Original Articles

RHEUMATISM IN INDIA

By E. H. VERE HODGE, M.D. (Cantab.) M.R.C.P. (Lond.)

LIEUTENANT-COLONEL, I.M.S.

Civil Surgeon, Jalpaiguri

CURRENT medical literature on diseases in the tropics or hot countries is, on the whole, silent on the subject of rheumatic infection though there seems to be ample evidence that the disease, as far as India is concerned, is by no means rare and is a potent, though not necessarily the most prominent, factor in the production of heart disease. There seems to be some diversity of opinion, though most practitioners consulted by the writer state that they have seen unmistakable cases of rheumatic fever, some of them in considerable numbers.

Clark (1930), after an exhaustive study of available records, suggests that the disease is practically non-existent in the tropics. He confines his observations to the strict geographical tropics and though he quotes the evidence of Calvert and Sutherland to the effect that they have seen rheumatic fever, chorea and nodules in Indian children, though they regard such conditions as rare, he disqualifies this evidence on the grounds of geographical limits. He further quotes Rogers as stating that out of 4,800 post-mortem examinations performed in Calcutta, only one showed rheumatic carditis. This observation is strikingly at variance with the clinical experience of most practitioners.

Other observers have of late years raised the question and concluded that rheumatic fever is responsible for a definite proportion of the heart disease which is unquestionably common. Basu (1925), investigating 446 cases of heart disease in Calcutta, attributed 21 to rheumatism. Hughes and Yusuf (1930) report 31 cases of heart disease admitted to the Mayo Hospital, Lahore, in seven months, of which 23 were mitral disease or mitral combined with aortic. They observed several cases of arthritis combined with carditis, responding, but less readily than European cases, to salicylates, and record the opinion that in the Punjab, rheumatic fever is an important cause of mitral stenosis. They have not seen chorea or nodules.

Stott (1930) from Lucknow states that acute rheumatic arthritis does attack young Indian adults and gives it as his opinion that unrecognized subacute infection is the probable cause of mitral disease in young Indians.

In Bengal the incidence of rheumatic fever is generally accepted. Enquiry from responsible practitioners in mofussil towns elicits the fact that the disease is rare, though they treat cases from time to time. The dispensary returns are misleading; the figures given under the heading of rheumatic fever treated in the out-patient departments are formidable and it would seem that the term is loosely applied to all forms of joint disease. There is, however, evidence that a certain proportion are genuine rheumatic fever with carditis. It must be remembered that in many of these institutions there is no accommodation for in-patients, so the treatment is out-door or none at all.

The writer has seen a number of unmistakable cases of rheumatic fever, in which the final result to the heart was often disastrous, in Chittagong, Chinsura and Darjeeling. Cases in the latter district need not be discounted on the grounds that a hill-station does not come into the discussion. Admittedly the climate is peculiarly provocative of manifestations in the infected child, but practically all cases gave a history of previous attacks in the plains or a family history that some close relation had suffered in the plains.

In India, the difficulties in assessing the prevalence of rheumatic infection are manifest. Many fully authenticated cases of rheumatic fever are recorded in different provinces, but there is no record of infections other than of the more obvious type.

On the analogy of European experience, the juvenile type should be more common. In this type the joint pain and swelling is often slight or fleeting, pyrexia is not marked and the full significance of the case is apt to be realized only when some degree of carditis is established. Further, carditis may be of such a minor degree that it passes unnoticed until, with the passage of years, some definite disability of the heart is established and by this time, the other rheumatic manifestations, often far from prominent, have been forgotten. Until such time as children's clinics are established in India and the poorer classes, among whom rheumatic infection is likely to occur, have realized the importance and value of medical advice in what are apparently minor degrees of ill-health, so long shall we be unaware of the prevalence of rheumatic infection in this country and so long will many cases come under treatment only when the heart is failing.

