CONCEALED PREEXCITATION AND WOLFF-PARKINSON-WHITE PATTERN POSSIBLY RELATED TO MAHAIM FIBERS

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One case of concealed preexcitation and four cases of Wolff-Parkinson-White (WPW) pattern (including one questionable case) in which the electrocardiographic (ECG) manifestations were highly suggestive of the conduction through Mahaim fibers or septal bypass are described. ECGs of Case 1 did not demonstrate preexcitation but showed a peculiar arrhythmia consisting mainly of the sequence of trigeminy and quadrigeminy due to the occurrence of "supraventricular premature beat" which is preceded by inverted P wave and often showed aberrant conduction of right bundle branch block (RBBB) pattern and occasional anterograde conduction failure. Atrial reciprocal rhythm (ARR) related to the presence of concealed bypass which is essentially capable only of retrograde conduction, that is, a concealed preexcitation, was the most likely mechanism. Intermittent conduction through the left-sided Mahaim fibers was the most likely mechanism of aberrant conduction of ventricular reciprocal beat (VRB). Case 2 was type A WPW pattern associated with short PR interval. After daily intravenous drip infusion of digitalis (Cedilanid), which has been generally considered to depress the atrioventricular (A-V) conduction and accelerate the anomalous bypass conduction, PR interval gradually prolonged to 0.16 sec, while type A WPW pattern with decreased size of initial delta wave remained. This finding was considered to favor the mechanism of either combination of A-V nodal bypass and left-sided Mahaim fibers or left-sided Mahaim fibers alone rather than left-sided (type A) Kent bundle. Case 3 was believed to be type A WPW pattern due to anomalous conduction through left-sided Mahaim fibers. However, concomitant RBBB could not be wholly excluded in Cases 2 and 3 (particularly, in Case 2). Regarding Case 4, it was impossible to label as type A WPW

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pattern without the aid of intracardiac recordings and pacing study because such a QRS pattern seems to have been labelled as atypical RBBB. Case 5 was considered to be atypical case associated with normal PR interval. WPW pattern of A-V junctional escape beats was indicative of the conduction through Mahaim fibers or septal bypass.

CLINICAL ECG studies of ARR and/or reciprocating rhythm in association with the WPW syndrome have not infrequently been described.¹⁻¹⁰ Recently, extrastimulus study with the use of intracardiac recordings has developed the observation of a newer entity of concealed WPW syndrome in which the anomalous A-V bypass is capable only of retrograde conduction.¹¹⁻¹⁷ However, possible examples of spontaneous ARR and/or reciprocating rhythm due to the existence of concealed retrograde bypass would appear to have extremely rarely been reported¹⁸ as compared to examples of ARR in which underlying mechanism is either functional longitudinal dissociation or dual pathways in the A-V junction. Further, ECG studies of WPW pattern or syndrome in which

conduction through Mahaim fibers or septal bypass is conceivable would appear to have extremely rarely been reported.¹⁹⁻²¹ This clinical ECG study includes one case of ARR due to concealed preexcitation in which RBBB type aberrant conduction of VRB was attributed to intermittent conduction through left-sided Mahaim fibers, three cases of WPW pattern possibly related to Mahaim fibers and one questionable case.

Report of Case 1

12 lead ECG (Fig. 1) and a 24 hour continuous monitoring ECG (Figs. 2 and 3) were obtained from a 57 year old obese black female who was admitted because of the sudden onset of lightheadedness with some vertigo associated

Fig. 1. 12 lead ECG of Case 1. Basic rhythm is sinus rhythm with rate of 96/min. PR interval and QRS duration are normal and there is no preexcitation pattern. PR interval and ventricular cycle length are shown by 1/100 sec.

