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CASE REPORT
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A case report of fulminant primary streptococcal pericarditis

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Background Bacterial pericarditis is a rare, rapidly progressive, and highly fatal infection, even with drainage and antibiotics. Gram-positive cocci, specifically *Streptococcus pneumoniae*, have been the most common cause of bacterial pericarditis from either haematogenous dissemination, or spread from another adjacent site of infection. Following the introduction of antibiotics in the 1940s and more recently the pneumococcal conjugate vaccine, the incidence has drastically decreased.

Case summary A previously healthy young male was diagnosed with acute pericarditis with no signs of haemodynamic compromise on initial presentation. Several hours later, he became unstable suffering from cardiac tamponade and septic shock. Despite urgent pericardiocentesis and drainage of purulent fluid, culture positive for streptococcus pneumoniae, multi-organ failure was eventually fatal.

Discussion We describe a rare case of primary *S. pneumoniae* purulent pericarditis leading to tamponade, septic shock, and death. Due to the high mortality rate of purulent pericarditis, a high index of suspicion is needed in order to initiate appropriate therapy with antibiotics and drainage.

Keywords Bacterial pericarditis • Streptococcus pneumonia • Pericardiocentesis • Case report

Learning points

- Primary streptococcus pneumonia pericarditis is very rare.
- Purulent pericarditis has a very high mortality rate.
- Broad-spectrum antibiotics and drainage are needed.

Introduction

Bacterial pericarditis with cardiac tamponade due to purulent pericardial effusion is rare in the modern antibiotic era. It is a rapidly progressive and highly fatal infection. Gram-positive cocci, specifically *Streptococcus pneumoniae*, are the most common cause of bacterial pericarditis in the setting of haematogenous dissemination or direct spread of infection from an adjacent site.

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Timeline

Day 0	Emergency department of another hospital with pleuritic chest pain
	Discharged on ibuprofen and colchicine
Day 1	Ongoing severe chest pain
On admission	Emergency department of our hospital.
	Afebrile
	Blood pressure 110/75 mmHg
	Heart rate 85 beats/min
	Respiratory rate 16/min
	Oxygen saturation 98% on room air
	Physical examination unremarkable
	Electrocardiogram (ECG): diffuse concave ST-elevation
	Transthoracic echocardiogram normal
	Chest X-ray normal
	Leucocytosis and high CRP
Day 2	Hemodynamic instability
	Blood pressure = 90/60 mmHg
	Heart rate = 120 b.p.m., respiratory rate = 22/min
	Oliguric.
	Distended jugular veins and Pulsus paradoxus
	ECG: sinus tachycardia, PR depression, diffuse concave ST-elevation
	Echocardiogram: moderate pericardial effusion with signs of cardiac tamponade
	Pericardiocentesis: 600 mL of purulent fluid
	Multi-organ failure (marble-like skin, confusion, renal, hepatic and pancreatic failure, myeloid suppression).
	Intubation. Cardiac arrest.

