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Paroxysmal bundle branch block a case of two to one right bundle branch block followed by intermit-tent right bundle branch block*

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Abstract

A case of paroxysmal bundle branch block, two to one right bundle branch block followed by intermittent right bundle branch block, which is associated with chronic cor pulmonale secondary to active, far advanced pulmonary tuberculosis, is presented. The incidence and mechanism of the paroxysmal bundle branch block have been discussed.

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PAROXYSMAL BUNDLE BRANCH BLOCK
A CASE OF TWO TO ONE RIGHT BUNDLE BRANCH BLOCK FOLLOWED BY INTERMITTENT RIGHT BUNDLE BRANCH BLOCK

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In cardiology clinic the cases of intraventricular block is not infrequently met with, especially with those of incomplete and those of complete right bundle branch block. As is well known, there is a great variety in the degree of the block, e. g., some of them may be very slight and the functional damage is transient, some others may be of intermittent and some cases may show the syndromes of alternating right and left bundle branch block[1,2], but two to one bundle branch block is very rare. Especially, in this country, we have no reported case of such a bundle branch block.

Recently, we have experienced a case which showed first complete right bundle branch block, followed by two to one right bundle branch block, in which complete right bundle branch block complex alternates incomplete right bundle branch block complex each together, and later changing to intermittent right bundle branch block, in which there are occurrences of intermittence between complete right bundle branch block and incomplete one, finally returning to complete right bundle branch block again. In this paper precise clinical findings of the patient is reported.

CASE REPORT

S. Y., a farmer, aged fifty-one years.

Clinical Diagnosis — 1. Pulmonary tuberculosis, far advanced, active, status, right thoracoplasty, 2. cor pulmonale, and (3) right bundle branch block.

Family History — His father died of pneumonia, mother of carcinoma of the stomach, eldest brother of carcinoma of the liver and elder brother of intestinal tuberculosis.

Past History — He experienced anal fistula, which was operated in August 1940. Past history about his heart disease was non-contributory except for occasional palpitation on exertion, which persisted for one and a half days sometimes.

History of Present Illness — Patient was first diagnosed as to have pulmonary tuberculosis in August 1939 and had a bedrest for over one year. He was hospitalized in because of pulmonary tuberculosis with hemoptysis from October 1942 to 1943. He had worked as a farmer until October 1956, when his pulmonary tuberculosis exacerbated and

he was given SM and PAS. In May 1958 he developed right exudative pleurisy, given SM, PAS and INH and in April 1960 he was admitted to Kokuritsu Okayama Ryoyosho (National Okayama Sanatorium) under the diagnosis of pulmonary tuberculosis, far advanced, active, with right old pleurisy.

Physical Examination — The patient was a slightly reduced male. The conjunctivae were slightly anemic. Radial pulsation was nothing significant. Heart was normal in size with regular rhythm, without any murmurs. Right lung had a dullness and diminished breath sounds associated with moist rales.

Chest X-ray showed two giant cavities with many small cavities in the right upper lung area and old adhesive pleurisy.

Laboratory Studies—Blood: R. B. C. 4,120,000 per cumm., Hb 14 gm. per dl., W. B. C. 8,000 per cumm., neutrophils 56 per cent, eosinophils 2 per cent, monocytes 2 per cent, lymphocytes 40 per cent. Urinalysis and stool examination were within normal limits. Red cell sedimentation rate was 65 mm. per hour. Vital capacity was 1,450 ml. and per cent vital capacity was 61 per cent. Sputum examination showed many acid fast bacilli on smear.

Hospital Course — In June and July 1960 he had right thoracoplasty, resected portion of the lst to 7th ribs, followed by chemotherapy. In January 1962 he was noticed a culous caries of the remained right 4th rib with cold abscess in the anterior area of right tuberthoracic wall which was curetted (Fig. 1.).

Fig. 1. A roentgenogram of the chest taken in January 1962, Status, thoracoplasty, right.

