TRANSIENT COMPLETE HEART BLOCK DURING ACUTE RHEUMATIC FEVER : A CASE REPORT

ONG PANG KOK M. RAMANATHAN

SUMMARY

We report a case of transient complete heart block with Stokes-Adams attack due to acute rheumatic fever. The patient was a 12-year old boy whose illness satisfied the criteria of acute rheumatic fever. A temporary transvenous cardiac pacer was necessary to tide him over the complete heart block. The rarity of this complication of acute rheumatic fever is highlighted.

INTRODUCTION

Complete heart block following acute rheumatic fever has rarely been reported. This is a report of acute rheumatic fever with documented complete heart block occurring in a 12-year old boy who recovered completely.

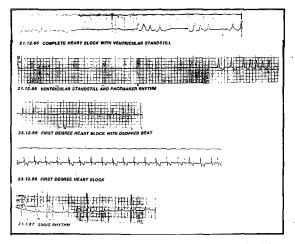
CASE PRESENTATION:

A 12-year old schoolboy was admitted to Malacca General Hospital in Dec. 1986 following three days of fever without chills or rigors. On the day of admission, he had recurrent episodes of loss of consciousness. He had no history of sore throat, joint pains or seizures and had been vaccinated against diphtheria.

On examination, he was pale, mildly febrile with blood pressure of 100/70 mm Hg and pulse rate 44/minute, regular and good volume. The jugular venous pulse was not elevated. He had

ONG PANG KOK, MBBS (Mal), MRCP(UK) M RAMANATHAN, MBBS (Mysore), MRCP(UK) Consultant Medical Department Malacca General Hospital 75400 Malacca. cardiomegaly and a loud ejection systolic murmur was heard at the apex. His throat was not injected. No other abnormalities were detected.

The ECG showed complete heart block with periods of ventricular standstill (Figure 1). The chest radiograph showed cardiomegaly with clear lung fields.



His haemoglobin was 9.6g% and the total white blood count was 17.2×10^9 /L with a differential count of 87% polymorphs and 13% lymphocytes. The erythrocyte sedimentation rate was 123 mm/ hour on admission. 81 mm/hour after 8 days, 13 mm/hour at three weeks and 15 mm/hour, at six weeks. His throat swab was negative for any pathogenic organism. The cardiac and liver enzymes, urea and electrolytes were normal. The anti-streptolysin-O titre was positive at 800 i.u./ml. While in the ward, the patient suddenly lost consciousness with blood pressure 40/20 mmHg and heart rate of 30 per minute. A temporary transvenous cardiac pacer was inserted under ECG control. He spontaneously recovered, with reversion of the complete heart block to sinus rhythm, through second degree heart block and first degree heart block respectively. The pacemaker was turned off without any complications at two days and the pacer wire was removed two days later.

The patient's fever subsided promptly with soluble aspirin. He was also given a course of penicillin. With complete rest in bed, he attained sinus rhythm and normalization of his ESR within six weeks. He was discharged with monthly injection of benzathine penicillin for rheumatic fever prophylaxis. On follow up one month after discharge, the patient remains well with normal sinus rhythm and normal erythrocyte sedimentation rate.

DISCUSSION

Our patient presented acutely with recurrent episodes of sudden loss of consciousness associated with bradycardia and hypotension due to complete heart block with long periods of ventricular standstill. This necessitated a temporary cardiac pacer to tide him over the acute stage of his illness.

The provisional diagnosis then was an acute viral infection with myocarditis and secondary complete heart block. Implication of a viral cause for the myocarditis depends on either isolation of the virus or a fourfold increase in the serum antibody titre. Unfortunately, these were not done in this patient. However, this patient's history and symptoms were not suggestive of a viral infection. Furthermore, the cardiac enzymes, namely, creatinine kinase, lactic dehydrogenase and aspartate transaminase were not elevated this excluded a myocarditis. His clinical features and subsequent investigations in fact, satisfied the criteria for acute rheumatic fever, i.e. definite evidence of carditis, with radiological and clinical cardiomegaly and apical systolic murmur, fever associated with elevated ESR and raised streptococcal antibodies (ASOT 1 : 800).

It is of great importance to differentiate between these two diagnoses, bearing in mind that an acute attack of rheumatic fever predisposes a patient to further involvement of the heart during subsequent attack and penicillin prophylaxis is required for life in some instances.¹

A recent report discussed an outbreak of acute rheumatic fever involving 74 patients, over an 18 month period.² The diagnosis was based on clinical manifestations meeting the modified Jones criteria. Of the 74 patients, 24 presented with single manifestation, 17 of which were carditis confirmed by auscultation. Overall incidence of carditis occurring alone and in combination with other manifestations was 67%. Electrocardiogram was done in 70 patients and 14 were found to have PR prolongation and no other conduction defects were described.

Complete heart block during acute rheumatic fever has been reported but it is rare and it is doubtful whether it is ever permanent. In first and second degree heart blocks, his bundle studies reveal that the site of reversible damage in conduction is proximal to the bundle of His.¹ Enhanced vagal tone, myocardial or pericardial irritation or endomyocardial ischaemia or inflammation could all have contributed to the development of the complete heart block. In a review of the literature, a case of acute rheumatic carditis associated with complete heart block and Wolff-Parkinson-White syndrome was reported to occur in a 13-year old boy who also satisfied the Duckett-Jones criteria for the acute rheumatic fever.³

ACKNOWLEDGEMENT

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