Case Report

A Case of Inconstant Left Bundle Branch Block Associated with Sepsis

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INCONSTANT bundle branch block is not a rare conduction. Since Lewis¹⁾ described transient bundle branch block initially in 1913, there have been many reports of transient or intermittent bundle branch block. The underlying diseases of them were mostly coronary and/or hypertensive heart disease. It has been occasionally reported that such block was associated with an infectious disease,²⁾⁻⁵⁾ especially with diphtheria.⁶⁾ Recently, we observed a case of inconstant left bundle branch block, which appeared during the course of sepsis and showed interesting electrocardiographic findings. This case is reported because of the rarity of the cause and the findings.

CASE REPORT

S.T., a 65-year-old man, was admitted to the Kanazawa University Hospital on December 3, 1962, complaining of fever.

The patient had been healthy till November, 1962, except that he had occasionally received a treatment of hypertension since 1960. On November 1, 1962, he suffered from nasal bleeding spontaneously, which continued thereafter for a week. Systolic blood pressure was 200 mm.Hg at first, and then it fell to 150 mm.Hg after a week. On November 29, he had fever of 37.3°C. In spite of the treatment with sulfonamide, chloramphenicol, kanamycin, and erythromycin, his temperature rose to 38.5°C on December 3. On the same day he was admitted to the Kanazawa University Hospital.

The patient had no history of suffering from syphilis, rheumatic fever, other infectious diseases, and angina pectoris.

Physical examination on admission revealed a well-developed man with dyspnea but no cyanosis. The temperature was 38.9°C, the respiration rate was 24, and the blood pressure was 130/68 mm.Hg. The pulse was regular with the rate of 88 and the radial artery wall was sclerotic. The tonsils were normal in appearance. No lymphnodes were palpable in the neck and axillae. The cardiac dullness appeared to be enlarged bilaterally, and a soft systolic murmur

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of Grade 2 was audible in the 4th intercostal space at the left sternal border. No rales were heard in the lung. The liver and spleen were not palpable. There were no ascites and no edema.

Laboratory examinations were summarized in Table I. The increased blood

Table I. Laboratory Data on Admission

Serum Wassermann reaction.....negative	Biochemical Study:
Blood sedimentation rate.....110 mm./1 hr.	Total protein..... 6.3 Gm./100 ml.
Urinalysis:	A/G.....0.42
Albuminuria +	NPN28.5 mg./100 ml.
Sedimentred cell 1/7-8 vf.	Na.....136mEq./L.
white cell 1/5-6 vf.	K3.9mEq./L.
Hemogram:	Cl98mEq./L.
R.B.C.395 × 10 ⁴	Total cholesterol.....155 mg./100 ml.
Hb. (Sahli)75%	Icterus index 2
W.B.C.23,100	BSP test (45')11%
Differential count	Zinc turbidity test7.4 u.
Neutrophils73%	Thymol turbidity test0.8 u.
Eosinophils 0%	SGO-T61.0 u.
Basophils 0%	SGP-T56.0 u.
Lymphocytes.....22%	C-reactive protein.....5+
Monocytes..... 5%	Blood culture35 colonies
	of <i>Alcaligenes faecalis</i> in 1 ml. of blood.

sedimentation rate, albuminuria, leucocytosis, slight impairment of liver function tests, positive C-reactive protein, and growths of bacteria on blood culture were chief findings.

The chest X-ray film revealed bilateral enlargement of the heart and the

Fig. 1. Chest X-ray film.

prominent ascending aorta (Fig. 1). The electrocardiogram on admission revealed intermittent left bundle branch block, prolonged A-V conduction, and left ventricular hypertrophy (Fig. 2). All QRS complexes were preceded by the sinus P wave with the prolonged P-Q interval measuring 0.24 sec. The

Fig. 2. Electrocardiogram (1), taken on December 3, 1962.

widened QRS complexes of left bundle branch block pattern were seen alternately with 2:1 variety among the normal QRS complexes. The preceding R-R intervals were the same of 0.68 sec. both in the normal and abnormal complexes. The normal QRS complexes in the left precordial leads revealed tall R with ST-T depression.

A diagnosis of sepsis was made. Otorhinopharyngological examination,

Fig. 3. Clinical course.

liver scintigraphy, cholecystography, and urine culture were performed, and it was considered that the primary focus of infection was probably located in the gallbladder and/or bile ducts. The patient was treated with chloramphenicol, penicillin, streptomycin, erythromycin, and kanamycin. The clinical course was illustrated in Fig. 3. The temperature fell to 36.6°C after 5 days, but slight

Fig. 4. Electrocardiogram (2), taken on December 4, 1962.

Fig. 5. Electrocardiogram (3), taken on December 5, 1962.

fever continued throughout the admission.

On the other hand the electrocardiograms showed various changes successively. On December 4, the second hospital day, it revealed atrial fibrillation and intermittent left bundle branch block (Fig. 4). The QRS complexes showed usually left bundle branch block pattern, but occasionally the complexes of

Fig. 6. Electrocardiogram (4), taken on December 7, 1962.