That heart disease is exceedingly prevalent all are agreed. Lieut.-Col. Chopra informs me that towards the end of the War he made the final examination of between 50,000 and 60,000 recruits and formed the opinion that at least 1 per cent. were suffering from some form of heart disease. Experience in hospitals confirms this view; patients with heart disease fill a considerable proportion of the beds. Again, those who have had occasion to examine candidates for government service cannot fail to be struck with the incidence of mitral disease,

often with a history of rheumatism. Arising from this point is the consideration as to how far we are to regard what we may call a typical mitral stenosis case as evidence of rheumatic infection. Remembering that mitral stenosis is a progressive lesion and that it may be years before it reaches a degree sufficient to cause real disability and that infantile rheumatism is a disease of indefinite manifestations, it is not surprising, particularly when dealing with the less intelligent of our patients, that we can get no history of a causative disease. Again, opinion is divided as to whether mitral stenosis is essentially of rheumatic origin. Horder (1926) in an analysis of 100 cases shows that only 64 gave a history of rheumatism. He writes 'Until we know more of the specific cause of rheumatism, we do not seem likely to get nearer to the solution of the problem of this special form of mitral sclerosis'. Brewer (1927), after an observation of 22,276 children of whom 952 were found to have heart disease, came to the conclusion that 'it would appear that, exclusive of congenital heart disease, there is one cause and one cause only, of permanent heart disease in children, namely, rheumatism, assuming, as seems probable, that chorea, post-scarlatinal rheumatism and acute nephritis in childhood are rheumatic affections'.

At the same time, it must not be forgotten what a large part in the diseases of this country is played by streptococcal invasions of varying virulence. It is this consideration which makes one cautious in diagnosing arthritis as rheumatic even in the acute stage. This particularly applies to the case of young women of the child-bearing age, who, though still within the age of liability to rheumatic fever, are also liable to acute or sub-acute arthritis of the rheumatoid type, often complicated by the development of apical murmurs and progressive anæmia. It is generally considered that bacterial endocarditis is of necessity fatal, but Libman (1925), in a series of 800 cases of bacterial endocarditis, found a small number of complete recoveries. He finds that the disease may occur in recurrent form and is of opinion that some cases of chronic valvular disease are due to this type of infection.

It may be that some cases of valve and muscle damage are of streptococcal origin and the prevalence of septic skin infections lends colour to this view.

It would seem wiser, therefore, to limit our deductions to children or adolescents in whom it would appear that the ætiological factor of mitral disease is predominantly rheumatic.

The possibility of the ætiological influence of other infective diseases calls for little comment. There is no evidence that the common infectious diseases lead to types of carditis resembling that caused by rheumatism, and the influence of scarlet fever, even if it can act independently of rheumatism, cannot be regarded as an important factor on account of its rarity. In two recent outbreaks in this country, one noted by Savage (1928) and a second seen by the writer, there is no record of the onset of scarlatinal carditis.

In the series of cases presented below, one or two are typical, the remainder are presented in detail on account of some point of particular interest. It is worthy of comment that, of the ten cases, no less than seven gave a definite family history of rheumatism and several a history that brothers or sisters had previously suffered. The list includes both Europeans and Indians, as the occurrence of rheumatism in the former, provided that they have not been out of India, is of equal importance in a discussion on the incidence of a disease which is in all probability governed more by climatic influence and the existence of infection, than by any racial differences of diathesis.

Among the cases described are the following unusual conditions :—

(1) Chorea in an Indian child, secondary to and following within three weeks on an attack of rheumatic fever. This is the only Indian patient in whom the writer has seen chorea and rheumatic nodules and, after consulting many observers, he has been unable to hear of a case. The child after developing endocarditis and pericarditis, finally died of partial heart-block. Poynton (1927) describes a case of complete heart-block with a ventricular beat of 36 at the onset of acute rheumatism.

(2) Rheumatic lung in the course of a second attack of rheumatic fever. The writer made this diagnosis on clinical findings. He is aware that this condition is disputed. On the one hand Wynne (1929), referring to Naish's findings of distinctive lesions in the lungs, considers that some cases, hitherto attributed to the pressure of an enlarged heart or pericardial effusion, are of the nature of rheumatic consolidation which, in its onset, induces very little alteration of the temperature or pulse rate, and frequently clears up without materially influencing the outlook. On the other hand, Findlay (1930) emphasizes the difficulty of distinguishing between consolidation and pleural effusion in children, and maintains that pure pulmonary lesions are caused by intercurrent infection.

