

Polymicrobial purulent pericarditis and peritoneal effusion in an immunocompromised patient with *Staphylococcus aureus* bacteraemia: a case report

Nino Cmor (1) 1, Eva Dora 1, Mitja Lainscak (1) 1,2, and Miha Tibaut (1) 1*

¹Division of Cardiology, General Hospital Murska Sobota, Ulica dr. Vrbnjaka 6, 9000 Murska Sobota, Slovenia; and ²Faculty of Medicine, University of Ljubljana, Ljubljana, Slovenia Received 10 February 2024; revised 8 May 2024; accepted 7 August 2024; online publish-ahead-of-print 13 August 2024

Background

Polymicrobial pericarditis is an extremely rare and lethal form of pericarditis. Prompt initiation of appropriate antimicrobial treatment and pericardial drainage are crucial.

Case summary

A 57-year-old immunocompromised male patient presented to the emergency department due to dyspnoea, chest pain, and fever lasting for 7 days. Following clinical, laboratory, and imaging work-up, he was found to have pericardial effusion with signs of tamponade. After pericardiocentesis through subxiphoid and apical approaches, 800 mL of gross purulent fluid was obtained. Blood and pericardial fluid cultures confirmed the diagnosis of polymicrobial purulent pericarditis (*Staphylococcus aureus* and *Bacteroides vulgatus*). Further work-up revealed minor peritoneal effusion, and paracentesis fluid culture revealed the presence of *S. aureus* and, additionally, *Candida albicans*. After treatment initiation with intravenous antibiotics, pericardial, drainage and supportive measures, the patient's condition initially improved despite the development of constrictive pericarditis. However, he suddenly deteriorated after 37 days of hospitalization and passed away after 51 days of hospitalization.

Discussion

To the best of our knowledge, this is the first report of purulent pericarditis and purulent peritoneal effusion in the settings of S. aureus bacteraemia with an absent primary infection focus. Clinicians should be aware of treatment options for purulent pericarditis and consider intrapericardial fibrinolysis, especially in patients not suited for more invasive pericarditis treatment.

Keywords

Polymicrobial pericarditis • Case report • Pericardiocentesis • Staphylococcus aureus bacteraemia • Tamponade

ESC curriculum

2.2 Echocardiography • 6.6 Pericardial disease • 6.1 Symptoms and signs of heart failure

Learning points

- Purulent pericarditis is a rare infection with unspecific clinical presentation.
- Polymicrobial purulent pericarditis is a condition with high mortality despite timely and aggressive antimicrobial treatment and effusion drainage.

Handling Editor: Rita Pavasini

Peer-reviewers: Dzan Horozic; Federico Marchini

Compliance Editor: Elton Luo

© The Author(s) 2024. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (https://creativecommons.org/licenses/by-nc/4.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact reprints@oup.com for reprints and translation rights for reprints. All other permissions can be obtained through our RightsLink service via the Permissions link on the article page on our site—for further information please contact journals.permissions@oup.com.

^{*} Corresponding author. Tel: +38625123408, Email: tibaut.miha@gmail.com

N. Cmor et al.

Introduction

Bacterial pericarditis, once common in clinical practice, is nowadays a rare entity due to widespread antibiotic use. $^{1-3}$ Diagnosis is based on pericardiocentesis sampling with macroscopic fluid examination, biochemical,

and microbiological analysis. The infection mainly spreads haematogenously but can also spread contiguously from the retropharyngeal space, thoracic space, or from below the diaphragm. We describe a case of polymicrobial purulent pericarditis caused by *Staphylococcus aureus* and *Bacteroides vulgatus* with a concomitant purulent peritoneal effusion.

Summary figure

Date	Event	Laboratory	Treatment
2 July 2022	Patient was admitted to the hospital due to acute respiratory failure, low blood pressure, fever and new onset atrial fibrillation.	Leukocytosis of 13,1x10°/L, C-reactive protein 200 mg/L and procalcitonin 5,1 mcg/L	The patient was started on empiric antibiotic therapy with parenteral ceftriaxone.
3 July - 5 July 2022	Echocardiography revealed a large, circumferential pericardial effusion with echocardiographic signs of tamponade physiology. Two pericardiocenteses with a subxiphoid and apical approach were performed.	The peripheral blood cultures revealed an infection with Staphylococcus aureus, while pericardial fluid cultures grew Staphylococcus aureus and Bacteroides vulgatus.	Intravenous flucloxacillin was added to the antibiotic treatment regimen.
8 July 2022	Acute abdominal pain occurred. CT of the abdomen revealed signs of paralytic ileus and a peritoneal effusion.		Ileus was successfully managed conservatively.
11 July 2022	A few millilitres of gross purulent peritoneal fluid were acquired with diagnostic paracentesis.	Cultures obtained from peritoneal fluid revealed an infection with Staphylococcus aureus and Candida albicans.	Intravenous micafungin was added to the antimicrobial treatment regimen.
22 July 2022	On echocardiography, signs of constrictive pericarditis with septal bounce were present without pericardial effusion or signs of valvular vegetation.		
11 July - 7 August 2022	Patient's condition gradually improved, and supplemental oxygen could be discontinued.		
8 August 2022	His status suddenly deteriorated. He became hypotensive and febrile and was in acute respiratory failure.	Leucocytosis of 25,7 x10°/L, C-reactive protein 72 mg/L and procalcitonin 1,2 mcg/L.	Non-invasive ventilation was started and intravenous meropenem was started.
22 August 2022	His clinical status further deteriorated, and after a total hospital stay of 51 days, he passed away.		

