Pediatr Cardiol 21:363–367, 2000
DOI: 10.1007/s002460010081

Pediatric Cardiology

Acute Pericarditis in Childhood: A 10-Year Experience

S. Roodpeyma, N. Sadeghian

Departments of Pediatrics and Pediatric Surgery, Taleghani Hospital, Shaheed Beheshti University of Medical Sciences, Tehran 19395, IR Iran

Abstract. Twenty children, aged 6 months to 13 years, with acute pericarditis admitted between 1987 and 1997 to a university hospital were analyzed retrospectively for their etiology, presentation, management, and prognosis. The most common types of pericarditis were purulent (40%), collagen vascular disease (30%), viral (20%), and neoplastic disease (10%). Most children presented with chest pain, fever, and tachypnea, but cardiac tamponade was not seen in any children. *Staphylococcus aureus* was the most frequent causative organism of purulent pericarditis and septic arthritis was the most common concurrent infection in the patients. Surgical drainage was performed for 11 cases, 9 underwent subxiphoid pericardial window, and 2 underwent thoracotomy. There was no constrictive pericarditis or reaccumulation of fluid after surgery. Two children died, one of staphylococcal septicemia and the other had a malignant mediastinal tumor. The remaining 18 made a complete recovery. We conclude that subxiphoid pericardial drainage is a simple, safe, and quick procedure and can be done easily in general hospitals by pediatric surgeons. The expensive facilities of cardiac surgeries are not needed.

Key words: Pericardial effusion — Subxiphoid pericardial window

Pericardial disease occurs in approximately 1 per 850 hospital admissions [13]. The most common causes of acute pericarditis in children include bacterial infection, viral pericarditis, connective tissue or collagen vascular disease, metabolic disease, neoplasms, postpericardiotomy syndrome, and idiopathic. All types of pericarditis are more common in males than in females and in adults compared with young children. The review by Gersony and McCraken [5] of 50 infants with purulent pericarditis indicated nearly equal sex distribution (56% males). Acute pericarditis is characterized by fever, chest pain, tachycardia, tachypnea, pericardial friction rub, and several electrocardiographic abnormalities. Echocardiography is the most sensitive method for diagnosis of pericardial effusion. Pericardial drainage is generally required for diagnosis and treatment of patients with pericardial effusion and often for the relief of associated cardiac tamponade [11]. With current operative techniques and perioperative management, the vast majority of patients with this condition can be operated on safely and expect improvement and survival [16].

The purpose of this study was to determine the etiology, clinical presentation, management, and outcome of acute pericarditis in children admitted to a general pediatric ward of a university hospital.

Patients and Methods

The medical records of all patients with a diagnosis of acute pericarditis admitted in the pediatric ward of Taleghani Hospital during a period of 10 years (1987–1997) were evaluated. The diagnosis of acute pericarditis was made on the basis of clinical manifestations of pericardial effusion confirmed by evidence of fluid collection on echocardiography. The total number of patients was 20: 11 boys and 9 girls with a mean age of 7.4 years (range: 6 months to 13 years). The majority of patients presented with chest pain, fever, and tachypnea. Clinical examination generally revealed a sick child with tachycardia, decreased heart sounds, or pericardial friction rub. Elevated jugular venous pulse and pulsus paradoxus were not common. The blood pressure was normal in all children and none had evidence of cardiac tamponade. The details are summarized in Table 1. An electrocardiogram, chest radiograph, and echocardiogram were performed in all cases. On M-mode echocardiography, with a small to moderate effusion, a "fluid space" was noted posteriorly. However, with largest effusions, fluid was seen anteriorly and posteriorly and the septal motion became grossly abnormal. Two-dimensional echocardiography revealed normal cardiac anatomy and identified circumferential pericardial effusions. Echocardiography was helpful to exclude other causes of an enlarged cardiac shadow especially myocardial dysfunction. Pericardiocentesis was performed in 17 cases. Specimens were taken from blood and pericardial fluid for microscopy, culture, and sensitivity. Pericardial aspirates were gram stained and cultured for bacteria as well as for mycobactrium tuberculosis. The fluid obtained was also estimated for sugar and protein levels and total cell counts. The laboratory findings on the pericardial fluids of 10 patients are shown in Table 2. Additional specimens

