Pediatr Cardiol 22:247–249, 2001
DOI: 10.1007/s002460010214

Pediatric Cardiology

Ventricular Aneurysm Complicating Neonatal Coxsackie B4 Myocarditis

S. Hoi-shan Chan, K.S. Lun
Department of Pediatrics, Queen Elizabeth Hospital, 30 Gascoigne Road, Kowloon, Hong Kong, China

Abstract. A premature neonate suffered from disseminated Coxsackie B4 infection. Myocarditis and a coexisting persistent ductus arteriosus became complicated with recurrent atrial tachycardia and severe heart failure. She survived with satisfactory cardiac function. Ventricular aneurysm was detected on follow-up echocardiography.

Key words: Coxsackie B — Myocarditis — Atrial tachycardia — Ventricular Aneurysm — Neonate

Ventricular aneurysms are well documented in adults, yet they are rarely reported in children [5]. We describe a premature neonate with disseminated Coxsackie B4 viral infection and myocarditis complicated by recurrent attacks of atrial tachycardia. She survived with recovery of ventricular function. Left ventricular aneurysm was demonstrated on follow-up echocardiography.

Case Report

The patient's mother suffered from low-grade fever, headache, and myalgia 1 week before delivery.

The patient was a 2380 g para 2 female baby born at 34 weeks' gestation by normal vaginal delivery. Apgar score was 5 and 8 at 1 and 5 minutes, respectively. She had mild respiratory distress after birth. Initial chest roentgenography was normal. She was treated with intravenous antibiotics empirically.

She developed fever on day 3. Bacterial cultures of blood, urine, and cerebrospinal fluid (CSF) were negative. Viral culture of CSF specimens, taken on day 3 and day 6, yielded Coxsackie B4 virus. Both CSF biochemistry and white cell counts were normal. Viral titers to enterovirus taken on day 3 and day 12 were 1:10 and 1:10, respectively. The results were possibly complicated by a large volume of blood product infusion for treatment of disseminated intravascular coagulopathy and a long period of continuous hemofiltration. Specific antibody titer to Coxsackie virus was not performed because of inadequate samples. Maternal viral serologic studies were performed at 2 and 4 weeks after onset of patient's illness, which demonstrated persistent elevated titers to Coxsackie B4 at 1:320 and 1:320, respectively. Maternal throat and stool viral cultures were negative.

Correspondence to: K.S. Lun

At day 6, the patient developed an acute narrow complex tachyarrhythmia of 300 beats/min. This was treated with adenosine. Echocardiography demonstrated a structurally normal heart, a 3-mm persistent ductus arteriosus (PDA), left ventricular end-diastolic dimension (LVEDD) of 1.8 cm, and ejection fraction (LVEF) of 63%. Both serum creatine kinase and CK-MB drawn immediately after the cardioversion with adenosine were elevated at 1410 IU/L and 33.5 μg/L (normal < 9.4), respectively. Serum creatine kinase increased to a maximal level of 2350 IU/L at day 13 and returned to a normal level of 52 IU/L at day 18. She suffered from another tachyarrhythmia at a rate 270 beats/min on day 7. Adenosine failed to terminate the tachycardia and cardioversion with 2 J direct current was required. Her illness was complicated with development of disseminated intravascular coagulopathy on day 7. Mechanical ventilation was needed as she developed severe tachypnea and poor perfusion. She required inotropic support using dopamine and dobutamine. Repeat echocardiography showed a dilated left ventricle with poor contractility (LVEDD = 2.2 cm; LVEF = 34%) and small pericardial effusion. No obvious regional wall motion abnormality was noted. Coronary anatomy appeared normal. During the subsequent 9 days, she developed nine attacks of tachyarrhythmia. These were treated with intravenous amiodarone, oral digoxin, and DC cardioversion. Review of ECGs with adenosine administration and esophageal ECG demonstrated the tachyarrhythmias were atrial tachycardias with rapid ventricular response.

The baby developed acute renal failure and required 14 days of continuous arteriovenous hemofiltration. Her ventricular function gradually recovered over 2 weeks. Echocardiography performed at day 20 revealed a LVEF of 58%. Ligation of PDA was performed on day 33 and she was weaned off the ventilator on day 38.

Echocardiography at 4 weeks of age revealed echodense areas involving the posterobasal region and part of the papillary muscle of the mitral valve (Fig. 1). The echodense areas showed diminished contractility. Left ventricular contractility was satisfactory on other regions. All previous echocardiography did not showed abnormality where the echodense areas developed. The last normal echocardiography at day 20 showed no abnormality of the ventricular wall. She was discharged at 2 months old.

