The changing face of rheumatism

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In recent years there has been a decline in the incidence of rheumatic fever and this has been accentuated since antibiotics became freely available for the treatment of throat infections. The rheumatic fever committee of the Royal College of Physicians reported in 1957 that the illness appeared less severe than in the past and that the immediate mortality was less. Despite this, it would be a mistake to think that acute rheumatism was not a commonly occurring condition. This presentation is a review of the incidence and some of the clinical and laboratory findings in acute rheumatism as observed in the M.R.C. Atheroma Unit of the Western Infirmary during the past seven years.

INCIDENCE AND DIAGNOSIS

Most studies of acute rheumatism have been designed to measure the therapeutic response to some form of treatment or to compare the incidence of carditis in comparable groups. Thus the criteria for inclusion as a case of acute rheumatism in such a trial have been designed to exclude the borderline or doubtful case. In this series, the criteria of Jones (1944) as modified for the cooperative clinical trial in 1955 and 1960 have been used (Table I). The criteria are in two groups, major and minor, and the diagnosis requires either one major and two minor,

or two major and one minor. Although such selection is essential if clinical material from different centres is to be compared, it is easy to overlook many cases of acute rheumatism and the true incidence may be obscured.

Table II shows the number of patients with acute rheumatism admitted to one hospital in Glasgow between 1954 and 1961. During this period 159

TABLE II

	PATIENTS WITH ACUTE RHEUMATISM ADMITTED TO WESTERN INFIRMARY 1954-61	
	Accepted	Rejected
M.R.C. Unit	139 + 20 ¹ = 159	140 (58)
Other wards	107 (unknown criteria)	

¹Twenty patients under 12 years of age

patients were accepted as cases of acute rheumatic fever, fulfilling the criteria. In the same period 140 patients were referred as rheumatic fever but rejected from the series. Analysis of the final diagnosis in this group of 140 patients is shown in Table III. Fifty-eight (38%) were considered to have acute rheumatism but had to be rejected from this series, usually because the arthralgia was confined to one joint or had settled before admission to hospital.

TABLE I

CRITERIA FOR DIAGNOSIS OF ACUTE RHEUMATISM¹

Major Criteria	Minor Criteria
1 Carditis (a) Development of an organic murmur (b) Change of heart size greater than 15% (c) Pericardial rub or effusion (d) Congestive cardiac failure in patients under 25 years in absence of other causes	1 Fever
2 Polyarthrititis Pain and either limited active movement or tenderness in two or more joints	2 E.S.R., not less than 15 mm. in 1 hr. (Westergren)
3 Chorea	3 Evidence of previous streptococcal infection (a) Culture with significant β haemolytic streptococci (b) Antistreptolysin O titre of at least 200 units/ml. (c) Reliable history of sore throat in four weeks before onset of rheumatic symptoms
4 Subcutaneous nodules	4 P.R. interval of 0.20 seconds or more
5 Erythema marginatum or annulare	5 Reliable history of rheumatic fever (as above) or evidence of pre-existing rheumatic heart disease

¹These are the criteria of Jones (1944) as amended for M.R.C. and American Heart Association combined trial (1955).

TABLE III
PATIENTS OVER 12 YEARS INVESTIGATED FOR
RHEUMATIC FEVER DURING SAME PERIOD BUT NOT
SATISFYING THE CRITERIA

	No. of Patients	
Acute rheumatism		
With chronic rheumatic heart disease	4	29
With previous rheumatic fever	5	
With erythema nodosum	1	
Others	19	
Subacute rheumatism		
With chronic rheumatic heart disease	5	29
With previous rheumatic fever	6	
Others	18	
Rheumatoid arthritis	11	
Gonococcal arthritis	5	
Reiter's syndrome	1	
Systemic lupus erythematosus	3	
Erythema nodosum	7	
Henoch-Schönlein syndrome	2	
Serum sickness	1	
Osteomyelitis	4	
Septic arthritis	2	
Arthralgia or arthritis of uncertain cause	15	
Osteoarthritis	6	
Gout	2	
Respiratory infection with arthralgia	16	
Acute pericarditis	2	
Miscellaneous	5	
Total	140	

Observation of this group suggests that many attacks of acute rheumatism cause so little arthralgia that a few aspirin tablets settle the discomfort and the patient never sees a physician.

The onset of rheumatoid arthritis may present acutely like rheumatic fever, but this group often have a positive Rose-Waaler test which confirms the correct diagnosis. Similarly the specific serological test for gonococcal arthritis and the L.E. cell test for disseminated lupus erythematosus help to establish the correct diagnosis. Erythema nodosum with a low antistreptolysin O titre should suggest sarcoidosis.

