Patent Ductus Arteriosus with Infective Endocarditis at Age 92

Tadashi Satoh and Naoki Nishida

Abstract

This report describes a 92-year-old woman patient with patent ductus arteriosus (PDA). She seems to be the oldest patient with PDA hitherto reported in the medical history. She developed infective endocarditis (IE) and congestive heart failure, and died at the age of 92. At autopsy, the PDA was found to the left of the origin of the left subclavian artery. Both left and right ventricles were hypertrophied with markedly dilated pulmonary arteries. IE involving the aortic valve extended to the sinus of Valsalva and pericardium, inducing pericarditis and cardiac tamponade. IE also resulted in systemic septic embolization.

Key words: patent ductus arteriosus, infective endocarditis

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Introduction

PDA is the failure of the duct between the pulmonary artery and aorta to close (Fig. 1). The duct normally closes within 3 weeks after birth. PDA is more frequently found in premature infants than in full-term babies, and more often in women than men (1). Most patients with PDA die before the age of 50 years unless treated surgically (2). Some patients, however, survive with little disability, and a few live a normal life span (3, 4). The oldest PDA patients in the literature were an 80-year-old woman in Japan (5) and a 90-year-old man in the U.S.A. (6). To our knowledge, the present patient is the oldest patient with PDA complicated with multivalvular disease to be reported in the literature.

Case Report

The case report of this patient was already reported in detail (7), and her brief medical record is as follows. She was born in 1905 and in 1970, she was examined at the Satoh Medical Clinic, Toyonaka, Osaka for a routine physical examination, and was diagnosed with multivalvular disease and was treated accordingly thereafter. At the age of 77, she was admitted to the National Cardiovascular Center, Osaka, Japan because of dyspnea on exertion and edema. The diagnosis of PDA and multivalvular disease (aortic stenosis, and mitral and tricuspid regurgitation) were made. The patient was thin, small and kyphotic. Her height was 140 cm, and her weight 35 kg. Her blood pressure was 160/84 mmHg, and her pulse was 72 beats/min. A cardiac examination revealed marked cardiomegaly, a grade 3 continuous murmur best heard at the 2nd intercostal space along the left sternal border, and a few live a normal life span (3, 4). The oldest PDA patients in the literature were an 80-year-old woman in Japan (5) and a 90-year-old man in the U.S.A. (6). To our knowledge, the present patient is the oldest patient with PDA complicated with multivalvular disease to be reported in the literature.

She developed several episodes of heart failure which were treated medically. Surgical management was not performed because of her age. She continued to do well until late in 1997 when, at the age of 91, she again began to develop congestive heart failure and was again admitted to the National Cardiovascular Center. Soon thereafter, however, her general condition deteriorated with low grade fever. Her level of alertness was reduced and she lapsed gradually into
unconsciousness. She had marked azotemia (serum urea nitrogen 95 mg/dl) and thrombocytopenia (platelet count 13,000/mcl), although her leucocyte count was within normal limits. She soon fell into a coma and her urine output decreased. She died on November 25, 1998, at the age of 92 years, four months, and 4 days.

**-autopsy report-

Patent ductus arteriosus was found between the left pulmonary artery and the descending aorta (Fig. 6). The inside diameter of the ductus was 5 mm. The heart weighed 450 gm. Both atria and right and left ventricles were dilated. The left ventricle was moderately hypertrophied and the right

**-figure 1-


**-figure 2-

Phonocardiographic tracings. A continuous murmur (CM) present at the pulmonary artery area peaked around the time of the second heart sound.

**-figure 3-

Chest x-ray showing an enlarged left ventricle occupying the apex with a dilated pulmonary trunk. Aortic knuckle is calcified.
Figure 4. ECG illustrated atrial fibrillation and ventricular hypertrophy.

Figure 5. Continuous wave Doppler flow in the ductus was recorded from the parasternal short-axis view. Flow above the baseline indicated flow from the aorta to the main pulmonary artery (upper recording). Color flow image of the parasternal short-axis view at the base demonstrated the ductus flow from the descending aorta (DA) to the main pulmonary artery (MPA)(lower). AO: aorta

ventricle was much more hypertrophied suggesting the existence of significant pulmonary hypertension. Histologically, the finding of the lung consisted of Heath-Edwards grade 1; (8) i.e., medial hypertrophy of muscular pulmonary arteries and muscularization of pulmonary arterioles. At the aortic end of the ductus, the ostium exhibited atherosclerosis. On the other hand, the pulmonary artery was markedly dilated. The aortic valve leaflets were thickened, fibrotic, and calcified. The aortic valve showed mild stenosis and was surrounded by yellowish, flattened atheromatous plaques. An infection involving the calcified aortic valve extended distally to the sinus of Valsalva through the left atrial wall and reached the pericardium, inducing pericarditis and cardiac tamponade (hemopericardium) (Fig. 7). Coagulated vegetations of infective endocarditis were noted on the mitral anterior leaflet, which was dome shaped without shortening. Proliferation of many gram-positive cocci with destruction of the aortic valve was detected. The vegetations of the infec-

Figure 6. The patent ductus arteriosus (PDA) is found between the left pulmonary artery (PA) and the descending aorta (DA).
Figure 7. Infective endocarditis of the aortic valve, showing massive destruction of leaflets with vegetation. Aortic annular abscesses extend distally into the left atrial wall and the pericardium, inducing pericarditis and cardiac tamponade (arrow) after rupture (small arrow). AO=aorta, PA=pulmonary artery, AV=aortic valve, LCA=left coronary artery.

