

Purulent Pericarditis Progressing to Constriction in Childhood: A Case Report

Briki. J^{1*}, Amelal. M¹, Idrissa. M¹, Eledrissi. A¹, Benlefqih. C¹, Rhissassi. J¹, Sayah. R¹, Laaroussi. M¹

¹Cardiovascular Surgery Department A, Ibn Sina University Hospital, Rabat, Morocco

DOI: <https://doi.org/10.36347/sasjs.2024.v10i08.012>

| Received: 27.06.2024 | Accepted: 03.08.2024 | Published: 21.08.2024

*Corresponding author: Briki Jihad

Cardiovascular Surgery Department A, Ibn Sina University Hospital, Rabat, Morocco

Abstract

Case Report

A rare disease with a high mortality rate, purulent pericarditis in childhood is a diagnostic and therapeutic emergency in order to avoid serious complications. It is recognized that the purulent pericarditis progresses to constriction, and treatment is based on surgery. We report the case of 9-month-old infant with purulent pericarditis that progressed to constriction.

Keywords: Pericardial effusion, pericardiocentesis, constriction, pericardectomy.

Copyright © 2024 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Purulent pericarditis (PP) remains a rare diagnosis in children with a dramatic natural history. It is a diagnostic and therapeutic emergency in which the combination of medical and surgical management has led to a significant reduction in mortality. The risk of progression to pericardial constriction is recognized, requiring close monitoring and appropriate treatment.

We present the case of a 9-month-old child, admitted for infectious respiratory symptoms, whose transthoracic echocardiography (TTE) revealed a pericardial effusion, and pericardial puncture yielded purulent fluid. The patient underwent surgical drainage and early partial pericardectomy. The child died of septic shock.

CASE PRESENTATION

The patient was a 9-month-old infant with no history of illness. He had been presenting with a productive cough for 20 days prior to admission, which had progressed to respiratory distress in a context of unquantified fever. On clinical examination, the patient was conscious, with a temperature of 37.8°C, BP 100-50 mmHg, heart frequency 64 bpm, SpO₂ 98%, polypnea 60 cycles/min, intercostal retraction and nasal flaring.

Auscultation revealed sibilant rales, and abdominal examination revealed an umbilical hernia with hepatomegaly.

Chest radiology revealed cardiomegaly with an cardiothoracic ratio at 0.79 and horizontalization of the ribs (Figure 1).

TTE showed a non-dilated, non-hypertrophied left ventricle, ejection fraction preserved at 70%. Non-dilated right heart chambers. Circumferential pericardial effusion measuring 12 mm in apical, 26 mm in anterior, 30 mm in posterolateral with fibrin deposition and no hemodynamic repercussions. Thickening of the pericardium. The inferior vena cava was slightly dilated (Figure 2).

Thoracic Computed Tomography Showed:

- Thickening and enhancement of the pericardial leaflets associated with a circumferential pericardial effusion measuring 24 mm in maximum diameter.
- Thrombus in the right atrium.
- Bilateral foci of condensation associated with infectious pneumopathy (Figure 3).

Biological findings included microcytic hypochromic anemia, predominantly neutrophilic hyperleukocytosis, C-reactive protein at 27, impaired liver function and thrombocytopenia.

The course was marked by the onset of a fever of 39°C, with moderate ascites, edema of the lower limbs and turgidity of the jugular veins, and in particular a worsening of the dyspnea. Surveillance TTE revealed worsening pericardial effusion with repercussions on the right cavities; invagination of the right atrium.

Pericardiocentesis was performed, yielding thick purulent fluid (100cc) with fibrin deposits blocking the drain (Figure 4).

Cytobacteriological examination of the puncture fluid which came back positive cultivating *Staphylococcus Aureus*.

Synergistic intravenous antibiotic therapy was started and then adjusted according to the bacteriological results.

Surgical drainage was indicated because of the purulent appearance of the puncture fluid and the worsening clinical hemodynamic impact of the effusion.

After induction, a vertical median sternotomy was performed. The pericardium was thick and retracted, with significant adhesions and more or less extensive areas of calcification. A partial pericardectomy was performed, along with drainage of a purulent fluid with fibrin deposits (Figure 5).

The patient was transferred to the pediatric intensive care unit, where her course was marked by rapid hemodynamic deterioration despite the best resuscitation measures, the onset of septic shock and multiple organ failure. Patient died after 72 hours.



