HEMATOMA OF THE INTERATRIAL SEPTUM ASSOCIATED WITH COMPLETE ATRIOVENTRICULAR BLOCK

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A 70-year-old man who had been treated for tongue cancer began to suffer from repeated syncopal attacks. His electrocardiogram indicated complete atrioventricular block. A permanent pacemaker was implanted. He was well for about 2 years after which time he developed dyspnea, suffered from general fatigue, and then suddenly died. A postmortem examination revealed a hematoma approximately 4×4cm in size in the interatrial septum, connected to a noncoronary sinus and rupture of the noncoronary leaflet, which compressed the tricuspid and aortic valves. This hematoma might have been related to the conduction disturbance caused by destructive compression on the conduction system, and his death by the rupture of the noncoronary leaflet.

HEMATOMA of the interatrial septum is very rare, with only 19 cases reported in the literature!¹⁻¹¹ It can occur from aortic valve stenosis!^{1,7} rupture of an aneurysm of the right coronary artery!⁴ a dissecting aneurysm of the ascending aorta!^{3,5,6,8-10} or blunt chest trauma!¹ In many cases, the hematomas were associated with atrioventricular conduction disturbance!^{3,6,7,9,10} because of their proximity to the A-V node and the bundle of His. We describe here a patient with complete atrioventricular block who was found to have a hematoma of the interatrial septum without the above mentioned causes at autopsy.

Key words:

Hematoma
Interatrial septum
Atrioventricular block
Permanent Pacemaker

CASE REPORT

A 70-year-old male whose chief complaint was a right submandibular tumor visited the Hyogo Medical Center for Adults on June 14, 1984. He had a previous history of hypothyroidism, a duodenal ulcer and hepatitis B. His electrocardiogram indicated a first degree atrioventricular block with a PR interval of 0.3 sec and left axis deviation (Fig. 1). As the tumor was diagnosed as tongue cancer, he underwent radiation therapy and chemotherapy from June 27 to September 28, 1984, and submandibular regional dissection on October 18, 1984.

Ten days later, on the night of October 28, syncopal attacks lasting several minutes began to occur repeatedly. Because the electrocardiogram revealed complete atrioventricular block (Fig. 1), on November 9, a

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Fig.1. Serial changes of the electrocardiograms.

Electrocardiogram taken on June 14, 1984 shows first degree atrioventricular heart block and left axis deviation, another taken on October 30, 1984 shows complete atrioventricular heart block with complete left bundle-branch block shaped escaped beats and another taken on October 14, 1986 shows pacemaker rhythm.

Paper speed at 25 mm/sec in the upper 6 strips and 12.5 mm/sec in the lower 3 strips, indicating the heart rate was 79 bpm, 29 bpm and 70 bpm respectively.

In the lowest strip, pacing spike fluctuation is probably due to the effect of respiration. Though the third beat appears to be lacking a pacing spike, a very small spike is visible before it. Besides, the timing and shape of this beat being the same as the other beats, this beat may also be a paced beat. Therefore, pacing failure is deniable.

Fig.2 Chest roentgenogram taken on October 14, 1986 shows old pulmonary tuberculosis, bullae and indistinct opacity in the right lung.

permanent pacemaker was implanted with an endocardial lead positioned in the right ventricle. An electrocardiogram showed transient recovery to first degree atrioventricular block and left axis deviation on November 14, 1984, but it showed pacemaker rhythm on May 26, 1985.

He did well postoperatively for about 2 years when he began to develop dyspnea and suffered from general fatigue in early October 1986. On October 14, 1986, an electrocardiogram showed the pacemaker rhythm as before (Fig. 1), and a chest X-ray revealed old pulmonary tuberculosis and bullae in the right upper lobe and indistinct opacity in the right lower lobe (Fig. 2).

Despite treatment with furosemide, one tablet (40 mg) a day from that day onward, he did not improve. On physical examination on October 25, 1986, in spite of abnormal findings such as drowsy consciousness, bilateral myotic pupils, poor light reaction and a weak tendon reflex, no marked abnormal finding was observed in his cardiorespiratory system. He had blood pressure of 155/100 mmHg, a regular pulse of 70 bpm, a clear heart and normal breathing sounds.