RHEUMATIC CARDITIS

When we consider the incidence of carditis in the 139 adolescents or young adults accepted for this series,

TABLE IV
REPORTED INCIDENCE OF CARDITIS IN
ACUTE RHEUMATISM

Series	Incidence (%)
Jones and Bland (1952)	65
Co-operative clinical trial (1955)	51
Lieber and Holoubek (1956)	56
U.S. Army (Bates, 1958)	56
British Army (Slater and Rosenbaum, 1959)	16 (E.C.G. 31%)
R.A.F. (Malpas and Landon, 1960)	52
Bywaters and Thomas (1961)	46
Begg, Kerr, and Knowles (1962)	18 (E.C.G. 59%)

25 (18%) had carditis and there were no deaths. This incidence continues the decline in carditis reported in recently published papers on acute rheumatism (Table IV) although it is not so obvious if E.C.G. changes alone are accepted as a manifestation of carditis.

The histological reports on auricular appendages biopsied at operation for mitral valvotomy might suggest that more stress is put on even minor changes in the electrocardiogram. Catto, Smith, and Taylor (1952) examined 25 auricular appendages removed at operation for mitral valvotomy, the age range of the patients being 24 to 46 years, and found that 15 (60%) of the auricular appendages contained Aschoff bodies. Seven of the 15 patients concerned gave no history of rheumatic fever and in the remainder the most recent attack had been 10 years previously. None of these patients had any evidence of acute rheumatism before operation, and in all the Westergren E.S.R. was less than 10 mm. in the first hour. This type of study has been extended by McKeown (1953), who in 17 necropsies on 92 cases of mitral stenosis has shown that where Aschoff bodies are present in the auricular appendages they were also found in the left ventricle. This raises the vexed question of the significance of these observations. Most pathologists would accept the Aschoff body as a manifestation of active rheumatic carditis but Enticknap (1953), for example, feels that this is not proven.

Further, there is the interesting syndrome reported by Abrahams and Brigden (1961) from Nigeria where rheumatic fever is rare, of mitral incompetence, pulmonary hypertension, and endomyocardial fibrosis. At necropsy in some of these cases Aschoff bodies were observed in the myocardium. Meantime, the pathologist should not put too much stress on the clinicians' inability to detect active carditis in such cases.

The recent observations of Kaplan and Meyers (1962) of an immunological reaction between group A streptococcal cells and the myofibrils of human heart tissue in a young patient dying of acute rheumatic fever is consistent with an autoimmune carditis and raises the question of the part played by the Aschoff body in such a process.

ARTHRALGIA

Polyarthritides was the presenting symptom in 92 of the cases in this series and in six cases was associated with erythema nodosum.

LABORATORY INVESTIGATIONS

The erythrocyte sedimentation rate is the most sensitive measure of the degree of rheumatic activity

available. In this series the E.S.R. (Westergren) had fallen to less than 10 mm. in the first hour in 88% of patients within eight weeks of treatment. Patients appear to be discharged from hospital with a lower E.S.R. than in the pre-antibiotic era, but in some instances this is due to more accurate diagnosis.

In this series acute rheumatic fever confirmed the seasonal peak in January and appears to be related to the incidence of streptococcal sore throats. We cultured group A, β haemolytic streptococci in 33% of 139 cases, which is high considering that most patients had received penicillin before admission to hospital. The antistreptolysin O titre was above 200 units in 70% of patients and in all cases associated with erythema nodosum was 1,250 units or more. The antistreptolysin O titre often takes six months or longer to fall below 200 units and this delay may be related to high dosage of penicillin (Carter, Bywaters, and Thomas, 1962). The titre can be raised in a case of mitral stenosis, presenting first with auricular fibrillation of recent onset and suggesting that active rheumatic carditis is present.

TREATMENT AND PROPHYLAXIS

With two exceptions, all patients in this series were treated with salicylate and penicillin. The response of the arthralgia to aspirin in moderate dosage (blood level 30 mg. per 100 ml.) is swift and impressive, but the decline in the development of permanent carditis in the patient with acute rheumatism is due to the negligible recurrence rate today. This is the most significant change in rheumatic fever and would appear to be due to the correct use of penicillin. We have maintained all our cases on oral penicillin V, 125 mg. b.d., for three to five years, and have had no recurrence in any patient who remained on this régime.

The long-term use of penicillin in patients with rheumatic fever with established valvular disease of the heart raises special problems, as the *Streptococcus viridans* about their teeth will be penicillin-resistant. Any dental manipulation without a change in antibiotic exposes these patients to the risk of subacute bacterial endocarditis. We have seen several instances of this sequence of events in Glasgow and it has been fully reported by Garrod and Waterworth (1962). These patients must be warned to have a change of antibiotic to cover any period of dental treatment.

CONCLUSION

The patient is fortunate if the onset of rheumatic carditis is associated with arthralgia and he sees his physician before valvular disease is established. The problem today is how to prevent the development of latent carditis which first appears before the physician has established valvular disease of the heart.

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