Case Report

An Aged Case of Trifascicular Block with Special Reference to a Histological Study of the Conduction System

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SUMMARY

A histological study by serial sections of the conduction system was performed in a case of 84-year-old female with a diagnosis of hypertension and complete atrioventricular (AV) block. The course of her electrocardiogram was divided into 6 stages as follows: 1) left ventricular hypertrophy (11 years before), 2) complete right bundle branch block (C-RBBB) and no axis deviation with PQ interval of 0.20 sec (10 years before), 3) C-RBBB with right axis deviation (2 years before), 4) advanced AV block with occasional ventricular capture, 5) C-RBBB with left axis deviation for 10 days' duration (1 year and 3 months before), and 6) complete AV block with the idioventricular rhythm with RBBB type (terminal 1 year).

There were moderate fibrosis in the AV node with partial destruction of conduction cells and the same fibrotic changes in the proximal portion of the AV bundle. The proximal portions of posterior and anterior fascicles of the left bundle branch were completely interrupted by the marked endocardial fibrosis. The right bundle branch showed sporadic fibrosis in the first portion, but in the second portion the conducting cells were thoroughly destroyed. The above-mentioned findings were compatible with the trifascicular block reported by Rosenbaum in 1969, in both the electrocardiographic and histological findings.

Additional Indexing Words:

Trifascicular block Bifurcation of AV bundle Complete AV block
RBBB with left anterior fascicular block RBBB with left posterior fascicular block Hemiblock

IN 1969 Rosenbaum¹⁾⁻³⁾ postulated the concept of 'trifascicular block' which assumed that the entire intraventricular conduction system is trifasci-

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cular, consisting of the right bundle branch and 2 fascicles of the left bundle branch.

Combination of permanent and intermittent blocks in these 3 fascicles gives rise to different varieties of conduction disturbance. Since then a lot of clinical cases in this category has been accumulated.⁴⁾⁻⁷⁾ But there have been few histopathological studies. In this communication we report a case of trifascicular block and investigated histological changes of the conduction system, comparing with her electrocardiograms.

CASE REPORT

A 66-year-old female (N.F.) entered the Yoiku-in Tokyo municipal Home for the Aged and enjoyed an eventful life. At the age of 73 her electrocardiogram showed left ventricular hypertrophy. After 1 year the electrocardiogram showed complete right bundle branch block with PQ interval of 0.20 sec (Fig. 1a). At the age of 81 she was noticed to have hypertension for the first time. In next winter, she was admitted to the Yoiku-in Hospital because of bronchopneumonia for 2 months. In this time the electrocardiogram revealed the appearance of right axis deviation besides complete RBBB (Fig. 1b). After discharge she had been hypertensive and treated for it at the outpatient clinic. One year later (83 years) she complained of

Fig. 1. Electrocardiograms a) taken at 73 years old, showing left ventricular hypertrophy with normal sinus rhythm and RBBB with no axis deviation, b) taken at 82 years old, showing complete RBBB with right axis deviation, and c) taken at 83 years old, advanced AV block with occasional ventricular capture.

Fig. 2. Electrocardiogram, showing complete AV block in the terminal 1 year.

Fig. 3. The chest X-ray films a) before and b) after the appearance of advanced or complete AV block.

an episode of vertigo, nausea and precordial discomfort with a short duration of syncope. At this time she was noticed to have bradycardia of 40/min and admitted to the hospital for the second time, with chief complaints of lightheadedness and vertigo. On admission, her consciousness was clear, pulse rate 30/min, and blood pressure 218/80 mmHg.

Fig. 4. a) Atrioventricular node (N), showing moderate fibrosis and patent AV node artery (EvG $\times$ 13). b) The penetrating portion of the AV bundle (B) (EvG $\times$ 13).

Fig. 5. a) The branching portion of the AV bundle (B) and the posterior fascicles of the left bundle branch (LB), revealing complete interruption of conducting cells (between arrows) (HE $\times$ 20) by subendocardial and summit fibrosis (F). b) The anterior fascicles of the left bundle branch (LB) (arrows), showing fatty metamorphosis (F) in slightly distal portion (EvG $\times$ 40).

Auscultation of the chest showed an increased first heart sound without any murmurs and no râles. There was neither edema nor palpable organs. Electrocardiograms showed advanced AV block with occasional ventricular captures. And the electrical axis showed right axis deviation or normal axis at the early stage but during clinical course the electrical axis was deviated to the left (Fig. 1c).