Correspondence to: S. Roodpeyma

Table 1. Summary of the main features of the patients and their management

Patient no.	Sex, age	Etiology	Associated illness	Pericardial tap	Medical treatment	Surgery
1	F, 11 years	SLE	Optic neuritis	−	Steroid	−
2	F, 12 years	Purulent	Septicemia, DIC	+	Antibiotic	+
3	M, 6 years	Purulent	Pneumonia, septic arthritis	+	Antibiotic	+
4	M, 12 years	Purulent	Osteomyelitis, septic arthritis	+	Antibiotic	+
5	M, 12 years	Purulent	Septic arthritis	+	Antibiotic	+
6	M, 5 years	Viral		−	NSAID	−
7	F, 3 years	Neoplasm	Mediastinal mass	+	Chemotherapy	+
8	F, 12 years	Viral		+	NSAID	−
9	F, 10 years	Viral		+	NSAID	+
10	F, 5 years	JRA	Pleural effusion	+	Steroid	−
11	F, 12 years	SLE	Pleural effusion	+	Steroid	−
12	M, 6 years	Purulent		+	Antibiotic	+
13	F, 7 years	JRA		−	Steroid	−
14	M, 6 years	JRA		+	Steroid	−
15	M, 6 months	Purulent	Septic arthritis, osteomyelitis, pneumonia	+	Antibiotic	+
16	F, 8 years	RF	Mitral regurgitation	+	Steroid	−
17	M, 13 years	Purulent	Septic arthritis, pyelonephritis	+	Antibiotic	+
18	M, 4 years	Purulent	Empyema	+	Antibiotic	+
19	M, 9 months	Neoplasm	Mediastinal mass	+	Chemotherapy	+
20	M, 2.5 years	Viral		+	NSAID	−

F, female; M, male; SLE, systemic lupus erythematosus; JRA, juvenile rheumatoid arthritis; RF, rheumatic fever; DIC, disseminated intravascular coagulation; NSAID, nonsteroidal anti-inflammatory drug.

Table 2. Results of the laboratory findings on the pericardial fluids ($n = 10$)

Case no.	RBC (μl)	WBC (μl)	PMN (%)	MN (%)	Sugar (mg/dl)	Protein (g/dl)	Culture
2	10,000	80	78	22	158	2.5	Negative
4	900	3,000	70	30	90	4.8	*S. aureus*
5	1,000	4,800	76	24	78	5.1	*S. aureus*
9	400	55	8	92	165	1.8	Negative
11	110	150	52	48	102	2.7	Negative
12	2,000	6,300	90	10	94	6.2	*S. aureus*
15	1,600	9,200	85	15	37	5.8	Negative
16	50	1,600	54	46	94	3.1	Negative
17	10,000	40,000	82	18	62	6.2	*S. aureus*
18	3,600	19,000	80	20	88	5.6	Negative

RBC, red blood cells; WBC, white blood cells; PMN, polymorphonuclear; MN, mononuclear.

were taken from a synovial joint or osteomyelitic abscess when relevant. Eight patients had purulent pericarditis which was evidenced by pus on pericardial aspirates, predominant polymorphonuclear cells on microscopy, and/or a positive culture. Parapericardial drainage was performed in these cases by a pediatric surgeon. The method of drainage was subxiphoid pericardial window. All children with purulent pericarditis were started on parenteral antibiotics, usually a combination of an antistaphylococcal agent (vancomycin) and a third-generation cephalosporin. Treatment was generally continued for a period of 4 weeks. The six patients with collagen (vascular) disease presented with the clinical manifestations of the original disease (e.g., arthritis, rashes, renal/optic involvements, and pleural effusion) plus signs of pericardial effusion. They were treated with steroids. None of the patients presented with signs of Kawasaki disease. Viral pericarditis was diagnosed in patients in whom other causes of pericardial effusion had been excluded and specific viral studies were not available. These patients received nonsteroidal antiinflammatory agents. Two patients with mediastinal mass had been referred as cases of congestive heart failure in whom clinical examination revealed signs of pericardial effusion. Echocardiographic examination showed evidence of pericardial fluid and the presence of a compressing mass which was confirmed by CT-Scan. These patients underwent surgery to debulk the tumor. One of them died and the other one was started on chemotherapy.

