GSC Biological and Pharmaceutical Sciences
eISSN: 2581-3250 CODEN (USA): GBPSC2
Cross Ref DOI: 10.30574/gscbps
Journal homepage: https://gsconlinepress.com/journals/gscbps/

(CASE REPORT)

Check for updates

Methicillin-Sensible *Staphylococcus aureus* (MSSA) Pericardial Effusion Causing Cardiac Tamponade: A case report

Hendianto [1,*], Felicia Aviana [1], Ida Sri Iswari [2], Ni Luh Indrayani [2] and I Nyoman Wiryawan [3]

[1] *Clinical Microbiology Specialist Program, Faculty of Medicine, Udayana University, Denpasar, Indonesia.*
[2] *Department of Clinical Microbiology, Prof DR I.G.N.G Ngoerah General Hospital, Denpasar, Indonesia.*
[3] *Department of Cardiology and Vascular Medicine, Prof DR I.G.N.G Ngoerah General Hospital, Denpasar, Indonesia.*

GSC Biological and Pharmaceutical Sciences, 2024, 28(01), 030–035

Publication history: Received on 27 May 2024; revised on 02 July 2024; accepted on 04 July 2024

Article DOI: https://doi.org/10.30574/gscbps.2024.28.1.0249

Abstract

Background: Methicillin-Sensible *Staphylococcus aureus* (MSSA) as the pathogen of the pericardial space is an uncommon case that can be fatal if untreated. The underlying disease accompany with infection that lead to cardiac tamponade can increase mortality rate of the patient.

Case description: We present a 29 years old male patient with severe dispnea who was found to have cardiac tamponade secondary to a purulent pericardial effusion. He was also had end stage renal disease with regular hemodialysis. The diagnosis was suggested by clinical context, imaging, pericardial fluid analysis and was confirmed by culture. MSSA were isolated from 2 times pericardial fluid, peripheral blood and double lumen catheter exit site swab that were growth in blood agar only. Identification and susceptibility to antibiotics was assessed by *Vitek2 Compact* automated system (*BioMerieux*), represent sensitive to penicillin, cephalosporin, quinolone, aminoglycoside except macrolides. Administration of cefazolin intravenous for 5 days and also pericardial drainage result in a full recovery for the patient.

Discussion: Purulent pericardial effusion is a rare condition that carries a high mortality rate as it can rapidly progress into cardiac tamponade. In developing countries, *Mycobacterium tuberculosis* is the most frequent cause of acute pericarditis followed by *Haemophilus, Staphylococcus and Streptococcus*. Prior to the advent of antibiotics, *Staphylococcus aureus* takes role as the predominant pathogen, this event was common as the result of hematogenous seeding such as catheter related hemodialysis in this patient.

Conclusion: Prompt diagnosis of purulent pericardial effusion also initiation of appropriate antibiotic and pericardial drainage treatment are the mainstays of successful management of this rare but potentially lethal case.

Keywords: *Staphylococcus aureus;* MSSA; Pericardial effusion; Cardiac tamponade

1. Introduction

Staphylococcus aureus is responsibe for numerous infection cases, it could be relatively mild to life-threatening. The primary reservoir for staphylococci is the human nares, with colonization also occuring in skin surfaces. As with most infections, the development of staphylococci infection is initiated when a breach of the skin or mucosal barrier allows staphylococci access to adjoining tissues or the blood stream. Less common bacteria success to invade the pericaridum when the bacterial flora has been exposed by antibacterial use and when the immune system is compromized.

* Corresponding author: Hendianto

Purulent pericardial effusion is the most serious manifestation of bacterial pericarditis, the appearance is gross pus in the pericardium or microscopically as purulent effusion. The condition is acute with fever, shortness of breath that suddenly can be severe and chest pain with the less common. Purulent pericarditis is always fatal if untreated. The mortality rate in treated patients is 40%, and death is mostly due to cardiac tamponade, sepsis, systemic toxicity, cardiac decompensation, and constriction, meanwhile the mortality in untreated patients approach 85%[1].

Acute bacterial pericarditis is rarely encountered in the modern antibiotic era. Before that, in developing countries and HIV patients, *Mycobacterium tuberculosis* is the most frequent cause of acute pericarditis followed by *Haemophilus, Staphylococcus and Streptococcus* (rheumatic pancarditis). Two large reviews of the bacteriology of purulent pericarditis in the antibiotic era found that *Staphylococcus aureus* was the most common pathogen, acccounting for 31% of cases in one series and 22% in the other[2][3].