Case presentation

A 41-year-old Caucasian male, without previous medical history, presented to the emergency department of another hospital with pleuritic substernal chest pain, radiating to the neck; his vital signs were reportedly normal. After evaluation, an initial diagnosis of low-risk acute pericarditis was established-absence of fever, pericardial effusion, and haemodynamic instability—and he was discharged on ibuprofen and colchicine, according to ESC Guidelines for pericarditis management.[1] The following day, due to persistent severe chest pain, he visited our emergency department. On admission, his temperature was 36.8°C, blood pressure (BP) of 110/75 mmHg, heart rate (HR) 85 beats/min, respiratory rate (RR) 16/min, and oxygen saturation 98% on room air. His physical examination was unremarkable. The electrocardiogram demonstrated normal sinus rhythm and diffuse concave ST-elevation (*Figure 1*) and the chest X-ray was normal. The transthoracic echocardiogram did not reveal pericardial effusion and the biventricular function was normal, without regional wall motion abnormalities. Initial laboratory results demonstrated leucocytosis and elevated C-reactive protein CRP (*Table 1*); our infectious disease specialist attributed these findings tentatively to viral Coxsackie infection, despite lack of symptoms consistent with upper respiratory tract infection; there was an enterovirus outbreak, including many Coxsackie cases, at the time. We, therefore, continued his treatment with ibuprofen 600 mg every 8 h and colchicine 0.5 mg every 12 h, adding an opioid analgesic (tramadol), considering him a case of low-risk acute pericarditis, as per ESC guidelines, which propose a short course of non-steroidal anti-inflammatory drugs, with the adjunct of colchicine for the prevention of recurrences.[1] Approximately 10 h later, despite the symptomatic improvement, he became haemodynamically unstable (BP = 90/60 mmHg, HR = 120 b.p.m., RR = 22/min), and he reported oliguria over the previous 6 h. On examination, distended jugular veins and pulsus paradoxus were found. His new electrocardiogram demonstrated sinus tachycardia with PR depression and diffuse concave ST-elevation. His arterial blood gases (ABGs) were pH 7.32/pCO_2 17.6 mmHg/pO_2 78.5 mmHg on nasal cannula 3 L/min, with lactate 7.5 mmol/L (normal value < 1 mmol/L). His new echocardiogram revealed moderate size (18 mm), semi-clear echo space between the epicardium and the parietal pericardium, indicating non-loculated pericardial effusion; in addition, there were signs of cardiac tamponade, including right ventricle and right atrium diastolic collapse and inferior vena cava plethora (*Figure 2*). We, therefore, proceeded with pericardiocentesis, via the subxiphoid route, and 600 mL of purulent fluid was drained (*Figure 2*). Despite complete pericardial drainage and with no evidence of effusive-constrictive pericarditis (absence of septal bounce and pericardial thickening), the patient remained haemodynamically unstable, even after 500 mL of normal saline administration, with signs of multi-organ failure (marble-like skin, confusion, renal and hepatic failure, and myeloid suppression) (*Table 1*). Subsequently, the patient became hypoxic and was, therefore, intubated but soon after suffered cardiac arrest. His initial rhythm was pulseless electrical activity and cardiopulmonary resuscitation (CPR) protocol was immediately initiated, with spontaneous circulation achieved within 5 min. At this point his ABGs were pH 7.122/pCO_2 72.8 mmHg/pO_2 86.0 mmHg on 100% O_2 administration, with lactate 19 mmol/L (normal value < 1 mmol/L). Despite noradrenaline and dobutamine i.v. infusions, the patient suffered again a cardiac arrest, and CPR was applied for 55 min, but without success. His pericardial and blood cultures were positive for *S. pneumoniae*.

Discussion

Bacterial pericarditis is a rarity, with an incidence of 1/18 000.[2] It is a highly fatal infection with mortality rates reaching 100% if left untreated,[3] and in half of the cases is diagnosed post-mortem.[2,3] Mortality rate remains as high as 40%, even when treated promptly, mainly due to cardiac tamponade, constriction, and septic shock.[2]

Diagnosis of purulent pericarditis is challenging, due to its infrequent occurrence, which leads to very low clinical suspicion. Predisposing risk factors include immunosuppression, alcohol abuse, and chest wall trauma.[4] In most cases, there is haematogenous dissemination or spread from an intrathoracic infection.

Our patient, with primary *S. pneumoniae* pericarditis, had no predisposing factors; he was immunocompetent, and he had no prominent signs of infection at another site. Unfortunately, this extremely rare condition—only very few cases of primary *S. pneumoniae* pericarditis have been reported in the literature[5]—led to cardiac tamponade and septic shock, which despite urgent pericardial drainage, was fatal. Pericardial drainage may be insufficient