Main laboratory data were as follows: The urine gave a positive test for protein; the sediment contained 10 to 20 white cells per low power field. The red blood cell count was 315×10^4 , and the white cell count 6,600 with normal differential. The total protein was 5.6 Gm per 100 ml, the urea nitrogen 27.5 mg, the sodium 150 mEq, the potassium 4.2 mEq and the chloride 118 mEq per liter. The cholesterol was 211 mg per 100 ml and the liver function test was almost normal. The serological test for syphilis was negative. A clinical diagnosis of advanced AV block was made, which was treated by β -stimulator with no effect. One month after admission the electrocardiogram showed development of complete AV block (Fig. 2). The chest X-ray films revealed significant increase of the cardiothoracic ratio after the appearance of advanced AV block on electrocardiogram (Fig. 3).

In her further clinical course congestive heart failure with edema on lower extremities, palpitation and dyspnea on exertion grew up and she died of bronchopneumonia 1 year and 3 months after admission.

Autopsy was performed. Heart weight was 440 Gm with few coronary sclerosis and the valves were almost normal. The wall of the left ventricle was thicken-

Fig. 6. a) The first portion of the right bundle branch (arrows) with some fibrosis but still retaining conduction cells (HE $\times 40$). b) The second portion of the right bundle branch (arrows), disclosing complete destruction of conducting cells with only some elastic fibers (EvG $\times 40$).

ed with the presence of scattered fibrosis. The other main pathological findings besides the heart were bronchopneumonia of left lower lung and congestion of the liver and the spleen. A histological study of the conduction system was performed by the serial sections according to the method of Lev,⁸⁾ as in the previous studies.⁹⁾⁻¹¹⁾ The serial sections were stained by hematoxylin and eosin, elastica van Gieson, and periodic acid Schiff, toluidine blue or colloidal iron stain for acid mucopolysaccharides. The AV node showed presence of moderate fibrosis and diminished specialized conduction cells, which, however, were an incomplete lesion (Fig. 4a). The AV nodal artery was, however, patent. The AV bundle revealed that in the penetrating portion the similar fibrotic change was present as in the AV node (Fig. 4b). Changes of the central fibrous body were those as found in the general aged cases, and the substance consisting of acid mucopolysaccharides was not so accumulated. On the other hand, in the proximal portion of posterior radiation of the left bundle branch there revealed almost complete interruption of conducting cells by subendocardial and summit fibrosis (Fig. 5a). The anterior radiation of the left bundle branch also showed the same lesion, associated with the increased adipose tissue (Fig. 5b). The right bundle branch showed marked fibrotic changes in the first portion (Fig. 6a) and in the second portion the conduction cells were almost completely destroyed (Fig. 6b).

DISCUSSION

The concept of trifascicular block was first developed from the close follow-up studies of electrocardiograms,¹⁾ and 8 subtypes of trifascicular block were classified by combining complete or incomplete block of the 3 fascicles,^{2),3)} but its morphological basis was not obtained in their 6 cases due to lack of the autopsy data.³⁾ Other histological studies of trifascicular block have not been fully accumulated. In 1969, Harris et al¹²⁾ reported a 46-year-old female of trifascicular block due to smoldering myocarditis. Electrocardiogram showed left posterior hemiblock (LPH), 2nd degree AV block with complete RBBB and LPH and complete AV block. Histological findings well corresponded to ECG and especially marked fibrosis was found at the septal summit. Tricot¹³⁾ reported diffuse amyloid deposition in myocardium as well as in the conduction system in a case, aged 69, who showed complete RBBB with alternating right and left axis deviation (type 5 of Rosenbaum). Smithen et al¹⁴⁾ reported a 62-year-old case with trifascicular block, consisting of permanent left anterior fascicular block and intermittent block of the left posterior fascicle and the right bundle branch leading to advanced block (type 7 of Rosenbaum). Histologically there demonstrated damages to the right and left bundle branches with the normal AV node and AV bundle.

