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Case Report

Transient sinus node dysfunction following exertional heat injury: A case report

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ABSTRACT

Heat-related illnesses (HRIs) continue to affect people worldwide, with the incidence expected to rise due to global warming. The cardiovascular system can be impacted either as part of a multisystem involvement or in isolation. Various electrocardiographic changes have been reported, with only one previous case of sinus node dysfunction in the literature. Here we report a case of transient sinus node dysfunction in a patient following exertional heat injury, which was managed with conservative measures. The patient recovered without complications. This is the second documented case of sinus node dysfunction in the context of heat injury. The patient made a full recovery, and this case highlights the importance of recognizing heat-related cardiac disturbances and managing them appropriately.

Key words: Cardiovascular system, Electrocardiogram, Global warming, Heat-related illnesses, Sinus node dysfunction

INTRODUCTION

Heat-related illnesses (HRIs) cover a spectrum of conditions ranging from heat exhaustion to heat stroke. Heat stroke, also known as sunstroke or *coup de chaleur*, is one of the oldest known medical conditions and continues to impact humans globally.[1] With rising global temperatures, these illnesses are likely to become more common. HRIs can affect multiple organs, including the heart.

Commonly affected organs include the kidneys (causing rhabdomyolysis and acute kidney injury) and the liver (causing acute liver failure). Catecholamine release due to heat stress initially increases inotropy and chronotropy, leading to an elevated cardiac output. If this response is sustained, it can contribute to stress-induced cardiomyopathy.[2]

Electrocardiographic (ECG) changes in HRIs include sinus tachycardia, supraventricular arrhythmias such as atrial fibrillation, atrial flutter, ectopic atrial tachycardia, both QT shortening and prolongation, pathological Q-waves, bundle branch blocks, ischemic ST-T changes, and other nonspecific ST-T changes.[3,4] In rare instances, sinus node dysfunction and complete heart block have been reported.[5,6] This case report presents the second documented case of transient sinus node dysfunction following a heat-related injury.

CASE REPORT

Patient information

A 50-year-old male with a history of hypertension (on telmisartan) presented with malaise for 2 days, followed by dizziness and oliguria (reduced urine output) on the day of hospitalization. The patient had been working outdoors in a hot and humid environment in northern India. There was no history of chest pain, palpitations, syncope, dyspnea, fever, or abnormal sweating, and no similar symptoms in the past.

Clinical findings

Upon arrival, his blood pressure was 97/52 mmHg (right arm, supine), and his pulse was 40 beats per minute, regular. His temperature and respiration were normal. Systemic examination was unremarkable. He was resuscitated in a cool room with intravenous fluids. His blood pressure and oliguria gradually improved; however, he continued to experience bradycardia.

Investigations

An ECG taken on arrival showed a heart rate of 40 beats per minute with a low atrial rhythm in the first ECG and

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isorhythmic atrioventricular (AV) dissociation in the second ECG [Figure 1-2]. Laboratory investigations revealed a normal hemogram with pre-renal azotemia. His admission creatinine was 1.38 mg/dL, which rose to 1.67 mg/dL the next day before settling at 0.80 mg/dL (reference range: 0.6-1.3 mg/dL). Electrolytes, liver function, thyroid function, and coagulation parameters were normal. Cardiac biomarkers (troponin-I and NT-proBNP) and a 2D echocardiogram were also normal. His heart rate gradually recovered, and ECG on day 2 showed normal sinus rhythm after resuscitation with fluids in a cool environment [Figure 3].

Diagnosis

A diagnosis of sinus node dysfunction was made, and secondary causes were excluded based on normal electrolytes, thyroid profile, and echocardiogram. To further assess the chronotropic response and AV conduction to rule out intrinsic sinus node or conduction pathway disease, the patient underwent a treadmill test. During peak exercise, the heart rate achieved was more than 90% of the maximum heart rate (age-adjusted) and showed no conduction defects.

Therapeutic intervention

The patient returned to sinus rhythm by the second day. A 24-hour Holter monitor did not show any arrhythmias, sinus pauses, or conduction blocks. Given the possibility of heat injury, the patient was managed conservatively with rest in an air-conditioned room and intravenous fluids.

Follow-up

The patient has been followed up as an outpatient for more than 6 months with no recurrence of symptoms.

