



## Case Report

## Sudden infant deaths from undiagnosed ventricular septal defect – Report of two autopsy cases



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## ABSTRACT

Ventricular septal defect (VSD) has a relatively non-aggressive clinical course; either spontaneous closure or causing congestive heart failure treatable with surgical intervention. We present two autopsy cases of sudden infant deaths from clinically undiagnosed VSDs. Case 1 was an 18-day-old boy. As the deceased coughed and became limp after feeding, he was carried to a hospital. Heart murmur was not auscultated there, so he was brought back to home. He presented severe dyspnea and then he was pronounced dead the next day. Case 2 was a 3-week-old boy. Any abnormality was pointed out at physical examination, although his mother felt his wheeze. He developed respiratory arrest at home in the morning and then he was confirmed dead at the hospital. Heart weights of these babies were heavier than mean weights of each normal development. There were perimembranous VSDs in both cases. Histology revealed that the pulmonary arterial walls were thickened. We diagnosed the cause of death in these cases was cardiac collapse with pulmonary hypertension due to VSD. Congenital heart diseases can be diagnosed as early as before birth, because echocardiograph and fetal echography are prevalent in these days. Most VSDs can be noticed by systolic murmur even today. We consider that the failure of initial clinical diagnosis of VSD in primary physical assessment could lead unexpected sudden death. These two cases reminded us to the importance of auscultation which is conventional but as one of the indispensable measure to find a clue for the congenital abnormality.

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## 1. Introduction

Ventricular septal defect (VSD) is one of the most common congenital cardiac diseases. Some authors recently reported higher incidence as 43.6–46.3 [1,2], although the incidence of VSD have been reported between 2 and 6 per 1000 live births in the past [3,4]. Researches in Japan have found 6.0–24.4 [5–8]. Majority of VSD cases has relatively non-aggressive clinical courses; either spontaneous closure or causing congestive heart failure treatable with surgical intervention. Systolic heart murmur usually triggers an awareness of VSD [9]. Some patients, on the other hand, can be diagnosed as VSD earlier using echocardiography or lately after showing symptoms of heart failure. Here we report two autopsy cases of VSD that were not diagnosed antemortem.

## 2. Cases

## 2.1. Case 1

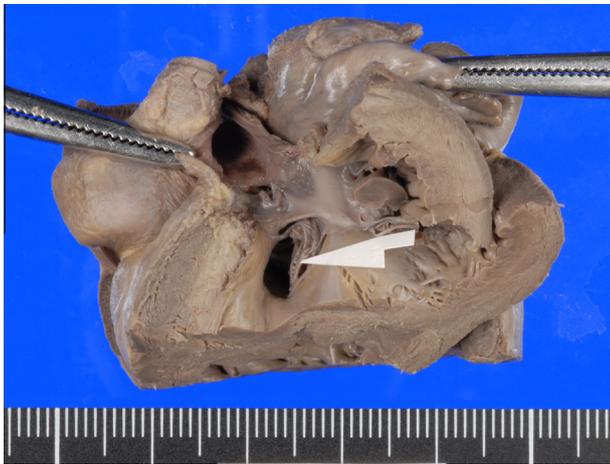
The patient was an 18-day-old boy. He was born at 40 weeks and 2 days of gestation with a weight of 2904 g, and had no notable

previous or family history. He experienced coughing after feeding in his 17th day. He was carried into an emergency unit because he manifested weakness. Heart murmur was not auscultated during physical examination according to the medical record. Any problem was not pointed out except tachypnea and peripheral coldness. His mother breastfed him twice after getting home, coming back on the same day. The next day, he cried. He hardly had any feeding due to dyspnea. When he was hospitalized again, he was with cardiopulmonary arrest. He was announced dead after an hour despite a resuscitation.

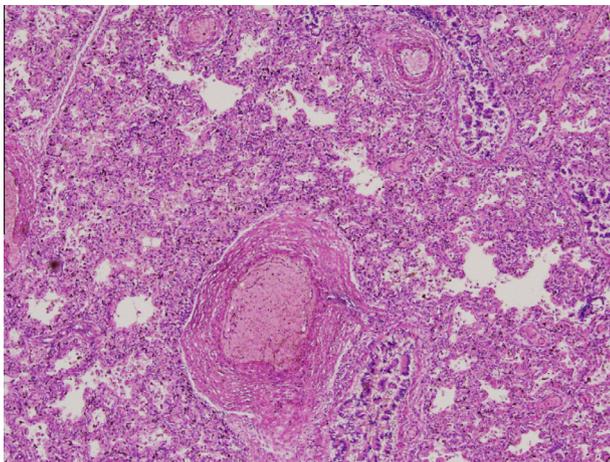
A medicolegal autopsy was performed approximately 30 h after death. He was 51 cm tall and weighed 3.0 kg. Physical constitution and nutrition were within normal limits. Light reddish-purple postmortem lividity was highly expressed on his back. There was no injury except small superficial abrasion in his face and some needle marks in his extremities. Cerebrospinal fluid was clear and light yellow in color. The left and right pleural cavities contained 5 and 6 ml of cloudy orange colored fluid, the pericardial sac contained 7 ml, and there was 15 ml in the peritoneal cavity. Main organs were generally engorged with blood. The left and right lungs weighed 32 and 51 g, respectively. The heart weight was 42 g. The left ventricle measured 0.6 cm in thickness and the right 0.4 cm. The diameter of aortic valve was 0.45 cm. The right

