorax.¹⁴ Guided by echocardiography, this is a relatively safe and effective procedure.¹⁵

Surgical drainage may be indicated in patients with intrapericardial bleeding, clotted haemopericardium or a difficult needle drainage insertion.¹ Pericardectomy has become uncommon due to its high operative risks.³ Recently, pericardial window either by subxiphoid route, anterior thoracotomy or by thoracoscopy has become the preferred option.^{3,16} This procedure creates a communication between the pericardium and the left pleural space where to ensure permanent drainage, the pericardium is stitched to the lung.¹⁶ The advantages being a relatively short and small-scale operation that can also be performed under local anaesthesia (for subxiphoid route) and allowing biopsies to be performed if diagnosis is needed.^{15,16}

Owing to the lack of a larger, prospective, randomised trials, there is currently no consensus on the most appropriate management and so the decision is very patient–physician dependent.¹⁰

Learning points

- ▶ Maintain a high index of suspicion in patients presenting with acute cardiac failure and hypotension of unknown aetiology to avoid delay in the diagnosis of cardiac tamponade.
- ▶ Bloody pericardial effusions associated with tamponade are most commonly associated with iatrogenic causes, malignancy, complications of myocardial infarction and idiopathic disease.
- ▶ Underlying malignancy must always be considered, especially if recurrent tamponade and other causes are unlikely.
- ▶ Pericardiocentesis is life-saving and offers immediate symptomatic relief as well as diagnosis. Pericardial window is preferred for recurrent tamponade due to its low operative risks and short procedure time.
- ▶ Prognosis is poor in patients with malignant tamponade, so early diagnosis and treatment is essential for patient care.

Contributors The authors were involved in all aspects of the case report write-up.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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