On physical examination in April 1962, the patient was remarkably emaciated. The color of the face was somewhat pale. No temperature elevation. Pulse rates about 80 per minute. Respiration 20 per minute, deep and rapid on walking. No cyanosis on finger nails, nor clubbing fingers. There was slight jugular venous dilatation. Thorax of status, post thoracoplasty, right. An old operative scar in the right lower anterior chest. Lungs showed dullness and diminished breath sounds in the right lower area. Prominent moist rales with occassional rhonchi in the right upper and middle areas. There was prolonged expiration on the left lung. The edge of the dullness of the heart was markedly displaced to right. Rhythm was regular and grade III systolic murmurs were audible at the apex. P_2 was stronger than A_2. There was no palpable liver, nor pitting ankle edema. Blood pressure was 120 systolic, and 80 diastolic. Vital capacity was 1,100 ml. and per cent vital capacity 31 per cent. Red cell sedimentation rate was 50 mm. per hour. R. B. C. was 4,500,000 per cmm., Hb 15.3 gm., W. B. C. 7,500 per cmm., neutrophils 76 per cent, lymphocytes 22 per cent, eosinophils 2 per cent, Urinalysis and stool examination were within normal limits. Many A. F. B. were seen on smear in sputum examination.

Electrocardiograms:

May 28, 1960 — (This is the first Ecg in the sanatorium.) Regular sinus rhythm in 75. Semi-vertical heart position. P-R interval 0.18 sec.. P wave 0.09 sec. in width and 2 mm. in height in lead II. Same P waves were seen in III and aVF. There were no other remarkable pathological findings.

June 3, 1961 — Regular sinus rhythm in 83. Vertical heart position. Marked clockwise rotation. Tall slender P with 2.7 mm. in height and 0.1 sec. in width in II, III and aVF. An rSr's' complex with QRS interval of 0.12 sec. in width in V_1. Same QRS interval in I, III, aVR, aVL and V_2. A wide S wave in I and rSr' in aVR. The above findings were suggestive of pulmonary P and right bundle branch block.

December 7, 1961 — Regular sinus rhythm in 79. P wave of 3 mm. in height and 0.12 sec. in width in II, rSr's' complex with 0.14 sec. of QRS interval in V_1 and V. A. T. of 0.06 sec.. Otherwise no remarkable changes.

April 13, 1962 (Fig. 2 and 3.) — There were some variations of QRS complex in each lead and these variations were revealed more distinctly in the right precordial leads. There were three kinds of QRS complexes in V_{3R} and V_{4R}, i. e., rsR' with 0.13 sec. (V. A. T. 0.06 sec.) in width, 6.5 mm. in height with inverted T, rsR's' with 0.13 sec. (V. A. T. 0.08 sec.) in width, 12 mm. in height, depressed ST and inverted T and 0.08 sec. (V. A. T. 0.05 sec.) in width, 5.5 mm. in height with elevated ST and inverted T in V_{3R}. Two kinds of QRS complexes were noted in V_{5R} and V_{6R}, i. e., qR with 0.12 sec. (V. A. T. 0.07 sec.) in width and 8 mm. in height with inverted T and qR with 0.08 sec. (V. A. T. 0.03 sec.) and 3 mm. with inverted T in V_{5R}. The complete right bundle branch block complex alternates with the incomplete right bundle branch block each together and the tracings seen in V_{3R} and V_{4R} are assumed a transition process from complete bundle branch block to alter nating (two to one) bundle branch block which is a phenomenon between complete and incomplete bundle branch block.

On April 13, 1962, the patient developed temperature elevation followed by cough, sputum expectoration and shortness of breath. Physical examination revealed jugular venous dilatation, rapid heart rate, increasing moist rales with rhonchi. Venous pressure in the ante-cubital vein showed 17 cm H_2O. He was given digosin 0.5 mg intravenously and

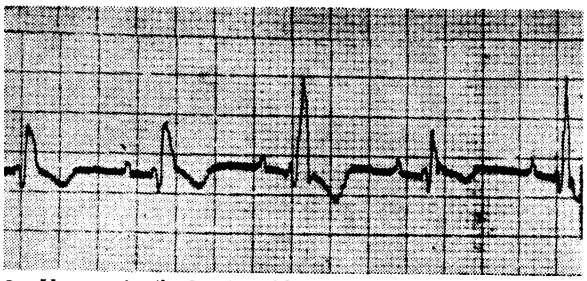

Fig. 2. V_{3R} on April 13, 1962. Note three kinds of QRS complexes.