She remained asymptomatic and required no medication since 3 months old. Serial echocardiography demonstrated ventricular aneurysm at left posterobasal segment with paradoxical expansion during systole (Fig. 2). Contractility of other ventricular segments appeared normal. She had no neurological sequelae up to 12 months follow-up.

Discussion

Ventricular aneurysm occurs rarely in children and mainly in those with infarction associated with coronary

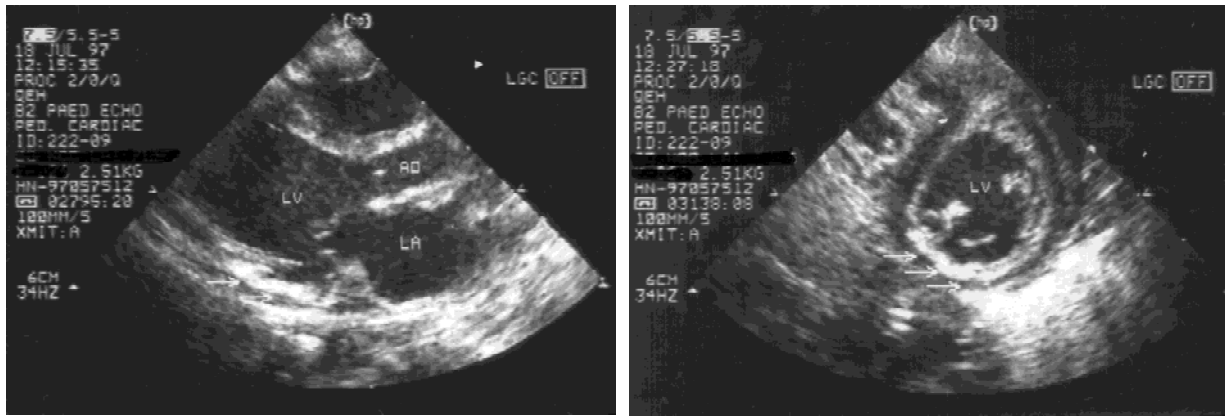

Fig. 1. Echocardiography (**Left,** parasternal long axis view; **right,** parasternal short axis view), performed at 4 weeks of age, demonstrated echodense areas over the posterobasal region of left ventricle and part of the papillary muscles of the mitral valve. *AO,* aorta; *LA,* left atrium; *LV,* left ventricle.

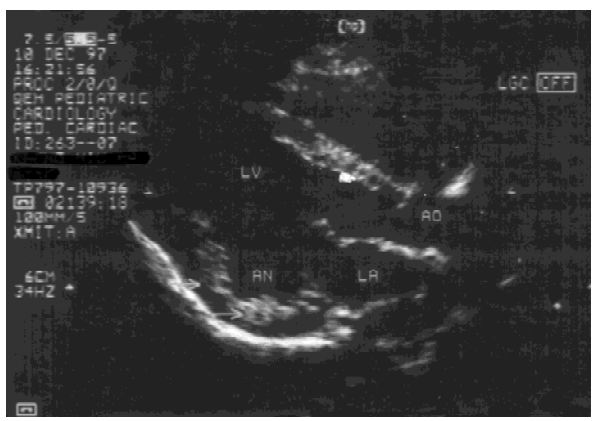

Fig. 2. Echocardiography (parasternal long axis view), performed at 6 months of age, showed ventricular aneurysm at posterobasal region of left ventricle. On real time, the aneurysm showed paradoxical expansion during systole. *AN,* aneurysm; *AO,* aorta; *LA,* left atrium; *LV,* left ventricle.

artery trauma or anomalies [5]. This is the first known reported case of ventricular aneurysm formation after viral myocarditis in a neonate. The formation of the ventricular aneurysm was demonstrated on serial echocardiography (Figs. 1, 2).

Ventricular aneurysm after viral myocarditis is rare in humans. Goudevenos et al. reported the first case of ventricular aneurysm after Coxsackie B4 myocarditis in a 39-year-old man with no evidence of myocardial infarction [2]. The relationship has been illustrated in two animal studies [1, 3]. Coxsackie B1 or B4 virus induced severe transmural myocarditis in the animals, resulting in aneurysmal dilatation of a fibrosing scar. Coronary vessels were patent and without vasculitis in the affected animals.

Although our patient had good recovery of ventricular function, ventricular aneurysm may result in complications. Possible complications include ventricular arrhythmia, mural thrombus formation, or progressive heart failure [3, 7]. Aneurysm may result in hemodynamic deterioration and may require surgical therapy. Although the incidence of these complications in ventricular aneurysm after myocarditis is unclear, our patient requires close follow-up. Fortunately, there has not yet been evidence of these complication.

