

Asymptomatic Right Ventricular Perforation by a Temporary Transvenous Pacing Lead in an Infant

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A 3-month-old male infant received transvenous temporary pacemaker for acquired complete atrioventricular block. However, a right ventricle perforation by the pacing lead was found incidentally when the patient was receiving permanent pacemaker implantation nineteen days after temporary pacing. There was no pacemaker dysfunction, active bleeding or hemopericardium. The temporary pacing lead was extracted and the site of perforation was sutured. This case is unique because of the patient's unusual clinical presentation, and we suggested that echocardiography should be performed cautiously to check the pacing catheter tip daily, even if asymptomatic and/or normal function of pacemaker.

Key Words: Complete atrioventricular block • Right ventricle perforation • Transvenous pacing lead

INTRODUCTION

Acquired complete atrioventricular block (CAVB) in infants and children is rare, and congestive heart failure can occur rapidly after acute manifestation of CAVB.^{1,2} The emergency treatment for CAVB is infusion of adrenergic agonists and temporary pacing. Then, a permanent pacemaker is implanted if there is persistent symptomatic CAVB. Herein, we report a rare complication of temporary pacing for CAVB in a 3-month-old infant.

CASE REPORT

A 3-month-old male infant had a birth history of

G1P1, gestation age of 38 weeks, birth weight of 2900 gm and no perinatal insult. The child's mother did not have any systemic or autoimmune diseases. The infant, who had decreased urine output, was inactive, irritable, crying and pale in appearance for one day, and was thereafter referred to our hospital, a tertiary referral center. After his arrival at our pediatric emergency room, his heart rate was approximately 60-70 beats per minute, blood pressure was 61/31 mmHg and capillary refilling time was more than 3 seconds. Electrocardiogram on admission showed CAVB with wide QRS wave (Figure 1A). Subsequent to preliminary patient testing, laboratory data revealed Creatine-phospho-kinase and Creatine-phospho-kinase MB form of 1138 and 111 u/l, respectively, troponin-I of 24 ng/ml, prohormone of brain natriuretic peptide of more than 35000 pg/ml and the absence of anti-Ro/SSA and anti-La/SSB antibodies; additionally, patient viral studies were all negative. The results of studies for maternal autoimmune diseases, including C3, C4, antinuclear antibody, rheumatoid factor and extractable nuclear antigen (SSA/anti-Ro, SSB/Anti-La, Smith and ribonucleic protein) were negative. He was admitted to our neonatal intensive care unit under the diagnosis of acute myocarditis with CAVB and cardiogenic shock. Echocardiography upon admission re-

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vealed profound bradycardia, cardiomegaly, left ventricular systolic dysfunction with ejection fraction/fractional shortness (EF/FS) of 31%/14% when the patient arrived and 52%/24% one day after hospitalization, moderate tricuspid/mitral regurgitation and minimal pericardial effusion. The diagnosis and treatment were based on the guidelines of the Taiwan Society of Cardiology for Heart Failure.³ Atropine, dopamine, isoproterenol and epinephrine were initially administered, but bradycardia and hypotension persisted. Transcutaneous pacing was performed, and then a 4F PACEL bipolar temporary pacing catheter (St. Jude Medical, Minneapolis, MN, USA) was placed via the left femoral vein under fluoroscopic guidance and continuous electrocardiogram monitoring by an experienced pediatric cardiologist. The position of the catheter tip and the pacemaker function were normal as established by posterior-anterior view of chest radiograph (Figure 1B). Patient chest radiograph (lateral view) that was obtained initially in the catheterization laboratory revealed neither identified abnormalities nor an unduly tense catheter (Figure 1C). The patient heart rate was determined

to be approximately 100-120 beats per minute, with a pacing power of 3 mA and a capture pacing threshold of 2 mA. His vital signs became stable after the initiation of transvenous temporary pacing; however, CAVB persisted more than 2 weeks. Follow-up echocardiography showed left ventricular systolic function with EF/FS of 74%/36% after use of a temporary pacemaker commenced. The function of the temporary pacing was normal during this period, and a permanent pacemaker was implanted nineteen days after transvenous temporary pacing. During the procedure, the tip of the temporary pacing wire was seen incidentally out of the right ventricular anterior wall, after the pericardial membrane was opened (Figure 2). However, there was no active bleeding or hemopericardium, and the temporary pacing lead was extracted and the site of perforation was sutured. Follow-up echocardiography showed no pericardial effusion, and left ventricular systolic function with EF/FS of 79%/46% after permanent pacemaker was implanted. The patient was discharged to his home 5 days after operation. Follow-up echocardiography 3 months later revealed normal left ventricular systolic function without cardiomyopathy.

