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Sudden infant death due to Lactococcal infective endocarditis

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ABSTRACT

Infective endocarditis (IE) of infants is rare, most of which occur associated with congenital heart disease or its cardiac surgery. We experienced a case of sudden death of a four-month-old male infant without congenital heart disease. It was elucidated by postmortem examination that the dead had suffered severe IE, which led him to death. In the microbiological genetic analysis using histological section, the pathogen causing inflammation in the present case was identified as *Lactococcus lactis* subspecies, although Staphylococci have been reported to be common and important one. Previously reported infectious diseases by *Lactococcus lactis* subspecies were all adult cases and this is the first report of an infantile death due to Lactococcal IE according to our knowledge. Any fatal disease may be included in sudden death cases targeted for forensic autopsy, even if it is rare. It is expected for forensic pathologists that they note such case and share each experience among themselves and other medical fields to develop a strategy for prevention.

1. Introduction

Infective endocarditis (IE) of children is much less than that of adults. It has been reported that most of the pediatric IE patients suffer congenital heart disease, the state of its surgery, or other preexisting heart disease, and infantile IE without congenital heart disease or preexisting heart disease is to be extremely rare [1–6]. From the aspect of etiologic agent, Lactococcal IE is very rare and only few adult cases were reported so far [7–9], while most frequent one is *Staphylococcus* species [1,2,4–6,10–12].

The hospitalization rate for IE of children was variable among published series [1–5]. Day et al. described that infants had formed one of the bimodal peaks (<1 year, $n = 545$, 36.8%) in the series of 1480 IE cases under 21 years of age [1], while Ishiwada et al. showed that infants and neonates were only 16 (8.5%) of 188 IE children [5].

In this article, we described a case of a four-month-old male infant without congenital heart disease as well as any preexisting heart trouble who had suddenly died due to Lactococcal IE just before hospitalization.

2. Case history

The patient was four-month-old male infant. He was born healthily, with 48 cm height and 2982 g body weight, without any physical abnormality at 37 weeks of pregnancy. He was the second child of the parents, and his brother had been grown healthy. The baby had been fed only with mother's milk and grew well. He took medical examination monthly and was vaccinated regularly. The last medical check was taken 10 days before death and he had no problem.

On the day of the death, his mother took the baby to a doctor at 10 a.m., because of cough and slight diarrhea continuing for a couple of days. According to the medical record, his body temperature was 36.8 °C at the hospital. Levine I systolic murmur that had not been audible at the past medical checks, was noticed. The respiratory sound was clear, and abdomen was soft. No clinical signs of Kawasaki's disease were documented. Cardiothoracic ratio was 48% in expiratory phase and no remarkable findings were found on chest X-ray. Color Doppler-echocardiograph showed mosaic signal as suspected regurgitation of the mitral valve. White blood cell counts, platelet, kalium and C-reactive protein had increased, while red blood cell count and creatinine had decreased. (Table 1). Although the medical doctor documented the necessity of following-up the baby's clinical course, only some medicines for cold (an expectorant, antibiotics (Cefditoren pivoxil), syrup for cough) were prescribed and the patient went home.

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Table 1
Laboratory data at the first-visit hospital.

White blood cell count	106.0	$10^2/\mu l$
Neutrophils	43.6	%
Lymphocytes	51.6	%
Others	4.8	%
Red blood cell count	367	$10^4/\mu l$
Platelets	64.0	$10^4/\mu l$
C-reactive protein	3.46	mg/dl
Kalium	5.9	mEq/l
Creatinine	0.21	mg/dl

Around 9 p.m., body temperature of the baby rose to 37.7 °C and his respiratory condition went worse. The parents took him to another hospital in neighboring city taking for two hours by their own car, because the hospital he had visited in the morning and other clinics in his residential area had denied accepting the ill baby in that night. He fell in cardiopulmonary arrest just on arrival at the hospital. Resuscitation was immediately performed, but not successful.

3. Autopsy findings

The deceased was well developed infant with 65 cm height and 6.5 kg body weight. Neither facial congestion nor conjunctival petechiae were observed. No injury was found at both external and internal examination. Exudates in left thoracic, right thoracic and abdominal cavities were 20 ml, 30 ml and 100 ml, respectively. The heart was 30 g in weight, without anomaly, such as ventricular and atrial septal defect, patent ductus arteriosus, patent foramen ovale and so on. No gross aneurysm of coronary artery was observed. There were some small patchy subpericardial bleedings on left heart. Right atrial and ventricular cavities showed moderate dilatation (Fig. 1). Ruptures of tendinous cords of the mitral valve were not noticed. In the stomach and duodenum, there was a small amount of coffee-like residuum but no curds. A little curds was found from lower part of the jejunum to the rectum. All other organs had no remarkable findings except moderate congestion.