Table 1. Eight Patients with PDA ≥ 75 Years of Age (5-7, 16-20)

<table>
<thead>
<tr>
<th>First Author (Year)</th>
<th>Age/Sex (Year)</th>
<th>Pulmonary Artery (s/d) (mmHg)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fishman (1951)</td>
<td>75/F</td>
<td>–</td>
<td>Am Heart J 41: 762, 1951.</td>
</tr>
<tr>
<td>Present case (1997)</td>
<td>91/F</td>
<td>–</td>
<td>–</td>
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F: female, M: male, s: systolic pressure, d: diastolic pressure.

tive endocarditis involving both aortic and mitral valves were the sources of emboli resulting in systemic septic embolization or so-called shower emboli. Emboli were noted in various organs including the thyroid, adrenal, kidneys, liver, and myocardium.

Discussion

The PDA is the third most common congenital cardiovascular anomaly, comprising approximately 10% of congenital anomalies or about 1 or 2 in 3,000 live births (7). PDA was first described in the third century by Galen (9). Gibson noted it to be associated with a continuous murmur (10). By 1938, advances in surgical techniques enabled Gross and Hubbard to perform the first surgical closure (11). Today, presentation in adult life is rare since the lesion is usually diagnosed and closed surgically in infancy or early childhood. Marquis et al (12) reported that 804 patients with PDA were seen in Edinburgh and 37 of them reached the age of 50. Campbell found that 34% of PDA patients died before the age of 40 and 61% before the age of 60 (2). In Japan, 5 PDA patients over 60 years of age have been reported and all were woman (5). The oldest known man patient with PDA lived to the age of 90 years in the USA (4); the oldest woman patient lived to the age of 80 years in Japan (5). We gathered information on PDA patient who lived >75 years of age reported between 1951 and 1996 (Table 1). The mean age of the patients was 83 years; 7 were women and 1 was a man. The woman to man ratio was high; it reflects the tendency for women with PDA to survive longer than men with PDA. These reports demonstrate that PDA is compatible with survival to an old age, but the natural history of PDA much more commonly involves substantial morbidity and mortality in an early to midlife. Hemodynamically significant adult PDA has typically been associated with inexorable cardiac disorder, including congestive heart failure, infective endocarditis, and pulmonary hypertension. Investigators found that atrial fibrillation, advanced left ventricular hypertrophy, and marked cardiomegaly were common in those elderly patients.

When viewed from the clinical course of the present patient, the diagnosis of PDA was not made until she was 76 years old, and the characteristic symptoms of aortic stenosis first appeared when she was 83. IE complicating PDA is now a rare occurrence (14). The basis of this IE was due to multivalvular disease (Table 2). IE primarily affects cardiac valves (15). Cardiac conditions that cause turbulent flow across a valve have been found to predispose patients to IE.
Table 2. Findings of Chest X-ray, ECG, and Echocardiogram

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<tr>
<td>X-ray (CTR)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Aortic regurgitation</td>
<td>66%</td>
<td></td>
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<tr>
<td>Aortic stenosis</td>
<td></td>
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<tr>
<td>Mitral regurgitation</td>
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<tr>
<td>Tricuspid regurgitation</td>
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<tr>
<td>PA systolic pressure</td>
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Table 3. Predisposing Conditions Associated with an Increased Risk of Endocarditis

<table>
<thead>
<tr>
<th>More common</th>
<th>Less common</th>
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<tbody>
<tr>
<td>Mitral valve prolapse</td>
<td>Rheumatic heart disease</td>
</tr>
<tr>
<td>Degenerative valvular disease</td>
<td>Hypertrophic obstructive</td>
</tr>
<tr>
<td>Intravenous drug use</td>
<td>Cardiomyopathy</td>
</tr>
<tr>
<td>Prosthetic valve</td>
<td>Pulmonary–systemic shunt</td>
</tr>
<tr>
<td>Congenital abnormalities</td>
<td>Coarctation of the aorta</td>
</tr>
<tr>
<td>(valvular or septal defect)</td>
<td>Cyanotic congenital heart disease</td>
</tr>
</tbody>
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(Insert Table 3 here)

Degenerative mitral and aortic leaflets were markedly affected in this case. Complications of IE include congestive heart failure, emboli, myocardial abscess, bacteremia, multiple organ failure, and death.

As shown in the autopsy, abscesses developed direct extension of active infection into the rings of supporting connective tissue around the valves. From there, abscesses extended into the adjacent atrial wall and ruptured into the sinus of Valsalva and the pericardium, inducing pericarditis and cardiac tamponade (hemopericardium). Vegetations of IE are a source of emboli, particularly during the active phase of the disease. Embolization is clinically evident in 11 to 43% of patients (14); however, the pathologic evidence of emboli at autopsy is found more frequently (45 to 65%) (13). Silver noted that emboli from left-sided endocarditis passed into the systemic circulation whereas those from right-sided endocarditis passed into the lungs, and larger vegetations (≥10 mm) are also associated with a higher frequency of emboli (13). Her final cause of death was sepsis and heart failure.

As to her long-lived PDA, even though the patient had such a serious cardiac handicap and high risk with PDA and multivalvular disease, medical management including the strict adherence to medication and her lifestyle, particularly her dietary habits had all contributed significantly to her longevity.

Acknowledgement

Most importantly, the authors thank Dr. C. Yutani, Chief, Division of Clinical Laboratory, National Cardiovascular Center, Osaka, Japan, for the autopsy report. Without the assistance acknowledged above, there would be no case report.

References

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