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Introduction

Lyme disease has been associated with junctional ectopic tachycardia (JET) and fascicular tachycardia in the past; however, we believe this case report is the first known reported case of a patient with Lyme disease presenting as fulminant myocarditis and cardiac arrest. Our patient is a previously healthy 12-year-old male subject who presented with cardiac arrest and JET, ultimately secondary to fulminant myocarditis from Lyme disease. With antibiotic and antiarrhythmic treatment, sinus rhythm returned, and ultimately he had full recovery of his cardiac function. His initial cardiac magnetic resonance imaging (MRI) showed diffuse delayed gadolinium enhancement along the interventricular septum, with near resolution 5 weeks later on repeat imaging. It is likely that edema and/or inflammation near the His bundle resulted in JET.

Case report

The patient is a 12-year-old previously healthy boy with a recent history of participation in an outdoor camp for 2–3 weeks who began to gasp for air while riding as a passenger in a car, with subsequent cyanosis and cardiac arrest, following participation in recreational outdoor activities earlier that afternoon. On arrival at a medical facility, cardiopulmonary resuscitation was initiated for pulseless arrest from ventricular fibrillation, with return of spontaneous circulation achieved after 8 minutes of cardiopulmonary resuscitation, including defibrillation, intubation, and intravenous (IV) epinephrine and lidocaine administration. After subsequent transfer to a pediatric intensive care unit at an academic center, he continued to suffer from extreme hypoxemia and challenging ventilation, with persistent tachyarrhythmias, concerning for JET with periods of fascicular tachycardia (Figures 1 and 2). Empiric ceftriaxone was initiated. He was transferred to our facility for possible extracorporeal membrane oxygenation support, and continued on high-dose dopamine, norepinephrine, and ceftriaxone. For acute management of the arrhythmia, he was started on procainamide 30 mcg/kg/min after a bolus of 5 mg/kg. He was later started on amiodarone 10 mg/kg/day infusions after loading doses were administered, for hypotension felt to be secondary to the procainamide. An echocardiogram identified normal segmental anatomy with severely depressed global left ventricular systolic function with an ejection fraction of 30%, and moderately depressed right ventricular systolic function. No regional wall motion abnormalities were noted. Brain imaging was concerning for global cerebral edema and moderate hypoxic injury to the basal ganglia, hippocampi, and primary motor and visual cortices.

With the addition of IV amiodarone, there was further improvement in his rate control and intermittent sinus rhythm, prompting a taper of procainamide over the following 3 days. Conversion to sinus rhythm and improvement in ventricular function occurred within 4 days of presentation, avoiding the need for extracorporeal membrane oxygenation support. A cardiac MRI supported the diagnosis of myocarditis with increased signal intensity on early gadolinium enhancement and late gadolinium enhancement imaging of the midanteroseptal segment and basal inferior segment of the left ventricle (Figure 3A–D). IV immunoglobulin was administered after obtaining the first cardiac MRI study. Viral polymerase chain reaction testing was negative for enterovirus, influenza, cytomegalovirus, and Epstein-Barr virus. Antibody testing for Lyme disease (enzyme-linked immunosorbent assay) was strongly positive for IgG antibodies, with a confirmatory IgM Western blot positive for Lyme disease. Ceftriaxone was continued for a 21-day treatment course. Vasoactives were weaned as his cardiac function recovered, with normalization of his ejection fraction within a week of presentation. He continued to suffer the ill effects of hypoxic ischemic brain injury, with altered mental status, agitation, and dysautonomia.

KEYWORDS Junctional ectopic tachycardia; Myocarditis; Lyme carditis; Sudden cardiac arrest; Cardiac MRI; Delayed gadolinium enhancement
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KEY TEACHING POINTS

- Junctional ectopy tachycardia is rare in children, especially when not associated with congenital heart surgery. Myocarditis should be considered in patients presenting with rare arrhythmias such as junctional ectopic tachycardia.
- Lyme disease is known to affect the atrioventricular node and can lead to junctional ectopic tachycardia.
- Junctional ectopic tachycardia associated with Lyme disease may have a good prognosis for the return of normal sinus rhythm, specifically after treatment with appropriate antibiotic therapy.

After discontinuation of procainamide, the IV amiodarone was transitioned to enteral dosing of 7.5 mg/kg/day with good rhythm control. No further arrhythmia was observed, with taper of the oral amiodarone over the course of 3 weeks. Repeat cardiac MRI demonstrated near-resolution of the previously noted early and late gadolinium enhancement (Figure 3E–H). The patient had normalization of his ventricular function with resolving abnormal findings on the repeat cardiac MRI, and no further arrhythmias after weaning off amiodarone. He continues to suffer from the effects of anoxic brain injury and requires long-term rehabilitation from his neurologic injury.