In our case the electrocardiograms showed at the first time RBBB with normal electrical axis or right axis deviation (1.5 years' duration) and then transiently left axis deviation. The rhythm was changed from sinus rhythm

to advanced AV block with occasional ventricular capture and finally to complete AV block. Above-mentioned changes of ECG suggested that the right bundle branch was completely blocked, while both anterior and posterior fascicles of the left bundle branch might be incompletely interrupted, which might be classified as type 5 of trifascicular block described by Rosenbaum.^{2),3)} Histological examination of the conduction system revealed that the site of block was the bifurcation of the AV bundle consisting of proximal portion of both anterior and posterior fascicles of the left bundle branch and the second portion of the right bundle branch. Bifurcation to the right bundle branch (the 1st portion) was fibrotic, but the interruption was not complete.

Histological examination of chronic complete AV block with or without electrocardiographic features of trifascicular block and RBBB associated with left axis deviation, demonstrated that the site of interruption was mainly the branching portion of the common AV bundle and the proximal portion of the branches.^{9),11),15)-21)} The etiology of the trifascicular block was not certain, but the similarity of the histological findings to those in the chronic AV block¹¹⁾ and RBBB with left axis deviation⁹⁾ suggested the close relationship of the etiology with aging changes by the sustained mechanical strain to the conduction system, and not with the coronary sclerosis, as previously reported.²⁰⁾

REFERENCES

1. Rosenbaum MB et al: Intraventricular trifascicular blocks. The syndrome of right bundle branch block with intermittent left anterior and posterior hemiblock. *Am Heart J* **78**: 306, 1969
2. Rosenbaum MB et al: Intraventricular trifascicular blocks. Review of the literature and classification. *Am Heart J* **78**: 450, 1969
3. Rosenbaum MB, Elizali MV, Lazzari JO: The hemiblocks. New concepts of intraventricular conduction based on human anatomical, physiological, and clinical studies. Tampa Tracings, Oldsmar, Florida, 1970
4. Nohara Y et al: Block ECG. *Naika* **28**: 849, 1971 (in Japanese)
5. Rosselot E et al: Trifascicular block treated by artificial pacing. *Am J Cardiol* **26**: 6, 1970
6. El-Sherif N: Intraventricular trifascicular block. *Brit Heart J* **33**: 626, 1970
7. Steiner C et al: Electrophysiologic documentation of trifascicular block at the common cause of complete heart block. *Am J Cardiol* **28**: 436, 1971
8. Lev M, Widran J, Erickson EE: A method for the histological study of the atrioventricular node, bundle and branches. *Arch Path* **52**: 73, 1951
9. Sugiura M et al: Histological studies on the conduction system in 14 cases of right bundle branch block associated with left axis deviation. *Jap Heart J* **10**: 121, 1969
10. Sugiura M et al: Pathohistological studies on the conduction system in 8 cases of complete left bundle branch block. *Jap Heart J* **11**: 55, 1970
11. Sugiura M et al: Histological studies on the conduction system in 8 cases of A-V conduction disturbances. *Jap Heart J* **11**: 460, 1970
12. Harris R, Siew S, Lev M: Smoldering myocarditis with intermittent complete A-V block and

- Stokes-Adamssyndrome. A histopathologic and electrocardiographic study of " trifascicular " bundle branch block. *Am J Cardiol* **24**: 880, 1969
13. Tricot R et al: Bloc trifasciculaire au cours d'une amylose cardiaque primitive. *Arch mal coeur* **63**: 1278, 1970
 14. Smithen C et al: Trifascicular block—An electrocardiographic, hemodynamic and pathological correlation. *J Electrocardiol* **4**: 145, 1971
 15. Lev M: Anatomic basis for atrioventricular block. *Am J Med* **37**: 742, 1964
 16. Blondeau M, Lenègre J: Bloc de branche et bloc auriculoventriculaire complet. *Arch mal coeur* **57**: 1, 1964
 17. Harris A et al: Aetiology of chronic heart block. A clinicopathological correlation in 65 cases. *Brit Heart J* **31**: 206, 1969
 18. Davies MJ, Harris A: Pathological basis of primary heart block. *Brit Heart J* **31**: 219, 1969
 19. Davies MJ: Pathology of conducting tissue of the heart. Butterworths, London, 1971
 20. Sugiura M, Ohkawa S, Okada R: Aging of the central fibrous body with a special reference to conduction disturbance. *Proc 8th Internat Congr Gerontol* **11**: 23, 1969
 21. Unger PN et al: The concepts of " masquerading " bundle-branch block. An electrocardiographic-pathologic correlation. *Circulation* **17**: 397, 1958