A young man with atrioventricular dissociation: something to worry about or just a false alarm?

Lim Lee Yee, MRCP

Department of Internal Medicine, Queen Elizabeth Hospital II, Kota Kinabalu, Sabah, Malaysia

SUMMARY

Atrioventricular dissociation is an abnormality of the cardiac electrical conduction when the atria and ventricles beat independently. It often manifests as a life-threatening bradycardia in complete atrioventricular heart block where the atria and ventricles beat asynchronously. This is usually caused by acute coronary events in the older population with cardiovascular risk factors and requires a prompt treatment to stabilize the hemodynamics by cardiac pacing. In rare cases, the atria and ventricles can beat synchronously despite atrioventricular dissociation. This type of atrioventricular dissociation occurs in younger population without cardiovascular risk factors especially in athletes. It is not life-threatening and usually does not require treatment or monitoring. We report a young man who presented with a syncopal attack due to a heat stroke after a marathon run, with an incidentally found atrioventricular dissociation in his electrocardiogram, which resolved by physical exertion.

INTRODUCTION

Syncope is a common presentation to the Emergency Department (ED) with a wide spectrum of possible aetiologies. The main aim of the initial approach to syncope is to distinguish it between life-threatening and benign causes of syncope.[1] Important aetiologies of syncope include cardiogenic causes such as cardiac arrhythmia and structural abnormality leading to cardiac outflow obstruction, and neurological causes such as seizure and vertebrobasilar vascular insufficiency, while vasovagal attack is one of the benign causes. Apart from history taking and physical examination, initial investigations for a syncopal attack consist of an electrocardiogram and a brain computed tomography to rule out cardiogenic and neurological causes respectively.

About 90% of patients with cardiogenic syncope have abnormal electrocardiogram findings, thus an electrocardiogram gives a good diagnostic value as an initial assessment tool for syncope.[2] Nevertheless, patients with non-cardiogenic syncope can have incidental abnormal electrocardiogram findings. It is important to rule out cardiogenic syncope as it is associated with an increase in all-cause mortality and morbidity. Further investigations with echocardiogram to look at structural abnormality, functional stress tests with electrocardiogram or echocardiogram to look at inducible ischemia, and Holter monitoring are crucial to confirm or rule out cardiogenic causes to guide the management of syncope.

CASE REPORT

A 17-years-old man presented with a syncopal attack after a marathon run. During the syncopal attack, he fell and landed with outstretched arms without any witnessed tonic-clonic movement of limbs or significant injury. He regained consciousness within a few minutes but could not recall any incident prior to the syncope. He was active in track and field for the past 3 years, with no medical illness and previous hospitalisation. He did not have any family history of heart diseases or seizures.

He had a brief period of confusion upon presentation to the ED, which resolved subsequently with a full Glasgow Coma Scale. His physical examination revealed that he was febrile with a body temperature of 38.5°C and tachycardic with a heart rate of 130 beats per minute, evidenced by sinus tachycardia on cardiac monitoring. His haemodynamic status was otherwise stable with a blood pressure of 132/56 mmHg and other systemic examination was unremarkable. His blood investigations showed myositis and acute kidney injury with elevated serum creatinine kinase, alanine transaminase, aspartate transaminase, and creatinine at 2107 U/L, 942 U/L, 500 U/L, and 180 µmol/L respectively. Other laboratory investigations, particularly his electrolytes and urinalysis were unremarkable.

He was treated for heat stroke with myositis and dehydration after a marathon run. With adequate intravenous hydration, his clinical condition improved with resolution of fever and tachycardia. His electrocardiogram revealed an isorhythmic atrioventricular dissociation with a heart rate of 50 beats per minute (Figure 1). Otherwise, he was asymptomatic, and an echocardiogram confirmed no cardiac structural abnormality with normal sized all cardiac chambers and a good left ventricular systolic function with an ejection fraction of more than 55%. A functional stress test with a 6-minute-walk-test revealed that he had no issue completing more than 700 m in 6 minutes, with a resolution of atrioventricular dissociation to sinus bradycardia in electrocardiogram with a heart rate of 56 beats per minute (Figure 2). He was discharged well after a 3-day-duration of hospital stay when his blood investigations normalised.