Case report

A 57-year-old immunocompromised male patient presented to the emergency department due to dyspnoea, chest pain, and fever lasting for 7 days. The patient's medical background encompassed a history of chronic obstructive lung disease, non-Hodgkin lymphoma in remission, and metastatic squamous cell lung carcinoma, for which oncological treatment has been planned but has not yet commenced. He had a long-term tracheostomy, which was constructed because of metastases in the hypopharynx. He lived alone, was ambulatory, and able to carry out work of a light or sedentary nature but still needed daily help with household chores. He had no relevant drug history.

On physical examination, the patient was hypotensive (90/60 mmHg), tachycardic, and febrile. His heart rhythm was irregular, and heart sounds were muffled. Breath sounds were decreased over the right basal lung field, and lung crackles were present bilaterally. There were no apparent signs of infection, and the surroundings of the tracheostomy were not visibly inflamed.

A 12-lead electrocardiogram showed new-onset atrial fibrillation with a rapid ventricular response of 170 beats per minute. Chest radiography revealed right-sided pleural effusion and an enlarged cardiac silhouette. Emergency echocardiography demonstrated a large, circumferential pericardial effusion of 3–4 cm in diastole with echocardiographic signs of tamponade, including diastolic right ventricular collapse (*Figure 1/* Supplementary material online, *Video S1*). Laboratory findings were remarkable for leucocytosis of $13.1 \times 10^9/L$, C-reactive protein of 200 mg/L, and procalcitonin of 5.1 µg/L. Urinalysis was unremarkable.

He was treated with supplemental oxygen and was given crystalloid solutions parenterally. After obtaining blood cultures and urine samples for microbiological analysis, empirical intravenous antibiotic therapy with ceftriaxone (2 g once daily) was initiated. Amiodarone-induced pharmacological conversion effectively reinstated sinus rhythm, accompanied by a subsequent rise in blood pressure. The decision for pharmacological cardioversion was based on the patient's immunocompromised status and developing sepsis, which necessitated a cautious approach. After initial stabilization, he was admitted to the high-dependency unit.



Figure 1 Emergency echocardiography with large pericardial effusion with echocardiographic signs of tamponade. RV, right ventricle; RA, right atrium; LV, left ventricle; LA, left atrium. *Pericardial effusion.

An urgent pericardiocentesis through a subxiphoid approach was performed with drainage of 500 mL of purulent fluid (Figure 2A). A subsequent echocardiographic examination revealed a persistent pericardial effusion of 3 cm in the diastole localized around the lateral wall of the left ventricle (see Supplementary material online, Video S2). A second pericardiocentesis was conducted through an apical approach to facilitate drainage of the residual effusion (Figure 2B). Both drains were left in situ (Figure 3). The pericardial catheter drainage exhibited a gradual decrease, leading to the removal of the catheter after 5 days, during which a total volume of 800 mL was drained. Staphylococcus aureus and B. vulgatus were identified from peripheral blood cultures and pericardial fluid. Following a microbiological examination, intravenous flucloxacillin at a dosage of 2 g four times daily was incorporated into the antibiotic regimen for targeted therapy. Thoracocentesis was performed, draining 1500 mL of transudative and sterile fluid from the right pleural space. His clinical status initially improved, and his demand for oxygen therapy decreased.

On the sixth day of hospitalization, acute abdominal pain occurred. A computed tomography scan showed minor peritoneal effusion but no signs of acute bowel obstruction or perforation. It was concluded that abdominal pain was due to paralytic ileus, which was successfully managed conservatively. Paracentesis was performed for diagnostic purposes, and microbiological cultures revealed the presence of *S. aureus* and *Candida albicans* in peritoneal fluid. Intravenous micafungin (100 mg once daily) was added to the antimicrobial therapy. The effectiveness of antimicrobial treatment was confirmed with additional sets of blood cultures, which remained sterile. After 32 days, the antibiotic treatment was discontinued. Control echocardiography showed signs of constrictive pericarditis with septal bounce and no pericardial effusion or valvular vegetation (see Supplementary material online, *Video S3*).

