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CASE REPORT
Other

Recurrent haemorrhagic pericardial effusion due to idiopathic pericarditis: a case report

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Background Haemorrhagic pericardial effusion (PE) has been described in pericarditis due to infection, neoplasm, collagen vascular disease, uraemia, pericardial inflammation after acute myocardial infarction, trauma, irradiation, and idiopathic pericarditis. Patients with large haemorrhagic PE develop recurrence or constrictive pericarditis (CP) frequently as complication without being treated intensively.

Case summary A 22-year-old female patient with a previous episode of pericarditis with severe PE was admitted for acute pericarditis. Three days before, she was evaluated at the emergency department and presented normal laboratory workup and no significant findings in the transthoracic echocardiogram (TTE). A new TTE showed severe PE and laboratory work-up showed low haemoglobin levels. Fifteen days later, due to slow evolution, a left anterior mini-thoracotomy pericardial window procedure was performed finding minimal haemorrhagic PE with clots. We performed a complete work-up for a cause without significant findings and treated intensely to prevent recurrence or CP.

Discussion This is a case of recurrent haemorrhagic PE due to idiopathic pericarditis. Physicians should perform an intensive workup in order to find the cause because of its clinical implications and possible treatments. An intensive treatment must be initiated as soon as possible to prevent recurrence or CP.

Keywords Haemorrhagic pericardial effusion • Idiopathic pericarditis • Constrictive pericarditis • Case report

Learning points

- Presence of haemorrhagic pericardial effusion (PE), haemodynamic compromise, or pleural effusion is more common in patients with tuberculous pericarditis or in malignant pericarditis. However, it can also be caused by idiopathic pericarditis.
- Patients with large haemorrhagic PE not receiving intensive therapy often develop recurrence or constrictive pericarditis.

Introduction

Pericardial effusion (PE) is a common finding in everyday clinical practice. Haemorrhagic PE has been described in pericarditis due to infection, neoplasm, collagen vascular disease, uraemia, pericardial inflammation after acute myocardial infarction, trauma, irradiation, and idiopathic pericarditis.[1] There are also cases of haemorrhagic pericarditis described in patients taking glycoprotein IIb/IIIa inhibitors or anticoagulants. Patients with large haemorrhagic PE develop

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recurrence or constrictive pericarditis (CP) frequently as complication without being treated intensively.

Timeline

5 months prior to presentation	Admission for chest pain in another centre. Equivocal diagnosis of acute pericarditis. No pericardial effusion (PE).
	Symptomatic treatment with nonsteroidal anti-Inflammatory drugs (NSAIDs) for a week.
4 months prior to presentation	Admission for recurrent acute pericarditis with severe PE. Haemoglobin of 9.9 g/dL.
	Treatment with ibuprofen and colchicine for 3 months.
3 days prior to presentation	She presented recurrence of chest pain. A transthoracic echocardiogram (TTE) showed mild PE.
	Haemoglobin of 12.6 g/dL and normal C-reactive protein (CRP) value.
	New treatment with Ibuprofen 800 mg three times a day (t.i.d.) and colchicine was initiated.
Upon presentation in emergency room	Hypotension and tachycardia. Severe chest pain. CRP of 18.2 mg/dL and haemoglobin of 10.6 g/dL.
	TTE showed severe PE with collapse of the right atrium.
	Change to Aspirin 1000 mg t.i.d. and colchicine.
12 days after	Persistence of moderate PE with fibrin content. Constrictive pericarditis (CP) physiology in control TTE. Chest X ray showed important left pleural effusion.
	Haemoglobin 8.8 mg/dL.
15 days after	Pericardial window with biopsy and drainage was performed. Minimal haemorrhagic PE with clots. Adhesions between epicardium and pericardium.
19 days after	Clinically stable. TTE with mild PE. No CP physiology.
	Discharged with Ibuprofen 800 mg t.i.d. and colchicine.