Figure 1: X-Ray Shows Cardiomegaly

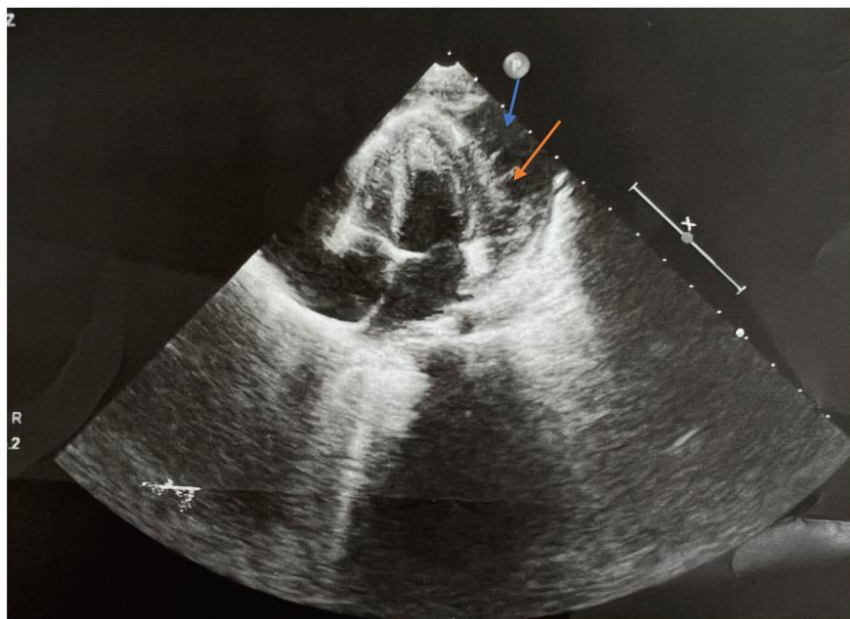


Figure 2: TTE image showing large pericardial effusion with fibrin deposit

➤ Blue narrow: pericardial effusion

- Orange narrow: fibrin deposit

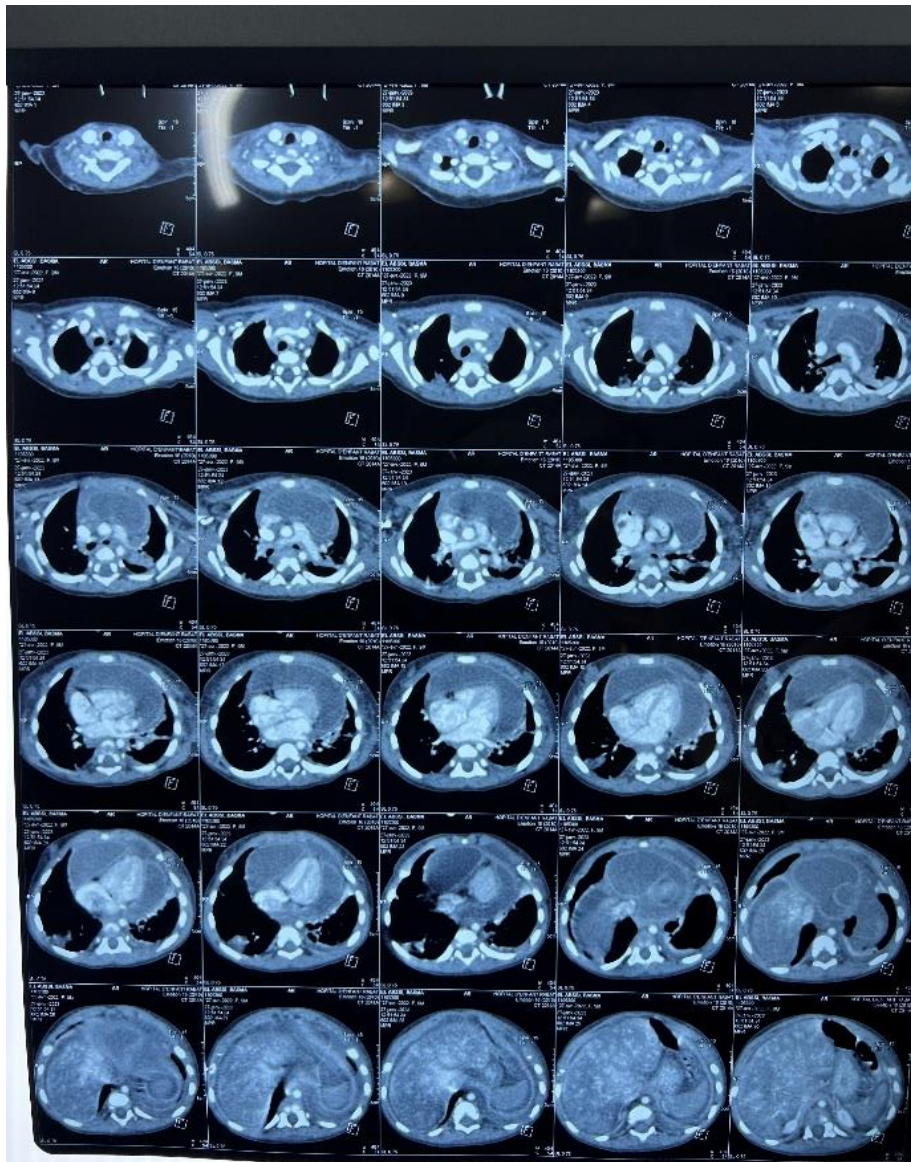


Figure 3: Image of thoracic CT scan showing circumferential pericardial effusion



Figure 4: purulent liquid drained by pericardiocentesis

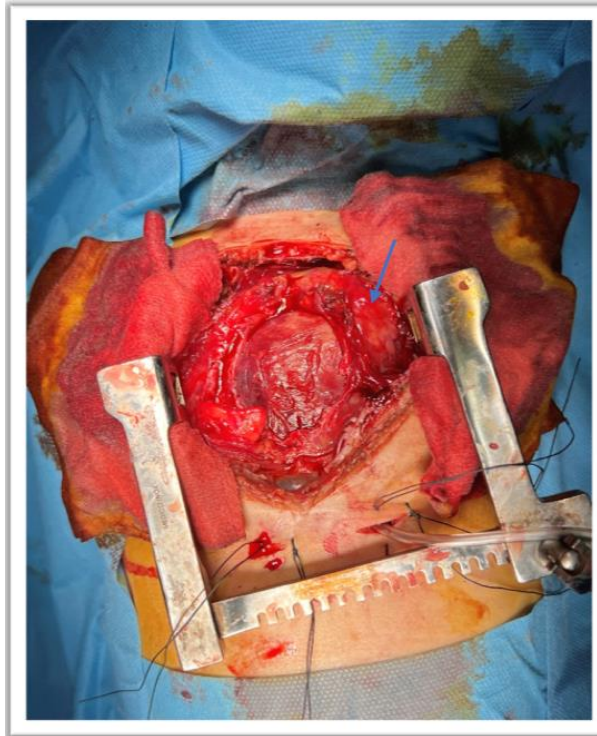


Figure 5: Operative view showing pericardial thickness and adhesences

DISCUSSION

Primary purulent pericarditis is rare in children, often associated with infection at another site, the contamination of which is either by direct extension, or haematogenous spread from a distant site [1-3]. Certain factors have been reported as predisposing patients to purulent pericarditis, such as immunosuppression, malignancy, pre-existing pericardial disease or previous cardiac surgery [4].

Infectious lung disease, septic arthritis, osteomyelitis, skin infections and septicemia are the most frequent associated infections in cases of purulent pericarditis according to studies by Abdelhak *et al.*, and thebaud *et al.*, [5, 6].

In the Western literature prior to the antibiotic era, streptococcus was the most common causative agent of PP, but since the antibiotic era, *Staphylococcus aureus* has become the most common causative agent [7, 8]. A number of series have reported *Haemophilus influenza* as an increasingly important cause of purulent pericarditis [9-11].