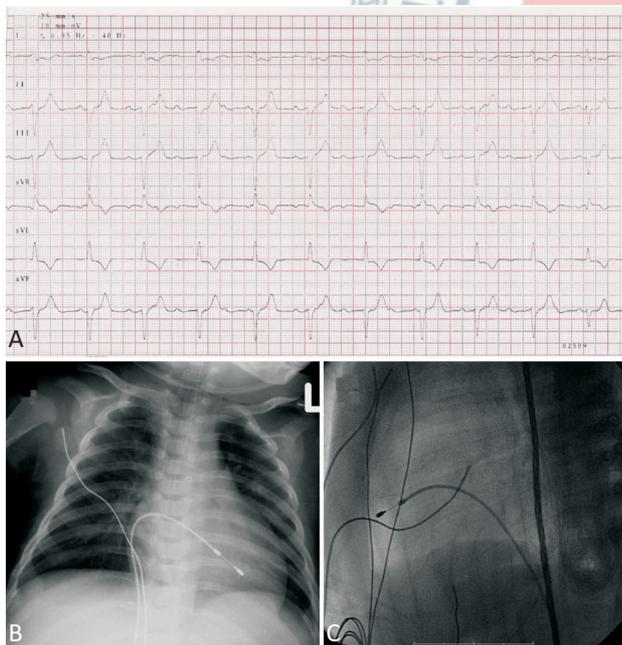


Figure 1. (A) The electrocardiogram on admission showed complete atrioventricular block with wide QRS wave, and the heart rate was 67 beats per minute. (B) The comparison with our previous chest X-ray before permanent pacemaker implantation revealed the position of the catheter tip was normal and unchanged. (C) The lateral view of chest X-ray revealed neither identified abnormalities nor too tense catheter.

DISCUSSION

Myocardial perforation has been reported to occur in 0.06% of patients receiving transvenous temporary

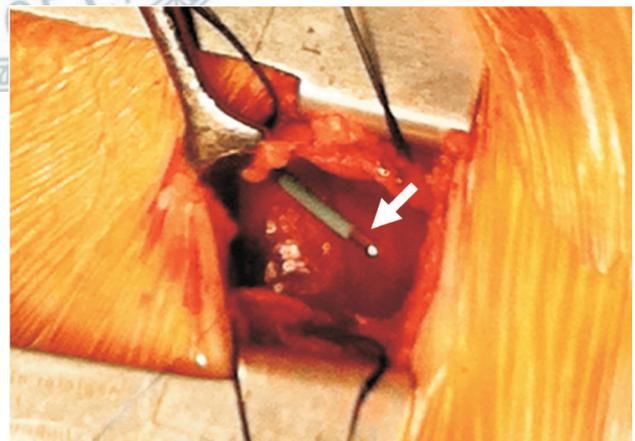


Figure 2. The tip of the temporary pacing wire was seen incidentally out of the right ventricular anterior wall (white arrow) after the pericardial membrane was opened. There was no active bleeding or hemopericardium.

pacing.⁴ Although this complication usually occurs during implantation or the first 24 hours after implantation, in this patient it occurred in a subacute condition, from 24 hours to one month after temporary pacing was initiated. The causes of leads perforation typically include early perforations which generally occur during intracardiac catheter manipulations, or during dislodgement of intracardiac catheters. The late stage cardiac perforation is characterized by the presence of necrotic tissue surrounding the catheter, which itself results in perforation of the heart chamber.⁵ The risk factors of cardiac perforation after pacemaker implantation include old age, use of corticosteroids, temporary pacing and active fixation leads.⁶ Lead perforation also can occur in infants and children and may result in morbidity or mortality.⁷ Furthermore, hemopericardium with cardiac tamponade or hemothorax could follow the myocardial perforation. Chest radiography is the first choice for detection of this complication, but misdiagnosis might occur if the lead tip is contained within the cardiac silhouette.⁸ Two-dimensional echocardiography or multidetector computed tomography can be used to confirm myocardial perforation.⁹ In our patient, no pericardial effusion was detected by transthoracic echocardiography during the period when temporary pacing was used. We did not use echocardiography routinely to check the tip of the pacing catheter because the pacemaker function was normal and there were no symptoms of myocardial perforation. Because the wall of the right ventricle is thin in young infants, a pacing lead may cause myocardial perforation. Although this complication is rare, it can lead to potentially fatal complications. Pacemaker lead perforation is a recognized complication of lead implantation. Therefore, we suggest that transthoracic echocardiography be performed cautiously,

with the tip of the pacing catheter checked daily, even if the patient is asymptomatic and/or the pacemaker function is normal.

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