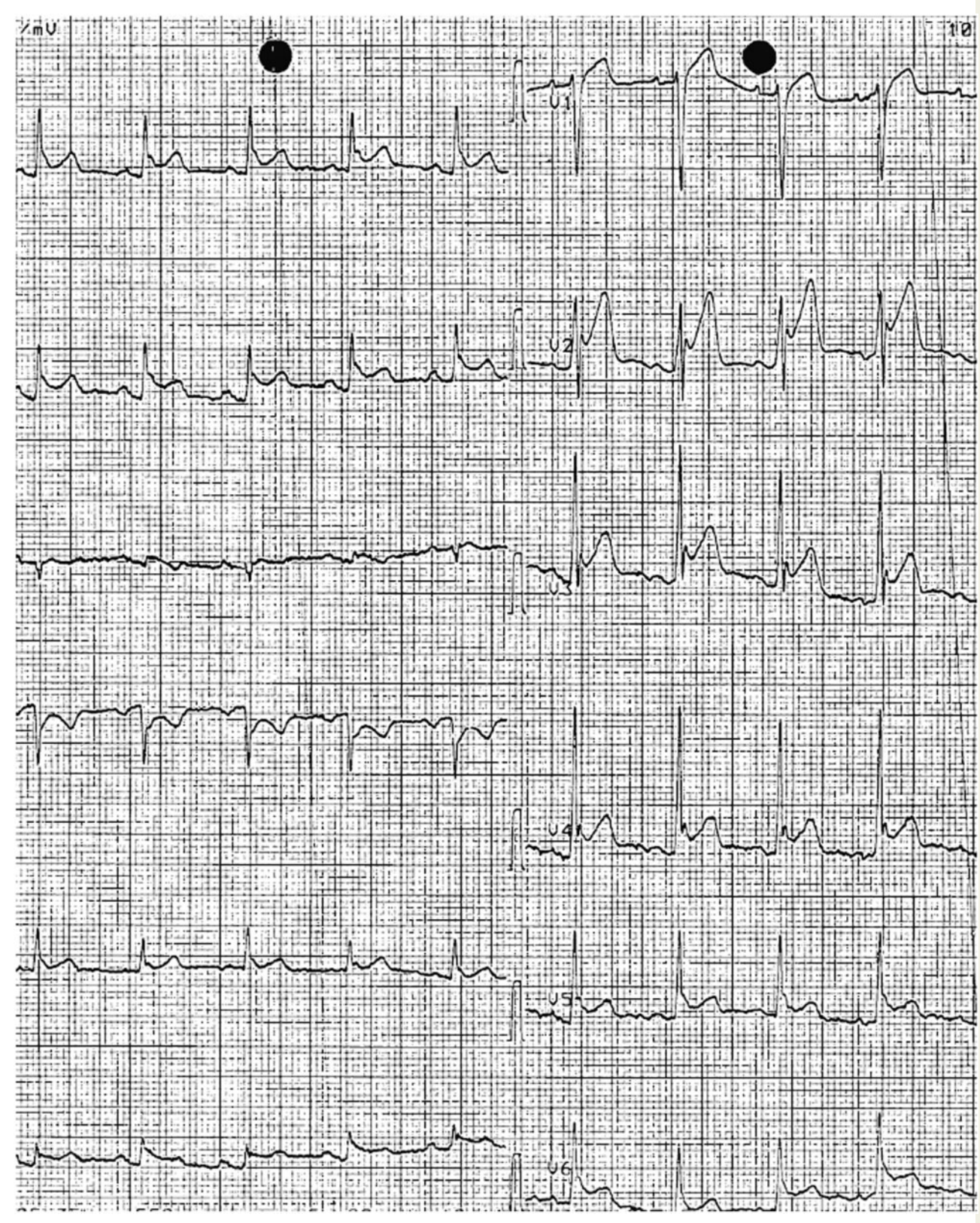

Figure 1 Electrocardiogram at presentation.