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**Fig. 1.** Case 1. Perimembranous ventricular septal defect (VSD) sized 0.7 cm × 0.6 cm.



**Fig. 2.** Case 1. Microscopic finding of the lung (H&E staining, ×40). The media of pulmonary arterioles was thickened.

ventricle was moderately dilated. There was a perimembranous VSD sized 0.7 × 0.6 cm (Fig. 1). Cardiac blood was liquidity without clot. Histological examination revealed severe congestion of the lungs. The media of pulmonary arterioles was thickened (Fig. 2). The iron staining (Berlin blue staining) of macrophages in the lungs was negative. Ventricular muscles in both sides, especially right ones were thickened. Endocardiums of both the ventricles and septum were mildly thickened. Myocardial abnormality such as myocarditis was not observed. The sinus node, atrioventricular node and the bundle of His were all normal. The liver was markedly congested. There were a few small lipid droplets in hepatocytes. The brain, thyroid gland and the kidney were normal expect mild congestion. No appreciable bacteria was detected in cultivation tests of blood and spinal fluid. There was no significant increase in titers of major antibodies in serum. Serum procalcitonin level was below 0.02 ng/ml and CRP concentration was 0.1 mg/ml.

## 2.2. Case 2

The patient was a 3-week-old boy. He was born at 38 weeks and 3 days of gestation with a weight of 3016 g. The baby and his mother were discharged from the hospital on his 5 days of life. Any abnormality was not pointed out during pregnancy or at physical examinations after birth. His mother noticed his wheeze



**Fig. 3.** Case 2. Perimembranous ventricular septal defect (VSD) sized 0.3 cm.

in several days after discharge. In his 24th night, he slept flat on his back. His mother breastfed once at midnight. She noticed his respiratory arrest next morning. Although he was taken to a hospital by ambulance and cardiopulmonary resuscitation was performed, he was confirmed dead.

A medicolegal autopsy was performed approximately 1 and half day after the death. He was 52 cm tall and weighed 3.4 kg. Physical constitution and nutrition were within normal limits. Reddish-purple postmortem livid was moderately observed on his back and limbs. There was no injury except needle marks at his limbs. Cerebrospinal fluid was clear with slight red. The left and right pleural cavities contained 4 and 2 ml of light reddish brown and light yellow brown colored fluid, respectively. Pericardial sac contained 2 ml of light reddish brown colored fluid. There was 5 ml of light brown fluid in the peritoneal cavity. The left and right lungs weighed 44 and 50 g, respectively. The heart weight was 36.6 g. The left ventricle wall measured 0.7 cm in thickness and right 0.5 cm. There was an ostium secundum-type atrial septal defect (ASD) sized 0.5 cm. The diameter of aortic valve was 0.51 cm. There was a perimembranous VSD sized 0.3 cm (Fig. 3). Cardiac blood was liquidity without clot. Histology revealed marked thickening of the media of pulmonary arterioles. The iron staining (Berlin blue staining) of macrophages in the lungs was negative. Ventricular muscles, especially right ones were thickened. Conducting system including atrioventricular node was normal.

Arachnoid membrane of the brain was thickened and the endothelial cells formed cubic. Liver was almost normal except mild congestion. There was no aberration in other organs. There was no inflammation in his lungs although serum influenza A and RS viral antibody titers elevated mildly. No appreciable bacteria was detected in cultivation tests of bold and swab from the nose and the bronchus.

## 3. Discussion

Growth and development of both the patients were sufficient. No lethal injuries or major abnormalities except VSD were found. There were no significant results in serum viral antibody titers and cultivation tests of blood and spinal fluid or swab from the respiratory tract, as far as we investigated. No inflammatory lesion was found in any organ in either of the cases. Possibility of fetal infection was thus excluded.

The hearts weighed 42.0 g in case 1 and 36.6 g in case 2, which were heavier than mean heart weight in one-month-old infants (23 g) [10] or in infants with body weight of 3000–3499 g (28.9 g) [11]. In case 1, the VSD area was 0.33 cm<sup>2</sup>. Body surface

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