Prognosis of pericarditis after acute myocardial infarction

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Out of 195 patients with acute myocardial infarction, 22 (11.3%) developed a pericardial friction rub. The diagnosis of the postmyocardial infarction syndrome was made in none of these. The course of the 22 patients with pericarditis was compared with that of a consecutive series of 50 patients without pericarditis. As a group, those with pericarditis had a greater rise in SGOT and higher incidence of arrhythmias and heart block, cardiac failure, and cardiogenic shock. A greater number had high ESR and abnormal chest films. All these differences were significant, but there was no significant difference in the mortality between the two groups. The complications were attributed not to the pericarditis, but to the extensive myocardial damage. Two patients with pericarditis who had been given anticoagulants developed haemopericardium which probably contributed to their death. It is concluded that (a) though pericarditis is indicative of severe myocardial infarction it does not increase the mortality; (b) a friction rub persisting for 3 days or more should be considered as a bad prognostic sign; and (c) in the presence of pericarditis in very ill patients with extensive infarction, anticoagulants should be discontinued since there might be a danger of haemopericardium.

The incidence of pericarditis after acute myocardial infarction, as it is diagnosed clinically by the auscultation of a pericardial friction rub, varies according to several workers (Table 1). The diagnosis of pericarditis is usually made clinically by the finding of a pericardial friction rub, which is usually a transient physical sign, developing during the early days after acute myocardial infarction, and it hardly can be considered as a severe complication of the disease (Thadani *et al.*, 1971). The pericarditis of the postmyocardial infarction syndrome is more pro-

longed, may be recurrent, and appears later in the illness, commonly after the third or fourth week, its incidence being 3 to 4 per cent according to Dressler (1959). Since the establishment of coronary care units several workers have studied the severe complications and metabolic changes after acute myocardial infarction (Julian and Oliver, 1968), but no further clinical observations, except a recent paper (Thadani *et al.*, 1971), appear to have been made on pericarditis which is a relatively common complication of the illness. The present study was therefore undertaken in order to determine the incidence and prognostic and clinical significance of pericarditis after acute myocardial infarction. A very brief summary of the findings has already been published elsewhere (Niarchos, 1971).

Patients and methods

Out of 375 patients admitted to the coronary care unit during one year, 195 had definite acute myocardial infarction as diagnosed by the criteria of the World Health Organization (1959): typical history of chest pain, electrocardiographic evidence (Q waves and ST, T changes), raised serum aspartate aminotransferase (SGOT) and lactic dehydrogenase, and necropsy in fatal cases. SGOT was measured by a modification of the method of Babson *et al.* (1962). Details regarding the general policy of this unit have been described elsewhere

TABLE 1 Incidence of pericarditis after acute myocardial infarction

Series	No. of patients with acute myocardial infarction studied	Pericarditis (%)
Parkinson and Bedford (1928)	100 clinical; 83 necropsy	7 13
Rosenbaum and Levine (1941)	206	16
Wood (1968)	—	10
Thadani <i>et al.</i> (1971)	779	7
Present series	195	11.3

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(Salazar and McKendrick, 1970). The electrocardiogram is monitored in the patient's room and in the nurses' central station and can be recorded permanently on paper. Anticoagulants, heparin 10,000 units every 6 hours and warfarin, are given routinely unless contraindications exist. The SGOT and lactic dehydrogenase are monitored for at least three consecutive days after admission. Serial 12-lead electrocardiograms are recorded daily for several days. A portable (1.5 m) antero-posterior chest x-ray is taken on admission and daily thereafter. Patients are examined three times daily by a senior house officer and at least once by the registrar and a senior cardiologist. The diagnosis of pericarditis was made clinically; the presence of the pericardial friction rub was agreed in all cases by at least two examiners including a senior cardiologist. In some of the patients the friction rub was confirmed by a means of a phonocardiogram (Fig. 1).

FIG. 1 Pericardial friction rub in a patient with posterior infarction.

The pericarditis group was compared with a 'control' group of 50 consecutive patients (out of the total 195), without pericarditis. The sex ratio, mean age, admission delay, and extension of the infarction on the electrocardiogram were similar in the two groups. The incidence of complications, laboratory findings, and mortality between the two groups were analysed statistically. Since the numbers involved are small, the Yates' correction for the χ^2 values was used (Hill, 1967).

Results

Pericardial friction

A pericardial friction rub was heard in 22 (11.3%) out of the 195 patients; 16 were men and 6 were women; their ages ranged from 36 to 75 years. The time of appearance of the rub from the onset of

symptoms varied (Fig. 2). In 2 patients it reappeared soon after resuscitation from cardiac arrest. The time that the friction rub was audible varied from 1 to 6 days (Fig. 3). From these two characteristics, the time of appearance did not influence the mortality, but the duration of the rub appeared to influence the mortality, since in the 4 patients who died

FIG. 2 Time of appearance of the friction rub from the onset of symptoms.

