Infective pericarditis in Nigerian children

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SUMMARY 53 children with infective pericarditis were seen at the University College Hospital, Ibadan, between 1967 and 1976. Their ages ranged from 10 days to 15 years but 53% of them were aged 5 years and below. Cough, fever, and breathlessness were the most common symptoms; cardiac decompensation was evident in over 30% of them, 23% had muffled heart sounds, but a pericardial friction rub was audible in only one. The main pathogens identified were *Mycobacterium tuberculosis* (11 cases), *Staphylococcus aureus* (11 cases), *Escherichia coli* (4 cases), *Pneumococcus* and *Pseudomonas* (3 cases each). Most of the patients had some other associated infection—such as, bronchopneumonia (12 cases), empyema thoracis (10 cases), lung abscess (10 cases), septicaemia (6 cases), and osteomyelitis (3 cases). Errors in diagnosis were common, the diagnosis having been missed in 72% of the cases identified at necropsy. Even if the correct diagnosis had been made during life and appropriate treatment given, the mortality rate (36%) was high. It is suggested that the onset of cardiac failure in any child with bronchopneumonia, empyema, or lung abscess should always arouse a suspicion of infective pericarditis.

Studies by many workers from developed countries on infective pericarditis suggest that this disorder is a rarity in childhood (Horan, 1957; Boyle *et al.*, 1961; Nadas and Levy, 1961; Benzing and Kaplan, 1963; Gersony and McCracken, 1967; D'Cruz *et al.*, 1970; van Reken *et al.*, 1974). Few studies on pericardial diseases have been undertaken by workers from the developing countries and these have concerned mainly adults (Schrire, 1964; Brockington and Edington, 1972; Carlisle and Ogunlesi, 1972; Brockington, 1974; Odi Assamoi *et al.*, 1975). To our knowledge there has been one study on pericardial diseases from Africa on children and adults and this suggested that infective pericarditis is more common among children in the developing countries than in developed ones (D'Arbella *et al.*, 1972). The purpose of the present study is to document the prevalence, clinical features, aetiological factors, and prognosis of infective pericarditis seen at the University College Hospital, Ibadan, during a period of 10 years.

Materials and methods

The study was prospective as well as retrospective. The prospective aspect comprised 14 children with infective pericarditis referred to the paediatric cardiac unit between January 1967 and December 1976. Clinical diagnosis of infective pericarditis in 11 of the 14 patients was confirmed by diagnostic pericardiocentesis which in each case yielded pus. The pericarditis in the remaining 3 children was of tuberculous origin, as confirmed by the pericardial exudate, a strongly positive tuberculin test, and satisfactory response to treatment with anti-tuberculous drugs. All 14 children underwent detailed evaluations, and their nutritional states were assessed from their weights, heights, mental states, muscle mass, subcutaneous tissues, and skin changes. Investigations carried out on each child were: blood count, haemoglobin genotype, culture of blood, pericardial or pleural aspirates, electrocardiography (ECG), and chest radiography. One patient underwent venous angiocardiology.

The retrospective study comprised 39 cases identified at necropsy during the same 10-year period. The necropsy diagnosis of these 39 cases was based on widely accepted criteria (Scotti, 1971). The clinical, laboratory, and necropsy findings of these patients were also reviewed.

Cases of pericarditis associated with cardiothoracic

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surgery, uraemia, malignancies, collagen disorders, endomyocardial fibrosis, or idiopathic cardiomegaly were excluded from the study. It should also be emphasised that the present series may not represent all the cases of infective pericarditis that passed through the hospital during the period as some of the patients might not have come to our attention.

Results

There were 53 patients (28 boys and 25 girls) and their ages ranged from 10 days to 15 years (mean 5.3 years). Two (4%) of them were neonates, 26 (49%) were between 1 month and 5 years, 16 (30%) between 6 and 10 years, and 9 (17%) were aged between 11 and 15 years.