Fig. 7. Electrocardiogram (5), taken on December 12, 1962.

the normal conduction were recorded. These normal QRS complexes were preceded by comparatively shorter R-R intervals similar to premature beat, but there were the widened QRS complexes preceded by more shorter R-R intervals. On December 5, the third hospital day, atrial fibrillation disappeared (Fig. 5). All QRS complexes showed left bundle branch block pattern and were preceded by the sinus P wave with the prolonged P-Q interval measuring 0.23 sec. No normal QRS complex was seen. On December 7, the fifth hospital day, the electrocardiogram showed intermittent left bundle branch block again (Fig. 6). Prolonged A-V conduction and left ventricular hypertrophy were also observed. The preceding R-R intervals of the normal QRS complexes were almost the same as that of the widened QRS complexes. On December 12, when fever had already become milder, left bundle branch block was no longer seen and the P-Q intervals recovered to 0.20 sec. (Fig. 7). However, the electrocardiogram of December 25 showed the prolonged P-Q intervals measuring 0.22 sec., which were observed thereafter till his discharge on February 6, 1963. The patient had never showed hypertension throughout the admission.

COMMENT

A case of inconstant left bundle branch block which appeared during the course of sepsis has been reported.

Two types are distinguished in inconstant bundle branch block; the intermittent and the transient.^{2), 7)} In the former the widened QRS complexes denoting bundle branch block alternate with the normal QRS complexes in an electrocardiogram, and in the latter the widened QRS complexes appear throughout a single electrocardiogram and disappear in later records.

It has been frequently reported that the appearance of such bundle branch block is related to the faster heart rate or the shorter R-R interval.⁷⁾⁻¹⁶⁾ This phenomenon is explained by the interrelation of the heart rate and the refractory period. On the contrary, other reports showed that slowing of the heart rate was followed by the appearance of bundle branch block in some cases.¹⁵⁾⁻¹⁷⁾ The effect of increased vagal tone is considered in these cases. In our case, bundle branch block had no relationship to the preceding R-R interval and showed the pattern of 2:1 block. Only 13 cases of 2:1 bundle branch block have been seen in the literature.^{2), 6), 8), 10), 13), 18), 19)} Furthermore, when the electrocardiogram revealed atrial fibrillation on the third hospital day, the QRS complexes showed mostly left bundle branch block pattern, but occasionally the normal QRS complexes were recorded following comparatively shorter R-R intervals as premature beat. Only two reports of similar cases with atrial fibrillation^{8), 20)} and seven reports of the same phenomenon with sinus rhythm^{2), 20)-23)} are available in the literature. In these cases two interpretations have been done; one is that the normal QRS complexes are

ventricular premature beat which arises from the interventricular septum just below the blocked region, and the another is that the impulse from the supraventricular region is normally conducted only during the supernormal phase of recovery of the injured bundle branch. The differentiation is more difficult in atrial fibrillation than in sinus rhythm because of the lack of the P wave. In our case the relationship of the QRS complex form to the R-R interval during atrial fibrillation is illustrated in Fig. 8. The normal QRS complexes are seen in the narrow area and seem to have some relationship to the supernormal phase. However, because of the minority of the normal QRS complex, we would reserve to decide which one of the two interpretations is conformed to our case.

Fig. 8. The relationship of the QRS complex form to the R-R interval during atrial fibrillation. Every QRS complex is plotted as one point in the diagram, the ordinate of which represents the R-R interval immediately preceding a QRS complex, and the abscissa of which represents the R-R interval preceding the R-R interval of the ordinate.

The underlying diseases of inconstant bundle branch block, which are available in the literature,¹⁾⁻⁴⁷⁾ are summarized in Table II. Coronary and/or hypertensive heart disease are most common underlying diseases, and only 4 cases of infectious diseases other than diphtheria are collected.²⁾⁻⁵⁾ It has been reported that about half of sepsis and some of other infectious diseases combined myocarditis.^{48), 49)} The inflammation or the edema of such myocarditis may have induced inconstant bundle branch block in our case. However, since inconstant bundle branch block associated with sepsis and various infections has been rarely reported and the prolonged A-V conduction persisted even after the healing of sepsis, it is considered that the block in our case might have also some relation to

Table II. Underlying Diseases of Inconstant Bundle Branch Block

Coronary and/or hypertensive heart disease	86 cases	59.7 %
Rheumatic heart disease	12	8.3
Thyrototoxicosis	6	4.2
Diphtheria	4	2.8
Other infectious diseases	4	2.8
Congenital heart disease	1	0.7
Others	20	13.9
Normal	11	7.6
Total	144 cases	100.0 %

the other factor such as coronary and/or hypertensive heart disease which were latent.

SUMMARY

A case of inconstant left bundle branch block which appeared during the course of sepsis is reported. The electrocardiogram showed 2:1 intermittent left bundle branch block and then the normal QRS complexes were seen following comparatively shorter R-R intervals among left bundle branch block with atrial fibrillation. The bundle branch block disappeared after alleviation of fever, but the prolonged A-V conduction persisted.

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