Table 1 **Laboratory parameters**

						Normal values
Time (h)	0	4	10	14	16	
WBC (k/μL)	26.1	30.6	3.3	1.8	0.7	4.0–11
Hb (g/dL)	14.7	13.9	12.8	11.9	11.9	12–17
PLT (K/μL)	346	328	214	124	80	140–400
INR	1.41	1.74	1.68		2.1	
LDH (mIU/mL)	317	250	214	185		135–214
SGOT (mIU/mL)	15	16	95	139		<32
SGPT (mIU/mL)	14	18	144	183		<33
CRP (mg/L)	115.3	501.3	578			<5
CPK (mIU/mL)	133	95	87	346		<170
CK-MB (ng/mL)	0.4	1	1,2	4		<3.1
TNI (ng/mL)	0.007	0.076	0.057	0.09		<0.01
Urea (mg/dL)	42	58	97	108		17–49
Creatinine (mg/dL)	0.8	1.2	2.9	3.8		0.5–0.9

WBC=white blood cells count, PLT=platelets count, INR=international normalized ratio, LDH=lactate dehydrogenase, CRP=C-reactive protein, SG0T=serum glutamic-oxaloacetic transaminase, SGPT= serum glutamic-pyruvic transaminase, CPK=creatine phosphokinase, CK-MB= creatine phosphokinase MB isoenzyme, TNI=troponine I