Case presentation

A 22-year-old female patient, with a previous episode of pericarditis with significant PE 4 months ago and no other comorbidities, was evaluated at the emergency department due to inspiratory and worse on lying down chest pain of 3 days duration without other symptoms. Electrocardiogram did not show significant findings. A transthoracic echocardiogram (TTE) was performed showing mild PE and laboratory determinations were normal including haemoglobin level of 12.8 g/dL (female 12–16 g/dL), except for a mild increase in C-reactive protein (CRP) value of 1 mg/dL (0–0.5mg/dL) (*Table 1*). She was discharged with ibuprofen 800 mg t.i.d. and colchicine 0.5 mg b.i.d.

Three days later, she was readmitted for persistence of chest pain. Upon arrival, she had tachycardia (115 b.p.m.), hypotension (85/55 mmHg), and temperature of 37.8°C. Cardiac auscultation was normal. Laboratory analyses were normal, except haemoglobin of 10.6 g/dL (12–16 g/dL), and CRP of 18.2 mg/dL (0–0.5mg/dL). A new TTE was performed showing global significant PE of with maximum diameter of 28 mm around lateral wall, collapse of the right atrium and without echocardiographic evidence of tamponade [variation of trans-mitral flow on Doppler with inspiration <25%, no collapse of right ventricle (RV), and normal inferior vena cava (IVC) diameter] (*Figure 1A* and *B*). Treatment with Aspirin 1000 mg t.i.d. and colchicine 0.5 mg b.i.d. was initiated.

An intensive work-up for a cause was performed. Consecutive laboratory assessment showed worsening of the anaemia with haemoglobin levels of (12–16 g/dL), increased CRP values up to 24.5 mg/dL (0–0.5mg/dL), and spontaneous coagulopathy with prothrombin time of 15.5 s (10–14 s). Autoimmunity study, quantiferon, and serologies were negative. In the previous admission for acute pericarditis with significant PE, she had also presented low haemoglobin levels of 9.4 g/dL (12–16 g/dL) which normalized after discharge. A chest computed tomography scan ruled out tumour masses or coronary lesions (*Figure 2*). She had a slow improvement without resolution of the PE and development of left pleural effusion, so the treatment was changed again to ibuprofen 800 mg t.i.d.

After the 12th day of treatment, a control TTE was performed showing a constrictive pericardial physiology with expiratory movement of interventricular septum towards the RV, IVC plethora, and persistence of moderate PE with abundant fibrin content (*Figure 1C* and *D*). Due to this finding, a left anterior mini-thoracotomy pericardial window with pericardium biopsy was performed, finding mild haemorrhagic PE with abundant clots and adhesions between the epicardium and the pericardium (*Figure 3*). Cytological examination of the pericardial and pleural fluid did not show malignant cells. The pericardial biopsy displayed inflammatory changes without evidence of malignancy. The analysis of the pericardial fluid revealed a haematocrit of 11.4% (<0.5%), white cell count of 2.99 $\times$ 10^3 µL (<1 $\times$ 10^3 µL) with 74.3% lymphocytes (20–45%), lactate dehydrogenase of 3733 U/dL (<200 U/dL), and glucose 73 mg/dL (74–106 mg/dL). Gram stain, Ziehl-Neelsen stain, and cultures were also negative.

Four days after the surgery, a new TTE showed mild residual PE and no CP physiology. She was discharged the next day with the same treatment. Three-months later, she remains asymptomatic and without PE or constrictive physiology in the TTE.

Discussion

Haemorrhagic PE has been described in pericarditis due to infection, neoplasm, collagen vascular disease, uraemia, pericardial inflammation after myocardial infarction, trauma, irradiation, and idiopathic pericarditis.[1,2] There are also cases of haemorrhagic pericarditis described in patients taking glycoprotein IIb/IIIa inhibitors or anticoagulants.