(3) Concurrent acute rheumatic arthritis and chorea with anginal attacks. In this connection the following quotation from Hutchison (1931) is of interest:— 'Rheumatic affections of the joints in the course of the disease (chorea) are fortunately rare, for the condition of a child who had both acutely inflamed joints and at the same time uncontrollable movements of the limbs, would indeed be far from enviable'. This was perhaps the most distressing case the writer has ever seen, as, in addition to the pains caused by the constant movement of the inflamed joints, the recurrent attacks of angina caused the child extreme agony.

(4) Infantile rheumatism with the insidious onset of carditis. This example is of importance in that it illustrates the type of case which we suspect to be responsible for more carditis than the more obvious arthritic type.

(5) Hemi-chorea with evanescent rheumatic signs in a European boy.

(6) Rheumatic mitral stenosis in a man of fifty, whose heart was further damaged by epidemic dropsy. This leads to the consideration as to whether epidemic dropsy with the accompanying separation of the muscle cells by œdema and occasional intra-muscular hæmorrhages may not result in permanent damage to the myocardium. The patient was subject to periodic paralytic seizures which may have been due to embolus,

but, in view of their evanescent character, were more probably of the nature of cerebral claudication.

(7) Chorea benefited by calcium lactate. That calcium is indicated in most cases of febrile nervous disturbance in children, as for instance in the low delirium of typhoid, has long been the conviction of the writer and the legitimacy of such treatment in chorea was confirmed by Warner (1930), who found that the blood and cerebro-spinal calcium content was lowered in chorea.

Cases 9 and 10 appeared at first to be of somewhat doubtful authenticity, but the writer finally came to the conclusion that they were of rheumatic origin.

Case 1.—D. S., Hindu female, aged 8. The child suffered from rheumatic fever three weeks ago. After a short intermission the temperature again rose. Shortly after, when seen in consultation, the child was found to be seriously ill with marked anæmia, enlarged heart and mitral regurgitation, and pre-choreic fidgety movements were noted. There was a history of recurrent sore throats and an elder sister was stated to have died of heart disease after rheumatic fever. After two weeks of irregular progress the child developed chorea. There was no improvement in the anæmia. Next day the patient complained of a persistent dry cough which was explained after twenty-four hours by the discovery of a pericardial rub. Two rheumatic nodules developed on the forehead.

From this point it was evident that the infection was gaining ground, there was an increase in the degree of anæmia, prostration was more marked, the fever persisted, choreic movements became more violent and the child complained of pain over the heart and in the epigastrium. The pulse increased in rapidity to 145. After ten days in this condition the child died. The practitioner who was present at the time stated that he was called about an hour before death and found the child in acute distress and that the pulse rate had slowed to 60.

Case 2.—S. B. G., Hindu boy, aged 7. The boy was first brought to the consulting room for general ill-health. He presented the typical picture of the rheumatic child. He was thin and markedly anæmic, and showed, in all his movements and in his posture even when sitting, an extreme languor. There appeared to be mitral incompetence. Enquiry elicited a history of an attack of rheumatic fever four months before with fever and joint inflammation lasting one week and it was evident that the rheumatic infection was still at work. Three weeks later, though at rest and taking salicylates, he developed a second attack of acute rheumatism with arthritis.

About a fortnight after the onset of this attack the writer saw him again and found him still the subject of progressive rheumatic invasion. Joint pains and swellings were slight, but there was persistent irregular pyrexia and marked anæmia. Auscultation revealed mitral incompetence and a pericardial rub. A fortnight later, at the next consultation, the condition was much the same save that there was a well-defined area of what appeared to be consolidation at the left back not extending to the base. There had been no alteration of the pulse, temperature or general condition during the development of this and the respiration was not affected. The patient made a slow but remarkable recovery. Some two months after, the only sign in the chest was that of a moderate degree of mitral incompetence.

