A Case Report of Ventricular Septal Defect in the Aged

Interrelation between Its Closing Process and Subacute Bacterial Endocarditis

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SUMMARY

A 70-year-old female with small ventricular septal defect complicated with subacute bacterial endocarditis was presented. The rare longevity and a pathogenesis of subacute bacterial endocarditis were discussed with special reference to hemodynamic stress by a rapid shunting jet stream passing through the small defect.

Additional Indexing Words:

Ventricular septal defect Geriatrics Closing process Subacute bacterial endocarditis

THE natural history of ventricular septal defect has been not so defined in spite of its urgent necessity for decision whether application of surgical repair or expecting the possibility of spontaneous closure. Among many case reports and reviews of literature in this field,¹⁾⁻⁶⁾ it was unanimously conceived that the ventricular septal defect could seldom survive the age of more than 60. It would, therefore, be valuable to present a 70-year-old case with ventricular septal defect complicated with subacute bacterial endocarditis and to discuss how the case could survive to this age.

CASE REPORT

The patient was a 70-year-old female with impressively small body height. She was admitted into the Tokyo Yoiku-in municipal home for the aged at the age of 65. At that time, it was known that she had a sharp systolic murmur with thrill, of which maximal point was at the 4th intercostal space of the left sternal border, moderate systemic hypertension and mild senile mental deterioration. She had been attending to the hypertension clinic as an out-patient during the following 4 years. At the age of 69, a high chill fever up to 39°C was first noticed. She was admitted to the Yoiku-in Hospital under suspicion of acute upper respiratory infection. Physical and laboratory examination revealed a systolic murmur (typical as Roger's

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Fig. 1. Chest X-ray film shows left ventricular enlargement and elongation of the aortic arch.

Fig. 2. ECG indicates left ventricular hypertrophy pattern.

murmur) without any sign of congestive failure, 214/90 mm.Hg in blood pressure and leukocytosis of 29,000. The chest X-ray film (Fig. 1) showed enlarged left side of the heart, elongation with calcification of the aortic arch and no clue of pulmonary congestion. The ECG indicated definite left ventricular hypertrophy (Fig. 2). The fever was quickly suppressed after a few day's treatment using antibiotics. However, her general condition became worse due to emaciation under feeding negativism with progressing senile dementia. At one month after the admission, high chill fever and leukocytosis of 21,500 recurred and subacute bacterial endocarditis complicated to the ventricular septal defect was strongly

suspected. She died under general weakness with intractable fever.

Autopsy findings: The heart was moderately enlarged. The right atrium was normal either in size or thickness. The right ventricle was almost normal in size at the inflow tract having convex ventricular septum towards lumen. At the outflow tract, the conus arteriosus pulmonum was, however, prominently dilated with almost normal wall thickness (Fig. 3a). The left atrium was of normal size and the left ventricle showed marked hypertrophy especially of the ventricular septum (Fig. 3b). The ventricular septal defect was situated at the lower part in the pars membranacea at the left side (Fig. 3b) and behind the crista supra-ventricularis (Fig. 3a) at the right side. The defect was 3.5×2.5 mm, and seemed to be divided into 2 parts by a bridging thick fibrous band which was continued to the chordae tendinae of the tricuspid valve. In the left ventricle, endocardium of the margin of defect was thickened and contracted towards the defect. In the

Fig. 3. (a) Right side view of the ventricular septal defect (arrow mark). Note prominent thickening around the defect. Ver. indicates a verrucous vegetation on the tricuspid valve.

(b) Left side view of the ventricular septal defect (arrow mark). There is a contracting tendency of the fibrous tissue around the defect.

Fig. 4. Microscopic view of the left ventricular side of small ventricular septal defect. Note focal endocardial fibroelastosis. $40\times$, Weigert van Gieson's stain.

right ventricle, fibrous endocardial thickening around the defect was strikingly thick, involving some chordae tendinae attached to the tricuspid valve. The tricuspid valvular tissue itself was also thickened around the defect especially at the commissure between the anterior and medial leaflets. A large relatively fresh organizing thrombus was formed at the commissure of the anterior and inferior leaflets (Fig. 3). A linear whitish endocardial thickening which started at the defect was oriented to the thrombus on the tricuspid valve. The histological examination revealed focal endocardial fibroelastosis around the defect (Fig. 4) and a few tiny valve-shaped projections of fibrous tissue in the defect swaying to right side. The tricuspid thrombus showed progressing organization with profuse appearance of polymorph-nuclear leukocytes, accumulation of red cells and exudation of fibrin.

Pathologic diagnosis: 1) Small ventricular septal defect situated at the pars membranacea with spontaneous narrowing tendency, 2) subacute bacterial endocarditis of the tricuspid valve.

DISCUSSION

The outlook for longevity of the cases with ventricular septal defect has been supposed poor, only 18% of cases having reached 30 years of age—a prognosis that is much worse than that of atrial septal defect or patent ductus, but better than that of Fallot's tetralogy.²⁾ On the other hand, the longest survival of ventricular septal defect cited by Selzer was the age of 79. It was also suggested that smaller defects may have no effect on circulatory dynamics and promise better prognosis unless a relatively high susceptibility (20%) to endocardial bacterial infection causes death of the cases.¹⁾ In our country, the authors could find no record of ventricular septal defect of the age of more than 60 during recent 7 years (1958–1964).⁷⁾ It is highly possible that our case is the oldest case with ventricular septal defect reported in Japan and also is one of rare cases in the world because there were only a few such cases reported by Mark et al.⁶⁾ and Bloomfield.⁵⁾

It has been well known that the life expectancy was undoubtedly enhanced by spontaneous closure of ventricular septal defects^{6), 8), 9)} whatever the closing process was; fibrous hyperplasia due to hemodynamic stress, adherence of the valvular tissue or bacterial or rheumatic inflammation.¹⁰⁾

In our case, the ventricular septal defect was small and was spontaneously narrowing due probably to persistent hemodynamic stress. Smaller defect and greater interventricular pressure gradient could make more accelerated shunting blood stream even though the shunting volume was not so much. The accelerated blood flow should reduce its side pressure to stimulate sub-endothelial connective tissue as well as abundant turbulent flow around the defect could. The focal endocardial thickening, valve-like structure in the defect^{11), 12)} and a linear flow line in the right ventricle might have resulted from

these kinds of phenomena. Furthermore, the accelerated narrow jet stream could make "jet impact"¹³⁾ wherever it hits. If the impact energy was large enough to give some harmful effect to coating tissue of the endocardium, bacterial infestation might easily occur at the impact point. In our case, elevated systemic blood pressure might increase the interventricular pressure gradient which made more rapid shunting stream to stimulate spontaneous narrowing of the defect and to intensify the jet impact to the opponent side. In addition, the contracting fibrous tissue around the small defect could make a directional change of the harmful jet stream to hit the tricuspid valve where the subacute bacterial endocarditis started. This hypothesis was endorsed by Bloomfield's description that small ventricular septal defect less than 3 mm. in diameter could be spontaneously narrowed and fibrous proliferation and frequently complicated with subacute bacterial endocarditis.⁵⁾ It was also noteworthy that many reported cases in the aged were complicated with systemic hypertension²⁾ just like this case.

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