Symptoms and signs. The commonest presenting symptoms were fever, cough, and breathlessness (Table 1). Five of the older children complained of retrosternal chest pain while 3 others, all aged less than 5 years, had febrile convulsions. Cardiac decompensation, as evidenced by tachycardia, weak peripheral pulses, raised jugular venous pressure, gallop rhythm, or hepatomegaly was present in over 30% of the children. Less than 25% of patients had muffled heart sounds, and pericardial friction rub was present in one. Kussmaul's sign was demonstrable in only one patient.

Nutritional state. Complete data on the nutritional state were available in 37 (70%) of the 53 patients. 10 (27%) of these 37 patients were marasmic while the remaining 27 (73%) were well nourished.

Chest radiography. Enlargement of the cardiac silhouette was present in 22 (82%) out of 27 patients in

whom the cardiac borders were clearly discernible. Three of these 22 patients with enlarged cardiac silhouettes had constrictive pericarditis of tuberculous origin. In one of the 22 patients pericardiocentesis yielded very little purulent aspirate in spite of clinical, radiological, and ECG signs of a large pericardial effusion. In order to assess the amount of the effusion a jugular venous angiocardiogram was carried out and it showed a large amount of pericardial fluid (Fig. 1). Another interesting chest x-ray which posed some diagnostic problem was that of an 11-month-old girl who presented with a history of fever, cough, breathlessness, and a swelling in the neck. On examination she was pyrexia (39°C) and had signs of cardiac failure and a left pleural effusion. In addition she had a tender and fluctuant mass which seemed to arise from the thoracic inlet. Although the mass did not move with swallowing, it increased in size whenever the child cried. The chest x-ray showed a large globular heart and a wide superior mediastinal shadow (Fig. 2). Diagnostic paracentesis of the left pleural cavity, pericardial space, and the cervical mass yielded pus. Post-paracentesis chest x-ray showed pneumomediastinum with the air extending into the soft tissue in the neck.

Fig. 1 Percutaneous venous angiocardiogram of a baby with purulent pericarditis. The pericardial effusion is demarcated on the right side by the opacified right atrium and the outer border of the cardiac silhouette.

Table 1 Symptoms and signs in 53 children with infective pericarditis

Symptoms	No. of patients	% of total	Signs	No. of patients	% of total
Fever	39	74	Pyrexia	53	100
Cough	35	66	Tachycardia	45	85
Breathlessness	28	53	Dyspnoea	36	68
Abdominal swelling	12	23	Hepatomegaly	35	66
Swelling of legs	12	23	Weak peripheral pulses	21	40
Anorexia	7	13	Pulmonary crepitations	21	40
Swelling of face	6	11	Splenomegaly	20	38
Vomiting	6	11	Peripheral oedema	19	36
Chest pain	5	9	Mucosal pallor	19	36
Diarrhoea	4	8	Raised jugular venous pressure	18	34
Vomiting	4	8	Muffled heart sounds	12	23
Convulsions	3	6	Ascites	10	19
			Gallop rhythm	6	11

Fig. 2 Chest x-ray of an 11-month-old child with pyopericardium and pyomediastinum. Note the soft-tissue shadow in the superior mediastinum.

It was thus concluded that the cervical abscess was an extension of a large pyomediastinum. Satisfactory recovery followed drainage of the pus collections and administration of erythromycin, digoxin, and hydroflumethiazide.

Other radiographic findings were bronchopneumonic consolidation (12 cases) and pleural effusion (10 cases). The pleural effusion was on the left side in 7 patients, on the right in 2 others, and bilateral in one. Pericardial calcification was evident in one patient with purulent pericarditis.

Electrocardiographic findings. Electrocardiograms were obtained in all 14 patients studied prospectively and in one of the necropsy cases. Flat or inverted T-waves, the commonest abnormalities, were observed in 10 (67%) of the 15 patients while 7 (47%) patients had dwarfed QRS complexes. Other ECG abnormalities included S-T depression (4 cases), bifid P-waves (2 cases), and frequent but unifocal ventricular premature contractions (1 case).

