THE SYNDROME OF RECURRENT IDIOPATHIC PERICARDITIS*

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Although idiopathic or 'non-specific' pericarditis is a recognized condition, the fact that the condition tends to recur is often forgotten. Recognition of this tendency is particularly important in practice because of the resemblance to recurring ischaemic heart disease.

The present paper deals with the clinical features and electrocardiographic findings in 8 patients who suffered repeated attacks of pericarditis of uncertain cause. A brief review of possible aetiological factors is presented.

CLINICAL FEATURES

The initial attack usually presents abruptly with severe, often excruciating pain felt across the chest, in the neck, shoulders, arms or upper abdomen. It is described as sharp or 'sticking', but may be burning or pressing. It may be aggravated by breathing or movement, and its severity may be altered by sitting forward (which frequently increases it) or lying on one side. This positional effect is, of course, unlike coronary vascular disease. Also, the patient does not usually present with a cold sweat and a shocked appearance, though this may occur. Martin described 19 cases of non-specific pericarditis, of which 4 presented in a shocked state—pale, sweating and with marked hypotension. Pethidine or morphine may be required for relief of the pain.

Subsequently, usually within 24 hours, a pericardial friction rub is heard, typically loud and widespread over the precordium. There is no correlation between the duration of the chest pain and the presence of the friction rub; the rub may be heard 3 or 4 hours after the onset of pain or it may be delayed for a few days. The chest pain may recur daily or last several weeks, but in spite of this the patient's general condition remains comparatively good. Fever, leucocytosis and a raised sedimentation rate may occur. In some cases a mild upper respiratory tract infection or fever precedes the attack of chest pain by a few days, or there may be preceding or accompanying pneumonitis. Sinus tachycardia or bradycardia may be noted. Of more importance is the occurrence of arrhythmias, though none were noted in the present group. Atrial and ventricular premature beats and atrial fibrillation have been reported.

The patient may suffer no further attacks; or second and subsequent attacks follow within months or even years. These recurrences are often milder, but the pain may be as severe as in the first attack.

In the present series all 8 patients developed recurrences starting at intervals from 3 weeks to 6 months after the initial bout. In 2 patients the relapses seemed to be related to a period of activity, e.g. after a session of heavy gardening in one and after a long bicycle ride in the other. Unlike angina pectoris, the pain did not occur during the effort but some hours later.

Relevant details of the present group of 8 patients are presented in Table I.

It can be seen that pleurisy was associated with pericarditis in 3 patients in the initial attack, and one patient was known to have gout. The drugs used in treatment are shown, but no claims are made in this regard. None of the patients was treated with steroids. Each patient suffered more than one attack, the intervals between episodes varying from 3 weeks to 6 months. Between attacks the patients all felt well and no clinical abnormalities were detected. However, the electrocardiograms in some remained abnormal, as discussed below.

Laboratory Findings

In the present series, laboratory investigations have been totally unrewarding, as has been the search for clues to

*Based on a paper presented at the Joint Biennial Congress of the Southern Africa Cardiac Society, the Association of Physicians of South Africa and the Society for Endocrinology, Metabolism and Diabetes of Southern Africa, Stellenbosch, October 1966.
TABLE I. AN ANALYSIS OF 8 CASES OF RECURRENT IDIOPATHIC PERICARDITIS