Fig.2. Two continuous strips (record A) in the top and five continuous strips (record B) in the bottom are some portions of a 24 hour continuous monitoring record. In the ladder diagram (A=the atria, A-V=A-V junction, V=the ventricle), R2 is a sinus beat initiating atrial reciprocal rhythm and R1 is a beat preceding it. P' is atrial reciprocal beat and R* designates ventricular reciprocal beat. Dashed lines illustrate the retrograde conduction to the atria through the concealed retrograde bypass. P' is invariably preceded by shortening of ventricular cycle, while the interval between the end of QRS(J) and P' is constant (approximately 200 msec). PR, P'-R* and R2-P' interval and ventricular cycle length are shown by 1/100 sec.

with epigastric distress and nausea. She had had episodes of postural dizziness before hospitalization but never this severe. 12 lead ECGs from 27-2-75 demonstrated normal sinus rhythm with "supraventricular premature beats" associated with occasional anterograde conduction failure. Although q waves can be observed in leads III and aVF in Fig. 1, examination of serial ECGs and past history were not indicative of inferior myocardial infarction and not suggestive of negative delta wave of WPW pattern. Fig. 4 indicates

the appearance of an isolated or a paired ventricular extrasystole(s) simulating left bundle branch block (LBBB) pattern during a treadmill exercise test monitoring (modified V5 lead), while the appearance of premature beat with inverted P wave (P') was not affected.

Report of Case 2

ECGs were obtained from a 76 year old emaciated Japanese male who was admitted

because of diabetes mellitus and asthenia. Fig. 5A shows 12 lead ECG of type A WPW pattern associated with short PR interval. He had no experience of paroxysmal supraventricular tachycardia. Following brief episode of reactive hypoglycemia, which was induced by oral hypoglycemic agent but was relieved promptly, the patient developed sinus tachycardia. Since elderly diabetic is especially sensitive to digitalis preparations whether long or short acting, parenteral Cedilanid 0.4 mg/day by intravenous drip infusion was initiated and the clinical condition improved. Fig. 6 depicts gradual prolongation of PR interval prior to the development of atrial fibrillation and discontinuation of digitalis. Atrial premature beats which occurred in different occasions before digitalization and one week after the discontinuation of Cedilanid) demonstrate varying degrees of preexcitation (Fig. 5B).

Report of Case 3 and Case 4

ECGs of 66 year old Japanese male who died because of pulmonary tuberculosis with advanced stage and severe malnutrition (Case 3) are shown in Fig. 7A. This case was believed to be WPW pattern due to Mahaim fibers. PR interval was 0.13–0.15 sec. ECG record during digitalization by using oral Lanatoside C 0.75 mg/day (from 3/13/78 to 3/27/78) was not available. ECGs of 61 year old Japanese female (Case 4) are shown in Fig. 7B for comparison with Cases 2 and 3. PR interval was 0.14 sec. There was no episode of paroxysmal supraventricular tachycardia in these cases.

Report of Case 5

Fig. 8 and Fig. 9 were selected from the ECGs of 61 year old Japanese female who was admitted because of congestive heart failure due to hypertension. Although PR interval was normal (0.16–0.20 sec) in this case, sinus beat always showed WPW pattern. Upright delta wave was pronounced in leads II, III, and aVF, and less pronounced in I, and V2–V6. In lead V1, delta wave was usually negative and occasionally positive. Fig. 9 depicts the spontaneous occurrence of A-V junctional escape beat (marked by star) simulating sinus beat. There was no previous episode of paroxysmal supraventricular tachycardia.