DISCUSSION

HRI can be exertional or non-exertional and includes heat exhaustion, heat injury, and heat stroke. When the core body temperature rises above 38.3°C without end-organ damage, it is termed heat exhaustion. Heat injury, which involves hyperthermia with end-organ damage (excluding neurological dysfunction), is referred to as heat stroke when the brain is involved. Treatment is primarily supportive, focusing on lowering the core body temperature, fluid resuscitation, managing electrolyte imbalances, and caring for affected organs.

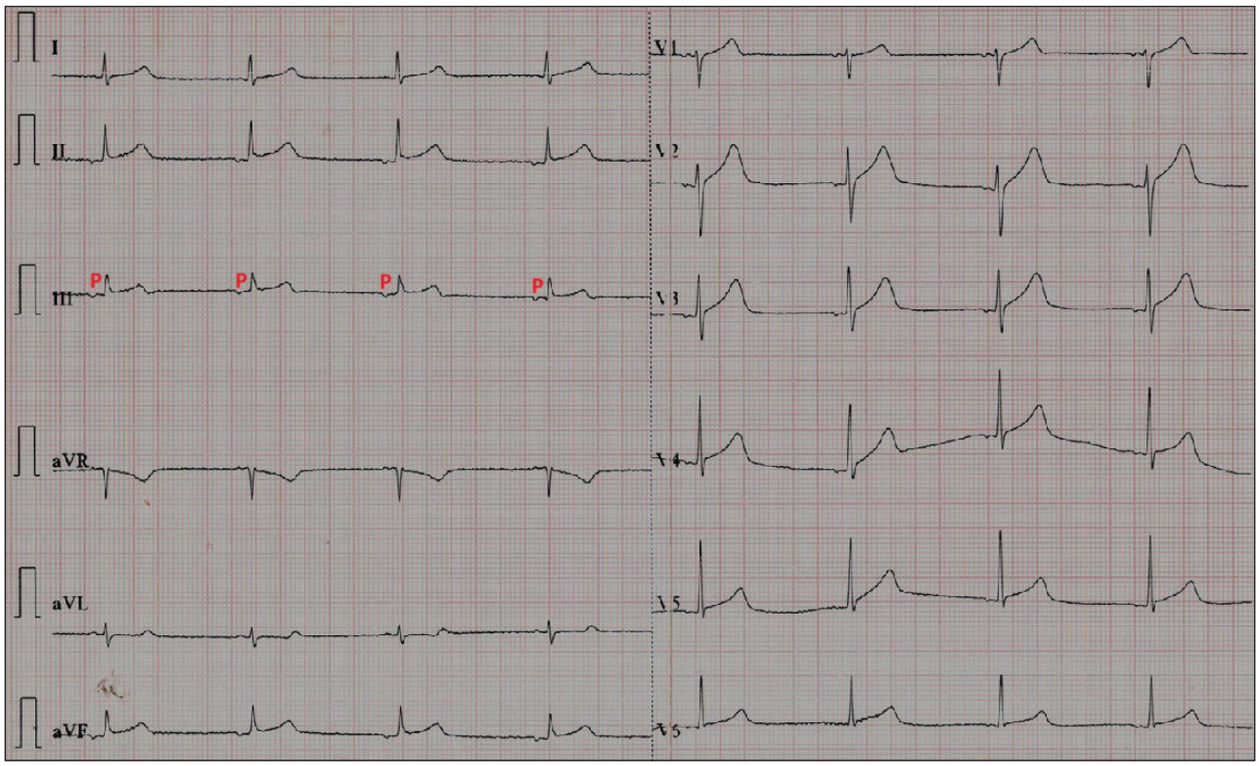

Figure 1: A 50-year-old man who presented with malaise, giddiness, and oliguria after exertional heat-related illness. His initial electrocardiogram showed low atrial rhythm with a heart rate of 40 beats per minute, with negative P in inferior leads as denoted by the red color letter "P".