Aetiological agents. Aetiological agents were identified on Gram-stain and cultures either of the aspirates or blood in 31 (58%) of the 53 patients. The commonest nontuberculous pathogens were *Staphylococcus aureus*, *Escherichia coli*, *Pneumococcus*, and *Pseudomonas aeruginosa* (Table 2). These organisms were isolated from the blood (6 patients), pericardial aspirates (9 patients), and pleural aspirates (4 patients). Three of the 4 patients with *E. coli* pericarditis were aged less than one year. Eight patients had histological evidence of tuberculosis in pericardial specimens obtained at necropsy while in 3 others the diagnosis of tuberculosis was made before

Table 2 Aetiological agents

Pathogen	No. of cases* (n=36)	% of all isolates
<i>S. aureus</i>	11	31
<i>M. tuberculosis</i> †	11	31
<i>E. coli</i>	4	11
<i>Pneumococcus</i>	3	8
<i>P. aeruginosa</i>	3	8
<i>Klebsiella</i> sp.	2	5
<i>Salmonella typhi</i>	1	3
<i>Dracunculus medinensis</i> (guinea-worm)	1	3

*4 patients each had more than one pathogen: (i) *S. aureus*, *Pseudomonas*, and *Klebsiella*; (ii) *S. aureus* and *Pneumococcus*; (iii) *Pneumococcus* and *Klebsiella*; (iv) *E. coli* and *Pseudomonas*.

†Histological proof only.

death. Guinea-worm was identified in the pericardium of one patient at necropsy.

Haematological findings. Total white blood counts were available in 19 patients, 14 (74%) of whom had a polymorphonuclear leucocytosis (neutrophils $>7.0 \times 10^9/l$; $7000/mm^3$). Anaemia (haematocrit $<25\%$) was present in 6 out of the 19 patients whose haematocrits were available. The haemoglobin genotype was determined in 21 patients; it was A in 18 patients, and A+S in 2 others. One patient had haemoglobin SC disease.

Associated conditions. 20 (38%) of the 53 patients either had empyema thoracis or an extensive lung abscess (Table 3). Five patients had both empyema and lung abscesses. The bronchopneumonia which was present in 12 patients was presumed to be bacterial in origin. Less common associated conditions were pyogenic meningitis, acute bacterial

Table 3 Associated conditions

Associated disease	No. of cases	%
Bronchopneumonia	12	23
Metastatic abscess (<2 cm in diameter)		
Lungs	7	
Brain	2	21
Kidney	2	
Empyema thoracis	10	19
Extensive lung abscess	10	19
Tuberculosis		
Pulmonary	4	
Meningeal	2	
Disseminated	2	17
Spinal	1	
Septicaemia	6	11
Osteomyelitis	3	6

endocarditis, myocardial abscess, omphalocele, congenital hydronephrosis, and left ventricular submitral aneurysm (one case of each).

Pathological types. 22 (42%) of the 53 patients had purulent pericarditis (Table 4); 13 of these 22 patients had some other intrathoracic suppurative lesions. Three patients with purulent pericarditis developed fatal cardiac tamponade, and at necropsy each of them had >300 ml pus in the pericardial cavity. 11 patients had serious pericarditis: 4 cases were tuberculous while the remaining 7 were idiopathic. Six of the 7 cases of constrictive pericarditis were also of tuberculous origin; the aetiology of the 7th case was unknown.

Antemortem diagnosis. Pericarditis was not suspected during life in 28 (72%) of the 39 cases identified from necropsy records; in most instances only the associated pulmonary infection was accurately diagnosed. Even when cardiac failure was recognised, as indeed was the case in 7 (25%) of the 28 cases, it was usually ascribed either to idiopathic cardiomyopathy or to acute cor pulmonale resulting from pneumonia or empyema.