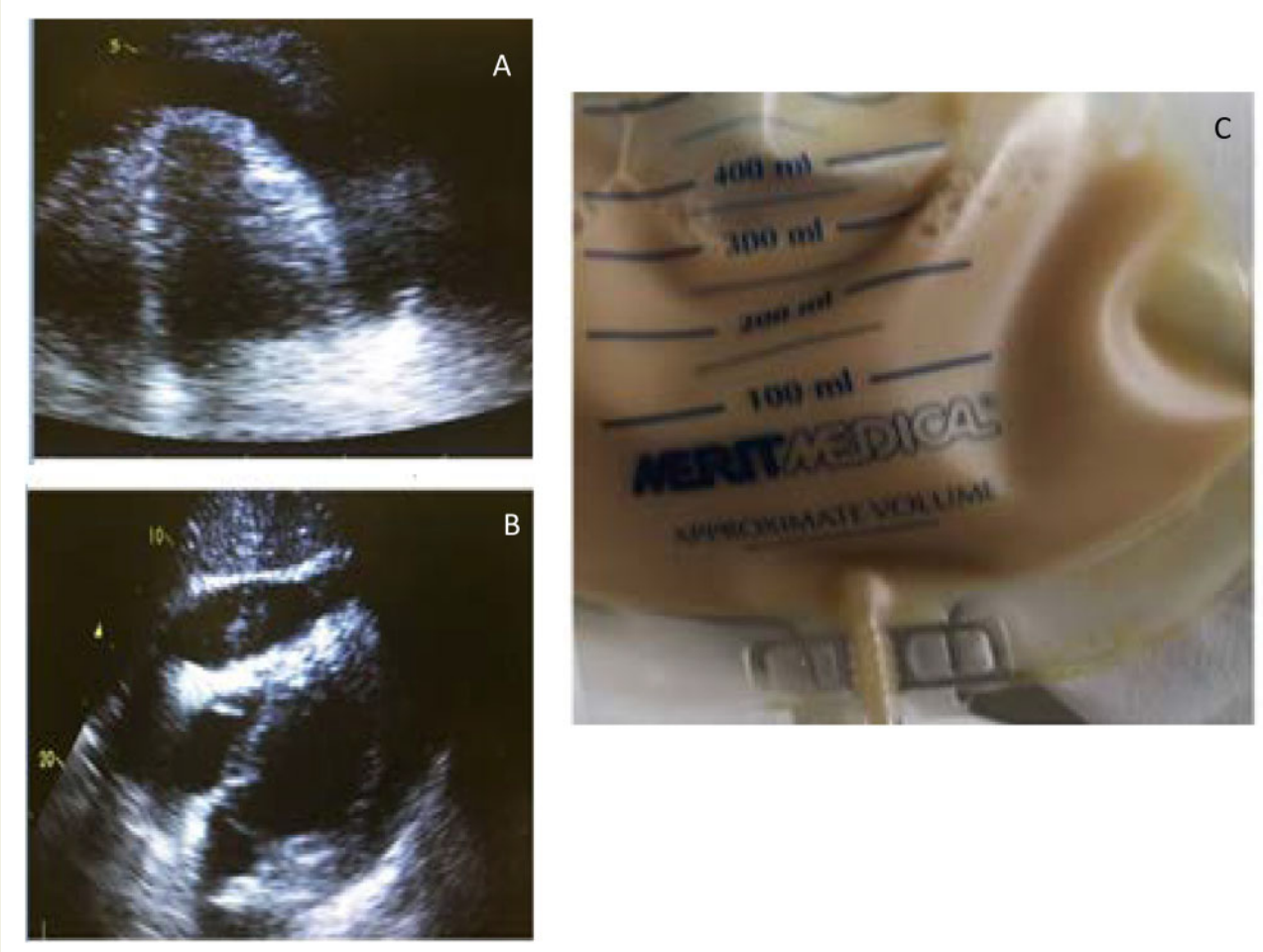

Figure 2 Transthoracic echocardiography and pericardial fluid. Motion frames in apical four-chamber view (A) and subxiphoid view (B) of a transthoracic echocardiogram revealing circiumferential pericardial. Effusion with right ventricular wall diastolic collapse suggestive of cardiac tamponade. (C) Purulent appearing pericardial fluid.

to evacuate fully the viscous and purulent effusion and a surgical approach is then indicated, including subxiphoid pericardiostomy with lavage and even total pericardiectomy.[6] Surgery may also prevent late complications such as persistent infection and constrictive pericarditis.[6] Although sepsis may have contributed to the fatal outcome, we believe that our patient succumbed from irreversible fulminant haemodynamic collapse, due to purulent tamponade, despite prompt pericardial drainage. Antibiotics were never administered, as the patient deteriorated very fast; we suspect that the clinical course would be no different if a single dose of the appropriate antibiotics was given.

Conclusion

Due to the high mortality rate of purulent pericarditis, a high index of suspicion is needed in order to promptly initiate appropriate therapy with broad-spectrum antibiotics and drainage.

Lead author biography

Eleni N. Bousoula, MD, MSc, PhD is a fellow interventional cardiologist at Onassis Cardiac Surgery Center. She studied medicine at Medical School of University of Athens. She earned her MD, MSc and PhD degrees from the same University. She worked for 3 years as a consultant cardiologist at the General Hospital of Tripolis and she completed 2 years fellowship in interventional cardiology. Her clinical and academic interests are interventional cardiology, acute cardiac care and heart failure.

Supplementary material

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

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

Consent: The patient was unable to provide consent as he died from his illness. The authors confirm that written consent for the submission and publication of this case, including images, has been obtained from the patient's next of kin in line with COPE guidance.

Conflict of interest: none declared.

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References

1. 2015 ESC Guidelines for the diagnosis and management of pericardial diseases: the Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC). Endorsed by: The European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J* 2015;**36**:2921–2964.
2. Sagrista-Sauleda J. Purulent pericarditis: review of a 20-year experience in a general hospital. *J Am CollCardiol* 1993;**22**:1661–1665.
3. Klacsmann PG, Bulkley BH, Hutchins GM. The changed spectrum of purulent pericarditis: an 86 year autopsy experience in 200 patients. *Am J Med* 1977;**63**:666–673.
4. Feinstein Y, Falup-Pecurariu O, Mitrică M, Berezin EN, Sini R, Krimko H et al. Acute pericarditis caused by *Streptococcus pneumoniae* in young infants and children. *Int J Infect Dis* 2010;**14**:e175–e178.
5. Patel H, Patel C, Soni M, Patel A, Banda V. Acute primary pneumonococcal purulent pericarditis with cardiac tamponade (a case report and literature review). *Medicine (Baltimore)* 2015;**94**:e1709.
6. Trpkov C, Nath E, Moon M, Windram J, Graham M. Fulminant pneumococcal pericarditis in a previous healthy patient. *Can J Cardiol* 2107;**33**:556e1–556e3.