A large PE, presence of haemodynamic compromise or pleural effusion is more common in patients with tuberculous pericarditis or in malignant pericarditis.[3–5] In patients with no apparent cause of PE at the time of diagnosis (40%), the presence of typical inflammatory

Table 1 Laboratory values measured at baseline and during the recent admission

Normal reference values	5 months prior to presentation	4 months prior to presentation	3 days prior to admission	Upon admission	12 days after
WBC (4000–11 000/μL)	8850	6940	10 450	6810	7470
Hb (female 12–16 g/dL)	11.6	9.9	12.8	10.6	8.5
Hct (36–48%)	36.3	31.3	38.6	32.1	25.6
TSH (0.5–5 IU/mL)	1.89	2.36	NA	0.99	NA
FT4 (0.8–2.3 ng/dL)	NA	1.04	NA	1.06	NA
Cr (0.6–1.2 mg/dL)	0.66	0.68	0.7	0.6	0.6
Troponin I (<0.08 ng/mL)	NA	<0.012	<0.012	<0.012	NA
CRP (0–0.5mg/dL)	NA	4.9	1	18.2	24.9
ALT (0–20 U/L)	16	12	NA	21	13
PT (10–14 s)	NA	13.8	NA	12.9	15.5
ANA (<1:80 dilution)	NA	1/160	NA	<1/80	<1/80
RF (0–14 UI/mL)	NA	5	NA	15	NA

ALT, alanine aminotransferase; ANA, antinuclear antibody; BUN, blood urea nitrogen; Cr, creatinine; CRP, C-reactive protein; FT4, free tetraiodothyronine; Hb, haemoglobin; Hct, haematocrit; NA, not available; PT, prothrombin time; RF, rheumatoid factor; TSH, thyroid-stimulating hormone; WBC, white blood cell count.

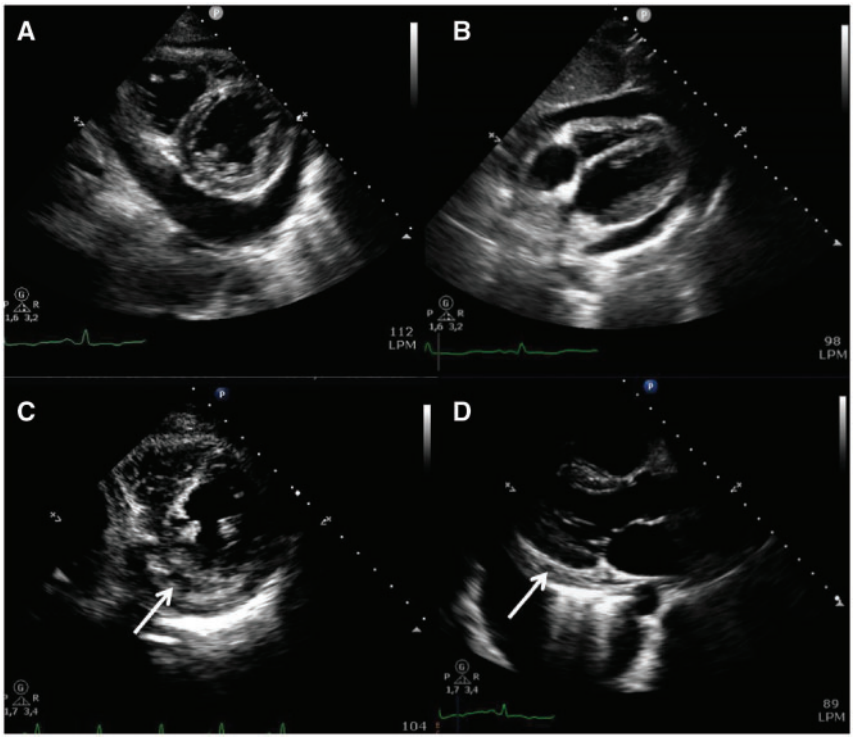

Figure 1 Transthoracic echocardiogram. (*A* and *B*) Short axis and subcostal view at admission. (*C* and *D*) Short- and long-axis view after 12 days of treatment showing fibrin content (arrow) in the pericardial space and left pleural effusion.

signs is predictive for acute idiopathic pericarditis, irrespective of the size of the effusion, and the presence or absence of tamponade.[6] We performed a complete workup for the underlying cause without any significant findings. It is difficult to establish a definitive bacteriologic diagnosis of tuberculous pericarditis, but since all bacteriologic tests including the pericardium culture and the quantiferon test were negative, we can rule out this possibility with reasonable certainty. The anaemia that the patient presented in both of the admissions for acute pericarditis could be explained by the haemorrhagic PE.