Results

The heart sounds were muffled in 13 cases and a friction rub was present in 7. Cardiomegaly was a constant feature in all patients. The electrocardiogram showed decreased voltage in 13 patients and a raised ST segment in 5 patients. Two patients with juvenile rheumatoid arthri-

tis had normal electrocardiograms. M mode and two-dimensional echocardiography showed the presence of pericardial fluid in all patients and the presence of a compressing mass lesion in the 2 patients with malignant neoplasms, who underwent further investigation by mediastinal CT-Scan (Fig. 1).

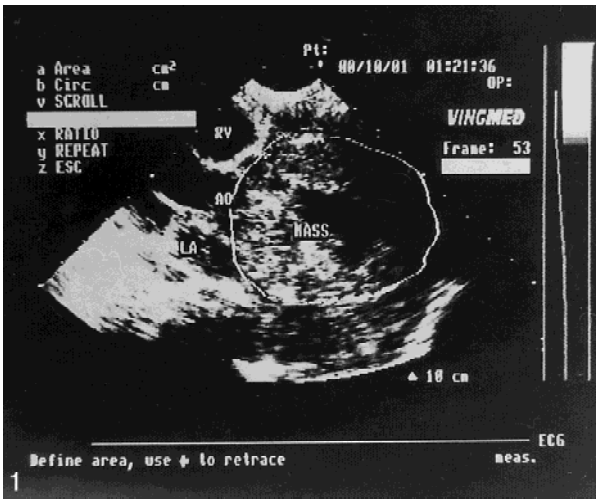
The etiology of pericardial effusion according to frequency was as follows: purulent or bacterial pericarditis, eight (40%); collagen vascular disease, six (30%); viral pericarditis, four (20%), and secondary to mediastinal mass invasion, two (10%). In patients with purulent pericarditis the preceding or concurrent infections were as follows: septic arthritis ($n = 5$), osteomyelitis ($n = 2$), pneumonia ($n = 1$), empyema ($n = 1$), and pyelonephritis ($n = 1$). Two patients had no evidence of other focal infection. *Staphylococcus aureus* was isolated from blood cultures in seven cases, and of these patients cultures of the pericardial fluid and of the synovial fluid were positive in four and three patients, respectively. In one patient who was already receiving several antibiotic agents started at the referring hospital, no organism could be cultured. A 12-year-old girl with staphylococcal septicemia (case 2) died 2 days after pericardial window operation due to disseminated intravascular coagulation and massive pulmonary hemorrhage.

There were six cases with collagen (vascular) disease. Three suffered from juvenile rheumatoid arthritis, two from systemic lupus erythematosus, and one from rheumatic fever. Two patients (cases 10 and 11) showed evidence of pleural effusion on chest x-ray (Figs. 3 and 4). One patient (case 16) had rheumatic mitral regurgitation in addition to pericarditis.

Four patients had viral pericarditis. One of them needed subxyphoid pericardial window due to deteriorating general condition, and the fluid was not purulent. All patients improved with nonsteroidal antiinflammatory drugs.

There were two cases with neoplastic disease. One patient (case 7) suffered from mediastinal germ cell tumor with pericardial invasion causing massive pericardial effusion. There was a marked improvement after surgical debulking of the tumor and subsequent chemotherapy. Another patient (case 19) was a 9-month-old boy with mediastinal lymphoblastic lymphoma which had invaded to the pericardium and brain. He underwent surgery for tumor debulking but died 6 hours after surgery.

Overall, 2 patients died and the 18 survivors remained well without evidence of recurrence or constrictive pericarditis for at least 2 years.



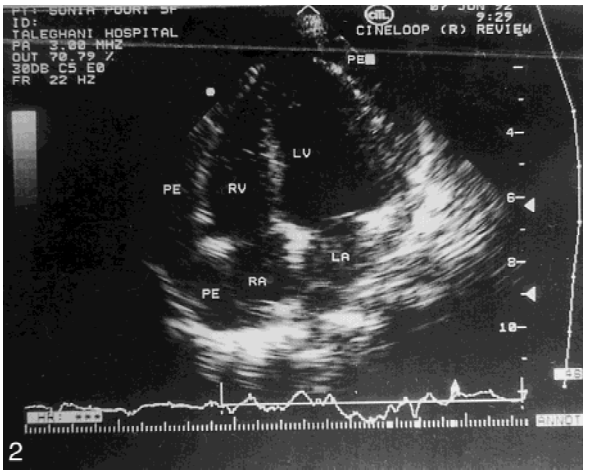
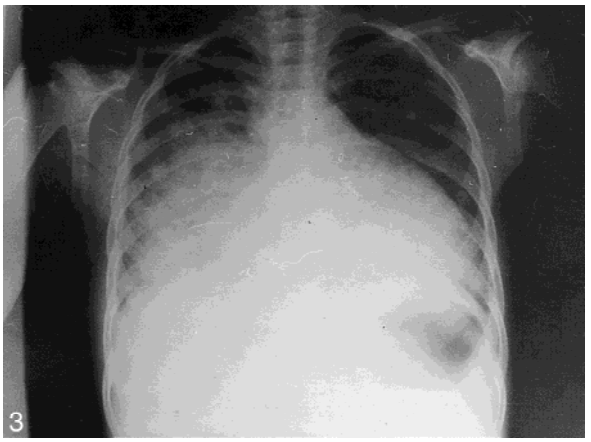