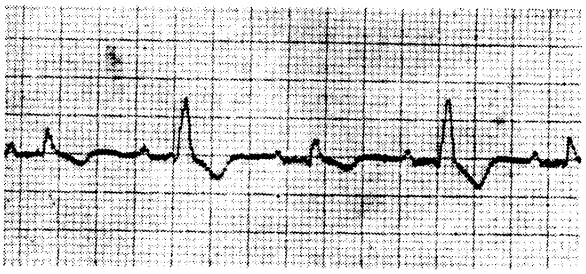

Fig. 3. V_{5R} on April 13, 1962. Complete right bundle branch block alternates incomplete right bundle branch block each together.

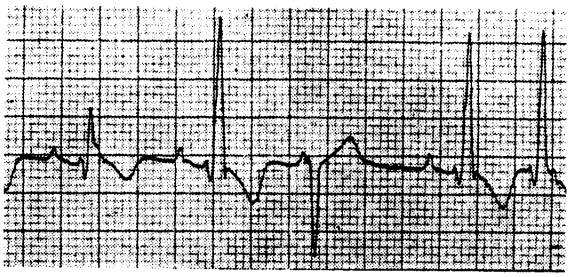

Fig. 4. V_{3R} on April 26, 1962. On digitalization.

digitoxin 0.3 mg for 3 days followed by 0.2 mg for 3 days and then 0.1 mg for 7 days. The irregularity of the heart beats had appeared.

April 26, 1962 (Fig. 4.) — Complete type of R. B. B. B. and incomplete type of R. B. B. B. appeared at random and were associated with ventricular premature contractions and supraventricular premature contractions. Digitalization was discontinued.

April 28, 1962 (Fig. 5.) — Regular sinus rhythm in 75. Alternating R. B. B. B. of complete and incomplete type appeared again.

May 16, 1962 and June 1, 1962 — No changes were seen.

June 29, 1962 (Fig. 6.) — Regular sinus rhythm in 79. Pulmonary P. Complete R. B. B. B. reappeared. There was no incomplete type, i. e., the alternating B. B. B. was no longer seen.

August 2, 1962 (Fig. 7.) — Regular sinus rhythm in 79. The incomplete type of complexes occasionally appeared among the complete type of complexes in each lead.

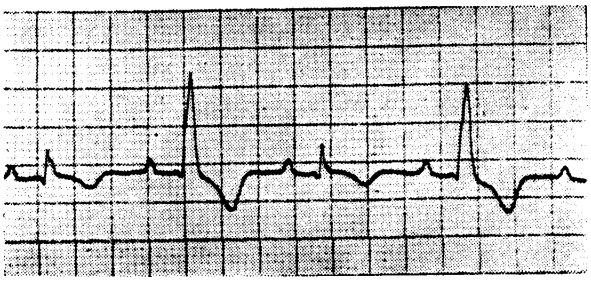

Fig. 5. Two days after discontinued digitalization.

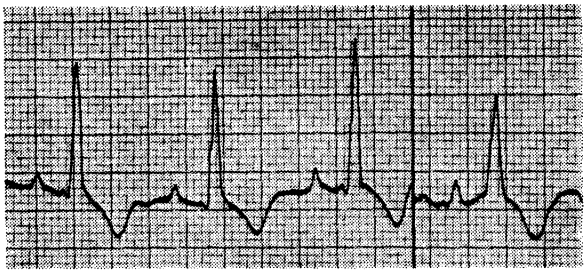

Fig. 6. V_{3R} (First 3 waves) and V_{4R} (Last one) on June 29, 1962.

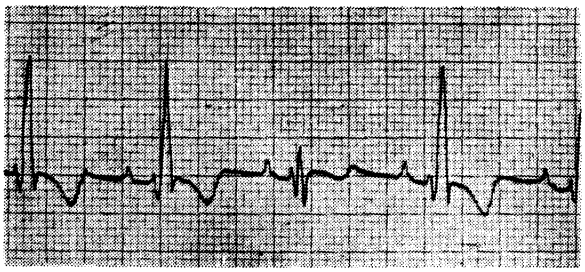

Fig. 7. V_{3R} on August 2, 1962. Incomplete type complex appeared.