Bacterial infections of the pericardial space are an uncommon cause of pericardial effusion. In the advent era of antibiotics, purulent pericarditis was more common and usually was the result of hematogenous or direct spread from an adjacent infection from another organs[4]. Here, we present a severe case of cardiac tamponade secondary to a purulent pericardial effusion in end stage renal disease (ESRD) patient with regular hemodialysis which was managed successfully with administration of intravenous antibiotics and pericardial drainage.

2. Case Description

A 29 years old male patient with history of hypertension, ESRD and had regular hemodyalisis since three months ago, came to emergency room with chief complaint dispnea. The symptoms began within 2 days before and getting worse until the present day. There was also chest pain and palpitation that he feel, every moment the episodes arised, it had no pattern of time. He was admitted in the day when he came and also had malaise, sweating, and extended purpuric exanthema. The patient also reported nausea at the onset, which was solved by spasmolytic treatment.

On the physical exam blood pressure was 110/70 mm Hg, the frequency rate was 154 bpm, respiratory rate was 24 bpm, axillary temperature was 36.8°C, peripheral capillary oxygen saturation was 95%, jugulary vein pressure +5 cm H2O, cardiac auscultation: S1 S2 normal, irregular, murmur (+), muffle heart sound (+). There is also purpura with partially confluent lesions on the neck, chest and trunk, with double lumen catheter (DLC) access on his right jugular vein. Laboratory results showed an inflammatory syndrome, slight leukocytosis, and impaired renal function, chest x-ray showed cardiomegaly with all chamber dilatation suspected pericardial effusion, then with echocardiographic examination showed severe circumfrential pericardial effusion with sign of tamponade and uncoordinated septal wall motion (Figure 1a and 1b).

Based on the facts before, the cardiologist performed pericardiocentesis to evacuated the pericardial fluid to prevent the condition become more massive and threatened the life of the patient, then pericardial fluid analysis and cultures were performed, meanwhile antibiotic treatment was hold due to there is no sign of sepsis also point of entry as evidence. On the night following treatment, fever and tachypnea were observed, his respiratory was stable also there was no vomiting. On the morning of the second day of treatment, the patient's pericardial fluid analysis was released out with the macroscopic was purulent fluid (Figure 2a) then microscopic cell was dominated by polymorphonucelar neutrophils, pericardial fluid culture was sent for the second time too. On the third day, fever was found in the patient, the first pericardial fluid culture identification and susceptibility to antibiotics was assessed by *Vitek2 compact* automated system (*BioMerieux*) informed the presence of MSSA which sensitive to cefazolin, then the antibiotic was administered.

Fever and other sign of bacterial infection make the cardiologist ordered a set of peripheral blood + DLC exit site swab culture to identify the risk factor of infection entry site and the spread. In following days, patient's condition going better and stable, surprisingly all the culture informed the same presence of MSSA (Figure 2b and 3), cefazolin was given until 7th day, also subxiphoid pericardiostomy was performed. On 6th-9th day, the patient made a full recovery also laboratory results were fine. His pericardial drain and chest tube were eventually removed, he was discharged on 10th day with a follow-up at the infectious disease clinic.

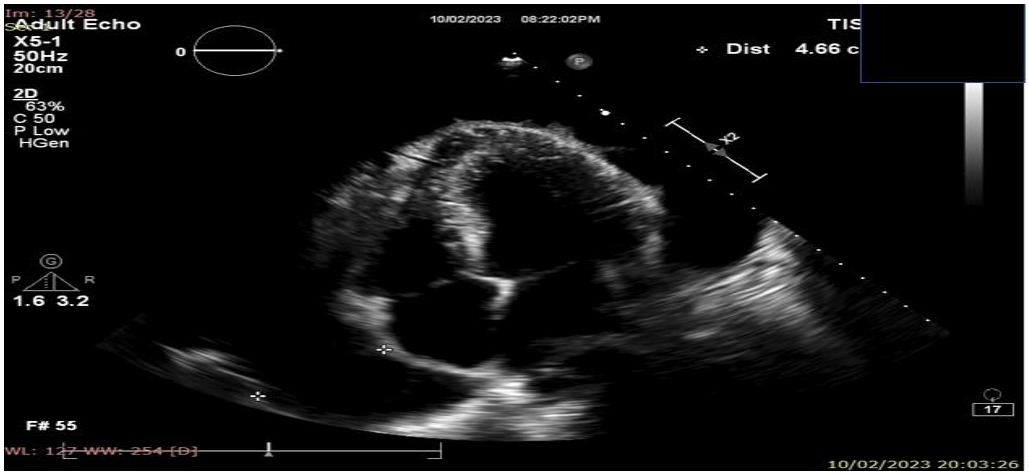

Figure 1a Severe circumfrential pericardial effusion with echo sign of tamponade, largest diameter 4.6 cm at posterior Left Atrium