DISCUSSION

The sinoatrial node is the major pacemaker in the heart. It initiates a propagation of electrical impulses in the heart from the atria to the ventricles via the atrioventricular node and His-Purkinje system, results in a synchronous

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Corresponding Author: Lee Yee Lim
Email: limleeyee88@gmail.com

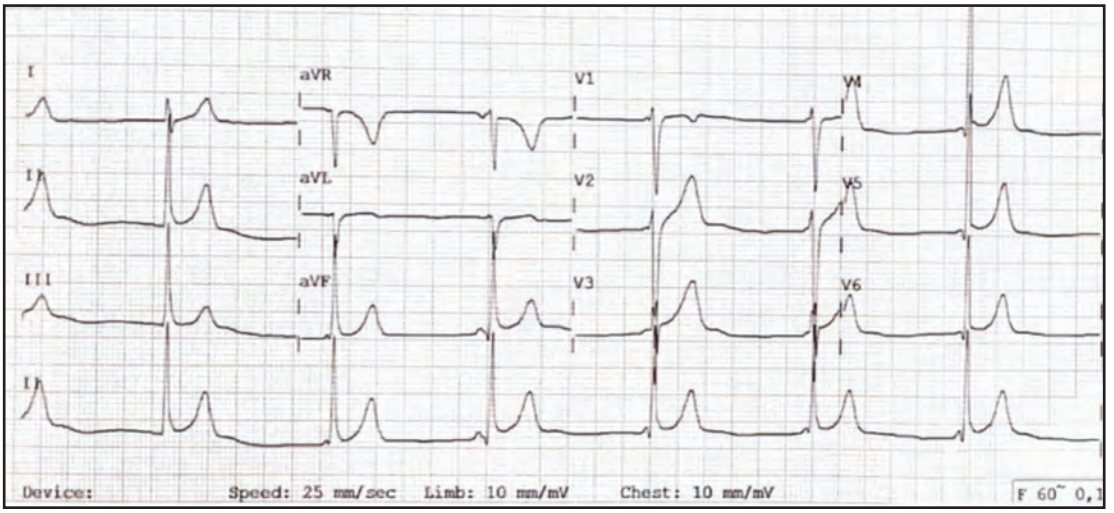

Fig. 1: A 12-lead electrocardiogram showed an isorhythmic atrioventricular dissociation, evidenced by a regular R-R interval with P waves "wander" from left to the QRS complex to "hide" within the QRS complex.