The clinical symptomatology of purulent pericarditis includes several non-specific signs, such as tachycardia, tachypnoea, fever, turgidity of the jugular veins and hepatomegaly, as well as other signs related to the associated infection.

Thoracic radiology usually shows cardiomegaly characteristic of pericardial effusion [7].

The diagnosis of pericardial effusion is confirmed by TTE. This is the key examination for rapid diagnosis, assessment of hemodynamic repercussions and detection of the state of cardiac tamponade, as well as looking for signs in favor of progression to constriction [12].

Purulent pericarditis is a therapeutic emergency; any delay can lead to dreadful complications, notably tamponade and septicemia [9, 13, 14].

The choice of antibiotics should be based on clinical indications. In the case of skin abscesses, osteomyelitis or pneumonia, a diagnosis of staphylococcal pericarditis is likely and treatment with penicillin and aminoglycoside, or vancomycin, is necessary.

Once the causative organism has been identified, antibiotics are adjusted [15].

The combination of medical treatment and pericardial drainage has been shown to reduce the mortality rate associated with purulent pericarditis to 20% and even less according to some authors [10], although the choice of the preferred pericardial drainage technique is still debated [9, 16].

The pericardium can be drained by pericardiocentesis or by surgical drainage. If pericardiocentesis does not relieve symptoms and signs of tamponade persist, immediate surgical drainage is indicated [17].