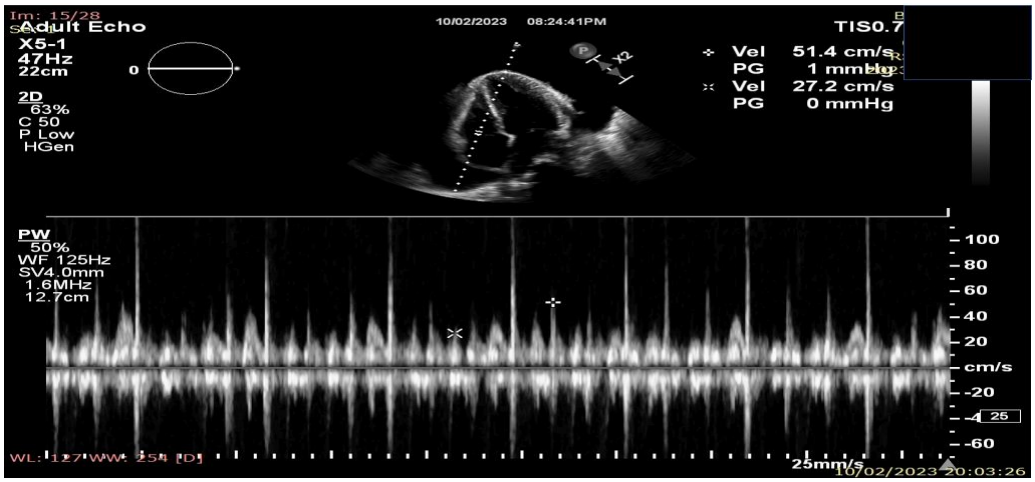

Figure 1b. Variability with inspirational at right ventricle, expect a 40% increase in TV in-flow velocity

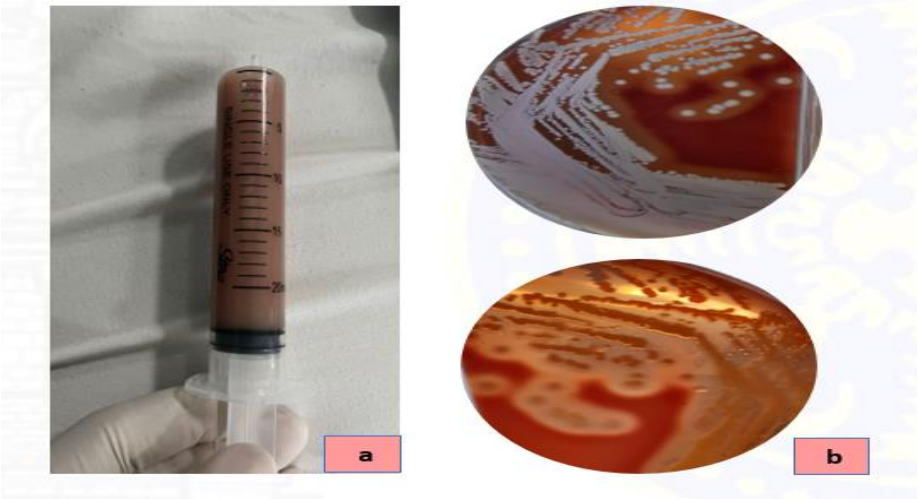

Figure 2 a. Purulent pericardial fluid b. medium to large (0.5–1.5 μm); smooth, entire, slightly raised, white colonies with beta-hemolytic on blood agar

