The Central African Journal of Medicine

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Volume Nineteen

JANUARY - DECEMBER

1973

SALISBURY : RHODESIA

The Central African
Journal of Medicine

Volume 19 FEBRUARY, 1973 No. 2

Pericarditis—A Five Year Study in the African

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A retrospective study of cases of pericarditis presenting to Harare African Hospital during the five year period 1967-1971 was carried out. This study excludes cases of pericarditis due to rheumatic heart disease and uraemia.

During the five year period there were 85 patients in whom a diagnosis of pericarditis or pericardial effusion was made (Table 1). Ten of these would appear on subsequent investigation or post-mortem findings to have been inaccurately diagnosed, and in 18 cases the diagnosis of pericarditis was established solely on clinical examination with no confirmatory investigations. These 28 cases have been excluded from this study. The report deals with the remaining 57 cases of pericarditis or pericardial effusion in whom the clinical diagnosis was confirmed by radiology, aspiration, surgery or post-mortem findings.

Table 1

PERICARDITIS (1967-1971)

HARARE HOSPITAL — SALISBURY

Total number of cases	85
Diagnosis subsequently disproved	10
Clinical diagnosis only	18
Confirmed diagnosis (all forms of pericarditis)	57
Constrictive Pericarditis	17

This table includes all cases which were listed in the records of Harare Hospital, Salisbury, Rhodesia, as pericarditis. There were 85 cases diagnosed clinically as pericarditis over the five year period, 1967-1971, but only 57 had confirmatory evidence. Of these 57 seventeen had constrictive pericarditis.

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Of these 57 cases, 17 were suspected of having constrictive pericarditis but in only five was the diagnosis confirmed at surgery or post-mortem. Nine patients refused surgery and were subsequently discharged.

AETIOLOGY (Table 2)

The aetiology of the condition was confirmed bacteriologically, histologically or on post-mortem findings in 30 of the 57 cases. Of the remaining 27 cases, 21 cases were provisionally diagnosed as tuberculosis and discharged on anti-tuberculous therapy. In 6 cases no aetiological factor was elicited.

Table 2

PERICARDITIS (1967-1971)

HARARE HOSPITAL — SALISBURY

AETIOLOGY (57 cases)

A. PERICARDITIS (All cases)

AETIOLOGY SUBSTANTIATED (53%)

(a) Tuberculosis	16 (53 per cent.)
(b) Acute Purulent	10 (33 per cent.)
(c) Malignant	2 (7 per cent.)
(d) Serous	2 (7 per cent.)
Total	30

AETIOLOGY UNSUBSTANTIATED (47%)

(a) ? Tuberculosis	21
(b) Unknown	6
Total	27

B. CONSTRICTIVE PERICARDITIS

(a) Tuberculosis	7 (41 per cent.)
(b) Undetermined	10 (59 per cent.)
Total	17

A table showing the aetiological factors operative or suspected in the 57 cases of confirmed pericarditis found in the records of the five year period (1967-1971) at Harare Hospital, Salisbury, Rhodesia.

Table 3

PERICARDITIS (1967-1971)

HARARE HOSPITAL — SALISBURY

Results of Pericardectomy

No.	Sex	Age	Aetiology	Adherence	Post-operative Progress	Result
				A. CONSTRICTIVE PERICARDITIS		
1	M	28	?T.B.	++	Died on Table — Haemorrhage	Died
2	F	25	T.B.	++	Uncomplicated progress	No symptoms 2 months
3	M	45	?	+	Uncomplicated progress	No symptoms 1 month
4	F	25	T.B.	++	Uncomplicated progress	No symptoms 1 month
				B. PERICARDIAL EFFUSION		
5	M	8	Acute purulent	Pus ++	Died on table — during bronchoscopy after pericardectomy	Died
6	M	27	T.B	Blood stained effusion	Respiratory distress. Ventilated 10 days.	Died 8 weeks post-operation. Intercurrent broncho-pneumonia.
7	M	24	T.B.	Blood stained effusion	Respiratory distress. Ventilated 4 days.	No symptoms 5 months.

A table to show the comparison between the results obtained from pericardectomy for constrictive pericarditis and for pericardial effusion with cardiac compression.

Of the 30 cases in whom the aetiology was confirmed, 16 were found to be due to tuberculosis, 10 were acute purulent pericarditides, two secondary to malignancy (one bronchogenic carcinoma and one lymphosarcoma) and two serous pericarditides (possibly of viral origin).

Seven cases of tuberculosis were confirmed amongst the 17 of constrictive pericarditis but no definite aetiological factor was elicited in the remaining 10 cases.

CONSTRICTIVE PERICARDITIS — CLINICAL FEATURES

The clinical features of the 17 cases of constrictive pericarditis were in the main fairly typical of the condition. The majority of patients presented complaining of cough, dyspnoea, chest pain and oedema. Their symptoms were usually of quite recent origin, the majority being between 3 weeks and 3 months duration.

Examination showed most cases to have oedema and ascites with a raised jugular venous pressure, pulsus paradoxus and hepatomegaly. Electro-cardiographs showed low voltage tracings and T-wave inversion. Radiology revealed a variety of features but pericardial calcification and absence of cardiac pulsation on cardiac screening were notable features in many of these cases. Other ancillary investigations which had been used were carbon dioxide insufflation, cardiac catheterisation and ultrasound.

An attempt was made to correlate the clinical features and the findings at operation. The three cases in whom a fairly marked degree of adherence of the pericardium was present showed atrial fibrillation and/or ventricular extrasystoles, whereas in the one case in which adhesion was not marked and in those in which a pericardial effusion was present, no disorders of rhythm were noted.

