CASE REPORT

The unmasking of a pyopericardium

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SUMMARY

Pyopericardium is a rare condition with a high mortality rate in which infection propagates in the pericardial space, leading to a pus filled pericardial effusion and cardiac tamponade, which can cause cardiogenic shock and death. We present a case of a previously healthy woman of 52, who was admitted with a severe lower respiratory tract infection that eventually led to a pyopericardium. The diagnosis of pyopericardium was delayed due to masking of symptoms by her underlying infection, sepsis and an upper gastrointestinal bleed that the patient suffered during the admission, requiring an emergency gastroscopy. The pyopericardium was considered when ST elevation was seen on an ECG and an ECHO discovered a large pericardial effusion causing tamponade. An emergency pericardiocentesis drained frank pus. Treatment with intravenous antibiotics, frequent pericardial drainage and a pericardectomy led to an excellent outcome, and a full recovery.

BACKGROUND

Since the advent of antibiotics, pyopericardium is a rare phenomenon.¹ It has a high mortality rate of 40% in treated patients, and near 100% in untreated patients, therefore it is important not to miss the diagnosis.²⁻³ In the current era of antibiotic resistance, we may see a resurgence of this rare infective condition. It is, for this reason, a relevant condition that is important to be aware of.

CASE PRESENTATION

A 52-year-old woman was referred from a primary care centre with a 12-day history of right-sided pleuritic chest pain and shortness of breath on exertion. Usually fit and well, she lived with her husband, had not travelled recently, was working at a supermarket and did not smoke or drink alcohol. Examination revealed coarse crepitations over the right lung base, tachycardia, a soft abdomen and normal heart sounds. Oxygen saturations were 94% on air with an elevated respiratory rate. Blood pressure was 112/64 mm Hg and the patient was febrile. Blood tests revealed a white cell count of $17.5 \times 10^9/L$ and C reactive protein of 411 mg/L. Urea was 15.1 mmol/L, creatinine and haemoglobin were in the normal range. A Chest X-ray revealed right basal consolidation (figure 1). The initial impression was a lower respiratory tract infection causing pleural irritation.

The patient was moved to a respiratory ward and started on intravenous antibiotics (tazocin 4.5 g three times a day), intravenous fluids and 28% oxygen.

Over the next 2 days she began to develop mild ankle oedema; the impression was oedema due to

Figure 1 Admission chest X-ray, note the right basal pneumonia.

sepsis and low albumin (of 26 g/L). Furosemide 40 mg was started and the intravenous fluids were slowed down.

On the second night, the patient had a large episode of malena. Blood pressure dropped to 88/50 mm Hg and she was tachycardic at 116 bpm. Haemoglobin was 6.1 g/dL and urea was 29.3 mmol/L, with normal creatinine. Four units of packed red cells were transfused and a diagnostic gastroscopy was organised the next day, which revealed a large 2.25 cm bleeding duodenal ulcer. This was injected with epinephrine and closed with clips. The antibiotics were continued intravenously, and a proton pump inhibitor was started. It was later thought that the duodenal ulcer was related to regular ibuprofen use leading up to the admission.

Haemoglobin remained stable; however, the patient's condition deteriorated. She began to report generalised abdominal pain and the ankle oedema was noted to be gradually worsening. Respiratory rate was 28 breaths/min and her saturations were 96% (on 28% oxygen). She was, however, haemodynamically stable. White cell count had risen to $20.8 \times 10^9/L$ and C reactive protein was still high at 168 mg/L. A chest X-ray on day 5 confirmed worsening right basal consolidation and small bilateral effusions (figure 2). Intravenous gentamycin was added to the treatment regime, and blood cultures were sent, which later could not identify an organism.

The patient's clinical condition did not improve. On day 7, her antibiotics were switched to ceftriaxone and clarithromycin, and she was planned for an ultrasound guided chest drain to relieve the pleural effusions. She was still reporting abdominal pain. Before the procedure could be carried out she became unresponsive, with a bradycardia and low

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Figure 2 Chest X-ray on day 5 of admission. Note the larger cardiac shadow in this image compared to figure 1.

systolic blood pressure of below 80 mm Hg. She was requiring high flow oxygen to maintain saturations, had cold peripheries and a high jugular venous pressure.

The patient was transferred to the intensive treatment unit (ITU), intubated, sedated and given inotrope support. She had a high central venous pressure of 16 mm Hg and low blood pressure of 93/67 mm Hg.

INVESTIGATIONS

A portable chest X-ray confirmed bilateral pleural effusions and pulmonary oedema, with a large cardiac shadow (figure 3).

An ECG performed in ITU showed saddle-shaped ST elevation (figure 4), which prompted the decision to carry out an emergency ECHO. This ECHO showed a large pericardial effusion (figures 5 and 6).

DIFFERENTIAL DIAGNOSIS

The diagnosis at this point was thought to be a pericardial effusion causing tamponade and cardiogenic shock, however, the cause of the effusion was unclear.

TREATMENT

An emergency pericardiocentesis aspirated frank pus from the pericardial space. The diagnosis of pyopericardium could now be made. The pericardial drain was left in situ for further drainage and intravenous antibiotics were continued. A total of 1.1 L of pus was drained in the first 24 h. Microscopy revealed a high

Figure 3 Portable chest X-ray on day 7 of admission in the intensive treatment unit. Note the large cardiac shadow, worsened right lower lobe consolidation and bilateral effusions.

white cell count comprising of macrophages and neutrophils, however, no specific organism could be grown in culture. Pleural fluid from a left chest drain grew nothing in culture.