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Race</th>
<th>Age (yrs.)</th>
<th>Attack No.</th>
<th>Years</th>
<th>Association</th>
<th>Lab. data</th>
<th>Drugs</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>WF</td>
<td>32</td>
<td>8</td>
<td>4</td>
<td>Mother</td>
<td>Max. SGOT</td>
<td>Tetracycline</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>WM</td>
<td>18</td>
<td>4</td>
<td>2½</td>
<td>Rheumatic fever</td>
<td>42</td>
<td>ESR++</td>
<td>Aspirin +</td>
</tr>
<tr>
<td>3</td>
<td>WM</td>
<td>51</td>
<td>5</td>
<td>2</td>
<td>Gout</td>
<td>30</td>
<td>Uric acid 7%</td>
<td>Butazolidine +</td>
</tr>
<tr>
<td>4</td>
<td>CM</td>
<td>40</td>
<td>3</td>
<td>3</td>
<td>Coryza</td>
<td>28</td>
<td>Anaemic</td>
<td>Tetracycline 0</td>
</tr>
<tr>
<td>5</td>
<td>WM</td>
<td>46</td>
<td>4</td>
<td>1½</td>
<td>Pleurisy</td>
<td>48</td>
<td>Butazolidine</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>WM</td>
<td>40</td>
<td>2</td>
<td>1</td>
<td>Pleurisy</td>
<td>40</td>
<td>ESR+</td>
<td>Butazolidine +</td>
</tr>
<tr>
<td>7</td>
<td>WF</td>
<td>25</td>
<td>3</td>
<td>1</td>
<td>Pleurisy</td>
<td>45</td>
<td>ESR+</td>
<td>Tetracycline 0</td>
</tr>
<tr>
<td>8</td>
<td>CF</td>
<td>30</td>
<td>4</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

possible aetiological factors. Mild leucocytosis with neutrophilia may be present. Anaemia is not a feature and its presence would suggest a more systemic disease. The erythrocyte sedimentation rate was elevated in 4 of the 8 patients, but it is a poor criterion of activity in the disease.

Serum enzyme studies may be useful. Agress and co-workers studied the enzyme changes in pericarditis in dogs after injection of abrasive powders and septic organisms. It was found that the degree of elevation of the SGOT level was related to the amount of sub-epicardial myocardial infarction produced, rather than the extent of pericardial inflammation. Studies in human cases by these authors have shown 8 cases out of 48 with raised SGOT, and then the highest level reported was 165 units. In general the absence of a rise in serial enzymes is characteristic of pericarditis, and the value of this observation is apparent. In severe pericarditis it is helpful to remember that values over 200 units are rarely if ever present without contributory disorder, whereas myocardial infarction of only moderate degree may easily exceed this level. The C-reactive protein test behaves erratically in pericarditis and the antistreptolysin-O titre (ASO) is rarely elevated. The fibrinogen polymerization test of Losner et al. is claimed to be more specific for the diagnosis of rheumatic fever and rheumatoid arthritis than the sedimentation rate, ASO or C-reactive protein. It is based on the observation that considerable amounts of fibrinogen are still detectable in the serum 2 hours after gross coagulation, when minimal amounts of heparin are added to whole blood. This has been found to be positive in non-specific pericarditis. It offers an additional advantage since it is not promptly suppressed by salicylates or by steroids, so it may be a useful guide to the duration of activity of the disease.

The Electrocardiogram

One of several patterns may occur, but the electrocardiogram may be unhelpful, with patterns varying from tracings to those indistinguishable from acute or chronic coronary artery disease. Most often the J point, the S-T segment and the T wave itself show alteration, and these changes are ascribed to underlying myocarditis which accounts for the 'current of ischaemia' and delay in sub-epicardial repolarization. As in myocardial ischaemia, S-T displacement is seen in the active stage, whereas T wave change may persist into the recovery stage and even for years after, if not permanently (Fig. 1).

The textbook case, then, presents displacement of the RST junction and S-T segment as the first change. This may occur in all the standard leads or just 1 lead or 2, depending on the electrical position of the heart. Standard lead III and aVF reflect the change in the vertically positioned heart, with the vector more parallel to the respective lead. Leads V2 - V4, overlying the cardiac muscle more directly, show S-T elevation in the majority of cases. It is said that the S-T segment retains the upward concave curvature of the normal S-T segment, but this is unreliable and it may exhibit a straight oblique course upward. The T waves may maintain their normal form initially. From this stage the ECG varies in pattern and duration of abnormality. The T wave may undergo a gradual change from low amplitude and notching to iso-electricity and ultimate inversion (Fig. 2). These T wave changes are generally seen in the same leads as the S-T segment alteration. However, only about a quarter of the cases show serial changes that are typical; in most, only abnormalities in the T wave amplitude or direction are seen. It is apparent then that a single electrocardiogram is not a firm basis for diagnosing the condition.