Discussion

Arrhythmias are common in acute viral myocarditis[1] and have been seen in up to 45% of patients.[2] Of these, nearly 80% of patients have ventricular tachycardia. In addition to ventricular tachycardia, ventricular fibrillation and complete heart block (CHB) are common, with supraventricular tachycardia, ectopic atrial tachycardia, and high-grade atrioventricular block seen less often.[2] In patients who survive acute fulminant myocarditis, resolution of their arrhythmia is common, with no inducible arrhythmias seen in a reported case series.[3] Prior case reports have described a neonate with

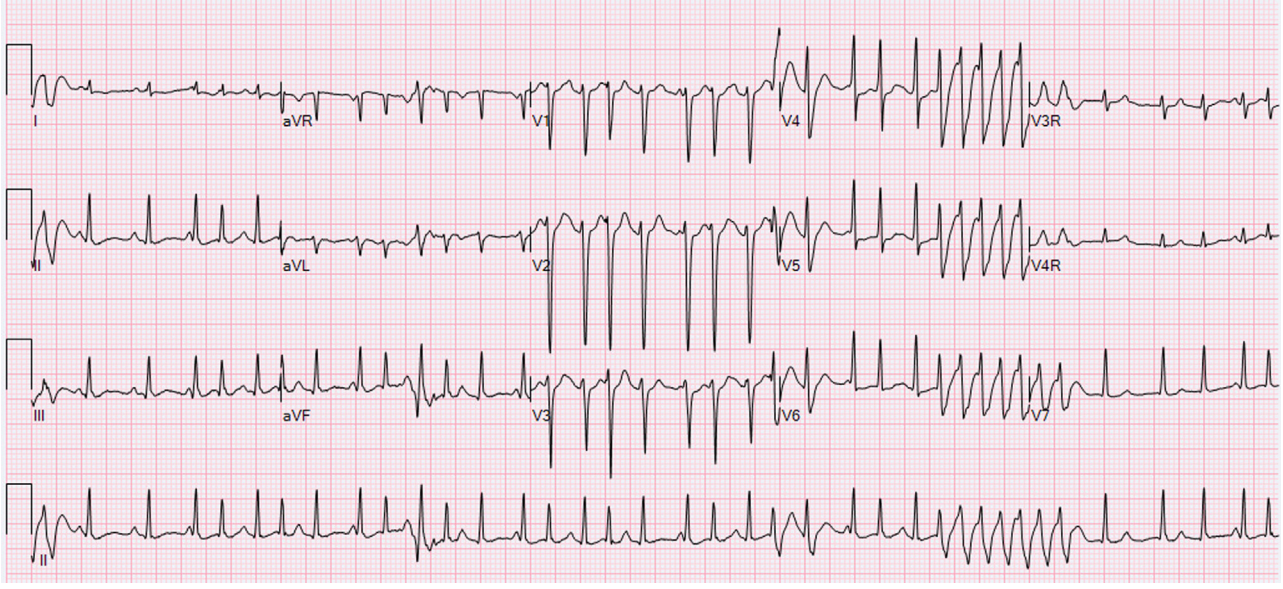

Figure 1 Electrocardiogram on initial presentation. Junctional ectopic tachycardia (JET) with aberrant intraventricular conduction vs independent fascicular tachycardia. Shortest cycle length during tachycardia was 160 msec. QRS morphology during JET was similar to that observed during sinus rhythm after resolution of tachyarrhythmia.