Transvenous pacing was required in ITU due to transient episodes of heart block. The pericardial drain required frequent aspiration to prevent further tamponade, and fluid balance was closely monitored to prevent overload.

Cardiothoracic surgeons performed a complete pericardectomy from an anterior approach. Pus was confirmed in the pericardial space with purulent tissue found in parts of the epicardial muscle. The heart was lifted out of the pericardial sac and debrided of all debris and purulent tissue, and then repositioned in the residual cradle of the pericardium.

The patient required continued intravenous antibiotics, diuretics and fluid management in ITU following her operation. After a further 6-day ITU stay she was transferred to a rehabilitation ward for physiotherapy as she had significant respiratory and skeletal muscle weakness from her prolonged period of sedation.

OUTCOME AND FOLLOW-UP

The patient required intensive physiotherapy and other than this recovered well. She was discharged from cardiology follow-up after 6 months as her symptoms had fully resolved.

DISCUSSION

Pyopericardium (sometimes called purulent pericarditis) is diagnosed when pus is drained from the pericardium or bacteria are grown from the pericardial space. It is rare, accounting for less than 1% of causes of pericarditis.⁴

The mortality rate can reach 100% if left untreated, due to cardiogenic shock as increasing amounts of pus in the pericardial space cause tamponade.³ ST elevation on an ECG may indicate pericarditis, but diagnosis is mostly made after an ECHO followed by aspiration of pus from the pericardial space.⁵ Other imaging such as CT and MRI can be used to aid diagnosis.¹ The condition generally presents with fever, chest pain, congestive cardiac failure, (pulsus paradoxus may be present indicating tamponade), and later with sepsis and cardiogenic shock.⁵ Central venous pressure may be high, with a low blood pressure (as was the case with our patient).

Diagnosis can be delayed due to the initial source of infection distracting from the underlying cardiac problem, and symptoms can be confused with acute coronary syndrome.⁶ The diagnosis of pyopericardium in our case was hindered by the complicated presentation, lack of chest pain, and the plausible explanation of symptoms with the diagnosis of pneumonia, sepsis and a bleeding duodenal ulcer. Peripheral oedema and pleural effusions may have alluded to the diagnosis, in addition to the fact that the cardiac shadow had increased in size throughout the chest X-rays performed during the stay (figures 1–3).

The likely reason for the lack of ability to culture an organism in the case of our patient was due to the high-dose antibiotics that the patient was receiving, which may have impeded growth of the organism in samples sent. The most common organism reported to cause pyopericardium in previous cases is *Staphylococcus aureus*.⁷ It has been reported that Gram-negative bacteria and fungi are becoming more frequent due to infection in immunocompromised hosts.⁷

Causes linked to pyopericardium are pneumonia, empyema, thoracic surgery and haematogenous spread through sepsis. More rare causes include perivalvular abscess rupture in endocarditis and spread along fascial planes from the oral cavity.^{1 5 6} Factors that can predispose to pericarditis include

Figure 4 ECG of our patient from intensive treatment unit on day 7 of admission showing saddle-shaped ST elevation in leads V4, V5 and V6.

immunosuppression, alcohol abuse and previous pericardial inflammation and fibrosis.^{5 6} Our patient had lost a lot of blood due to the upper gastrointestinal bleed (haemoglobin dropped to 6.1 g/dL) and subsequent massive blood transfusions given could have resulted in transfusion-related immune modulation, weakening the host's ability to mount an effective initial

immune response.⁸ In our patient's case, the source of infection could have been either haematogenous spread through sepsis, or from empyema, both caused by the pneumonia. Unfortunately, the colour of the pleural fluid was not documented and no organism was grown, so empyema cannot be confirmed.

Treatment is based around drainage of the pyopericardium, antibiotic therapy and pericardectomy.⁶ Complications of the pericardial catheter can include pneumothorax and cardiac or coronary laceration during insertion. The risk of these can be reduced with echographic guidance of catheter placement.^{9 10} The pericardial catheter is left in situ to allow further drainage and prevent an effusion recurring (which can occur in up to 75% of cases).¹¹ It is recommended that the drain is left until less than 25 mL a day is draining.¹¹

There is also literature that states that an epicardectomy may also be performed in order to liberate movement of the myocardium.¹ The 'turtle cage' technique has also been suggested as an alternative to epicardectomy, in which slits are made in the epicardium to allow movement of the fibrotic sections.¹² In our patient, an epicardial debridement was performed and had a good outcome.

Figure 5 Parasternal long-axis ECHO view of our patient's left ventricle, showing the large pericardial effusion, which was found to be a pyopericardium.

Figure 6 Another ECHO view of the pyopericardium, this time a parasternal short-axis view of the left ventricle.

Learning points

- ▶ Coexisting infections can mask symptoms of a more sinister pathology.
- ▶ When a previously healthy patient is becoming fluid overloaded, look for an underlying cause. Consider an ECHO.
- ▶ Saddle-shaped ST elevation, while most commonly caused by viral pericarditis, can have much more sinister causes that should be remembered.
- ▶ Comparison with previous X-rays is important; in this case the enlarging cardiac shadow could be seen from the first and second X-rays.
- ▶ Beware the patient with atypical chest pain, fever and cardiogenic shock.

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