Treatment and outcome. The 14 patients studied prospectively were managed with appropriate antibiotics, drainage of pus collections, digoxin, diuretics,

and blood transfusion in appropriate cases. Five (36%) of the 14 patients died; 4 of them had purulent pericarditis and one had tuberculous pericarditis. Thus the mortality rates were 36 and 33% respectively for purulent and tuberculous pericarditis. Age, sex, nutritional state, or duration of the illness before hospitalisation did not appear to affect the mortality. In 2 of the 5 fatal cases there was an associated large lung abscess and a massive empyema in 2 others. The 9 survivors have been followed up for periods varying between 18 months and 4 years. Eight of them have recovered completely (Figs 3a, b) while the remaining patient has persistent cardiac decompensation.

Discussion

The present series of 53 cases of infective pericarditis seen during a period of 10 years in children aged 10 days to 15 years is in accord with the 62 cases reported from Uganda by D'Arbella *et al.* (1972) in patients aged 0–20 years. In contrast, Simcha and Taylor (1971) reported only 5 cases seen at The Hospital for Sick Children, London, between 1952 and 1970. Workers from different parts of North

Fig. 3a Chest x-ray of a baby with purulent pericarditis; the cardiac silhouette is enlarged and globular.

Table 4 Types of pericarditis in 53 children

Type	Patients (n = 53)	%
Fibrinoid pericarditis	5 (1)	9
Effusive		
Serous	11 (4)	21
Purulent	22	42
Haemorrhagic	3	6
Adhesive pericarditis	5	9
Constrictive pericarditis	7 (6)	13

Figures in parentheses denote proportion of cases that were of tuberculous origin.

Fig. 3b Same patient as in Fig. 3a, 4 weeks later. The cardiac size had returned to normal after pericardial drainage and administration of antibiotics, digoxin, and diuretics.

America (Benzing and Kaplan, 1963; van Reken *et al.*, 1974; Strauss *et al.*, 1975; F. Jaiyesimi, unpublished data) have also stressed the rarity of infective pericarditis in childhood in these developed countries. It may thus be concluded from the present series, and that of D'Arbella *et al.* (1972) from Uganda, that infective pericarditis is a more common childhood disease in the developing countries than in developed ones. Poor sanitation, widespread malnutrition with its depressing effects on immune mechanisms (Chandra, 1972; Dossetor *et al.*, 1977), and the high incidence of bacterial respiratory infections (Abdel-Khalik *et al.*, 1938; Mimica *et al.*, 1971; Aderale, 1977; Silverman *et al.*, 1977), no doubt contribute to the high prevalence of infective pericarditis in children living in developing countries; but infective pericarditis is by no means a major childhood health problem in such countries. For example, it accounted for only 6% of paediatric cardiac disorders (A. U. Antia and F. Jaiyesimi, unpublished data), and for less than 1% of total paediatric admissions into University College Hospital, Ibadan, during this 10-year period. Similarly, during the same period 2079 necropsies

were carried out on children aged 15 years and below, and the incidence of infective pericarditis was 0.02%.

In the present as well as in other series (Horan, 1957; Al-Omari and Samarra, 1971; D'Arbella *et al.*, 1972; van Reken *et al.*, 1974; Odi Assamoi *et al.*, 1975) *S. aureus* was the most common aetiological agent. This organism has also been reported to be the commonest infectious agent in other conditions in our hospital (Aderale *et al.*, 1974; Montefiore *et al.*, 1974; Jaiyesimi, 1977). *E. coli* was the next most common pyogenic organism and this may be related to the number of infants in the present series. It is worth noting that the aetiological agent was unknown in 41% of the patients in the present study. D'Arbella *et al.* (1972) also reported a high incidence of idiopathic cases in their series. This high incidence of idiopathic cases may be attributed to several factors. Firstly, antibiotics might have been administered before the patients' admission into hospital. Secondly, a low index of suspicion would result in many cases being unrecognised and thus in failure to institute appropriate investigations. Thirdly, in most of our patients no virological studies were carried out.