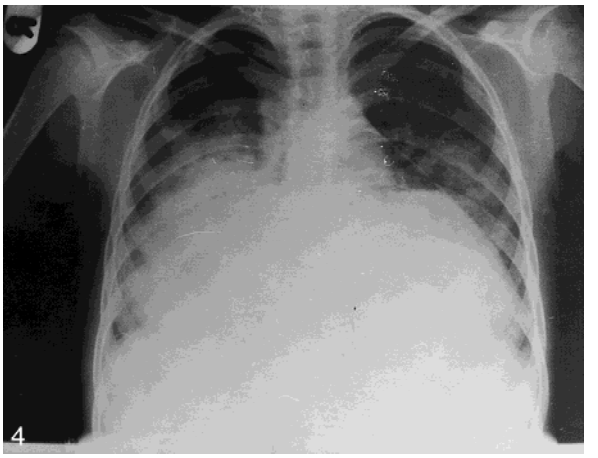
Fig. 1. Case No. 7: A 2-D echocardiogram showing a mass compressing the heart and producing a pericardial effusion.

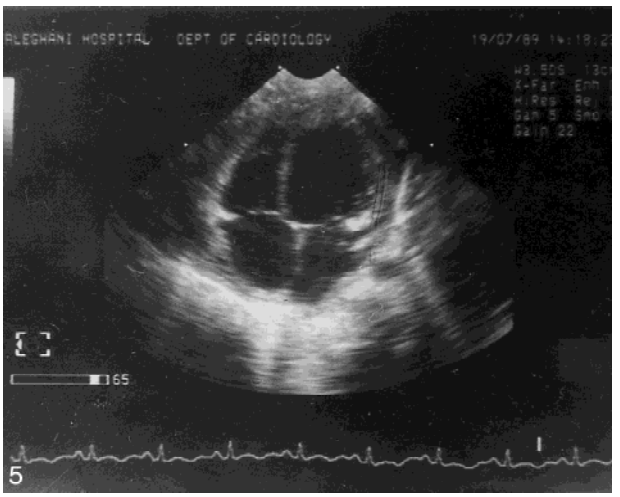
Fig. 2. Case No. 10: A four-chamber view showing the fluid on both the medial and the lateral aspects of the heart.

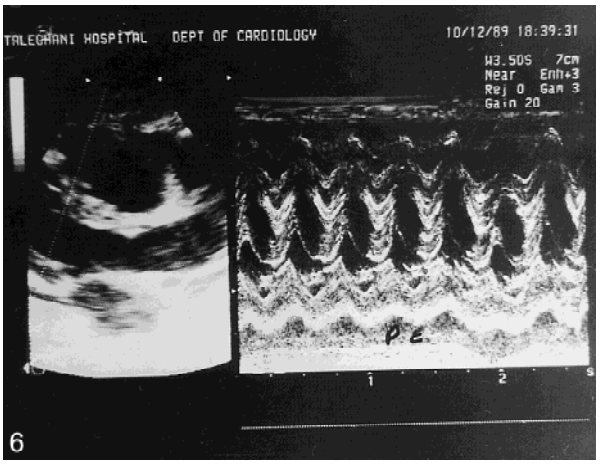
Discussion

Primary purulent pericarditis is rare. The disease is associated most often with infection from another site, with hematogeneous or direct spread to the pericardium. The most common concomitant site involved is usually the lung, especially for *S. aureus*, *Hemophilus influenzae*, and *Streptococcus pneumonia*. In patients with septic arthritis, osteomyelitis, or skin infection *S. aureus* is the usual causative organism of pericarditis [13]. In series from developing countries such as New Guinea [3], Zimbabwe [14], and Nigeria [2, 8], *S. aureus* remains the most common cause, and no cases of *H. influenzae* were described. In a series of 28 cases in South Africa, Weir and Joffe [19] reported only 1 case of *H. influenzae* purulent pericarditis. In a series of 12 cases in Malaysia, Majid and Omar [10] reported only 1 case of *H. influenzae* purulent pericarditis plus meningitis, and *S. aureus* was the most common causative organism (6 patients); they did not seen any cases of tuberculosis. Lack of tuberculosis pericarditis was also observed in studies by