Schwab and Herrmann enumerated the following guides which favour ischaemia rather than pericarditis when interpreting a single electrocardiogram: (a) S-T displacement more than 4 mm.; (b) deep T wave inversion; (c) monophasic wave formed by ST-T wave fusion;
Hull states that there may be steroids that can be used. Although the clinical picture in both was identical with idiopathic cases from tuberculous pericarditis. This point was made by Schrire who described the autopsy findings that surgery was required. Although the virus was not recovered, there occurred a 4-fold rise in neutralizing antibody titre against the Coxsackie virus. Less than 25 cases of constriction associated with acute pericarditis have been reported in the literature.

Of interest is the rapidity with which constriction may develop in this disease, compared with the slow onset of constriction in tuberculous pericarditis.

TREATMENT
Complete bed rest is called for in acute pericarditis. Usually within 2 - 3 weeks the condition has subsided and gradual ambulation can be advised. The present group of patients were all kept in bed initially for at least this period of time, but one cannot say whether prolongation of rest would have prevented recurrences. Analgesics are necessary during the first few days, and morphine or pethidine is usually required for the severe chest pain. Antibiotics should only be used for concurrent and complicating bacterial infections. Cardiac glycosides and quinidine may be required for the appropriate arrhythmias. Steroids are often dramatic in effect, producing rapid subsidence of symptoms and fever. However, their use is only warranted if the patient's general condition and temperature fail to respond to rest and analgesics. Obviously all bacterial causes for such failure must be excluded before steroids can be used. In cases that recur the use of a steroid may be warranted, but since such treatment carries its own complications it is not recommended for routine use.

DISCUSSION
Although the syndrome of acute idiopathic pericarditis is sometimes called 'benign', it is apparent that some patients develop pericardial constriction, and others pericardial effusions with tamponade. These complications have not developed in any of the present group of 8 patients. It is of interest to consider possible aetiological factors.

Pericarditis after operations on the heart (post-commissurotomy syndrome) has been attributed to reactivation of the rheumatic process, though there is more pain than one usually sees in rheumatic pericarditis and the tendency to relapse is far greater. There have been reports of heart antibodies found in the sera of patients after pericardiotomy and myocardial infarction. Ehrenfeld et al. in Israel, and Van der Geld in Amsterdam, have detected specific heart antibodies in a proportion of patients. How-

(d) Pardee T wave; (e) reciprocal S-T segment displacement in standard leads. Hull states that there may be exceptions to (a) and (b), but (c), (d) and (e) are rarely misleading.

Fig. 2. T wave inversion (case 2).

In the present group of 8 patients the electrogram patterns were capricious. One case showed the textbook phases in the initial attack, but subsequent episodes did not alter a persistently notched T wave. Four cases showed low amplitude or inverted T waves as the only change in the initial or subsequent attacks, and the remaining case showed S-T elevation initially, but a normal pattern thereafter in spite of severe chest pain in the relapses.

DIFFERENTIAL DIAGNOSIS
Recurrent attacks of pericarditis occur in the following associations:
1. Idiopathic pericarditis syndrome
2. Infectious pericarditis (incl. tuberculous)
3. Postmyocardial and pericardial injury:
   (a) Postinfarction
   (b) Postcardiotomy
   (c) Postradiation
4. Connective tissue disorders:
   (a) Rheumatic fever
   (b) Systemic lupus erythematosus
   (c) Rheumatoid arthritis
   (d) Serum sickness
5. Metabolic:
   (a) Uraemia
   (b) Myxoedema
   (c) ? Gout

