Accelerated idioventricular rhythm (AIVR) is usually considered a benign arrhythmia in childhood. It is defined as 3 or more consecutive ventricular beats at a rate 10%-15% faster than the prevailing sinus rate. It is differentiated from other forms of ventricular tachycardia because of the benign course, lack of haemodynamic compromise and occurrence in the absence of structural heart disease. AIVR can be associated with increased vagal tone, pregnancy, and drug toxicity; most commonly however, it is seen in the absence of a cause or structural heart disease in children.

Case Report

A 15-year-old boy who was previously healthy apart from attention deficit hyperactivity disorder presented to the general pediatric team with a 48-hour history of high-grade fever, short-term memory loss, and acute confusional state. This occurred on a background of a 3-week history of bloody diarrhea. He was on guanfacine for his attention disorder. He was also a high-level athlete and was evaluated by a cardiologist 2 years ago for sinus bradycardia and was discharged from cardiology after electrocardiogram (ECG) and Holter with a diagnosis of sinus bradycardia with first-degree heart block. On examination at presentation, his higher functions were altered with bradyphrenia, poor short-term memory, and attention deficits, and significantly changed from baseline. No focal neurologic deficits were elicited. Cardiac examination was completely normal apart from the abrupt changes in heart rates that were haemodynamically well tolerated. His abdomen was soft and nontender.

On telemetry he was seen to have 2 patterns of heart rate (see Fig. 1, A and B): one at 45 beats per minute and the second around 75 beats per minute. The 12-lead ECG confirmed that he was alternating between sinus bradycardia with first-degree block (Fig. 1A) and AIVR (Fig. 1B). His echocardiogram demonstrated normal anatomy with flow visualized into both coronary arteries and normal biventricular function. All left ventricular (LV) dimensions on echocardiogram (ECG) and Holter with a diagnosis of sinus bradycardia.

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Ethics Statement: The research reported has adhered to the relevant ethical guidelines.

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See page 431 for disclosure information.
dL). Cardiac magnetic resonance imaging (cMRI) with contrast was performed to rule out cardiomyopathy, coronary anomalies, and myocarditis. cMRI showed mild LV dilatation and preserved ejection fraction of 70%, with no evidence of myocarditis, edema, or scarring of the myocardium. The LV end diastolic index volume was 113 cc/m² (upper limit: 104 mL/m²). This mild dilatation of the left ventricle was attributed to physiological compensation for bradycardia in an athletic heart. The heart rate during scan was sinus at 46/min. A positron emission tomography scan of his heart was not performed because of unstable neurologic findings and no anticipated change in management. An MRI of the brain showed scattered punctate foci of diffusion restriction, consistent with small vessel vasculitis. Colonoscopy was suggestive of ulcerative colitis. Intravenous methylprednisolone was started. There was immediate resolution of the AIVR, and the patient went into remission for his ulcerative colitis, with moderate improvement of his neurologic symptoms as well. Figure 1C shows the resolution of the inflammatory markers.

His neurologic symptoms recovered slowly with time, and at 8 months of follow-up, he is back to his baseline neurologically. The patient has had two 24-hour Holters thereafter: 3 months and 8 months after discharge. He has also had 2 outpatient ECGs and numerous clinical assessments by 3 clinical services, all of which have recorded normal heart rates and/or documented sinus rhythm. Throughout his course, he was not on any medications suspected to cause AIVR. The Holter 2 years ago was also negative for AIVR, suggesting that the presentation was exclusively associated with the acute onset of his inflammatory bowel disease (IBD).

