CASE REPORT

Ectopic Atrial Tachycardia and Reversible Myocardial Dysfunction in a Child with Enterovirus 71 Infection: A Case Report

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The authors reported a rare case of ectopic atrial tachycardia (EAT) with reversible myocardial dysfunction associated with enterovirus 71 (EV71) infection. A previously healthy 11-year-old boy presented with progressive heart failure. An initial ECG revealed a regular narrow QRS tachycardia with abnormal P wave morphology. Echocardiography showed severely impaired left ventricular function (LVEF 20%). Viral study (PCR) in stool was positive for EV71. Treatment with inotropic support, amiodarone, and carvedilol resulted in gradual restoration of sinus rhythm over 10 days. Cardiac function returned to normal within three months. EV71 can cause EAT along with other complications and should be considered in patients with persistent tachycardia and cardiac dysfunction. Recognizing this arrhythmia led to proper management to control ventricular rate, then termination of the tachycardia and gradual restoration of ventricular function to normal.

Keywords: Enterovirus 71; Ectopic atrial tachycardia; Myocardial dysfunction; Myocarditis; Heart failure

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Ectopic atrial tachycardia (EAT) is one of the most common arrhythmias causing ventricular dysfunction in children. In most case of EAT, no identifiable etiology is found. While viral myocarditis is often thought to be the cause of EAT in some patients, there is little evidence of the association. The authors report a reversible myocardial dysfunction and heart failure concurrent with EAT in a child with enterovirus 71 (EV71) infection.

Case Report

The present study was approved by the Institutional Review Board (IRB), Faculty of Medicine, Chulalongkorn University (IRB No.033/64). A previously healthy 11-year-old boy presented with progressive dyspnea for one week with a history of an upper respiratory tract infection two weeks earlier. Physical examination on admission showed an alert child with tachypnea, tachycardia with a heart rate of 180 to 190 bpm, and normal blood pressure at 98/60 mmHg. There was lateral shifting of the apical cardiac impulse without significant cardiac murmur, crepitation in both lungs, slight hepatomegaly, and pitting edema on both legs. Neurological evaluation was normal.

Chest X-ray showed cardiomegaly with pulmonary venous congestion (Figure 1A). Echocardiography revealed normal cardiac anatomy including coronary arteries, marked dilatation of left ventricle with severely impaired left ventricular function as the left ventricular ejection fraction (LVEF) was 20%, and no pericardial effusion (Figure 1B). Electrocardiography (ECG) showed a regular narrow QRS tachycardia with long R-P and short P-R, rate 180 to 190 bpm, abnormal ST-T change in left precordial leads and an abnormal P wave morphology, consistent with EAT (Figure 2). Blood tests showed elevated troponin-T of 0.08 ng/mL when the normal is 0.013 to 0.025 ng/mL, and NT-pro BNP of 8,395 pg/mL when the normal is 25 to 160 pg/mL. Viral study (PCR) in stool was positive

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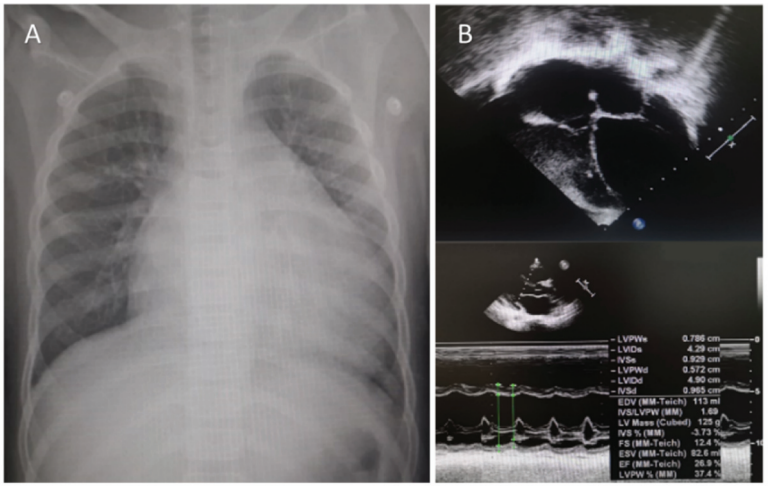

Figure 1. (A) Chest X-ray on admission showed cardiomegaly and pulmonary congestion. (B) Echocardiography on admission showed marked left ventricular (LV) dilatation (LV end-diastolic dimension 4.9 cm=+2.4 SD) with impaired left ventricular function, LVEF around 20%.