Case 3.—N. M., European girl, aged 10. She had never been out of India. The case was first seen by the writer on the seventh day of the disease. The general condition, with heavily furred tongue, headache and abdominal distension, suggested typhoid, but the pulse was unduly rapid, there was epigastric pain and tenderness, and examination of the heart revealed a well-marked systolic bruit. Temperature 103°F. Further enquiry elicited the fact that the child had suffered from rheumatic fever one year before in Calcutta. Three days later the temperature was 101.8°F. with a pulse of 120, there was pain and swelling of the left knee, pain and tenderness of the muscles of the neck and a papular rash over the sacrum, buttocks and left knee. On the eleventh day there was severe pain and stiffness in the throat with injection of the fauces.

At this point there was slow improvement with an intermission of two days, but a relapse set in abruptly with pain in the shoulder, severe headache, fever and a disproportionate pulse rate of 136. From the fourth day of the relapse, began a series of heart attacks lasting from a few minutes to an hour, with pain of such severity as to necessitate morphia. The pulse rate rose to 156, the lips and extremities became blue. The joint pains now extended to the shoulders, wrists and fingers while the abdominal pain, which had been a prominent feature all through, persisted. At this time signs of chorea became manifest in twitching movements of the fingers. The next development was an alteration of the mental outlook, the child became irritable and excitable. Following this was the development of inco-ordination of the muscles of speech and swallowing, leading to great difficulty in feeding. From now onwards to the end of the illness, thirty days after the relapse, the course was one of repeated heart attacks, totalling altogether nine, severe abdominal pain, choreic movements of intense violence, causing distress on account of the painful inflamed joints. The child became absolutely dumb and exhibited one very unusual feature, that is, choreiform movements of the muscles of the left eye-ball. As regards the condition of the heart, with the persistent rapidity of the pulse it was not easy to estimate what was going on, there was evident dilatation, with mitral and possibly aortic involvement. A fortnight before death pericarditis developed. Salicylates in high doses failed to relieve the joint pains and chloral pushed to the point of inducing cyanosis had only a temporary effect in quietening the movements, which were of such extreme violence that there was danger of the patient throwing herself out of bed. During the last week of the illness the anginal attacks were of daily occurrence. The patient died in a state of cyanosis and unconsciousness.

Case 4.—Hindu boy, aged 6½. The existence of heart disease was first noted during an attack of dysentery some three months previously. When first seen by the writer the heart was enlarged, the pulse persistently rapid and there was to be heard a loud systolic and diastolic murmur at the apex. Fever, 99°F.—100°F. had been recorded for fourteen days.

Enquiry failed to elicit evidence of anything but slight aching in one wrist joint, but there was a history of family incidence in that a maternal uncle contracted mitral stenosis at the age of 14. On first examination the child appeared to threaten chorea in that he was aimlessly fidgety, but after rest in bed this passed off. The fever persisted for some six weeks together with an accelerated pulse, but when last reported on, eight months after the first discovery of the heart disease, the child was in good health with a pulse rate of 90, which may be taken as within the normal for that age, but the systolic diastolic murmur persisted at the apex and the heart was enlarged.

Case 5.—G. B., English boy, aged 8. The patient was admitted in the first instance to a school infirmary for a septic throat. After fourteen days the throat had cleared, but the patient complained of pain in both knees and weakness of the right arm. There was no previous personal history of importance but the elder brother had suffered from rheumatism and chorea. Shortly afterwards the patient developed well-marked choreic movements in the left arm and leg, the trunk was scarcely affected, and the face and right limbs not at all. The paralytic element was well in evidence and the affected limbs were lacking in power. The movements of the arm on the bed resembled nothing so much as those of a freshly-landed fish. The case ran a somewhat tedious course, the movements continuing for over two months with occasional pain and

tenderness in the knee joints and low fever. The movements, except for occasional twitching of the trunk, remained strictly confined to the left side. The severity of the infection was demonstrated by the development of anæmia and mitral incompetence. The boy was moved down from the hills at the onset of the cold weather and the ultimate developments are unknown.

Case 6.—P. Ch., Hindu male, aged 47. He states that at the age of 12 he suffered from rheumatic fever and was considered to have made a good recovery, but the heart was further injured by epidemic dropsy. He considers that during adolescence he further strained his heart by over-indulgence in athletics. The mother suffered from rheumatic fever at an early age.