SURGICAL MANAGEMENT (Table 3)

During the five year period pericardectomy was undertaken in 7 cases. Four were cases of constrictive pericarditis and 3 had pericardial effusion with tamponade. Of the 4 cases of constrictive pericarditis one patient died during the operation as the result of uncontrollable haemorrhage due to a tear in the right atrium. The remaining 3 cases all made an uncomplicated post-operative recovery and were all asymptomatic one month after surgery.

In the 3 patients with pericardial effusion for whom surgery was undertaken the results were unexpectedly less satisfactory. One patient died during bronchoscopy after completion of an emergency pericardectomy for a staphyloccal pericarditis with tamponade, the pericardium being up to one inch thick with adherent fibrin. The second case required ventilation for 10 days following surgery and improved considerably, but was cachectic and died eight weeks later due to respiratory failure consequent upon widespread bronchopneumonia of very rapid onset. The third case required artificial ventilation for four days following pericardectomy but subsequently improved and was asymptomatic when seen five months later.

PROGRESS AND MORTALITY RATES (Table 4)

The prognosis is difficult to assess because of the inability to secure adequate follow-up of cases. Of the 16 proven cases of tuberculous pericarditis, 11 were discharged on antituberculous therapy and five died. The most disturbing feature noted in this review is that all 10 patients presenting with acute purulent pericarditides subsequently died.

Of the seven patients in whom a pericardectomy was performed three died (43 per cent.)

It is interesting to note that the post-operative morbidity and mortality was higher amongst the cases with pericardial effusion than amongst those in whom constriction was present, but this may well be because the operations were performed because of rapid deterioration despite conservative measures.*

Table 4

PERICARDITIS (1967-1971)

HARARE HOSPITAL — SALISBURY

PROGNOSIS

	Total	Discharge	Death
Confirmed Tuberculosis	16	11	5
Acute purulent	10	—	10

This table indicates the seriousness of acute purulent pericarditis. The 69 per cent. success rate with confirmed tuberculous pericarditis treated surgically/medically compares favourably with the 100 per cent. mortality amongst patients who had acute purulent pericarditis.

* In an earlier series of patients treated surgically in the same hospital by J.M.M. the reverse had been true where the effusion was not apparently pyogenic (or amoebic — one case) in origin, but in these cases rapid deterioration despite conservative measures was not occurring. Excluding pyogenic cases, the worst surgical results in this series were obtained where fibrous invasion of the myocardium had occurred.

DISCUSSION

The aetiology of constrictive pericarditis has been the subject of much discussion and although Tuberculosis has been incriminated as the major aetiological factor this is still controversial. Mohiuddin (1967) in reviewing 17 cases of constrictive pericarditis showed only one case to be of tuberculous origin. Madaras *et al.* (1967) found only 4 cases of tuberculosis out of 17 in whom cardiac decortication was performed. Histological study in their remaining cases revealed only non-specific chronic inflammation. McPhail *et al.* (1967) however, showed 18 out of 30 cases to be of tuberculous aetiology. Rooney *et al.* (1970) in reviewing the aetiological factors in constrictive pericarditis state that in autopsied cases of pericarditis where a specific cause was documented 11 per cent. of pericarditides were due to Tuberculosis. In our present series 16 of the 30 cases of pericarditis in whom the aetiology were confirmed were of tuberculous origin, as were 7 of the 17 cases of constrictive pericarditis. In the remaining 10 cases of constrictive pericarditis no aetological factor was determined.

In reviewing the results of surgical treatment in contrictive pericarditis Daymen *et al.* (1967) showed the operative and post-operative mortality rates to vary from 3-45 per cent. in different series. These workers had a 9 per cent mortality rate in their series of 22 cases operated upon at the Hammersmith Hospital over a period of 15 years. McPhail *et al.* (1967) had seven deaths in their series of 30 cases of constrictive pericarditis in whom pericardectomy was attempted, the causes of death being myocardial failure (4), inadvertent tearing of the inferior vena cava (2) and pulmonary embolism (1). In our series, pericardectomy was performed on 4 patients with constrictive pericarditis. One patient died as the result of a tear in the right atrium and the remaining 3 patients made good post-operative recoveries.

The place of steroid therapy in the management of pericarditis has been the subject of some discussion and Rooney *et al.* (1970) have shown that steroid therapy reduces the mortality rate in patients treated both medically and surgically. These workers feel that the suppression of pericardial reactivity through the use of steroids can minimise the accumulation of fluids and the induction of arrhythmias.

The high mortality rate of patients with acute purulent pericarditis is well recognized and Sanyal *et al.* (1970) in reviewing six cases of acute staphylococcal pericarditis, of whom four died, state that "this constitutes a medical emergency requiring immediate drainage of the pericardial sac by aspiration or surgically". In our present series, all the patients with this condition were treated with antibiotics and pericardiocentesis and in only one patient was a pericardectomy attempted. al Omari and Samarrai (1971) report three cases of acute pyogenic pericarditis treated successfully by broad spectrum antibiotics and drainage either by aspiration or pericardiostomy using a xiphisternal approach. It is suggested, therefore, that formal surgical drainage i.e. pericardiostomy will offer more adequate treatment than pericardial aspiration in this very serious condition.

SUMMARY

A retrospective five year study of 57 cases of pericarditis is presented. The aetiology, clinical features and results of surgical intervention are discussed. The presence of a 100 per cent mortality in cases of acute purulent pericarditis is noted.

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