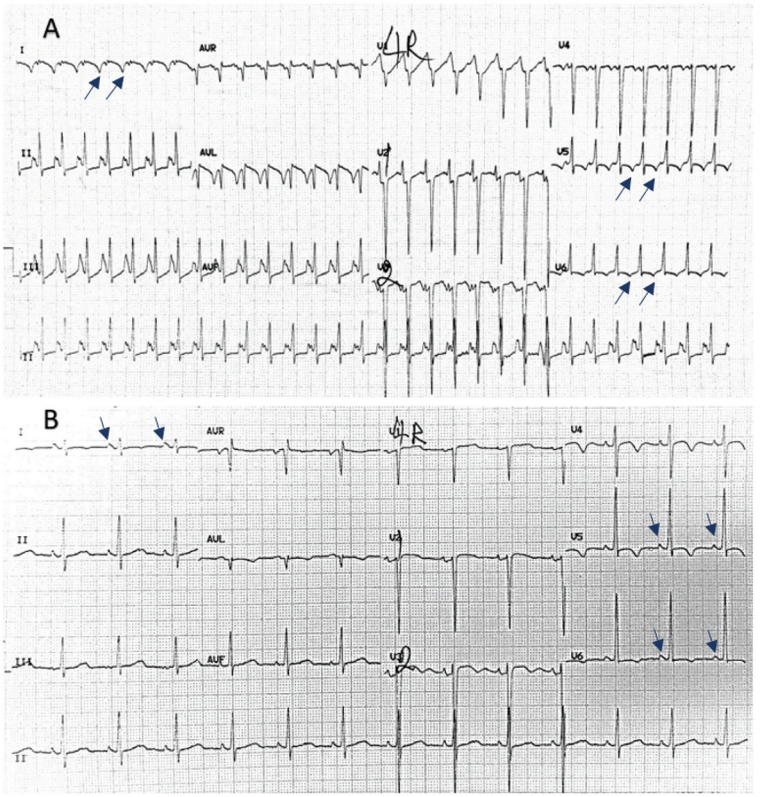

Figure 2. (A) The electrocardiography (ECG) on admission showed narrow QRS tachycardia with 1:1 AV association, long RP, short PR and abnormal P wave morphology (downward in lead I, V5, V6; arrows) consistent with ectopic atrial tachycardia (EAT). (B) ECG after restoration to sinus rhythm (note the P wave was upright in lead I, V5, V6; arrows).

for EV71. The patient was diagnosed as having acute viral myocarditis and EAT associated with EV71 infection.

Treatment

The patient was treated with dobutamine and milrinone infusion together with furosemide injection

to improve the symptoms of heart failure. Adenosine injection resulted in transient atrioventricular block without termination of the tachycardia. Amiodarone was given with 5 mg/kg bolus infusion in 60 minutes, followed by continuous infusion starting at 5 mcg/kg/minute. Six hours after amiodarone initiation, when the rhythm abruptly changed from EAT rate 170 bpm to sinus tachycardia rate 120 bpm, the patient suddenly developed hypotension, agitation, and signs of poor perfusion. Stopping amiodarone and increasing dobutamine restored his clinical stability. Amiodarone infusion was then restarted at lower dose with the aim to slow down the ventricular rate, resulting in gradual control of the ventricular rate and complete elimination of EAT 10 days later. Enalapril and carvedilol were later added after hemodynamic stability. Serial echocardiography revealed gradual improvement of ventricular function and all inotropic agents could be weaned off after three weeks of hospitalization. He was discharged one month after the episode with amiodarone, carvedilol, enalapril, furosemide, and spironolactone.