Interpretation of ECGs and Discussion

In Fig. 1, the interval between the P wave of sinus beat preceding the inverted P wave (P') to the sinus P wave following it is almost equal to twice the preceding sinus cycle in aVR—aVF (full compensatory pause) or less than twice the preceding sinus cycle in I—III, V1—V3 and V4—V6 (not full compensatory pause). The return cycle following P' exceeds the basic sinus cycle length. The premature beat preceded by P' terminates trigeminal or quadrigeminal sequence through Fig. 1 to Fig. 3 and very often shows aberrant conduction of RBBB pattern and occasional anterograde conduction failure (Figs. 2 and 3). They may suggest the following possibilities, for example, low atrial premature systoles with occasional anterograde block, A-V junctional premature systoles with persistent retrograde atrial capture and intermittent anterograde con-

Fig.4. A treadmill exercise test monitoring record (modified V5 lead) revealed the fixed QRS(J)-P' interval (around 200 msec) during sinus tachycardia (rate: 140–150/min). In the ladder diagram to illustrate the sequence of conduction in the intra-ventricular reentry loop (R L), three sites of block are postulated: proximal, intermediate, and distal (concealment). A=the atria, A-V=A-V junction, V=the ventricle, F=fusion beat. Ventricular cycle length is shown by 1/100 sec. Lower three strips are continuous.

duction failure, atrial or A-V junctional parasystole, and, finally, ARR.

In Fig. 2, the occurrence of P' after sinus beat is constant through the record and the interval between the end of QRS (J) of preceding sinus beat and P' is constant (approximately 200 msec) irrespective of the ventricular cycle length change through the record (Figs. 2 and 3). Further, sinus acceleration induced by a treadmill exercise test (140–150/min) did not affect the persistent occurrence of premature beat and QRS (J)–P' interval. This finding and the absence of atrial fusion beats negate the possibility of either atrial

parasystole or A-V junctional parasystole. Besides the “fixed” QRS (J)–P' interval irrespective of ventricular cycle length change, the absence of two consecutive “supraventricular premature beat” with P' and the absence of “supraventricular premature beat” after another premature beat with anterograde block would favor an argument against (low) atrial premature beat.¹⁸ The interval between P' and the following QRS complex of premature beat (P'–R* interval) often exceeds 0.12 sec and is less than or does not exceed A-V conduction time of immediately preceding sinus beat irrespective of the morphological change of

Fig.5. 12 lead ECG in the basal state of Case 2 shows type A WPW pattern with short PR interval (A). Atrial premature beats (marked by star) in different occasions (before digitalization and one week after the discontinuation of Cedilanid) demonstrate varying degrees of preexcitation (B).

Fig.6. After hypoglycemic attack (17/XII), intravenous drip infusion of Cedilanid (0.4 mg/day) was initiated (18/XII) and the PR interval gradually prolonged to 0.16 sec. Occasional atrial premature beat had PR interval of 0.22 sec. Note the morphological changes of WPW pattern (A). Cedilanid was discontinued after the development of atrial fibrillation (22/XII) (B).

Fig.7. Additional two cases (Case 3 in Fig.7A and Case 4 Fig.7B) are shown for comparison with Case 2.

Fig.8. Selected 12 lead ECGs of Case 5 demonstrate sinus beat with WPW pattern and normal PR interval. Note that QRS complex varies in lead V1 in different occasions.

Fig.9. ECG of Case 5. Basic rhythm seems to be sinus arrhythmia. PR interval of sinus beat measures 0.20 sec and QRS duration is 0.08 sec in lead II. The finding of leads III & aVR suggests the mechanism of second degree S-A block of Wenckebach type. WPW pattern of escape beat marked by star in lead aVF indicates anomalous conduction through the Mahaim fibers. Atrial activities marked by P2 and P4 demonstrate "ventriculophasic trapping" under the influence of preceding ventricular premature beat (VPB). Each of other atrial activities marked by P1, P3-4, and P6-8 appears to be showing "accrochage" and linked to escape beat. Long rhythm record obtained later demonstrated the validity of such an explanation (unpublished observation). Further, it was concluded that concealed discharge of the focus possibly situated in or near the origin of anomalous bundle due to retrograde conduction of the VPBs permitted the escape of the beat marked by star and the beat preceded by P6 (cycle: approximately 52/min, 1.16 sec).

premature beat. Furthermore, the occurrence of P' is invariably preceded by a shortening of ventricular cycle in Fig. 2. This finding in combination with the absence of atrial fusion beat is not compatible with general features of A-V junctional premature beat. Thus, ARR may be the most likely explanation but the absence of sufficient prolongation of preceding PR interval does not favor the mechanism of functional longitudinal dissociation in the A-V junction (or A-V nodal reentry). ARR related to the existence of concealed retrograde bypass, that is, a concealed pre-excitation, is the most likely mechanism of the fixed QRS (J)-P' relationship and the premature QRS complex (marked by R*) represents VRB.

