Pseudo second degree atrioventricular block with bradycardia

Successful treatment with quinidine

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Pseudo second degree atrioventricular block resulting from blocked His premature beats was successfully treated with quinidine. The diagnosis was proved by His bundle electrogram which showed both blocked and conducted His premature beats. The blocked His prematures produced second degree atrioventricular block by making the atrioventricular junction refractory. Quinidine abolished both conducted and blocked His extrasystoles. There has been no recurrence of arrhythmia during a one-year follow-up.

Langendorf and Mehlmán (1947) suggested that non-conducted junctional premature beats might produce second degree atrioventricular block by blocking the sinus impulse following the premature beat. They suggested that the block was caused by junctional extrasystoles which were concealed because of both anterograde and retrograde block. Rosen, Rahimtoola, and Gunnar (1970) showed, with His Bundle recordings, second degree atrioventricular block resulting from blocked His premature beats. Since there was no intrinsic abnormality of the atrioventricular conduction, they called the resulting block, 'pseudo-block'.

We report here a patient with bradycardia resulting from pseudo second degree atrioventricular block caused by blocked His premature beats: he was successfully treated with quinidine.

Case report

A 54-year-old man reported to the hospital complaining of tiredness and weakness. There was a history of heavy alcoholic intake, but the patient had discontinued drinking a few months before. There was no history of palpitation or dizziness. Physical examination revealed an irregular pulse, with a basic rate of about 70 beats a minute but with periodic slowing to almost 30 beats a minute. The blood pressure was 120/80 mmHg (16.0/10.6 kPa). There was no evidence of heart failure. The heart size was normal, no gallop rhythm was heard, and

there were no murmurs. The remainder of the physical examination was normal. The electrocardiogram showed supraventricular premature beats with aberration and episodes of apparent Mobitz type II block (Fig. 1a). Apart from the arrhythmia, the electrocardiogram was normal. The patient was admitted for further evaluation and treatment of the arrhythmia. Initially he was given tincture of belladonna, 1 ml, four times a day. The premature beats as well as periods of apparent second degree atrioventricular block were uninfluenced by this therapy. His bundle recordings were then made to clarify the nature of the arrhythmia.

Electrophysiological studies

The His bundle electrogram was recorded simultaneously with lead II of the surface electrocardiogram at a paper speed of 100 mm/s. Several hundred cardiac cycles were analysed. The majority of the beats recorded were sinus beats, with normal AH and HV intervals. The QRS complex of these sinus beats was normal in duration. Premature beats occurred at an average frequency of 3 to 5 per minute. These beats could be separated into two types according to the behaviour of the QRS complex. In the first type, there was a minor change in QRS morphology but without significant increase in its duration. The second type showed wide QRS complexes resembling ventricular ectopic beats. The simultaneous His electrogram, however,

FIG. 1 (a) Electrocardiogram before quinidine therapy. The top strip of the cardiogram shows premature beats with varying morphology of the QRS complexes. The second strip shows 2:1 atrioventricular block, followed by 3 conducted sinus beats with constant PR interval. The succeeding P wave is blocked producing type II second degree atrioventricular block. The His bundle electrogram (HBE) was recorded simultaneously with surface electrocardiogram at a paper speed of 100 mm/s. The recordings are labelled b, c, and d. (b) Apparent type II atrioventricular block is seen in the surface electrocardiogram. HBE shows H' with anterograde and retrograde block preceding the blocked atrial (A) deflection. (c) and (d) HBE clearly shows that the premature beats seen in the surface electrocardiogram are His extrasystoles conducted with varying degree of aberration since H' deflection precedes the V deflection of the premature beats. The A deflection representing the atrial activation by the sinus impulse follows the premature beat in (c). It is superimposed on H' of the premature beat in (d).

showed a consistent presence of a His spike preceding the V deflection in both types of premature beats localizing their origin to the His bundle (Fig. 1c, d). These two types of premature beats were His extrasystoles, one type with slight and the other with more aberrant ventricular conduction. Periods of second degree atrioventricular block with non-conducted normal P waves occurred at frequency of about 10 a minute. The surface electrocardiogram showed that the block was type II in nature and occurred without a change in PR interval of the preceding sinus beats. At no time was more than one beat blocked in succession. His bundle recordings during these periods showed spikes preceding the blocked A deflection (Fig. 1b). The latter corresponded to the non-conducted normal P waves. As the patient had conducted His premature beats throughout the recording, it is reasonable to conclude that these spikes represented His premature beats with both retrograde and anterograde block. These spikes will be designated as H'. H'-A interval varied between 94 and 304 ms with an average interval of 212 ms. Every blocked sinus beat was preceded by H'. The behaviour of the His premature beat was determined primarily by the interval between the H spike of the preceding sinus beat and the H'. The His extrasystoles with short H-H' intervals were blocked, while those with long H-H' intervals were conducted with slight aberration. The H-H' interval of the premature beats with greater degree of aberration fell between these 2 groups. These three types of His premature beats could clearly be separated into three groups, determined by the length of the H-H' interval (Fig. 2).

Hospital course

Quinidine sulphate, 200 mg, four times a day was given in an attempt to suppress the premature beats. Within 24 hours, the premature beats as well as periods of pseudo atrioventricular block were completely abolished and did not recur during a seven-day period of observation with continuous electrocardiographic monitoring (Fig. 3). Quinidine was then stopped, and within 12 hours the premature beats as well as the pseudo second degree atrioventricular block reappeared. The arrhythmia continued for 36 hours until quinidine therapy was restarted, at which time the arrhythmia again disappeared. During a one-year follow-up under quinidine therapy, there has been no recurrence of the arrhythmia.

Discussion

This case is an interesting example of successful

FIG 2 The effect of timing of H' on conduction to the ventricles. Abscissa: H-H' intervals; Ordinate: preceding H-H intervals. Note that His premature beats with short H-H' interval are blocked while those with long H-H' interval are conducted with slight aberration. His prematures with obvious aberration fall in between these two groups. The preceding H-H intervals do not appear to influence the behaviour of the His extrasystoles.

treatment of apparent second degree atrioventricular block with quinidine, a drug which normally would be considered contraindicated in atrioventricular block. It is important to recognize the 'pseudo' nature of the block. The diagnosis should be suspected when a patient presents with apparent type II atrioventricular block with narrow QRS complexes but who also shows junctional premature beats elsewhere in the electrocardiogram. The usual

FIG 3 The electrocardiogram recorded after quinidine therapy was started. The premature beats as well as periods of second degree atrioventricular block are no longer present.

settings for atrioventricular block, such as myocardial infarction and digitalis toxicity, may not be present. The presence of His premature beats showing both retrograde and anterograde block should be shown by His electrogram before embarking on a trial of quinidine.

In our patient, the reason for occurrence of His premature beats was not clear. They did not appear to be re-entrant beats, as the coupling interval varied. The premature beats occurred irregularly and did not appear to arise from a parasystolic focus. Blocked premature beats may arise from any part of the His-Purkinje system but the presence of conducted premature beats preceded by a His spike localized their origin to the His bundle. The interval between the H deflection of the preceding sinus beat and the H' determined the conduction of the premature beats to the ventricles. With shorter H-H' intervals the premature beats showed both retrograde and anterograde block, and consistently blocked the following sinus impulse by concealed

conduction within the atrioventricular junction. We did not encounter any sinus beats conducted to the ventricles with prolonged AH interval following the blocked H', as described by Rosen *et al.* (1970). The unique success of quinidine in abolishing the significant bradyarrhythmia in this patient eliminated the need for implanting a permanent pacemaker.

References

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