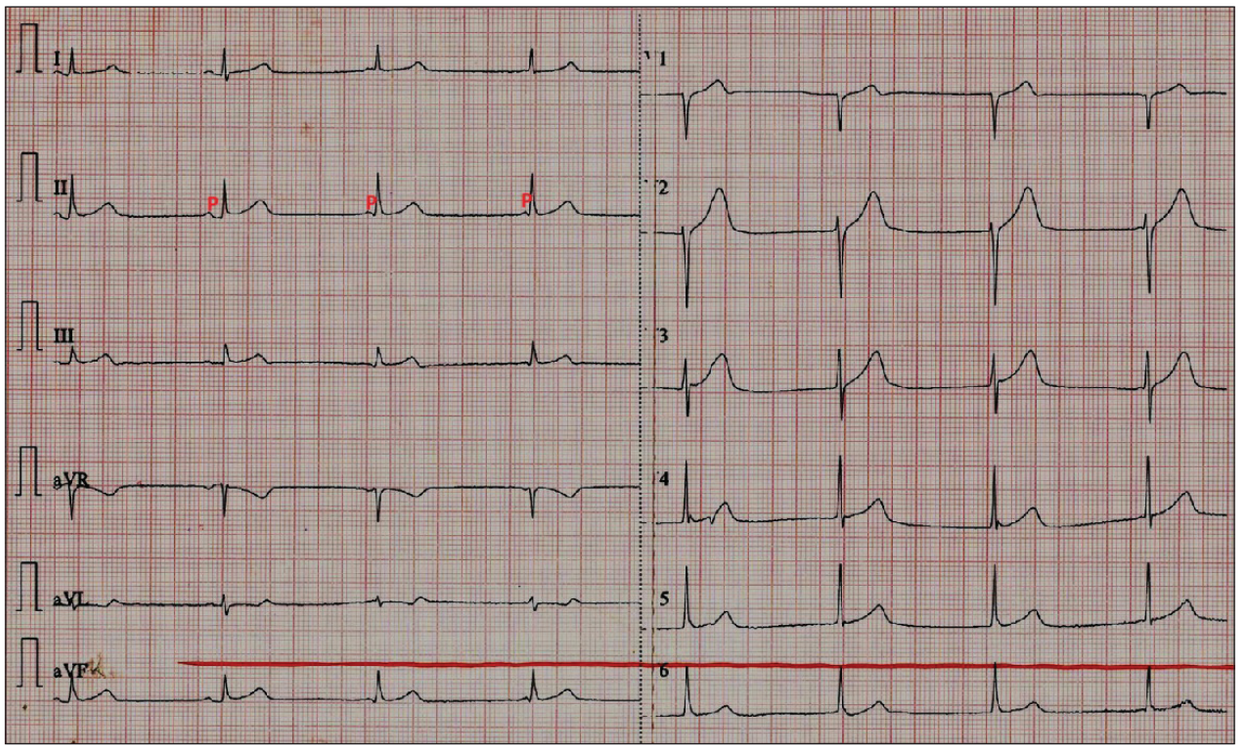

Figure 2: A 50-year-old man who presented with malaise, giddiness, and oliguria after exertional heat-related illness. His second electrocardiogram showed a heart rate of 40 beats per minute, suggestive of isorhythmic AV dissociation. P wave is denoted by the red color letter "P".