FIG. 3 Duration of the friction rub in 20 patients of the series.

it persisted for more than 3 days (mean 3.6), while in the survivors it persisted for up to 2 days (mean 1.4). This difference is significant ($P < 0.01$). In almost all patients the rub was audible in the left lower part of the sternum; its intensity and quality varied from patient to patient; it was heard during systole and diastole in 14 patients, during systole in 3, and during diastole in 1 only, its timing being uncertain in the remaining 4 who had murmurs as well.

Symptoms

Symptoms, pleuritic praecordial pain, and dyspnoea, occurred in 6 patients (Table 2); only in 5 out of the 22 patients the temperature rose above 37.7°C ; it ranged from 37.7 to 41.5°C .

Laboratory findings

All patients had an infarction, class I according to the World Health Organization classification. All patients who died had an extensive anterior or anteroposterior infarction (Table 3) in contrast to the survivors who had small posterior infarctions,

TABLE 2 Features due to pericarditis *per se*

	No. observed
Pericardial rub	
Mean duration 1.4 dy	14
Mean duration 3.6 dy	6
Pericardial pain	6
Dyspnoea	2
Haemopericardium	2
Specific electrocardiographic abnormalities	4
Pericardial effusion	2

TABLE 3 Type of infarction and mortality

	No. of patients	Died
A. Extensive	12	2
Anterior	> 18	> 4
Anterior + posterior	6	2
B. Not extensive		
Posterior	4	0
Total	22	4

but the difference was not significant ($P > 0.70$). All serial electrocardiograms were examined in detail for changes due to the pericarditis *per se* and not to the infarction, but in 4 patients only the characteristic ST rise was found (Fig. 4). The SGOT ranged from 168 to 488 units/ml, mean 333 units/ml in 21 patients. In the survivors the SGOT was below 300 units/ml (Table 4), but the difference was not significant ($P > 0.30$). The erythrocyte sedimentation rate (ESR) ranged from 20 to 121 mm in the first hour in 8 patients, being normal in the remainder. Leucocytosis, 10,000 to 16,000/mm³, occurred in 11 patients. The chest x-ray was available in 18 out of the 22 patients of the series. It was normal in 5, showed pulmonary oedema in 8, pulmonary venous congestion in 2, pleural fluid in 1, and pericardial effusion in 2 (Fig. 5).

TABLE 4 SGOT and mortality in 21 patients with pericarditis

SGOT (units/ml)	No. of patients	Died
100-199	4	0
200-299	3	0
300-399	7	1
400-500	7	3
Total	21	4

Complications

Arrhythmias and heart block occurred in 18 (82%) out of the 22 patients. Most patients developed more than one arrhythmia. In none did the appearance of the friction rub coincide with any arrhythmias. Left ventricular failure was mild in 4 patients treated with frusemide only and severe in 14 (62.6%) treated with digoxin and frusemide. In none of these patients was the left ventricular failure caused by the coexistent pericarditis. Cardiogenic shock occurred in 6 (27%) patients. In 2 patients it became worse with the appearance of pericarditis, and these died two and a half days later.

Mortality and necropsy

Four patients (18%) died in hospital from cardiogenic shock and secondary ventricular fibrillation. Two died suddenly at home during the follow-up period. All 4 patients who died in hospital were

TABLE 5 Findings in 22 patients with, and in 50 without pericarditis

	With pericarditis	Without pericarditis	P value
Anticoagulants	13	35	> 0.50
Arrhythmias and heart block	18	34	< 0.01
Heart block (2nd + 3rd) degree	12	4	< 0.01
Left ventricular failure	14	10	< 0.01
Cardiogenic shock	6	1	< 0.01
Haemopericardium	2	0	> 0.10
Mean SGOT/ml	333 units	180 units	—
ESR > 20 mm/1st hr	8	14	< 0.03
Leucocytosis			
> 10,000/mm ³	11	21	> 0.50
Temperature > 37.7°C	5	15	> 0.30
Abnormal chest film	13	16	< 0.01
Mortality	4	6	> 0.80
Stay in coronary care unit (mean)	8 dy	3.8 dy	—
Total stay in hospital (mean)	46 dy	28 dy	—

FIG. 4 Posterior infarction complicated by heart block and pericarditis producing the characteristic ST rise.

FIG. 5 Cardiomegaly due to pericardial effusion; confirmed by necropsy.

found to have an extensive infarction at necropsy. One had a congenital small right coronary artery. In 2 patients considerable amounts of haemorrhagic pericardial fluid were found, 300 and 370 ml, respectively. These were the patients who had deteriorated with the appearance of pericarditis. They had received anticoagulants along with the other 11 out of the 22 patients.