The purulent fluid may be thick, with fibrin deposits, which makes its evacuation by pericardiocentesis almost impossible; in this case surgical drainage is indicated [10].

Constrictive pericarditis is the final stage of acute or chronic inflammation leading to thickening of the adherent pericardium which impairs ventricular filling [18, 19]. Progression to constriction is rare, favored by poor socio-economic conditions, late diagnosis and prolonged delay in initiation of adequate treatment [20], which is consistent with our case that lingered for more than two weeks before landing in our department.

The European Society of Cardiology suggests that a pericardectomy or pericardial window is necessary in the treatment of purulent pericarditis in adults [21]. However, the literature on the need for these surgical procedures in children is inconclusive.

The correct timing of pericardectomy is a controversial issue; Thebaud *et al.*, in their study spanning 15 years of experience, indicate pericardectomy only when the effusion has been completely evacuated, with an average delay of 10 weeks before constriction occurs. They justify this approach by the inflammatory form that will strike the surgeon in the event of persistent effusion, without a cleavage plane, and pericardectomy will be difficult and incomplete. Close echocardiographic monitoring for several weeks is strongly recommended in order to detect this complication, which often develops insidiously [22].

In contrast to the 10-year retrospective study by Cakir *et al.*, in which 27.7% (5/18) of patients required early pericardectomy [1].

In our case, the purulent and thick appearance of the effusion fluid blocking the puncture catheter, as well as the worsening signs of hemodynamic repercussions, were in favor of surgical drainage. The symphysis found with thickening of the pericardial leaflets necessitated early partial pericardectomy, in a sub-acute inflammatory phase, which rendered the procedure incomplete.

In cases of fibrin accumulation, or when the pericardial fluid is very thick, the efficacy and safety of intrapericardial perfusion of fibrinolytic agents, such as Urokinase and Streptokinase, have been demonstrated in the work of Ekim *et al.*, and Ustünsoy *et al.*, to prevent progression to constriction [23, 24].

CONCLUSION

The diagnosis of purulent pericarditis should be made in the presence of an infectious syndrome and pericardial effusion. Early management is extremely important for good outcome. Although there is general agreement that surgical drainage is mandatory, the

approach, methods of drainage and extent of pericardial resection have been the subject of much debate.

Author's Contribution:

All authors have read and approved the manuscript.

B.J, A.M: main authors managed the patient.

I.M, E.A, B.C: co-author analyzed the patient data and was a major contributor in writing the manuscript.

R.J, S.R, and L.M: supervised the management of the patient, and revised the manuscript.

Conflicts of Interest: There are no conflicts of interest.

REFERENCES

1. Çakir, Ö., Gurkan, F., Balci, A. E., Eren, N., & Dikici, B. (2002). Purulent pericarditis in childhood: Ten years of experience. *Journal of Pediatric Surgery*, 37(10), 1404–1408. doi: 10.1053/jpsu.2002.35401. [PubMed] [CrossRef] [Google Scholar] [Ref list]
2. Levy, P. Y., Corey, R., Berger, P., Habib, G., Bonnet, J. L., Levy, S., ... & Raoult, D. (2003). Etiologic diagnosis of 204 pericardial effusions. *Medicine*, 82(6), 385–391. doi: 10.1097/01.md.0000101574.54295.73. [PubMed] [CrossRef] [Google Scholar] [Ref list]
3. Megged, O., Argaman, Z., & Kleid, D. (2011). Purulent pericarditis in children: Is pericardiotomy needed? *Pediatric Emergency Care*, 27(12), 1185–1187. doi: 10.1097/PEC.0b013e31823b44af. [PubMed] [CrossRef] [Google Scholar] [Ref list]
4. Rubin, R. H., & Moellering, R. C., Jr. (1975). Clinical, microbiologic and therapeutic aspects of purulent pericarditis. *American Journal of Medicine*, 59(1), 68–78. doi: 10.1016/0002-9343(75)90323-X. [PubMed] [CrossRef] [Google Scholar] [Ref list]
5. Abdel-Haq, N., Moussa, Z., Farhat, M. H., Chandrasekar, L., & Asmar, B. I. (2018). Infectious and noninfectious acute pericarditis in children: An 11-year experience. *Int J Pediatr*, 2018, 5450697.
6. Thébaud, B., Sidi, D., & Kachaner, J. (1996). Purulent pericarditis in children: A 15 year-experience. *Arch Pediatr*, 3, 1084–90.
7. Sinzobahamvya, N., & Ikeogu, M. O. (1987). Purulent pericarditis. *Arch Dis Child*, 62, 696–699.
8. Dupuis, C., Gronnier, P., Kachaner, J., Farru, O., Hernandez, I., Ducoulombier, H., & Vliers, A. (1994). Bacterial pericarditis in infancy and childhood. *The American journal of cardiology*, 74(8), 807–809.
9. Majid, A. A., & Omar, A. (1991). Diagnosis and management of purulent pericarditis. *J Thorac Cardiovasc Surg*, 102, 413–417.
10. Cheatham Jr, J. E., Grantham, R. N., Peyton, M. D., Thompson, W. M., Luckstead, E. F., Razook, J. D., & Elkins, R. C. (1980). Hemophilus influenzae purulent pericarditis in children: diagnostic and