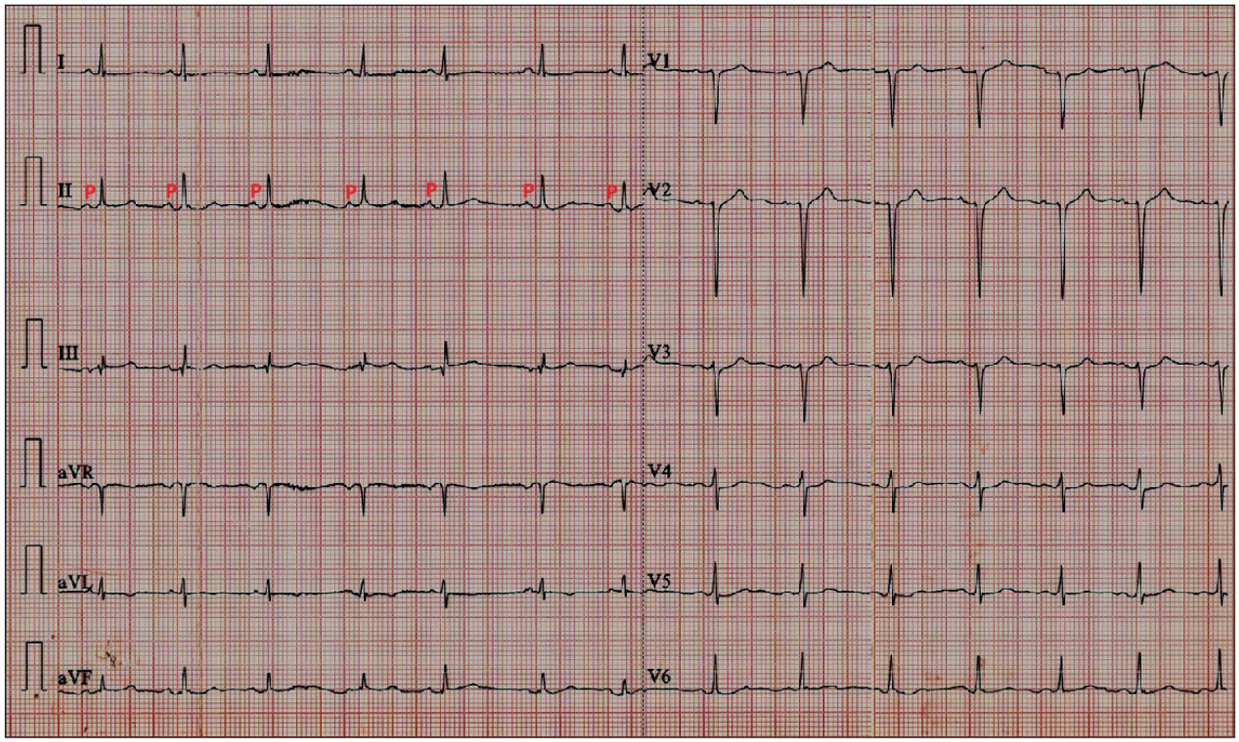

Figure 3: A 50-year-old man who presented with malaise, giddiness, and oliguria after exertional heat-related illness. His electrocardiogram on day 2 after resuscitation and recovery showed normal sinus rhythm with a heart rate of 80 beats per minute. P wave is denoted by the red color letter "P".

HRI can affect various organ systems, including the cardiovascular system. ECG changes in HRI should be interpreted with caution, as they may represent transient changes due to myocardial injury, catecholamine surges, electrolyte imbalances, or multi-organ failure.

ECG findings such as ST-T changes, which may resemble an acute ST-elevation myocardial infarction, ischemic ST-T changes, stress-induced cardiomyopathy, various bundle branch blocks, and cardiac arrest, have all been reported in the context of HRI.[2-4,7,8] Apart from bundle branch blocks, one case of complete heart block in a heatstroke patient with myocarditis has been reported, and the patient was managed with a permanent pacemaker.[6] Another case of transient sinoatrial dysfunction in heatstroke with metabolic acidosis (without hyperkalemia) has also been documented, managed with external cardiac pacing, rapid cooling, and supportive care.[5]

Our case would be classified as having exertional heat injury, with hypotension, acute kidney injury, and sinus node dysfunction. The pathophysiology of heat-related myocardial injury is not well understood, but it may involve a systemic inflammatory response triggered by hyperthermia, endotoxins, and interleukins, as well as protein denaturation and deoxyribonucleic acid breakage.[9] Stress-induced cardiomyopathy due to a catecholamine surge is another plausible mechanism.[2] Calcium's role in hyperthermia-related myocardial injury has been demonstrated in animal models.[10]

Sinus node dysfunction is rarely reported in the literature and may be under-recognized. Other causes of sinus node dysfunction, including intrinsic disease, were considered; however, normal biochemical parameters, a normal echocardiogram, and the recovery of sinus rhythm after resuscitation strongly suggested that heat injury was the primary cause. The normal results from a 24-hour Holter monitor and a normal chronotropic response during the treadmill test helped exclude intrinsic conduction pathway disease.

CONCLUSION

HRI can cause transient sinus node dysfunction, which can be identified through an electrocardiogram. The usual treatment of HRI, including hydration and cooling in a controlled environment, is important. If the patient recovers uneventfully, the insertion of a permanent pacemaker should be avoided.

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