Coxsackievirus B neonatal myocarditis is frequently associated with arrhythmia during the acute phase. Ventricular arrhythmia and supraventricular tachycardia have been described [4, 9]. Shah et al. reported the first case of atrial flutter after neonatal Coxsackie B2 myocarditis [8]. Our case further supports the association of arrhythmia with viral myocarditis. We believe that the ventricular aneurysm resulted from direct myocardial damage after myocarditis rather than the effect of recurrent atrial tachycardia.

Severe Coxsackie B infection in neonate is associated with high death rate. In Kibrick's review of 45 cases of neonatal Coxsackievirus B myocarditis, only 12 survived [6]. Our patient demonstrates the unique complication of recurrent attacks of atrial tachycardia and formation of left ventricular aneurysm after Coxsackie B myocarditis.

References

1. El-Khatib MR, Chason JL, Lerner AM (1979) Ventricular aneurysm complicating Coxsackievirus group B, types 1 and 4 murine myocarditis. *Circulation 59*:412–416
2. Goudevenos J, Parry G, Gold RG (1990) Coxsackie B4 viral myocarditis causing ventricular aneurysm. *Int J Cardiol 27*:122–124

3. Hoshino T, Matsumori A, Kawai C, Imai AJ (1984) Ventricular aneurysms and ventricular arrhythmias complicating Coxsackie virus B1 myocarditis of Syrian golden hamsters. *Cardiovasc Res 18*:24–29
4. Jack I, Townley RRW (1961) Acute aspetic myocarditis of newborn infant, due to Coxsackie viruses. *Med J Aust 2*:265–268
5. Johnsrude CL, Towbin JA (1998) Myocardial infarction in childhood. In: Garson A Jr, Bricker JT, Fisher DJ, Neish SR (eds) *The science and practice of pediatric cardiology*, 2nd edn, vol II, Williams & Wilkins, Baltimore, p 1973
6. Kibrick S (1961) Viral infections of the fetus and newborn. *Perspect Virol 2*:140–159
7. Leachman RD (1990) Acquired coronary artery disease. In: Garson A Jr, Bricker JT, McNamara DG (eds) *The science and practice of pediatric cardiology*, 1st edn, Lea & Febiger, Philadelphia, p 2283
8. Shah SS, Hellenbrand WE, Gallegher PG (1998) Atrial flutter complicating neonatal Coxsackie B2 myocarditis. *Pediatr Cardiol 19*:185–186
9. Yokota Y, Wada M (1989) Accelerated ventricular rhythm associated with myocarditis in a neonate. *Pediatr Cardiol 10*:178

DOI: 10.1007/s002460010225

Around *PediHeart*: Neonatal Bradycardia

We have all probably had the experience of being asked by neonatal or general pediatric colleagues to evaluate a term baby with a bradycardia. One *PediHeart* member described an otherwise well-appearing infant who had a heart rate while asleep of 55 bpm. An electrocardiogram (ECG) demonstrated a sinus bradycardia but was otherwise normal. The heart rate was 198 bpm when the baby was crying. The prenatal history and the infant's examination were normal. He was concerned and wanted to know if *PediHeart* readers had a sense for how slow was too slow?

The general consensus was that some newborns demonstrate higher vagal tone and/or less adrenergic tone than their peers, especially during sleep resulting in a lower heart rate. They believed that this was normal and did not portend a future catastrophe. Their impression was that nothing need be or should be done. Several respondents raised the long QT syndrome (LQTS) as a possibility, having seen similar presentations of LQTS in their own practice. In addition to the ECG already done, they recommended repeating one several weeks after discharge and checking the electrocardiograms of other family members. Other thoughts in addition to the long QT syndrome included neonatal hypothyroidism, central nervous system abnormalities, and unrecognized prenatal depression.

I liked this question because it concerned a common clinical situation that doesn't get much attention in the usual cardiology literature. Certainly a heart rate of 55 bpm in a term newborn falls well outside the normal range [1] and would prompt some type of evaluation. It's also interesting that we are asked to evaluate neonatal bradycardia in the 70–80 bpm range fairly often despite evidence that term newborns have a lower vagal to sympathetic tone ratio and a lower variability in heart rate than older infants or children [2]. There still seems to be a lot we don't know about this "routine" scenario.

Francis McCaffrey, M.D.
PediHeart Editor

References

1. Davignon A, Rautaharju P, Boiselle E, et al. (1979–80) Normal ECG standards for infants and children. *Ped Cardiol 1*:123–131
2. Massin M, von Bernuth G (1997) Normal ranges of heart rate variability during infancy and childhood. *Ped Cardiol 18*:297–302