Postmyocardial infarction syndrome

This syndrome was not diagnosed in any of the survivors, neither during their period in hospital, nor during the follow-up period, since in none of the patients did the pericardial friction rub recur or other signs and symptoms of the syndrome appear (Dressler, 1959).

Control group

The findings in the group without pericarditis are compared with those of the pericarditis group in Table 5. The latter had greater mean SGOT and included a greater number with arrhythmias and heart block, left ventricular failure, and cardiogenic shock, and these differences were significant ($P < 0.03$). No patient from the control group had haemopericardium at necropsy, but the difference was not significant ($P > 0.10$). There were significant differences in the number of patients with high ESR and abnormal chest films between the two groups ($P < 0.03$). There was no significant difference in the mortality between the two groups ($P > 0.80$).

The findings which were due to the pericarditis *per se* are summarized in Table 2.

Discussion

The 11.3 per cent incidence of pericarditis which complicated myocardial infarction in this series is within the range of 7 to 16 per cent reported by others (Table 1). It is logical to assume that it would probably be higher when looked for intentionally, since a pericardial friction rub is a transient physical sign of varying intensity and duration. Most of the patients developed pericarditis during the second, third, or fourth day from the onset of symptoms; it persisted longer in the very ill patients, from 3 to 6 days. Half of these died. The appearance of a friction rub soon after cardiopulmonary resuscitation (external cardiac massage, intracardiac injections, etc.), could be a simple coincidence or it could be due to trauma to the pericardium-epicardium. Pericarditis occurred in both anterior and posterior infarctions. All patients had a transmural infarction on the electrocardiogram, which probably had involved the subepicardial myocardium. The SGOT was greatly raised, above 400 units/ml in 5 out of 6 patients who died.

Regarding the symptoms, signs, and laboratory findings which were due to the pericarditis itself (Table 2), the rub should not be confused with the murmurs which commonly occur after acute myocardial infarction (Burch, DePasquale, and Phillips, 1968; Cheng, 1970). The pain should be differentiated from that due to pulmonary embolism, re-infarction, and the persisting pain of the infarction *per se*. Careful inspection of the electrocardiograms is necessary for the discovery of the pericarditic ST, T changes. Care should be taken to diagnose early a pericardial effusion and so prevent tamponade. This, however, is a difficult diagnosis since a plain portable chest film, especially in the presence of shock or left ventricular failure, is very unreliable. Cardiomegaly with sudden changes of the heart shadow which obscures the hila in the presence of clear lung fields is suggestive of pericardial effusion (Baron, 1971).

Anticoagulants were given to 60 per cent of the pericarditis group and to 70 per cent of the controls. It is unlikely, therefore, that the pericarditis was induced by anticoagulants. Haemopericardium occurred in two patients who became worse with the onset of the pericarditis and died in cardiogenic shock. This complication has been reported by Miller (1969), Fell *et al.* (1965), and Goldstein and Wolff (1951), but not by Thadani *et al.* (1971), who do not advise the discontinuation of anticoagulants in the presence of pericarditis.

The reported incidence of the postmyocardial infarction varies from less than 1 per cent to 3-4 per cent (Table 6); it was not diagnosed in this series, since none of the survivors developed a late pericarditis or the other characteristics and pulmonary manifestations of the syndrome. These, however, could be nonspecific and due rather to inflammation and left ventricular failure, common complications after myocardial infarction (Weiser *et al.*, 1962).

TABLE 6 Incidence of Dressler's syndrome

Series	No. of patients with acute myocardial infarction studied	Syndrome (%)
Dressler (1959)	—	3-4
Broch and Ofstad (1960)	500	1
Davidson, Oliver, and Robertson (1961)	500	< 1
Thadani <i>et al.</i> (1971)	779	3
Present series	195	Nil

More patients from the pericarditis group developed arrhythmias, heart block, left ventricular failure, and shock; a great number had high ESR and abnormal chest x-rays, and all these differences were significant. However, all these were due to the extensive infarctions and none was caused or became worse as a result of the pericarditis, except in the two patients who developed haemopericardium which probably contributed to their death. There was no significant difference in the mortality between the two groups.

It is concluded that pericarditis after acute myocardial infarction, though it is a sign of extensive myocardial damage, does not increase the mortality, and in that respect this study confirms the findings of Thadani *et al.* (1971). A friction rub which persists for more than 3 to 4 days should be considered as a bad prognostic sign. Anticoagulants might cause haemopericardium in very ill patients with extensive infarctions. It is suggested therefore that under those circumstances anticoagulants should be discontinued.

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