Various therapeutic options were discussed. Due to comorbidities, he was not an ideal candidate for an operative procedure in general anaesthesia. Intrapericardial fibrinolysis was considered a potential alternative, but we ultimately decided against this therapeutic option because of the relatively high risk of possible haemorrhagic complications. The patient remained haemodynamically stable, and his condition gradually improved. Supplemental oxygen was no longer necessary, marking a positive initial recovery. Furthermore, he demonstrated promising progress by being able to walk ~100 m.

However, after 37 days of hospitalization, his status suddenly deteriorated. Acute respiratory failure, hypotension, and pyrexia developed, and he needed non-invasive ventilation, fluid resuscitation, and vasopressors. Chest X-ray showed bilateral pneumonia and a right-sided pleural effusion. On bedside echocardiography, no pericardial effusion was present. Laboratory findings were suggestive of sepsis with leucocytosis of $25.7 \times 109/L$, C-reactive protein of 72 mg/L, and procalcitonin of $1.2 \, \mu g/L$. However, blood cultures obtained at that time remained sterile, and empirical intravenous antibiotic therapy with meropenem (1 g/8 h) was initiated. Sputum cultures showed *Pseudomonas aeruginosa* pneumonia. He further deteriorated, and after a total hospital stay of 51 days, he passed away. The cause of death was evident, and a post-mortem was not needed.

Discussion

Purulent pericarditis is a rare condition, with an estimated incidence of 1/18 000. Due to its atypical presentation and late recognition, diagnosis is still confirmed post-mortem in more than half of cases. Clinical symptoms are often unspecific; patients present with fever, chills, and tachycardia. Typical symptoms and signs, such as chest pain and pericardial friction rub, are frequently absent. It is more common in immunocompromised patients; identified risk factors are thoracic surgery, chronic kidney disease, immunosuppression, alcoholism, and neoplasms. 1,2,9

In our case, we confirmed the presence of *S. aureus* in blood cultures and cultures from peritoneal effusion. To our knowledge, this is the first

N. Cmor et al.



Figure 2 Pericardiocenteses through subxiphoid (A) and apical (B) approach with drainage of purulent fluid.



Figure 3 Pericardial drains *in situ*. The drainage from the pericardial catheter gradually decreased, and the catheter was removed after 5 days. A total of 800 mL was drained.

report of purulent pericarditis and purulent peritoneal effusion in the settings of *S. aureus* bacteraemia. Signs and symptoms of urinary or respiratory tract infection were absent. The clinical exam did not reveal any significant skin ulceration. Additionally, computed tomography revealed no clear focus of infection in the abdominal cavity. Imaging modalities, such as labelled leucocyte scintigraphy, were not available at our institution at the time of the patient's hospitalization, and we deemed

him unsuitable for transportation. The primary focus of infection was, therefore, not found. Similarly, a primary clinical focus is not found in 25% of *S. aureus* bacteraemia cases. ¹⁰

Staphylococcus, Streptococcus, Haemophilus species, and Mycobacterium tuberculosis are the most common causative agents of purulent pericarditis nowadays, while pneumococcus was responsible for the majority of cases in the era before the introduction of antibiotics. Although S. aureus is now the most common causative agent, certain gram-negative anaerobic microorganisms such as Bacteroides fragilis, Prevotella, Porphyromonas, and Fusobacterium are also reported as causes of purulent pericarditis. 11

Staphylococcus aureus and B. vulgatus were isolated from pericardial effusion, and to our knowledge, this is the first reported case of polymicrobial pericarditis with this combination of causative pathogens. Polymicrobial pericarditis is an extremely rare presentation of pericarditis.^{4–7,12} de Jong et al.⁷ reviewed 49 cases, emphasizing the shift towards gram-negative bacteria, fungi, and mycobacteria as pathogens for bacterial pericarditis since the worldwide use of antibiotics. An example is B. vulgatus, a gram-negative anaerobe, which, together with other Bacteroides spp., accounts for a significant proportion of the gut bacteriome.¹³

Since polymicrobial pericarditis has a reported mortality rate of up to 75%, ⁷ a very extensive diagnostic work-up for the primary infection focus is warranted. ^{4–7,12} Initial treatment should include empiric intravenous antimicrobial therapy and sufficient effusion drainage. ¹ Incomplete effusion drainage can lead to loculations with fibrin accumulation, predisposing the patient to constrictive pericarditis. ⁸ Current European guidelines emphasize the importance of complete drainage, suggesting surgical techniques to be employed only after adequate drainage cannot be achieved using intrapericardial fibrinolysis. ^{1,14}

Following pericardial and pleural drainages with appropriate antimicrobial treatment initiation, our patient clinically improved and was able to walk. At the same time, echocardiography revealed signs of constrictive pericarditis. Considering the patient's general condition and his comorbidities, he was regarded to be a poor surgical candidate, with radical pericardiectomy being an unsuitable treatment option. ^{1,14} Intrapericardial fibrinolysis was considered as a possible treatment option. Due to the possibility of complications in the form of major haemorrhage (e.g. haemorrhagic tamponade), ^{14,15} we decided that its risks outweighed the expected benefits and decided against it.