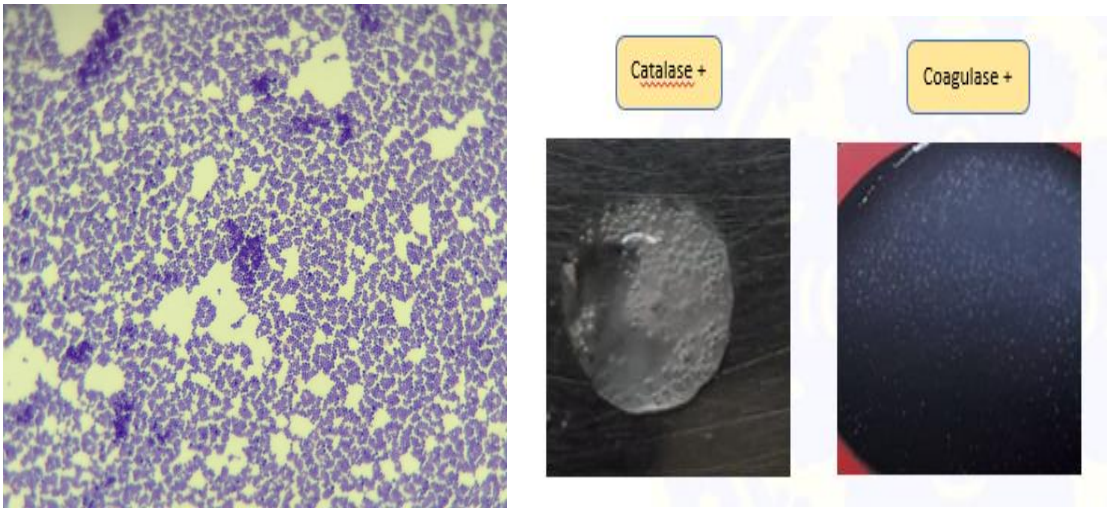

Figure 3 Gram-positive coccus with catalase positive (+) and coagulase positive (+)

3. Discussion

Purulent pericarditis is a rare life-threatening infection of the pericardial space, the cases were more likely happened in the pre-antibiotic era[5]. The pericardium that lacks compliance, and moreover expanding bacterial infection can rapidly increase pericardial pressures causing cardiac tamponade. The mode of infection spreading can be classified into: direct extension of chest wall infection, local extension from an intrathoracic process, open chest wall wounds (e.g.,injury or surgery), and hematogenous spread. The infection frequently found in patients who are immunocompromised, diabetes mellitus, alcohol abuse, and wide spread systemic infection[6].

Pericardial effusion may develop as a result of pericarditis. The etiological distribution of pericardial efussion of acute presentation with cardiorespiratory symptoms are: neoplasm (33%), idiopathic (14%), acute pericarditis (12%), trauma (12%), uremia (6%), post-pericardiotomy (5%) and infection (5%, 4% bacterial)[7]. The microbiology of purulent pericarditis mostly found with *Staphylococcus, Streptococcus, Haemophilus,* and *Mycobacterium tuberculosis* predominating, though atypical organisms such as *Candida* and *Salmonella* have been described, polymicrobial infections are uncommon[3][8]. Treatment should be initiated promptly with broad-spectrum antibiotics and source control.

Staphylococcus aureus is Gram-positive bacteria that are cocci-shaped and tend to be arranged in clusters that are described as "grape-like." On media, these organisms can grow in up to 10% salt, and the color appearance are often golden or yellow. These organisms can grow aerobically or anaerobically (facultative) and at temperatures between 18ºC-40ºC. Typical biochemical identification tests include catalase and coagulase positive. *S. aureus* are one the most common bacterial infections in humans and are the causative agents of multiple human infections, including bacteremia, infective endocarditis, skin and soft tissue infections, osteomyelitis, septic arthritis, prosthetic device infections, pulmonary infections, gastroenteritis, meningitis, toxic shock syndrome, and urinary tract infections[9].

The diagnosis of purulent staphylococcal pericarditis was based on the isolation of MSSA in purulent pericardial fluid. Theoretically, it could be caused by local transmission from the endocardial source or even caused by blood spread. Cardiac tamponade is extremely rarely diagnosed in the context of bacterial infections; in a series of 136 consecutive cases, only 5 patients had positive cultures out of which 2 were with *S. aureus* and other 3 were diagnosed with infective endocarditis[10].