The laboratory data taken the same day

suggested dehydration due to hyperglycemia, a RBC of $608 \times 10^4/\text{mm}^3$, total protein of 10.2 g/dl, urinary sugar of 2.0 g/dl, and blood sugar of 689 mg/dl. Furthermore, there were findings indicating an increased inflammatory reaction (WBC, $9800/\text{mm}^3$; ESR, 50 mm/h and CRP, 3.7 mg/dl), slight liver dysfunction (GOT, 75 IU/l; GPT, 80 IU/l and ALP, 276 IU/l) and increased catabolism or slight renal dysfunction (BUN, 45.3 mg/dl and serum creatinine, 1.5 mg/dl). There was, however, no significant abnormality in either a serum electrolyte level (Na, 146.4 mEq/l; K, 4.57 mEq/l and Cl, 104.7 mEq/l) or atrial blood gas measurements (pH, 7.422; PCO_2 , 39.7 mmHg; PO_2 , 87.4 mmHg; HCO_3 , 25.8 mEq/l; base excess, 2.0 mEq/l and O_2 saturation, 96.8%). Treatment was initiated by the administration of 500 ml of isotonic saline solution with 5 units of regular insulin twice a day. However, he suddenly lost consciousness next day and fell down while walking to a lavatory before lunch. The ambulance car arrived within 10 min, but his pulse was not palpable and respiration had stopped. An electrocardiogram taken at our hospital upon arrival showed ventricular fibrillation.

A postmortem examination revealed a globular hematoma approximately 4×4 cm in size of the interatrial septum which involved the lower half of the interatrial septum and compressed the tricuspid valve (Fig. 3 A). A short axis cross-section of the heart at the level of the mitral valve revealed that the hematoma involved the upper part of the interventricular septum (Fig. 3 B). Furthermore, it reached the intra-aortic space through a fissure in the wall of the noncoronary sinus (Fig. 3 C), causing a rupture of the noncoronary cusp (Fig. 3 D). Fig. 4 shows the location of the hematoma relative to adjacent structures and the rupture site.

Histological examination of the hematoma by hematoxylin eosin stain revealed that the hematoma stained heterogeneously, was surrounded by a wall composed of connective tissue, and that the normal myocardium around the wall was being compressed (Fig. 5 A). Staining the same site with Masson's trichrome stain showed only that the wall of the hematoma stained blue, indicating that

Fig.3 Macroscopic findings of the heart.

A: View from the right atrium and right ventricle shows a globular hematoma, 4×4 cm in size, (indicated by an arrow) located in the interatrial septum adjacent to the tricuspid valve. The lead of the permanent pacemaker is shown.

B: Short axis section at the level of the mitral valve.

Note the upper part of the interventricular septum is invaded by the hematoma which is indicated by an arrow.

The lead of the permanent pacemaker is shown.

C: Short axis section at the level of the coronary sinus.

Note the hematoma located between the right and left atrium communicates with the aortic lumen through the fissure of the noncoronary sinus as indicated by an arrow.

D: Short axis section at the level of the aortic valve viewed from the base side. Note the rupture site of the noncoronary cusp (indicated by arrows) destroyed by the hematoma.

RA=right atrium; RV=right ventricle; TV=tricuspid valve; LA=left atrium; LV=left ventricle; MV=mitral valve; PA=pulmonary artery; AO=aorta.

the inside had not yet organized and that it had been formed relatively recently (Fig. 5 B).

Other autopsy findings include (1) the post-therapeutic state of the floor of mouth cancer, (2) congestion of the liver and other organs, (3) atelectasis, (4) fibrosis of the thyroid gland, (5) chronic inactive hepatitis, (6) a postoperative state of the duodenal ulcer and (7) moderate atherosclerosis of the aorta and coronary artery.

DISCUSSION

Cases of the hematoma of the interatrial septum reported so far are summarized in Table I¹⁻¹¹ Although these reports mention several causes, none of them was found in our case. As shown in Fig. 6, the interstitial tissue of the right atrial wall near the interatrial septum communicates with an aortoatrial space, which is bordered by the noncoronary aortic sinus and membranous

Fig.4. Schematic illustration showing the location of the hematoma.

Arrows indicate the ruptured site.

RA=right atrium; RV=right ventricle; RVOT=right ventricular outflow tract;
LA=left atrium; LV=left ventricle; AO=aorta; RCC=right coronary cusp;
LCC=left coronary cusp; H=hematoma.

interventricular septum in close proximity to the bundle of His. Histologically, the atrial myocardium in this region and the interatrial septum are formed of loosely arranged muscle bundles, and the aortoatrial space is filled with loose areolar tissue. Hence, according to Yacoub⁹, hematoma of the right atrial wall and interatrial septum is easily formed if an adjacent high-pressure chamber or vessel such as the left ventricle, the noncoronary aortic sinus or the right coronary artery, ruptures into the aortoatrial space. In our case, considering there was no such a large artery in the interatrial septum or the aortoatrial space that could cause a large hematoma, the fact that the inside of the hematoma had no communication with other sites except the ruptured wall of the noncoronary sinus and the noncoronary cusp, indicates that the blood flowed from the intra-aortic space into the aortoatrial space through this rupture and then the interatrial septum leading to a hematoma. In addition, as no aortic regurgitation was detected the day before his death, the rupture of the noncoronary cusp, which apparently occurred immediately be-

fore death, was considered to have contributed little to the formation of the hematoma. Moreover, autopsy showed no evidence of a dissecting aneurysm, which would have occurred if the blood that flowed into the aortoatrial space had passed upward into the wall of the ascending aorta.