With this case, we highlight the importance of meticulous aetiological diagnostic work-up in polymicrobial purulent pericarditis and prompt initiation of appropriate antimicrobial treatment. Clinicians should be aware of adjunctive treatment options for purulent pericarditis and consider intrapericardial fibrinolysis, especially in patients not suited for surgical treatment options.

Lead author biography



Nino Cmor was born in Murska Sobota (Slovenia). After finishing Medical Faculty in Maribor (Slovenia), he started working as a junior doctor at General Hospital Murska Sobota (Slovenia). In 2020, he started specializing in internal medicine at General Hospital Murska Sobota. In 2023, he started residency in Cardiology at General Hospital Murska Sobota. His main areas of interest are invasive cardiology and heart failure.

Supplementary material

Supplementary material is available at $\it European Heart Journal-Case Reports online.$

Consent: The patient reported in this case is deceased. Despite the best efforts of the authors, they have been unable to contact the patient's next-of-kin to obtain consent for publication. Every effort has been made to anonymize the case. This situation has been discussed with the editors.

Conflict of interest: None declared.

Funding: This research received no external funding.

Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

References

- Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, et al. 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.
- Kondapi D, Markabawi D, Chu A, Gambhir HS. Staphylococcal pericarditis causing pericardial tamponade and concurrent empyema. Case Rep Infect Dis 2019;2019: 3701576.
- Kapačinskaitė M, Gabartaitė D, Šatrauskienė A, Sakaitė I, Maneikienė VV, Zorinas A, et al. A rare case of primary purulent pericarditis caused by Streptococcus constellatus. Medicina (Kaunas) 2023;59:159.
- Bhattarai M, Yost G, Good CW, White CF, Nepal H. Primary purulent pericarditis with cardiac tamponade due to oropharyngeal polymicrobial infection: a case report and literature review. Korean | Thorac Cardiovasc Surg 2014;47:155–159.
- Kanwal A, Avgeropoulos D, Kaplan JG, Saini A. Idiopathic purulent pericarditis: a rare diagnosis. Am J Case Rep 2020;21:e921633.
- Cvetko TT, Ljubic J, Ostricki B. Purulent pericarditis caused by methicillin-sensitive Staphylococcus aureus in an immunocompetent adult after COVID-19 pneumonia: a case report. Eur Heart | Case Rep 2023;7:ytad141.
- de Jong Y, van Loenhout RB, Swank DJ, Jansen CL, Sorgdrager BJ. Polymicrobial bacterial pericarditis and cardiac tamponade caused by pericardial penetration of an adjustable gastric band. BMJ Case Rep 2018;2018:bcr2017221589.
- Sagristà-Sauleda J, Barrabés JA, Permanyer-Miralda G, Soler-Soler J. Purulent pericarditis: review of a 20-year experience in a general hospital. J Am Coll Cardiol 1993;22: 1661–1665.
- Pankuweit S, Ristić AD, Seferović PM, Maisch B. Bacterial pericarditis: diagnosis and management. Am J Cardiovasc Drugs 2005;5:103–112.
- Tong SY, Davis JS, Eichenberger E, Holland TL, Fowler VG Jr. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin Microbiol Rev 2015;28:603–661.
- 11. Brook I. Pericarditis due to anaerobic bacteria. Cardiology 2002;97:55–58.
- Ased S, Payne AN, Yang C. Idiopathic hemodynamically unstable polymicrobial purulent pericarditis: a rare case presentation. J Am Coll Emerg Physicians open 2020;1:744–746.
- Zafar H, Saier MH Jr. Gut Bacteroides species in health and disease. Gut Microbes 2021;
 13:1–20.
- Augustin P, Desmard M, Mordant P, Lasocki S, Maury JM, Heming N, et al. Clinical review: intrapericardial fibrinolysis in management of purulent pericarditis. Crit Care 2011;15:220.
- Juneja R, Kothari SS, Saxena A, Sharma R, Joshi A. Intrapericardial streptokinase in purulent pericarditis. Arch Dis Child 1999;80:275–277.