- therapeutic considerations. *The Journal of Thoracic and Cardiovascular Surgery*, 79(6), 933-936.
11. Morgan, R. J., Stephenson, L. W., Woolf, P. K., Edie, R. N., & Edmunds Jr, L. H. (1983). Surgical treatment of purulent pericarditis in children. *The Journal of Thoracic and Cardiovascular Surgery*, 85(4), 527-531.
 12. Wendy, J. W. (1986). Echocardiographic features of a purulent pericardial peel. *Am Heart J*, 111, 990-992. Garvin, P. J., Danis, R. K., Lewis, J. E., & Willman, V. L. (1978). Purulent pericarditis in children. *Surgery*, 84(4), 471-475.
 13. Jaiyesimi, F., Abioye, A. A., & Antia, A. U. (1979). Infective pericarditis in Nigerian children. *Archives of Disease in Childhood*, 54(5), 384-390.
 14. Weir, E. K., & Joffle, H. S. (1977). Purulent pericarditis in children: An analysis of 28 cases. *Thorax*, 32, 438-443.
 15. Dupuis, C., Gronnier, P., Kachaner, J., Farru, O., Hernandez, I., Ducoulombier, H., & Vliers, A. (1994). Bacterial pericarditis in infancy and childhood. *The American journal of cardiology*, 74(8), 807-809.
 16. Allaria, A., Michelli, D., Capelli, H., Berri, G., & Gutierrez, D. (1992). Transient cardiac constriction following purulent pericarditis. *European journal of pediatrics*, 151(4), 250-251.
 17. FYFE, D. A., HAGLER, D. J., PUGA, F. J., & DRISCOLL, D. J. (1984, June). Clinical and therapeutic aspects of *Haemophilus influenzae* pericarditis in pediatric patients. In *Mayo Clinic Proceedings* (Vol. 59, No. 6, pp. 415-422). Elsevier.
 18. Troughton, R. W., Asher, C. R., & Klein, A. L. (2004). Pericarditis. *The Lancet*, 363(9410), 717-727. doi: 10.1016/S0140-6736(04)15648-1.
 19. Lange, R. A., & Hillis, L. D. (2004). Acute pericarditis. *The New England Journal of Medicine*, 351(21), 2195-2202. doi: 10.1056/NEJMcp041997.
 20. Sow, D., Fall, M., Kuakivi, N., BA, M., SARR, M., & MARTIN, S. (1985). Aspects de la péricardite purulente chez l'enfant à Dakar. *Bulletin de la Société Médicale d'Afrique Noire de Langue Française*, 29(1), 199-211.
 21. Maisch, B., Seferović, P., Ristić, A., Erbel, R., Rienmüller, R., & Adler, Y. (2004). Task Force members, ESC Committee for Practice Guidelines (CPG), Document Reviewers, Guidelines on the Diagnosis and Management of Pericardial Diseases Executive Summary: the Task Force on the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*, 25, 587-610. <https://doi.org/10.1016/j.ehj.2004.02.002>
 22. Thebaud, B., Sidi', D., & Kachaner, J. Les péricardites purulentes de l'enfant: 15 arts d'exp6rience Service de cardiopédiatrie, hopital Necker-Enfants-Malades, 149. rue de „Sevres, 75743 Paris cedex 15, France
 23. Ekim, H., & Demirbağ, R. (2004). Intrapericardial streptokinase for purulent pericarditis. *Surg Today*, 34(7), 569-72. doi: 10.1007/s00595-004-2773-x. PMID: 15221548.
 24. Üstünsoy, H., Celkan, M. A., Sivriköz, M. C., Kazaz, H., & Kilinç, M. (2002). Intrapericardial fibrinolytic therapy in purulent pericarditis. *European journal of cardio-thoracic surgery*, 22(3), 373-376. doi: 10.1016/s1010-7940(02)00258-0. PMID: 12204726.