In our patient, the purulent pericarditis likely caused by hematogenous spread which evidenced by MSSA cultured in blood, pericardial fluid drainage, and also DLC exit site swab. The detection of MSSA suggested the possibility of bacteremia from the skin puncture related to the catheterization and the spread of infection into bloodstream ended at the pericardial sac. Hemodialysis, thoracic surgery, chemotherapy, immunocompromise, and AIDS are all risk factors for purulent pericarditis[11]. The history of ESRD and regular hemodialysis with poor hygiene made the higher chance that mechanism happened in this patient. The presence of extended purpura exanthema raised the possibility of a toxic pathology process. Previously, there was reported that *Staphylococcus aureus* enterotoxin C (SEC) was the cause for a series of 3 fatal cases of purpura fulminans associated with sepsis[12]. Echocardiography remains the gold standard

tool for diagnosing pericardial disease and cardiac tamponade. Pericardiocentesis with echocardiography-guided is become the first line treatment in cardiac tamponade[13].

Pericardial fluid drainage is always performed when the patient presents with cardiac tamponade and when there is indication for purulent or potential massive pericardial effusion. Aggressive management is necessary as purulent pericarditis carries a high mortality risk, simple percutaneous drainage alone is usually insufficient and could cause the disease process to become a constrictive or persistent form of purulent pericarditis because of loculations and adhesions[14][15][16]. Classical subxiphoid pericardiotomy has the advantage of achieving more permanent and complete drainage than pericardiocentesis while avoiding the risk of sternal or pleural contamination and is useful in the critically ill patients where a thoracotomy may be too aggressive. Furthermore, it allows for mechanical breakdown of loculations and septations by the surgeon[17][18].

Evidence of MSSA systemic infections were isolated from 2 bottles of blood culture, pericardial fluid, and DLC exit site swab culture. The susceptibility to antibiotics was also assessed by *Vitek2 Compact* automated system (*BioMerieux*). *S.aureus* was the most frequently isolated pathogen in blood cultures and pericardial fluid in a group of 21 patients in which pericarditis was the revealing symptom of endocarditis; the aortic valve was mainly affected and 47% of the cases exhibited cardiac tamponade[19], similar to this presented case. Although drainage of the pericardial fluid is required for adequate source control and, in some cases, achieving hemodynamic stability also should not delay initiation of antibiotic therapy are important.

Empiric treatment should be involved an anti-staphylococcal agent with a cephalosporin and fluoroquinolone[8][19]. In selecting antimicrobial therapy, the ability of potential agents to kill the causative organism as well as the minimum inhibitory concentration (MIC; the lowest concentration that inhibits growth) need to be considered. In this case, antibiotic sensitivity test show that MSSA was sensitive to penicillin, cephalosporin, quinolone, aminoglycoside except macrolides. Empiric combination of an antistaphylococcal antibacterial and aminoglycoside, followed by tailored antibacterial therapy according to the results of pericardial fluid and blood cultures should be applied promptly[20].

4. Conclusion

Purulent pericarditis lead to effusion and cardiac tamponade is a rare condition that most physicians probably have not encountered before. Pericardium sac is inelastic and rapid accumulations of fluid process could rapidly increase pericardial pressures too, it's impacts can cause hemodynamic collapse even death. The microbiology diagnostic of purulent pericarditis cause of bacterial infection found that MSSA as the pathogen, through rapid diagnosis and initiation of appropriate antibiotic also pericardial drainage treatment are the mainstays of successful management of this rare but potentially lethal case.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that there is no competing interest regarding the manuscript.

Statement of informed consent

The patient himself gives consent for information about the history and condition (photograph and article) to appear in a journal article.

Funding

The authors are responsible for the study's funding without the involvement of a grant, scholarship, or any other funding resource.

Author Contribution

H and FA were involved in writing the manuscript. ISI, NLI and INW supervised and revised the manuscript. All authors prepare the manuscript and agree for this final version of the manuscript to be submitted to this journal.

References

[1] J. Sagrista-sauleda, J. A. BARRABIftS, G. Permanyer-miralda, J. Soler-soler, and F. Barcelona, "Purulent Perl'=~_l ~ IN: Review of a General Flosph''."

[2] R. H. Rubin and R. C. MOELLERING Jr, "Clinical, Microbiologic and Therapeutic Aspects of Purulent Pericarditis," 1975.

[3] P. G. Klacsmann, B. H. Bulkley, and G. M. Hutchins, "The Changed Spectrum of Purulent Pericarditis An 86 Year Autopsy Experience in 200 Patients."

[4] T. Keersmaekers, S. R. E. Elshot, and P. T. Sergeant, "Primary bacterial pericarditis," Acta Cardiol, vol. 57, no. 5, pp. 387–389, 2002, doi: 10.2143/AC.57.5.2005459.