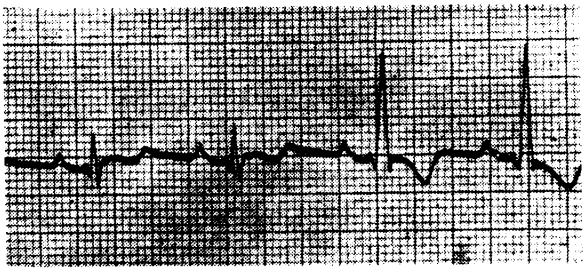

Fig. 8. V_{3R} on September 27, 1962. Spontaneous transition.

August 27, 1962 — All tracings showed complete R. B. B. B. again.

September 27, 1962 (Fig. 8 and 9.) — Incomplete type of R. B. B. B. complexes was seen in I, II, V_1, V_2, V_3, V_6 and V_{5R}. Complete type of R. B. B. B. complexes was seen

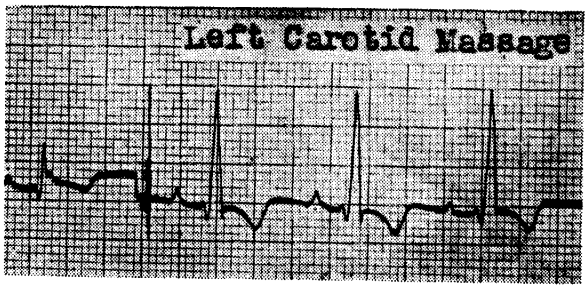

Fig. 9. V_{3R} on September 27, 1962. Transition by carotid massage.

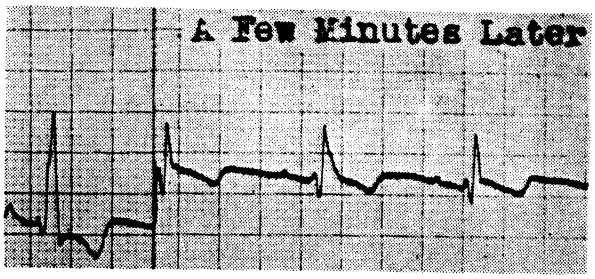

Fig. 10. V_{3R} on October 11, 1962. Spontaneous transition after a few minutes.

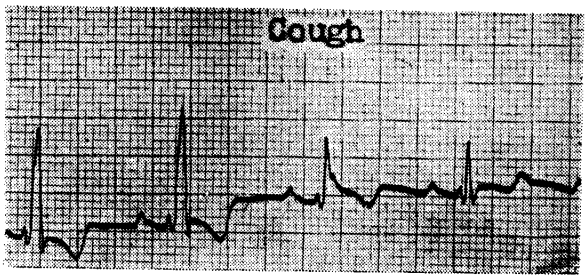

Fig. 11. V_{3R} on October 11, 1962. Transition by cough.

in III, aVR, aVF, aVL, V_4, V_5 and V_{4R}. In V_{3R} a spontaneous transition from incomplete type to complete type was noted. Also complexes of incomplete type changed spontaneously to that of complete type. Furthermore, complexes of incomplete type were induced to complete type by carotid massage and exercise.

October 11, 1962 (Fig. 10 and 11.) — All 16 leads showed complete R. B. B. B. but a few minutes later, V_{3R} showed incomplete type, further, a few minutes later, incomplete type in V_{3R} changed to complete type. Furthermore, an interesting finding is that intermittence occurred with coughing and exercise of the upper extremities from complete to incomplete, and from incomplete to complete.

October 25, 1962 — Regular sinus rhythm in 80. Complete R. B. B. B. was seen again. No incomplete complexes were longer appeared by cough, carotid massage or exercise of the upper extremities.

DISCUSSION

In 1927, N. STENSTROEM[3] reported first one instance of two to one bundle branch block as a brief transitional event, occurring between relatively normal bundle branch conduction and complete right bundle branch block. An excellent example of two to one right bundle branch block was, later becoming complete, reported in 1928 by R. F. LEINBACH and P. D. WHITE[4].

In 1938, W. J. COMEAU and P. D. WHITE[5] reported 71 cases of paroxysmal bundle branch block associated with heart disease in a review and an analysis of the literature, with thirteen new cases and notes upon the influence of the vagus. They included 6 cases of two to one bundle branch block and a case of three to one and four to one bundle branch block.

