Dear Editor

Brugada syndrome is a heterogeneous genetic channelopathy that predisposes to ventricular arrhythmias and sudden cardiac death (SCD). The electrocardiogram (ECG) findings only suggest a diagnosis of Brugada syndrome but are not confirmatory. The ECG pattern is known to vary with time in some patients and many have a normal baseline ECG [1,2]. There are two distinctly described ECG patterns, type I Brugada is characterized by ST elevation (≥2mm) with a “coved” pattern or a T-wave which is inverted with an upward convexity, in other cases the elevated ST segment descends and rises again forming a “saddleback” type pattern, this is called the type II Brugada. The electrophysiological and clinical manifestations of this syndrome have been found to be secondary to various factors [3-10]. Of these, mutations in cardiac sodium channel SCN genes (SCN5A and SCN10A) are the most well-known [11]. Since all affected families do not have these mutations, it is believed that other mutations in sodium channel genes or mutations in non-sodium channel genes may also cause Brugada syndrome [12-15].

There have been some cases with a normal baseline EKG where in a Brugada pattern was induced by factors such fever, infection, cocaine use and even medication, particularly sodium channel blocking agents. With SCD being a common presenting symptom, it is critical to make an early diagnosis and institute preventive treatment such as an implantable cardioverter defibrillator. Our case described below is of a patient who presented to the emergency room (ER) with a fever and was found to have a pattern of ST elevation without any other cardiac symptoms.

The patient is a 71 year old male who presented to the emergency room with fever (103.5 F). An ER ECG (Figure 1) showed RBBB, coved ST elevation in leads V1-V4 with T-inversion characteristic of Type I Brugada syndrome. He was urgently taken for left heart catheterization, which revealed normal coronaries. An echocardiogram performed two days after this showed normal left and right ventricular function. His EKG went back to his baseline pattern of RBBB/LAFB after fever resolution (Figure 2) and a diagnosis of type I Brugada syndrome unmasked in the setting of fever was made. He was offered defibrillator implantation, but he opted for conservative management.

Upon asking for a prior history of syncope, he reported two events; one when he was seventeen and another in his thirties. Both times he was in a hot environment and experienced a prodrome of feeling confined and crowded before briefly losing consciousness, suggestive of a vasovagal etiology and did not seek medical attention at the time.

Brugada syndrome triggered by fever is known to predispose to malignant ventricular arrhythmias. Other factors

Figure 1:

Figure 2:
known to induce a Brugada pattern include cocaine, electrolyte imbalances, drug overdose and medication affecting sodium channels. Several cases similar to the one described here have been shown to be triggered by fever. One study showed that such patients are at a very high risk of developing malignant ventricular tachycardia during the same acute event that triggered the Brugada pattern ECG changes [8]. With this mode of presentation that can be fatal, it is imperative that such patients are treated for fever or taken off the offending medication as soon as a Brugada pattern is identified. With the syndrome being a genetically inherited entity, screening and education of family members in the risks and causative factors must be included along with treatment of the affected patient.

References