[5] D. Meier, M. Kirsch, S. D. Qanadli, O. Muller, D. Fishman, and C. Trana, "Case report of an unusual combination of purulent pericarditis and false aneurysm of the ascending aorta," J Cardiothorac Surg, vol. 13, no. 1, Jan. 2018, doi: 10.1186/s13019-018-0699-4.

[6] D. Kondapi, D. Markabawi, A. Chu, and H. S. Gambhir, "Staphylococcal Pericarditis Causing Pericardial Tamponade and Concurrent Empyema," Case Rep Infect Dis, vol. 2019, pp. 1–5, Jul. 2019, doi: 10.1155/2019/3701576.

[7] S. Ben-Horin, I. Bank, V. Guetta, and A. Livneh, "Large symptomatic pericardial effusion as the presentation of unrecognized cancer: A study in 173 consecutive patients undergoing pericardiocentesis," Medicine, vol. 85, no. 1, pp. 49–53, 2006, doi: 10.1097/01.md.0000199556.69588.8e.

[8] S. Pankuweit, A. D. Risti´c, P. M. S. Seferovi´c, and B. Maisch, "Bacterial Pericarditis Diagnosis and Management," 2005.

[9] S. Y. C. Tong, J. S. Davis, E. Eichenberger, T. L. Holland, and V. G. Fowler, "Staphylococcus aureus infections: Epidemiology, pathophysiology, clinical manifestations, and management," Clin Microbiol Rev, vol. 28, no. 3, pp. 603–661, 2015, doi: 10.1128/CMR.00134-14.

[10] C. Sánchez-Enrique et al., "Cause and Long-Term Outcome of Cardiac Tamponade," American Journal of Cardiology, vol. 117, no. 4, pp. 664–669, Feb. 2016, doi: 10.1016/j.amjcard.2015.11.023.

[11] A. Kaye, G. A. Peters, J. W. Joseph, and M. L. Wong, "Purulent bacterial pericarditis from Staphylococcus aureus," Clin Case Rep, vol. 7, no. 7, pp. 1331–1334, Jul. 2019, doi: 10.1002/ccr3.2224.

[12] G. R. Kravitz, D. J. Dries, M. L. Peterson, and P. M. Schlievert, "Staphylococcal Purpura Fulminans • CID 2005:40 (1 April) • 941 Purpura Fulminans Due to Staphylococcus aureus." [Online]. Available: https://academic.oup.com/cid/article/40/7/941/372566

[13] Y. Adler et al., "2015 ESC Guidelines for the diagnosis and management of pericardial diseases," European Heart Journal, vol. 36, no. 42. Oxford University Press, pp. 2921–2964, Nov. 07, 2015. doi: 10.1093/eurheartj/ehv318.

[14] G. K. Sethi, R. M. Nelson, and C. B. Jenson, "Surgical management of acute septic pericarditis," Chest, vol. 63, no. 5, pp. 732–735, 1973, doi: 10.1378/chest.63.5.732.

[15] H. Has¸im, U. ¨ Stünsoy, A. Celkan, C. Sı ˙vrı ˙koz, H. Kazaz, and M. Kılınç, "Intrapericardial fibrynolytic therapy in purulent pericarditis." [Online]. Available: www.elsevier.com/locate/ejcts

[16] M. Corachan, P. Poore, G. P. Hadley, and A. Tanner, "Purulent pericarditis in Papua New Guinea: report of 12 cases and review of the literature in a tropical environment," 1983.

[17] N. Becit, Y. Ünlü, M. Ceviz, C. U. Koçoğullari, H. Koçak, and Y. Gürlertop, "Subxiphoid pericardiostomy in the management of pericardial effusions: Case series analysis of 368 patients," Heart, vol. 91, no. 6, pp. 785–790, Jun. 2005, doi: 10.1136/hrt.2004.037499.

[18] C. P. Petcu, R. Dilof, C. Bătăiosu, and P. D. Petcu, "Case Report Purulent Pericardial Effusions with Pericardial Tamponade-Diagnosis and Treatment Issues," 2013.

[19] L. H. Katz, S. Pitlik, E. Porat, P. Biderman, and J. Bishara, "Pericarditis as a presenting sign of infective endocarditis: Two case reports and review of the literature," Scand J Infect Dis, vol. 40, no. 10, pp. 785–791, 2008, doi: 10.1080/00365540802169106.

[20] L. J. Goodman, "Purulent Pericarditis," Curr Treat Options Cardiovasc Med, vol. 2, pp. 343–350, 2000.