In the microscopic examination, severe endocarditis was observed on tricuspid and mitral valves (Fig. 2). The main inflammatory cells were neutrophils, though lymphocytes were also intermingled. Atrial endocarditis was stronger and more widely spread in the left than the right side. A small fibrin thrombus infiltrated with many neutrophils was adhered on the right atrial endocardium. Vegetation was not observed. Such acute inflammation expanded to inferior area of atrial septum, membranous part of ventricular septum and superior area of muscular part of ventricular septum (Figs. 3 and 4). Bacteria could not be detected on the histological section by Gram staining. No arteritis was observed in coronary arteries. There were a few macrophages in the pulmonary alveoli, but not inflammatory cells. Scattered lymphocytic infiltration was seen in sinusoids in the liver. Other organs had nothing worthy to be recorded histologically.

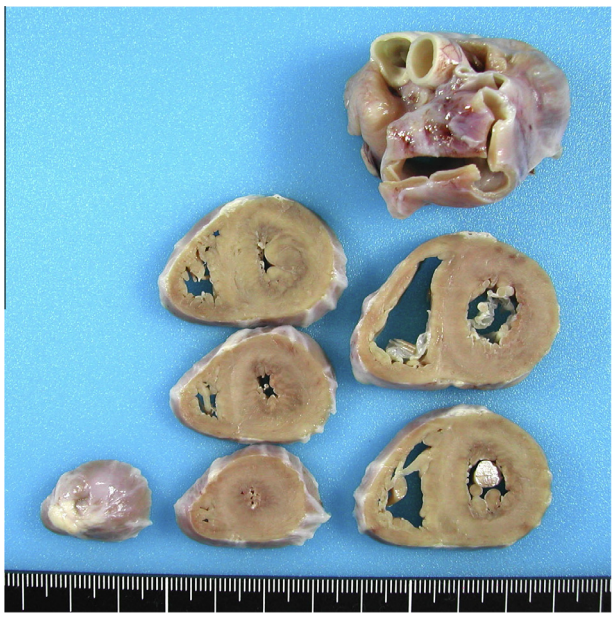

Fig. 1. Macroscopic finding of the heart. The right ventricle was expanded moderately in a sectioned surface.

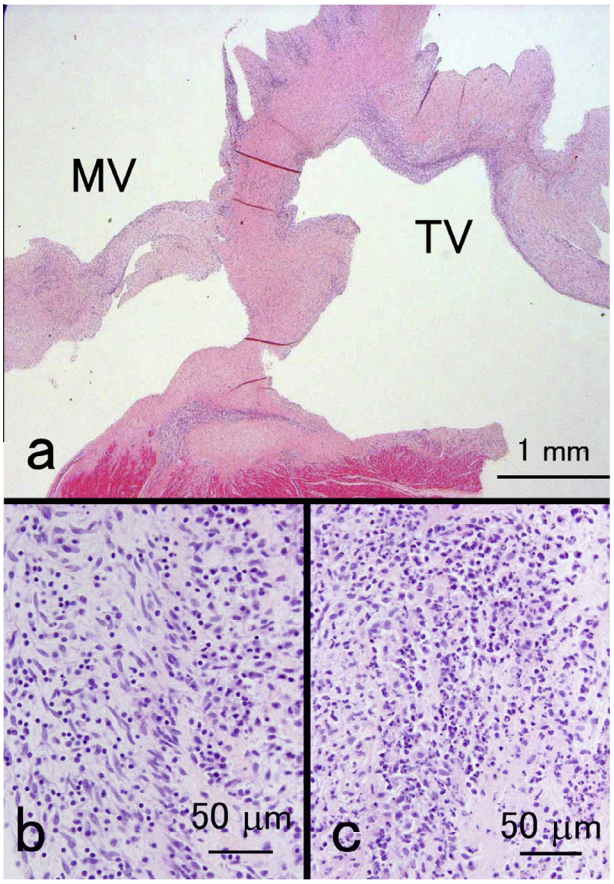

Fig. 2. Histological findings of mitral and tricuspid valves. Both valves were slightly thickened by acute inflammation (a). Amounts of neutrophils were infiltrated on mitral valves (b) and tricuspid valve (c) (MV: mitral valve, TV: tricuspid valve) (Hematoxylin–Eosin (HE) staining).

4. Laboratory examinations

Antigens of Influenza virus (A and B), RS virus and Adenovirus were not detected from the surface of pharyngeal and respiratory mucosa by immunological examination. Titer of anti-viral antibodies in the serum for Adenovirus, Parainfluenza virus (types 1–3), RS virus, Coxsackie virus (group A type 9, group B types 1–6), Mumps virus, Measles virus, Herpes zoster virus, Herpes simplex virus, Cytomegalovirus, and Echo virus (types 5, 11, 16, 18, 19, 22 and 24) were all within normal limits. In the microbiological examination by culture, only *Acinetobacter baumannii* and *Serratia marcescens* were detected from left and right bronchus respectively.

Bacterial polymerase chain reaction (PCR) examination and DNA analysis for identification of the pathogen were performed as follows: genomic DNA was obtained from the