Several workers have stressed the ease with which cases of infective pericarditis are unrecognised before death (Poynton, 1908; Poynton, 1934; Horan, 1957; Boyle *et al.*, 1961). In the present series the diagnosis was missed in 72% of cases that came to necropsy. This high incidence of misdiagnosis may be due to the nonspecific nature of the presenting symptoms and the scarcity of characteristic signs. For instance, a pericardial rub is audible in only 15 to 25% of cases (Benzing and Kaplan, 1963), while the intensity of the cardiac sounds may be normal in the presence of cardiac tamponade (Williams and Soutter, 1954; Nadas and Fyler, 1972). Occasionally some of the characteristic signs may be obscured by those of a coexisting bronchopneumonia, lung abscess, or empyema, as was indeed the case in most of our patients. Similarly, the clinical diagnosis of effusive tuberculous pericarditis is fraught with pitfalls: the same globular, 'murmurless' heart may be due to endomyocardial fibrosis and the pericardial fluid chemistry may be similar in both endomyocardial fibrosis and tuberculous pericarditis (Antia, 1968; Somers *et al.*, 1971). In such clinical situations a right ventriculography may be the only method of differentiating between these two conditions.

The spectrum of associated conditions in the present series is similar to that described by previous authors (Boyle *et al.*, 1961; Benzing and Kaplan, 1963; van Reken *et al.*, 1974). However, the extensive pyromediastinum which was found in one patient has not, to our knowledge, been previously reported.

The factors which have been reported to have adverse effects on the prognosis in purulent pericarditis are overwhelming infection (Horan, 1957), and inadequate evacuation of the pus within the pericardial cavity (Al-Omari and Samarraï, 1971). In the present series prognosis appeared to have been adversely affected by septicaemia, coexisting suppurative lung disease, and misdiagnosis with failure to institute appropriate treatment. Rooney *et al.* (1970) suggested that prognosis in tuberculous pericarditis might be adversely affected by delay in diagnosis and treatment, as well as by the mechanical constraints which pericardial inflammation imposes on myocardial compliance. As only 3 of our prospectively-studied patients had tuberculous pericarditis we cannot make any valid statements on the prognosis in that category of patients.

The failure to recognise many cases of infective pericarditis before death highlights the need for a high index of suspicion on the part of clinicians, particularly those who practise in the developing countries where the disease is relatively more common. The onset of cardiac failure in any child with bronchopneumonia, empyema, or lung abscess should always arouse a suspicion of purulent pericarditis. In addition to frequent clinical and radiographic evaluations, a diagnostic pericardial tap should be mandatory in all such patients; and a venous angiogram may be required occasionally to demonstrate a loculated pericardial effusion. The need for these invasive procedures, however, can be obviated by echocardiography, this being a non-invasive and perhaps the most sensitive method of detecting pericardial effusions (Feigenbaum *et al.*, 1966; Moss and Bruhn, 1966; Feigenbaum, 1972; Meyer and Kaplan, 1972). Furthermore the ease with which echocardiography can be repeated renders it invaluable in assessing the course of a pericardial effusion.

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Change of reference style

From January 1980, the *Archives* will change its style for citing references and will follow the number system. We are making this change in order to conform with the 'Vancouver style' of uniform requirements for manuscripts submitted to biomedical journals.

Previously the *Archives* has used the Harvard reference system. Its advantages and disadvantages compared with those of the number system have seemed evenly balanced, but the editors have been aware of the burden imposed on authors and their secretaries by the different styles which different journals require. Therefore, with the unanimous agreement of the editorial committee, we have decided to support a move whereby a large—and increasing—number of major medical journals

will accept manuscripts presented in one agreed style.

The system numbers references consecutively in the order in which they are first mentioned in the text. References are identified in the text by arabic numerals. For further details see instructions to authors inside the front cover, and the references cited.^{1,2}

From now onward all manuscripts submitted should accord with the new style.

¹ International Steering Committee of Medical Editors. Uniform requirements for manuscripts submitted to biomedical journals. *Br Med J* 1979; **1**; 533-535.

² Lancet. The Vancouver style. Uniform requirements for manuscripts submitted to biomedical journals. *Lancet* 1979; **1**; 429-430.