The possible role of latent left-sided (type A)

Kent bundle with anterograde unidirectional block as a retrograde bypass or the role of retrograde bypass emanating from the left ventricle in reciprocating tachycardia of a concealed WPW syndrome has been suggested.²² Further, the negativity of P wave in lead I during reciprocating tachycardia in WPW syndrome has been considered to be indicative of the left-sided bypass.²³ Furthermore, one recent study in which most of anomalous bypasses of WPW syndrome were found to be located in left free wall of the heart has demonstrated that the retrograde conduction time of a concealed WPW syndrome often approximates to around 200 msec.²⁴ Nevertheless, it is difficult to be certain whether the region to which the bypass reaches is the left atrium, or whether it is near the coronary sinus from the

finding of the negativity of P' in leads II, III and precordial leads.

The aberrant conduction with "fixed" RBBB pattern which demonstrates constant P'—R* interval (approximately 130 msec) in this particular case seems to be not related to the change of coupling interval or preceding ventricular cycle. In Fig. 2, P'—R* interval of VRB with RBBB type aberrant conduction is fixed and not prolonged as compared to the varying P'—R* interval of VRB with normal (or slightly aberrant) configuration. The latter reveals maximum prolongation to approximately 160 msec (top strip of the record B in Fig. 2).

Thus, the absence of prolongation of P'—R* interval regarding the VRB with RBBB type aberration appears to strongly suggest the conduction through the left-sided Mahaim fibers²⁵⁻²⁸ rather than functional RBBB.

On the other hand, the pauses after atrial reciprocal beat (P') are either fully or not fully compensatory through the record. The pause which is not fully compensatory may represent the outcome of discharge of sinus node and resetting of the sinus cycle by the atrial reciprocal beating. Full compensatory pause might reflect either undisturbed sinus impulse formation due to S—A entrance block, or emergence of the next sinus impulse permitted by the delay of the retrograde impulse in reaching the sinus node²⁹ and, the emergence of sinus impulse could not affect the atria. However, it is conceivable that some degree of depression of sinus node automaticity or an addition of other factors could slightly lengthen the pause.

In Fig. 4, an occurrence of an isolated and a paired ventricular extrasystole(s) with fixed coupling interval to the preceding VRB upon shortening of ventricular cycle represents the reversed rule of bigeminy.²⁹ If we assume the mechanism of intraventricular reentry as the genesis of these extrasystoles, it may be possible to suppose as follows. The ladder diagram to illustrate the sequence of conduction in the intraventricular reentry loop (R L) follows the illustration of Levy et al.³⁰ The shortened ventricular refractoriness resulting from shortened ventricular cycle may render the conduction speed through the reentry circuit appropriate (presumably relatively slower) enough to permit intraventricular reentry, producing reentrant beat coupled to preceding VRB. Development of ventricular extrasystole with LBBB pattern would imply an achievement of reentry through the

circuit to the right bundle branch system. A paired extrasystoles may reflect two consecutive circus movements (manifest intraventricular reentry), while absence of extrasystole after VRB would imply an abortive reentry or a concealment.