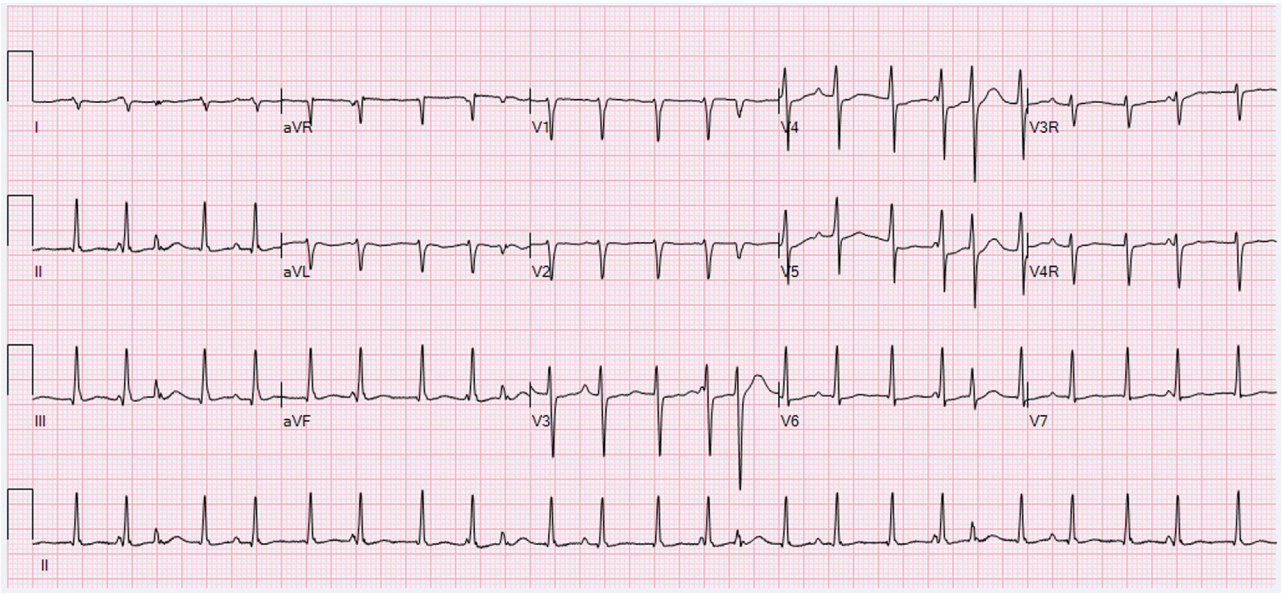

Figure 2 Electrocardiogram (ECG) with slow junctional ectopic tachycardia (JET). This ECG, obtained after the administration of amiodarone and procainamide, showed stabilization of the JET rate. AV dissociation with sinus capture beats, nonspecific T-wave changes, and prolonged QTc (484 msec) were evident.

enterovirus myocarditis and CHB that progressed to JET on an isoproterenol infusion,[4] Lyme carditis with initial fascicular tachycardia in a fatigued 42-year-old man,[5] and JET in an otherwise asymptomatic 3-year-old diagnosed with Lyme carditis.[6] To our knowledge, this is the first reported case describing JET as the predominant rhythm at acute presentation of fulminant myocarditis and out-of-hospital sudden cardiac arrest.

JET most commonly occurs in children during the early postoperative period after repair of congenital heart disease.[7,8] It is thought to be related to ischemia/reperfusion or physical injury to the cardiac conduction tissue related to prolonged operative times, and/or surgical trauma to the conduction system.[7] JET is seen less often as a congenital arrhythmia, but when present, has a strong association with cardiomegaly and heart failure.[9]

It is likely that JET developed in our patient secondary to myocardial inflammation near the His bundle, in a similar fashion to postoperative JET.[7,8] *Borellia burgdorferi* infection has a predisposition to the AV conduction axis, so that the most common arrhythmia is AV block. Inflammation in the AV node and intermittent AV block can be associated with JET. In fact, the occurrence of JET is a good predictor for spontaneous recovery of AV conduction following the onset of postoperative CHB.[10] Unique to our case in support of this theory was evidence of both early and delayed gadolinium enhancement on cardiac MRI,[11] seen scattered throughout the ventricular septum, including the basal septum. With resolution of the JET and normalization of left ventricular function, antiarrhythmic medications were successfully discontinued. Given that his JET resolved within 4 days of starting ceftriaxone, and his left ventricular function improved within a week of restoration of sinus rhythm, it is hypothesized that the arrhythmia resolution was predominantly owing to antibiotic treatment of Lyme carditis. Before a decision was made as to whether to implant a cardioverter/defibrillator device, a repeat cardiac MRI was performed. This study revealed near-resolution of the previously noted early gadolinium enhancement and late gadolinium enhancement imaging, supporting the decision to defer implantable cardioverter/defibrillator implant. In subsequent follow-up for 8 months, the patient has had no further episodes of arrhythmia and on last evaluation continued to have normal ventricular function.