The reporting cases of transient or intermittent bundle branch block are not uncommon, but few cases of two to one bundle branch block have been reported in the Japanese literature.

Incidence: Association of paroxysmal bundle branch block with coronary and hypertensive heart disease is the largest group. The organic basis of the block in these cases is probably either arteriosclerotic narrowing in the coronary vessels supplying the conduction tissue, with or without partial permanent damage to a bundle branch or the direct or indirect effects of an acute coronary thrombosis, changed in the conducting fibers accompanying failure of the myocardium may be the primary or an additional factor in the production of the branch block.

Transient bundle branch block has been repeatedly attributed to acute infections and particularly to an active rheumatic process. Diphtheria on rare occasions can cause this type of transient conduction disturbance. With an active rheumatic infection in the presence of chronic rheumatic heart disease it is unjustifiable to attribute a transient bundle branch block solely to a specific action of the acute process of the conducting tissue and to ignore the effects of the chronic lesion.

It seems that infection usually acts largely through its effects on the whole myocardium including the bundle branches rather than specifically on a bundle branch or the blood supply of that branch.

Transient or intermittent bundle branch block may occur less frequently in younger individuals with chronic heart disease of rheumatic origin.

Temporary bundle branch block has been said to appear occasionally during the administration of drugs in the treatment of heart disease, particularly digitalis and quinidine[6].

Temporary bundle branch block may occur in cor pulmonale secondary to pulmonary diseases: emphysema, bronchial asthma, tuberculosis, silicosis,

bronchiectasis, pulmonary fibrosis and so on. In this condition dilatation of the right heart is the most important factor[7].

It is well known that the vagus nerves supply fibers to the S-A and A-V nodes and that carotid sinus pressure acting reflexively through the vagi may produce varying degrees of block in either node. The vagus had also been credited with the ability to depress conduction through the bundle branches and increase in vagal tone does not produce sustained inhibition of bundle branch conduction. Variations in vagal tone affect the conduction only by decreasing or increasing the cardiac rate to such an extent that the depressed branch becomes capable or incapable of transmitting impulses.

Mechanism: It is not necessary that complete block exist in one branch in order that impulses be forced to travel through the opposite branch and the myocardium to reach the affected ventricle. Conduction by this pathway may be necessary when one branch is only depressed. So long as the conduction time through the damaged branch is greater than that through the intact branch plus the myocardial pathway between the two ventricles, the affected ventricle is activated by an impulse traveling through the latter channels, and bundle branch block complexes result. Whenever the conduction time through the damaged branch passes the critical level and becomes shorter than that through the channel just mentioned, normal QRST complexes appear. It is therefore apparent that if conduction through the affected branch is close to this critical zone, small changes in the conductivity of the depressed branch result in sudden and complete changes in the form of the ventricular complexes. Such small changes in conductivity result, for example, from the increase or decrease in diastolic rest due to slight alternations in heart rate.

In instances of two to one, three and four to one bundle branch block[8], by a mechanism similar to that in partial A-V dissociation, the conduction of one or two impulses through the damaged branch increases the refractory period of that branch so that the succeeding impulse is delayed beyond the critical level and a branch block complex appears. The resulting rest permits the normal conduction of the next one, two or three complexes.

When severe myocardial failure or coronary thrombosis is the cause of temporary bundle branch block, conduction through the affected branch is severely depressed or even temporarily abolished. As improvement occurs, the potential conduction time becomes progressively shorter but branch block complexes remain until it passes below the critical level at which time normal complexes suddenly appear.

The complexes of the intermediate type which have been recorded during transitions probably occur when the conduction time through the damaged branch falls within the critical zone and is approximately equal to that through

the intact branch plus the myocardium. The ventricle on the affected side is then partly activated by impulses which have passed along both routes.

SUMMARY

A case of paroxysmal bundle branch block, two to one right bundle branch block followed by intermittent right bundle branch block, which is associated with chronic cor pulmonale secondary to active, far advanced pulmonary tuberculosis, is presented. The incidence and mechanism of the paroxysmal bundle branch block have been discussed.

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