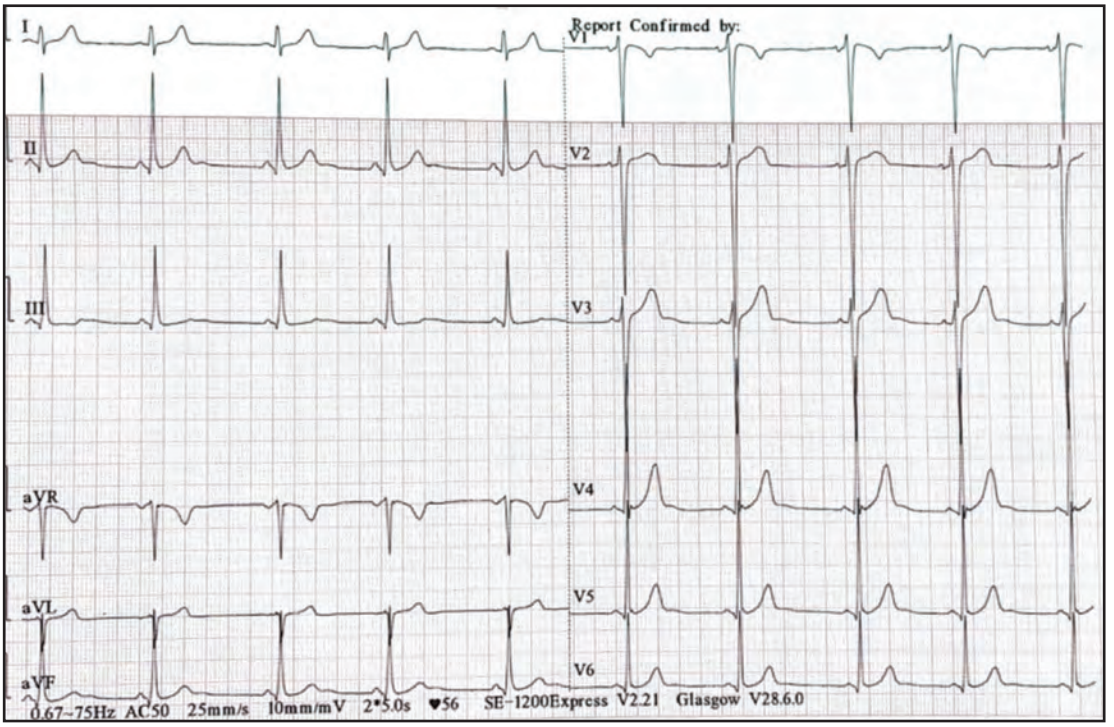

Fig. 2: A 12-lead electrocardiogram showed sinus bradycardia with short PR intervals of 104 milliseconds, evidenced by a regular R-R interval with fixed PR intervals.

depolarisation of the atria and ventricles to ensure an effective cardiac contraction. In cases when the pacing rate of sinoatrial node is low or any conduction disturbance of impulses from sinoatrial node to the atrioventricular node, the intrinsic pacemaker cells located at the atrioventricular junction will be the major pacemaker to pace the ventricles at a rate of 40 to 60 beats per minute. This rhythm is known as a junctional rhythm.

An atrioventricular dissociation occurs when the atria and ventricles are paced by different pacemakers, hence beat independently. Three theories of atrioventricular dissociation were described in the literature, which are atrioventricular dissociation by default, atrioventricular dissociation by usurpation, and a complete heart block. In atrioventricular dissociation by default, a slowing of sinoatrial node as the dominant pacemaker leads to the dominance of an independent ventricular pacemaker. Atrioventricular dissociation by usurpation describes an acceleration of the ventricular pacemaker which surpasses the intrinsic atrial rate of the sinoatrial node. A pathological blockage of the atrioventricular node occurs in complete heart block, which prevents the conduction between the atria and ventricles, and results in independent rhythms.[3]

In cases of atrioventricular dissociation with bradycardia, it is crucial to rule out complete heart block and drug toxicity such as digitalis toxicity as urgent treatment is required to prevent fatal complications.[4] However, in our present case, the young man was an athlete who was active in track and field for 3 years without previous hospitalisation. His incidental abnormal electrocardiogram findings did not fit into the clinical conditions to suggest a cardiogenic syncope. A normal echocardiogram and a reversion of atrioventricular dissociation to sinus bradycardia after a 6-minute-walk-test confirmed his bradycardic atrioventricular dissociation was physiological. Bradycardia is common amongst athletes with sinus arrhythmias, junctional rhythms, atrioventricular conduction delays, as well as isorhythmic atrioventricular dissociation such as present case reported in the literature.[5]

Isorhythmic atrioventricular dissociation happens when the sinus rate and the junctional rate are nearly similar, but they are beating independently. It is a physiological condition for those with low resting heart rate, especially young athletes, whereby a low sinus rate that falls within the range of the intrinsic junctional rate will lead to co-dominance of both sinus and junctional pacemaker. In physiological conditions where the atrioventricular node is not diseased, any simple activities which can increase the sinus rate will reverse the condition back to sinus rhythm.

CONCLUSION

The young man in our present case had an "abnormal" electrocardiogram which is completely normal. It is an incidental physiological finding that does not warrant any further investigation and he should be allowed to continue his activities in sports without restriction. Abnormal electrocardiogram findings in patients presented with cardiac associated symptoms such as syncope often result in difficulties in decision-making and diagnosis by the attending medical personnel. Good clinical acumen and appropriate investigations are crucial to ensure a proper diagnosis and management.

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