Constrictive pericarditis is one possible serious complication of recurrent pericarditis. The true prevalence of CP is unknown but it is observed in 0.2–0.4% of patients who have undergone cardiac

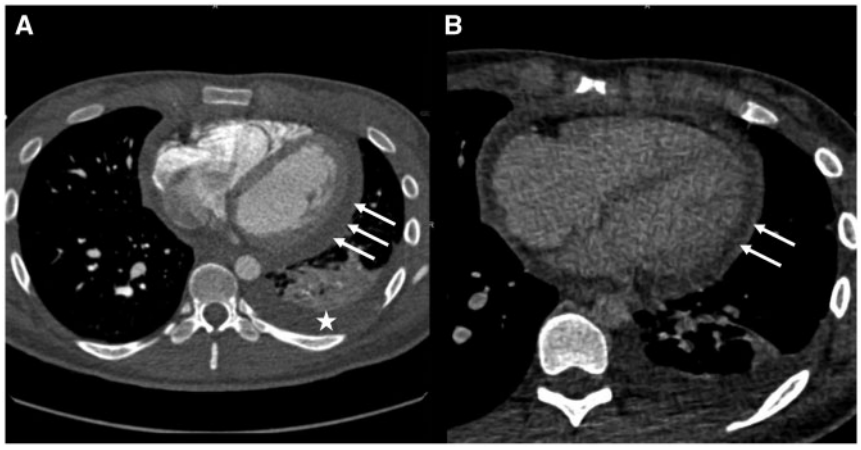

Figure 2 Thoracic computed tomography scan. (*A*) Axial view showing pericardial (arrows) and pleural effusion (star) with compressive atelectasis. (*B*) Late pericardial enhancement (arrows).

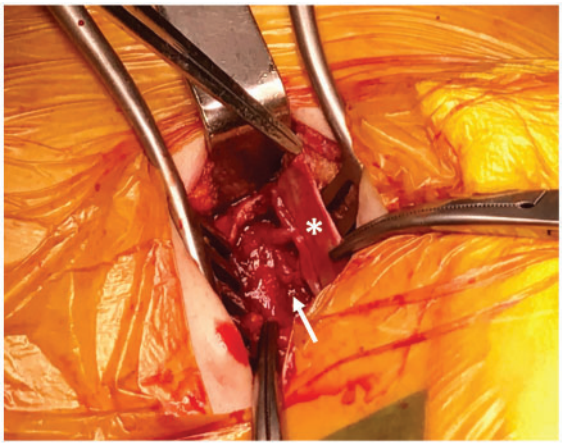

Figure 3 Pericardial window showing mild haemorrhagic pericardial effusion with clots (arrow) and the inflammation of the pericardium (asterisk).

surgery or have had a pericardial trauma or an inflammation due to a variety of aetiologies.[7] Constrictive pericarditis can occur after virtually any pericardial disease process, but only rarely follows recurrent pericarditis. The most common reported causes in developed countries were idiopathic or viral (42–49%).[8] Patients with large haemorrhagic PE developed recurrence or CP frequently as complication without being treated intensively.

Pericardial drainage procedures can be performed for diagnostic or therapeutic purposes and are not justified on a routine basis in patients without haemodynamic compromise.[5] Effusions of unknown origin are one of the exceptions to this rule and should be performed for diagnostic or therapeutic purposes. Physicians should perform an intensive workup in order to find a cause and initiate intensive treatment as soon as possible to prevent recurrence or CP.

Conclusion

This is a case of recurrent haemorrhagic PE due to idiopathic pericarditis. Physicians should perform an intensive workup in order to find the cause because of its clinical implications and possible treatments. An intensive treatment must be initiated as soon as possible to prevent CP.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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