Follow-up and outcomes

The patient was doing well during the follow-up visits. The ventricular function returned to normal in three months after treatment. All antiarrhythmic medications were gradually weaned off over a two-year period without a recurrence of the EAT or ventricular dysfunction.

Discussion

Well known as the cause of several childhood diseases such as hand-foot-mouth disease and herpangina, enterovirus is one of the common etiologies of viral myocarditis in children[1]. EV71 is unique among all strains of the enterovirus as several epidemics in the past indicated that it is associated with varieties of cardiopulmonary and neurologic manifestations, such as encephalitis, myocardial dysfunction, non-cardiogenic pulmonary edema, and autonomic dysfunction[2,3]. While pulmonary edema is the most common cause of death in children with EV71 infection, the causes of this are still unclear[2]. Myocardial dysfunction, non-cardiogenic pulmonary edema secondary to encephalitis or surge of catecholamine from autonomic dysfunction have been suggested as the etiologies[4-7].

EAT is a term that generally refer to atrial tachycardia as a result of increased automaticity of atrial tissue. In cardiac cellular electrophysiologic term, this is a result of increased slope of the phase 4 in the cardiac action potential. In case of incessant EAT, the patient can develop heart failure as a result of tachycardia-induced myocardial dysfunction. The treatment of choice in symptomatic patients is to control ventricular rate or EAT with either medications or RF ablation. EAT is one of the most common sustained tachyarrhythmia causing heart failure in children and the prognosis is generally good with proper treatment[8].

In most case of EAT in children, no etiology is found, and the condition can sometimes be self-limited, especially in infants[8]. Acute viral myocarditis has been implicated in some patients with EAT or junctional tachycardia[1,9]. To the authors' knowledge, there has been no previous report of EV71 as a cause of EAT in children and there is a clear clinical implication for recognizing this arrhythmia in patients with EV71 infection. As EV71 has been shown to cause myocardial dysfunction, pulmonary edema and autonomic dysfunction, elevated heart rate in these patients may be mistaken for sinus tachycardia and EAT may be missed if ECG is not carefully looked at. In these patients, it may be difficult to distinguish direct myocardial injury from myocarditis versus tachycardia-induced cardiomyopathy, or both, as the cause of myocardial dysfunction, especially with elevation of cardiac troponin[10] like in the present case. Nevertheless, missing the EAT or not treating it would likely result in the delay or failure of improvement of ventricular function. An elevation of cardiac enzyme represents myocardial injury that can also be present in either myocarditis or persistent tachycardia as seen in various type of tachyarrhythmia[11]. As controlling heart rate is the treatment of choice for tachyarrhythmia-induced dilated cardiomyopathy, distinguishing between EAT and sinus tachycardia is important for the appropriate management, which may include antiarrhythmic medications or RF ablation. Gradual reduction of ventricular rate may be needed in some patients with severely depressed ventricular function to avoid hypoperfusion as a result of abrupt decrease in heart rate as illustrated in the present case.

Conclusion

EV71 infection can be the cause for EAT and myocardial dysfunction in children. Electrocardiography is the diagnostic key for EAT and should be carefully examined especially in patients presented with cardiac dysfunction and persistent tachycardia. Recognizing this arrhythmia led to proper management to control the rhythm and

to allow gradual restoration of ventricular function to normal.

What is already known on this topic?

EAT is an uncommon cause of tachyarrhythmia in children. There is no specific etiology found in most cases, however, EAT can occur in association with acute viral myocarditis. However, to the authors' knowledge, there has been no previous report of EV71 as a cause of EAT in children.

What this study adds?

The authors emphasized the clinical significance to recognize this tachyarrhythmia in the patients with EV71 infection particularly those presenting with persistent tachycardia and heart failure, as this is a treatable condition and failure to diagnose it would likely lead to a progressive heart failure, death, or the need for heart transplantation.

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Conflicts of interest

The authors declare no conflict of interest.

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