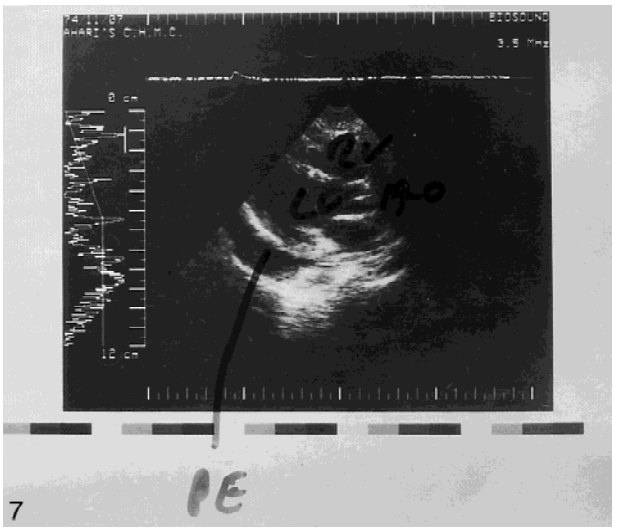


Fig. 3. Case No. 11: Chest radiography of a 12-year-old girl with systemic lupus erythematosus showing severe cardiomegaly and pleural effusion.

Fig. 4. Case No. 11: Chest radiography of a 12-year-old girl with systemic lupus erythematosus showing pneumopericardium after pericardial tap and pleural effusion.

Fig. 5. Case No. 12: A four-chamber view showing the fluid on the lateral aspect of the heart.

Fig. 6. Case No. 15: M mode and 2-D echocardiogram showing echo-free space posteriorly.

Fig. 7. Case No. 18: Long-axis view of 2-D echocardiography showing echo-free space between the posterior left ventricular wall and the parietal pericardium.

Ballal et al. [1] from Oman and Sinzobahamvya and Ikeogu [14] from Zimbabwe. In a study of 43 cases of bacterial pericarditis in children in France, Dupuis et al. [4] showed that *S. aureus* was the most frequent cause of infection. In the majority of the previous studies the condition was always secondary to a septic focus elsewhere, usually staphylococcal pneumonia. In our group of children purulent effusion was the most common form of pericardial involvement, *S. aureus* was the most frequent cause of infection, and septic arthritis was the most com-

mon concomitant illness. We have not seen any cases of tuberculosis.

Collagen (vascular disease) is another cause of pericardial involvement in children. Pericarditis occurs in acute rheumatic fever as a component of pancarditis, and it is a common manifestation of juvenile rheumatoid arthritis. Pericarditis is the most common cardiac lesion of systemic lupus erythematosus. Pericarditis in collagen disease does not need surgical drainage and usually heals well with medical treatment alone. In our series there were six cases with collagen disease, all of whom improved with steroid therapy alone.

The viruses that most commonly cause acute pericarditis are Coxackie virus group B and echovirus type 8. Viral pericarditis appears to be uncommon in young children. There are no clinical features that distinguish active viral pericarditis from idiopathic pericarditis, and it is likely that many cases of community-acquired idiopathic pericarditis are due to unrecognized viral infections [9]. In our study there were four patients with diagnosis of viral pericarditis, and the causative organism remained unknown.

Neoplastic pericardial effusion results from direct invasion of the pericardium. This malignant involvement is seen in patients with Hodgkin's disease, lymphoma, and leukemia. In our report there were two cases of mediastinal malignancy with invasion to pericardium.