In Case 2, daily intravenous administration of 0.4 mg of Cedilanid by drip infusion for four days caused to gradually prolong the short PR interval to normal level (0.16 sec) prior to the development of atrial fibrillation, while WPW pattern with decreased size of initial delta wave and taller later deflection remained (Fig. 6A). Occasional atrial premature beat had PR interval of 0.22 sec. The varying configuration of WPW pattern during atrial fibrillation (Fig. 6B) contrasts with the observation that, in general, established RBBB in the presence of atrial fibrillation would not tend to show morphological alteration of QRS complex upon ventricular cycle length change.

Although the effects of digitalis on various anomalous bypasses have not yet been established with certainty, the drug has been generally considered to depress the normal A-V conduction and often accelerate the conduction through the bypass.³¹⁻³⁴ The gradual prolongation of PR interval after the use of digitalis negates total A-V bypass of left-sided (or type A) Kent bundle. Thus, we postulate the mechanism of either combination of A-V nodal bypass (presumably atrio-nodal bypass)²⁷ and left-sided Mahaim fibers or left-sided Mahaim fibers alone. Digitalization could induce conduction disturbance (block) through the atrio-nodal bypass in addition to A-V nodal conduction delay. Rather prominent initial delta wave of WPW pattern with rsR' configuration in V1 and RsR' configuration in V2 in association with short PR interval in the basal state (Fig. 5A) may indicate the preexcitation occurring predominantly through the left-sided nodo-ventricular bundle.²⁸ Further, concomitant RBBB can not be wholly excluded.³⁵⁻³⁶ On the other hand, WPW pattern with decreased initial delta wave in association with normal and constant PR interval after the administration of digitalis (Fig. 6A) may indicate different degree of preexcitation possibly due to impaired conduction through both the A-V node and nodo-ventricular bundle. It can be observed that atrial premature beat with prolonged PR interval in Fig. 5B resembles the WPW pattern (with decreased size of initial delta wave) which appeared after digitalization to some extent. This may also reflect different degree of preexci-

tation due to A-V nodal conduction delay and impaired conduction through nodo-ventricular bundle. Alternative possibility would postulate the presence of left-sided nodo-ventricular bundle which has longer effective refractory period than the A-V node and resultant anomalous conduction through left-sided His-ventricular bundle.²⁷ This explanation related to the possibility that multiple Mahaim fibers (i.e., nodo-ventricular and His-ventricular bundle tracts) might be present in this case. However, it is quite difficult to distinguish conduction through multiple functioning pathways from varying degree of preexcitation through one bypass.

Case 3 and Case 4 simulate Case 2, except that PR interval is always normal (Fig. 7A and Fig. 7B). In Case 3, normal PR interval negates preexcitation through total A-V bypass of Kent bundle. Thus, we believe that preexcitation is occurring through the left-sided Mahaim fibers. It seems that concomitant RBBB is unlikely, although such a possibility cannot be wholly excluded. Case 4 cannot be labelled as type A case with certainty because this QRS configuration appears to have been considered to be atypical RBBB.

On the other hand, surface ECG studies of WPW pattern or syndrome in which the occurrence of A-V junctional escape beat with WPW pattern indicates the anomalous conduction through the Mahaim fibers would appear to have extremely rarely been described.¹⁹⁻²¹ Fig. 9 of Case 5 depicts such an instance. The A-V junctional escape beats in aVL & aVF have less pronounced delta wave and taller terminal portion as compared to the QRS of sinus beat.

In conclusion, in Case 1, VRB in ARR of concealed preexcitation demonstrated an intermittent aberrant conduction with fixed RBBB pattern. It was not related to the change of coupling interval or preceding ventricular cycle. Such a peculiar phenomenon seems to be best accounted for by intermittent conduction through the left-sided Mahaim fibers. The observation of Case 2 suggests that the combination of A-V nodal bypass (atrio-nodal or atrio-His) and left-sided Mahaim fibers instead of total A-V bypass of left-sided (or type A) Kent bundle is necessary to be considered as a genesis of type A WPW pattern or syndrome with short PR interval in certain instances. This is the first preliminary study to suggest that administration of digitalis preparations would be helpful in distinguishing them.

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