Discussion

AIVR is usually a benign and well-tolerated arrhythmia. This arrhythmia is commonly noted during the reperfusion phase of myocardial infarction in adults. AIVR can occur frequently in pediatric hearts without any structural abnormalities in which case it usually carries a good prognosis.1,2 There have been a few case reports of AIVR having a malignant course; however, these children had confounding factors including status post cardiac arrest; having QT interval of 600 milliseconds or reduced ventricular function at presentation.3 Stress cardiomyopathy was excluded in the absence of specific

Table 1. The known causes of AIVR are listed

| Category          | Causes                                                                 |
|-------------------|------------------------------------------------------------------------|
| A                 | Idiopathic                                                              |
| B                 | Cardiac pathology                                                      |
|                   | Post-myocardial infarction                                              |
|                   | Post-cardiac surgery                                                    |
|                   | Cardiomyopathies                                                       |
|                   | Dilated cardiomyopathy                                                 |
|                   | Arrhythmogenic right ventricular cardiomyopathy                        |
|                   | Arrhythmogenic inflammatory cardiomyopathy                             |
| C                 | Drugs                                                                   |
|                   | Digoxin, desflurane, halothane, acetone                                 |

history, signs of shock, ECG signs of ischemia, abnormal echocardiogram, or cMRI. In our case, there was no known etiology for this rise in troponin, other than inflammation. It is clear that his AIVR, troponin, and CRP all followed a decreasing trend with initiation of treatment, suggesting a correlation between generalized inflammation and AIVR. As

Novel Teaching Points

- AIVR is considered a benign arrhythmia of childhood defined as 3 or more ventricular beats at a rate 10%-15% higher than the sinus rate.
- It may be seen in association with myocardial infarction, cardiomyopathies, drugs, or high vagal tone.
- AIC is a condition where systemic inflammation causes arrhythmias and reduction in cardiac function.
- AIVR may be seen in the AIC subgroup, and a careful search for inflammation needs to be done in the symptomatic patient presenting with AIVR.
- Resolution of AIVR may be seen with the resolution of systemic inflammation.

Figure 1. (A) Sinus bradycardia with first-degree heart block. (B) Wide complex rhythm with right bundle branch block and superior axis. Arrows mark dissociated sinus P waves. (C) Resolution of inflammatory markers with steroids. Arrow marks steroid administration.

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| C                 | Drugs                                                                   |
|                   | Digoxin, desflurane, halothane, acetone                                 |
the troponin normalized, the rhythm did so as well. IBD is known to be associated with heart failure, arrhythmia, and perimyocarditis. However, our patient had no evidence of pericardial irritation or myocarditis, recognizing that focal, subclinical myocarditis cannot be excluded. In arrhythmogenic inflammatory cardiomyopathy (AIC), arrhythmias can be both atrial and ventricular with increased arrhythmia burden seen during IBD flare-ups. Although atrial fibrillation is the arrhythmia commonly implicated, other arrhythmias including atrioventricular block, ventricular arrhythmias, and increased P and QT dispersions have been recorded in literature. Various pathologies like electrolyte disturbance, high circulating inflammatory mediators, changes to the autonomic nervous system and long term sequels on the myocardial structure have been postulated as possible mechanisms. Two studies looking at AIVR in the infant age group including one where the tachycardia was noted in utero reported excellent prognosis with spontaneous resolution with age. A ventricular rate of approximately 10%-15% above the sinus rate has been proposed as a diagnostic feature. The mechanism is believed to be due to excess automaticity originating from abnormal calcium channels affecting phase 4 of the cardiac action potential. AIVR in association with postoperative congenital heart disease also carries a good prognosis. The known causes for AIVR are listed in Table 1.

AIC is a term describing a group of patients presenting with nonischemic inflammation of the myocardium with reduced ejection fraction, documented ventricular arrhythmia, and changes on cardiac positron emission tomography scan. IBD is known to be associated with AIC. Our patient’s troponins were significantly elevated suggestive of myocardial damage. It is theoretically possible that the AIVR was pre-existing and undetected, but the relatively frequent assessments since the index hospitalization have not identified AIVR. To our knowledge, an association of AIVR with IBD has not been previously reported. The haemodynamics in our patient remained stable through the AIVR phase, and resolution of the arrhythmia was seen with resolution of the systemic inflammation.

Conclusion
AIVR may be a presenting rhythm in acute generalized inflammatory states like IBD. We postulate that the generalized inflammation may precipitate AIVR and that treatment of the inflammation leads to resolution of AIVR in these cases.

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