The possibility that the hematoma was formed during the pacemaker implantation procedure is low because the hematoma did not communicate with the right atrium or ventricle, and all causes except one, blunt chest trauma, reported to date were the abnormalities due to high-pressure vessels.

Although it was found that the outside of the hematoma was covered with the connective tissue wall indicating that the hematoma was old, the inside of the hematoma was thought to have formed recently because it was not yet organized. From this finding it can be postulated that the blood had recently flowed into the old hematoma and the large hematoma, disclosed during the autopsy, was newly formed.

As to the cause of his sudden death, the possibility of respiratory or circulatory disturbance such as airway obstruction, mas-

Fig.5. Microscopic findings of the hematoma.

A: Hematoxylin-eosin stain, $\times 6.6$.

The hematoma on the left side of the picture is surrounded by a connective tissue wall around which the atrial muscle fibers are compressed.

B: Masson's trichrome stain, $\times 6.6$.

No fiber is stained inside the hematoma, indicating it had not yet organized and was relatively new.

sive embolism or massive extravasation was ruled out because there were no such findings at autopsy. The possibility of pacing failure is unlikely because the pacing lead was fixed well to the right ventricular wall at autopsy and the electrocardiogram recorded 13 days before his death showed normal pacing rhythm. Hyperglycemia noted 1 day before his death, which may have resulted from furosemide induced impaired glucose tolerance, was not considered to be the direct cause of death because (1) the interval from the onset of unconsciousness to death was too short to suggest death from diabetic coma, (2) the

absence of urine ketone bodies excluded the possibility of chemical coma, which refers to the dangerous state of progressive diabetic ketoacidosis without disturbance of consciousness, and (3) fluid supplementation with insulin therapy had been started. On the other hand, the rupture of the noncoronary cusp may have caused sudden death through acute systemic circulatory failure or ventricular fibrillation from hypoperfusion of myocardium.

As to the atrioventricular block, considering the above mentioned locational relations and histological characteristics of the interatrial septum, the aortoatrial space and

TABLE I REPORTS OF INTERATRIAL HEMATOMA

Author	ECG	Etiology
Maguire, 1887	None	Aortic stenosis; bacterial endocarditis
Dittrich	None	Unclear
Wood, 1931	A-V block (presumed)	Dissecting aneurysm
Marano, et al, 1945	Sinus rhythm with ventricular ectopics	Aneurysm of the right coronary
Nissim, 1946	None; heart rate 36 bpm	Dissecting aneurysm
Pick, et al, 1953	A-V block	Dissecting aneurysm
Orbison, et al, 1956	Intermittent A-V block	Aortic stenosis; bicuspid, calcified aortic valve
Giraud, et al, 1958	Sinus rhythm	Dissecting aneurysm
Yacoub, et al, 1972	A-V block	Dissecting aneurysm
	A-V block	Dissecting aneurysm
	A-V block (presumed)	Dissecting aneurysm
	A-V block (presumed)	Dissecting aneurysm
Gaetano, et al, 1979	A-V dissociation	Dissecting aneurysm
	A-V dissociation	Dissecting aneurysm
	Complete A-V block	Dissecting aneurysm
	First degree A-V block	Dissecting aneurysm
	Clinical data not available	Dissecting aneurysm
	Clinical data not available	Dissecting aneurysm
Steven, et al, 1987	Intermittent atrial fibrillation	Blunt chest trauma

the conduction system, it is possible that many cases reported to date had some conduction defects (Table I)^{3,6,7,9,10}. In this case, as well, the histological examination near the hematoma could not identify the conduction system because of the marked destruction caused by the compression from the hematoma, indicating that the block might have been brought about by the hematoma. Moreover, the atrioventricular block which had occurred 2 years and 4 months before his death might have been caused by the hematoma because the occurrence of the hematoma could be considered old and no other cause of the block could be detected. The fact that the escaped beats on the electrocardiogram recorded on Oct. 30, 1984 showed a complete left bundle branch block shape, might indicate that the influence of the hematoma reached the left bundle branch. However,

as we could not examine the conduction system, there remains the possibility of other major causes such as sclerosis of the left side of the cardiac skeleton¹² and Lenègre's disease.¹³

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Fig.6: Schematic illustration of a longitudinal section through the noncoronary cusp showing the relative locations of the noncoronary sinus, the aortoatrial space, the right atrial wall and the bundle of His. The arrow shows the path of rupture from the noncoronary sinus into the right atrial wall through the aortoatrial space.

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