Pericardial drainage is generally required for diagnosis and treatment of patients with pericardial effusion and often for associated cardiac tamponade. It must be emphasized that antimicrobial therapy alone is insufficient for the successful treatment of purulent pericarditis. Experience from the past few years suggests that excellent results can be obtained when adequate surgical drainage and antibiotic therapy are combined [12, 15]. Various effective techniques of pericardial drainage are available, each with different advantages and disadvantages. Insertion of a subxiphoid tube drain is a relatively minor procedure. It is more suitable for removal of thin pus and has been used with good results [11, 12, 15, 17]. The technique of pericardial window and pleural drainage has also been used [6, 7, 18]. Our preferred method of pericardial drainage is the subxiphoid approach. Surgical subxiphoid pericardial drainage provides a simple, safe, and expeditious treatment of most symptomatic pericardial effusions. This technique allows rapid access to the pericardium and is associated with low morbidity and excellent long-term results. Early pericardiostomy with a wide-bore tube to allow free drainage is effective in preventing constriction. None of the survivors in our series developed constriction, which usually occurs in the first postoperative year.

In view of our findings, we believe that although some cases of acute pericarditis may require anterior pericardiectomy, the technique of the subxiphoid window for the drainage of pericardial effusion is safe and effective in most patients and can be performed easily by a pediatric surgeon.

References

1. Ballal N, Vyas H, Noveli V (1991) Acute purulent pericarditis in Omani children. *J Trop Pediatr 37*:232–234
2. Bentley SJ, Andy JJ, Adeyomo A (1979) Staphylococcal pericarditis with pyopericardium. *Trop Geogr Med 31*:525–529
3. Corchran M, Poore P, Hadley GP, Tanner A (1983) Purulent pericarditis in Papua New Guinea: report of 12 cases and review of the literature in a tropical environment. *Trans R Soc Trop Med Hygene 77*:341–343
4. Dupuis C, Gronnier P, Kachaner J, et al. (1994) Bacterial pericarditis in infancy and childhood. *Am J Cardiol 74*:807–809
5. Gersony WM, McCraken GH (1967) Purulent pericarditis in infancy. *Pediatrics 40*:224–232
6. Hoier-Madsen K, Saunamaki K, Wulff J, Kjoller S, Jensen C (1985) Purulent pericarditis in children. *Scand J Thorac Cardiovasc Surg 19*:185–188
7. Iggo R, Higgins R (1988) Bilateral empyema and purulent pericarditis due to *Haemophilus influenzae* capsular type b. *Thorax 43*:582–583
8. Jaiyesimi F, Abioye AA, Antia AU (1979) Infective pericarditis in Nigerian children. *Arch Dis Child 54*:384–390
9. Lorell BH (1997) Pericardial disease In: Braunwald E (ed) *Heart Disease. A Textbook of Cardiovascular Medicine.* Saunders, Philadelphia, pp 1505–1534
10. Majid AA, Omar A (1991) Diagnosis and management of purulent pericarditis, experience with pericardiectomy. *J Thorac Cardiovasc Surg 102*:413–417
11. Moores DW, Dziuban SW Jr (1995) Pericardial drainage procedures. *Chest Surg Clin North Am 5*:359–373
12. Mueller XM, Tevaearai HT, Hurni M, et al. (1997) Long-term results of surgical subxiphoid pericardial drainage. *Thorac Cardiovasc Surg 45*:65–69
13. Pinsky WW, Friedman RA, Jubelirer D, Nihill MR (1992) Infectious pericarditis In: Feigin R, Cherry JD (eds) *Textbook of Pediatric Infectious Disease.* Saunders, Philadelphia, pp 377–386
14. Sinzobahamyya N, Ikeogu MO (1987) Purulent pericarditis. *Arch Dis Child 62*:696–699
15. Thebaud B, Sidi D, Kachaner J (1996) Purulent pericarditis in children: a 15 year experience. *Arch Pediatr 3*:1084–1090
16. Tuna IC, Danielson GK (1990) Surgical management of pericardial diseases. *Cardiol Clin 8*:683–696
17. Vassilopoulos PP, Nikolaidis K, Filopoulos E, Griniatsos J, Efremidou A (1995) Subxiphoidal pericardial window in the management of malignant pericardial effusion. *Eur J Surg Oncol 21*:545–547
18. Vigneswaran WT, Hardie R, Ferguson C, Faichney A (1985) Cardiac tamponade due to lancefield group A β-haemolytic streptococcal pericarditis. *Thorax 40*:549–550
19. Weir EK, Joffe HS (1977) Purulent pericarditis in children: an analysis of 28 cases. *Thorax 32*:438–443