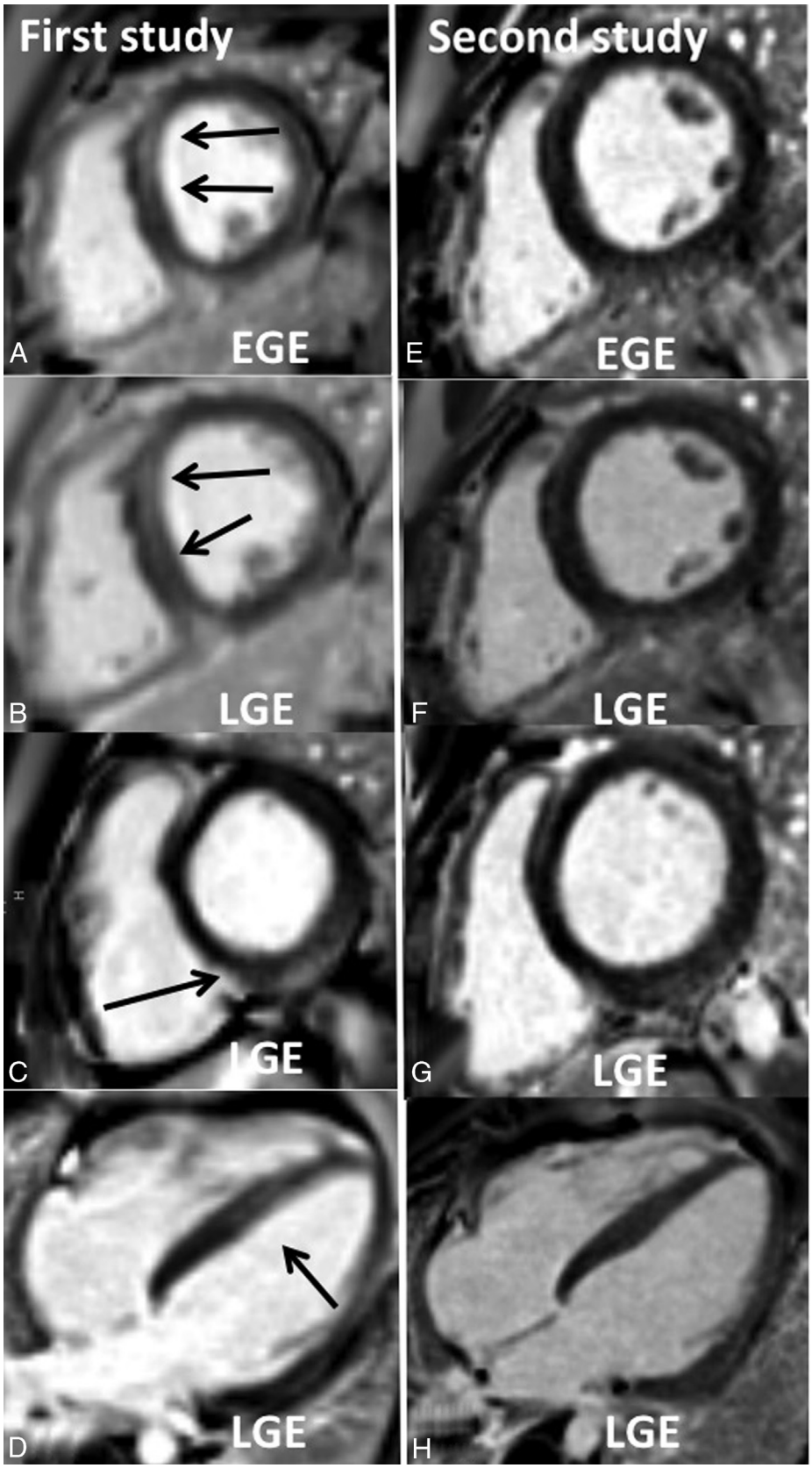

Figure 3 Magnetic resonance images on presentation (A, B, C, D) and 5 weeks after treatment with intravenous immunoglobulin and ceftriaxone (E, F, G, H). **A:** Early gadolinium enhancement (EGE) of the interventricular septum. **E:** Improved EGE of the interventricular septum. **B, C, D:** Late gadolinium enhancement (LGE) of the interventricular septum. **F, G, H:** Improved LGE of the interventricular septum.

Conclusions

Myocardial inflammation and edema in the area of the His bundle places a patient with myocarditis at risk for junctional/fascicular ectopic tachycardia, and may resolve with time, similar to postoperative JET. Tachycardia-mediated ventricular dysfunction should resolve with time after return of sinus rhythm, but over a more prolonged time period than observed in our patient.[12] A diagnosis of Lyme-associated myocarditis should be considered in patients with arrhythmias uncommon for patients with structurally normal hearts, such as JET and fascicular tachycardia.

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