The heart is enlarged, mitral stenosis present, but there is a fair degree of functional competence. In June 1928, while bicycling, the patient suddenly developed paralysis of the right arm and leg with complete loss of speech. Judging from the complete and rapid recovery from this attack and the course of the subsequent attacks, it would seem that the condition was one of cerebral claudication. After twenty-four hours the power of speech returned, but recovery in the arm and leg was slower and it was fifteen days before full power returned. Even after this, any prolonged exercise in writing induced mental exhaustion. In March 1929 there was a recurrence, this time affecting the right leg only, of seven days duration. The third attack in August 1930 took the form of loss of memory.

The fourth attack in April 1931 was manifest paralysis of the left leg which persisted in some degree for two months but ultimately passed off. At this time advanced cardiac failure was evident; this persisted until the patient's death in August. During the last month of life there was a mental change culminating in definite insanity with hallucinations.

Case 7.—A. H., European girl, aged 10. The patient had suffered from a mild attack of chorea some six months previously, preceded by an erythema all over the body. The mother and one cousin are known to have suffered from rheumatic fever. The child had suffered from measles and pneumonia at the age of three and the tonsils had been removed at the age of seven. The present attack is severe and began while the child was at a boarding school. The movements were violent and speech was lost. In addition, there was definite psychic alteration in the direction of irritability. Anæmia was progressive, but throughout the illness there was no evidence of cardiac involvement. Progress was slow under the routine treatment with chloral, bromides and arsenic and there was but slight improvement after a month's treatment. At this time, calcium lactate, grs. 9 a day for four days in each week, was ordered. From this point there appeared an abrupt amelioration in symptoms so that after two months the child was practically well.

Case 8.—Mahommedan boy, aged 12. Five months previously the boy was attacked suddenly with fever, and swelling and pain in the wrists, elbows and knees. The patient complained of a sore throat. The attending physician diagnosed rheumatic fever. The duration of the disease was 21 days and the boy made an apparently good recovery.

The present condition is as follows:—The boy is slightly anæmic, there is a soft blowing murmur at the apex with a reduplicated second sound. The pulse when he is lying is 88, standing it is 82, and on very slight exertion 92. It is suspected that the infection is still at work.

Case 9.—Hindu female, aged 23. The patient has had three children. Generalized pain all over the body followed by pain flitting from joint to joint. The throat was painful and swallowing became difficult, but there was no sign other than redness of the fauces. There was irregular fever rising to 104°F. After five days of treatment with sodium salicylate, grs. 40 per day, the symptoms abated but recurred and persisted for a month in spite of sodium salicylate administered in doses of grs. 120 per day. During this time the patient developed a progressive anæmia and an apical murmur of increasing intensity. Ultimately she made an apparently complete recovery save that the apical systolic murmur persists.

Case 10.—Hindu male, aged 22. Admitted to Chinsura Hospital for acute febrile arthritis of 10 days duration. The right ankle and both knees are inflamed and tender with effusion.

There is a history of multiple boils all over the body for the last two months but no pyorrhœa or tonsillitis.

Heart.—First sound, soft blowing murmur at the apex.

Under salicylate treatment the joints subsided in five days and the patient consequently refused to stay in hospital. Fifteen days later he was again admitted to hospital in the same condition as before. The relapse had started on the day following that on which he had left hospital. After three days the temperature which had not been above 100°F. fell to normal and the pulse which had shown no marked acceleration fell to 60 and subsequently rose to 64.

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ALEPOL IN THE TREATMENT OF LEPROSY

By AMBA DATT BHANDARI, M.S.M.F.

Medical Officer-in-Charge, Out-patient Department, District Hospital, Gorakhpur

Of the various preparations of hydnocarpus oil used in the treatment of leprosy, Alepol—a sodium salt of a selected fraction of the fatty acids of hydnocarpus oil—is one. Alepol can be given intramuscularly or subcutaneously. For the last year in our leprosy clinic I have been trying subcutaneous injections of Alepol with fairly good results. Cases are treated as out-door patients twice weekly on Wednesdays and Sundays. The following table shows the symptoms of some of the cases who are attending as out-door patients and are being given Alepol injections. I have only cited in the following table those cases who