By exclusion one arrives at the diagnosis of idiopathic pericarditis, but one can make a positive diagnosis as well in the right setting. The distinction from tuberculous pericarditis presents the greatest difficulty.
ever, as with thyroid antibodies, so with heart antibodies, evidence has been presented that heart antibodies are the result and not the cause of the damage. Ehrenfeld’s suggestion is that cardiac damage of rheumatic, traumatic or ischaemic origin leads to antigenic change which in susceptible individuals is manifested by antibody production. Continued exposure to such altered antigen seems necessary for detectable antibody response. So if heart antibodies do occur in idiopathic pericarditis as found by Van der Geld in 2 cases, they are likely to have occurred post hoc and do not necessarily establish that the disease is an allergic manifestation.

Virus infection is suggested by the fever and the not infrequent accompaniment of pleurisy or pneumonitis. Several studies have failed to show a single virus to predominate as the cause of sporadic cases, though virus antibodies to a high titre have been demonstrated in individual cases. Usually the Coxsackie group B virus is implicated, but infectious mononucleosis and lymphogranuloma have also been associated. Hypersensitivity can cause pericarditis; cases have been reported after tetanus antitoxin injection and associated with drug allergy. Also eosinophilic infiltration of the pericardium has occurred with the pulmonary infiltration with eosinophilia syndrome. These possible aetiologies do not apply in most cases, of course.

One other disease state of interest as a possible cause of recurrent pericarditis is gout. Paulley et al. reported 3 cases of acute pericarditis with relapses which they attributed to gout. The evidence for this was the association of acute gout with the heart disease in 2 patients and a family history of gout in the third. They claimed that all 3 responded well to treatment with Butazolidin or Colchicine. They suggested that pericarditis in uraemia might be due to uric acid crystals rather than to urea. It is of interest that parakeets with renal lesions have been found to have gout, some with urate deposits on the pericardium. One patient of the present group had a history of gouty arthritis, and the uric acid level of the serum was raised. It would be foolhardy to label this gouty pericarditis, but the disease should be kept in mind as a possible aetiological factor although the evidence for this is far from proven.

SUMMARY

Six cases of recurrent pericarditis of non-specific aetiology are presented. The difficulty in distinguishing this syndrome from ischaemic heart disease is emphasized. A brief review is given of the electrocardiographic findings and possible aetiological factors.

I wish to thank Prof. V. Schrire of the Cardiac Clinic, Groote Schuur Hospital, for his advice.

REFERENCES


THE FAMILY PHYSICIAN OF THE FUTURE*

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The subject chosen for my remarks in this paper would suggest that, by some mystic means, I have been privileged to take a glimpse into the future and to forecast the position of general practice in the years ahead. Oh, that such were possible! Despite this inability to forecast with accuracy the ultimate role of the general practitioner in the provision of future medical care, it behoves us as present-day general practitioners to project our thinking beyond the present time.

In my country the College of General Practice of Canada is being invited, at an unprecedented rate, to accept an increasing responsibility to ensure the position of the family doctor as a vital force in Canadian medicine. We are faced with a number of disturbing questions:

- Are we as general practitioners prepared to accept this responsibility?
- How capable are we of predicting the future position of the general practitioner in a rapidly changing world society?
- What information do we have that would assist us in making these predictions?

What are some of the forces working for and against the inevitable changes that must take place?

I shall attempt to answer some of these questions. My limited knowledge of the general practice of medicine in South Africa, suggests to me that our problems are somewhat similar. It is possible that we may find common solutions.

In recent years, the many educational, social, political and economic changes affecting modern society, have had, and will continue to have, a profound influence on the practice of medicine. Nowhere are these changes more apparent than in general practice itself. In my view, we may best predict our future position by a review of these changes.

Without question, the greatest change that has taken place in Canadian medicine during the past 20 years, can be seen by examining our programmes of medical education. The accumulation of medical knowledge during this period has prompted many dramatic changes in our medical educational programmes, at both the undergraduate and graduate levels.

1. We have witnessed the creation of a number of new medical schools, and expansion of existing ones.

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