Status Epilepticus as an Initial Presentation of Brugada Syndrome: A Case Report

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Patients presenting with convulsions are sometimes incorrectly treated for epilepsy, as these symptoms may be manifestations of underlying cardiac disease. Brugada syndrome, associated with an elevated risk of developing fatal arrhythmic events, is a rare disorder characterized by a unique electrocardiographic pattern. The typical clinical presentation mainly involves syncope of unclear cause or sudden death. Seizures are uncommon clinical manifestations of Brugada syndrome, and reports of status epilepticus as initial presentation of Brugada syndrome are extremely rare. In this case report, we present a male patient with a typical pattern on 12-lead electrocardiography, following resuscitation for generalized convulsive status epilepticus.

Key Words: Brugada syndrome, status epilepticus
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Status epilepticus is usually defined as continuous seizure activity lasting 30 minutes or more, or intermittent seizure activity lasting 30 minutes or more, during which time consciousness is not regained [1]. This condition may be attributed to many causes, including cerebral hypoxic damage [2]. Patients presenting with convulsions are sometimes mistakenly treated for epilepsy, although these symptoms may be a manifestation of hypotension caused by bradycardia [3]. These symptoms may also be seen in patients with hemodynamically compromising ventricular tachycardia (VT) or ventricular fibrillation (VF), presumably resulting from cerebral hypoperfusion [4].

Brugada syndrome, which is unrelated to structural cardiac abnormality, is a rare electrical disorder characterized by a unique electrocardiographic pattern of right bundle branch block, and ST-segment elevation in right precordial leads (V1 to V3). There is a high risk of developing fatal arrhythmic events such as VT or VF. The clinical presentations are mostly syncope of unclear cause or sudden death. Seizure is an uncommon clinical presentation of Brugada syndrome, reported in only a few articles [4,5], and none of which note status epilepticus as the initial manifestation of the syndrome. In this article, we describe a male patient who presented with status epilepticus and typical pattern on 12–lead electrocardiography (ECG) after resuscitation, demonstrating generalized convulsive status epilepticus as the initial manifestation of Brugada syndrome.

Case Presentation

A 41-year-old male patient was brought to the local emergency department (ED) for generalized tonic-clonic movements. According to his co-workers, the patient was talking to them without any apparent discomfort, before starting to exhibit tonic-clonic movements that lasted for more than half an hour before arrival to the ED. During resuscitation, VF in ECG records (Figure 1) with seizure activities persisted until direct current (DC) shock was applied and sinus rhythm was restored. The patient was then transferred to the intensive care unit at another hospital as soon as vital signs were stabilized and maintained with medication. Initial physical examination was normal except

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for Glasgow Coma Scale (GCS) status of E1VEM2. Neurologic examination revealed positive light reflex, negative Doll's eye sign, hyperreflexic deep tendon reflex, and positive Babinski sign. The complete blood count and chemistry panel were within reference ranges, except for glutamate-oxalacetate transaminase and glutamate-pyruvate transaminase, which were 94 IU/L and 136 IU/L, respectively. Creatinine phosphokinase and creatinine phosphokinase isoenzyme MB were 378 IU/L and 65 IU/L, respectively. Troponin I was initially 0.123 ng/mL and subsequently peaked at 0.995 ng/mL. Sinus tachycardia with V1~V3 RSR' pattern and ST-segment elevation (Figure 2) was found on ECG records. Echocardiogram was normal. Chest X-ray revealed no cardiopulmonary abnormality, and brain computerized tomography showed diffuse brain edema with blurring sulci and no obvious local hypodense or hyperdense lesions. Electroencephalography several days later revealed generalized continuous voltage suppression, which was compatible with hypoxic encephalopathy. Supportive treatment was arranged for the patient, including tracheostomy, stress ulceration prevention and rehabilitation.

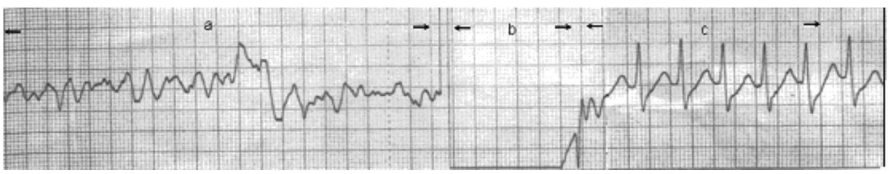

Figure 1.
a: Electrocardiography recording during the status epilepticus showing ventricular fibrillation.
b: Period of direct current shock.
c: Sinus rhythm was restored after direct current shock was applied.

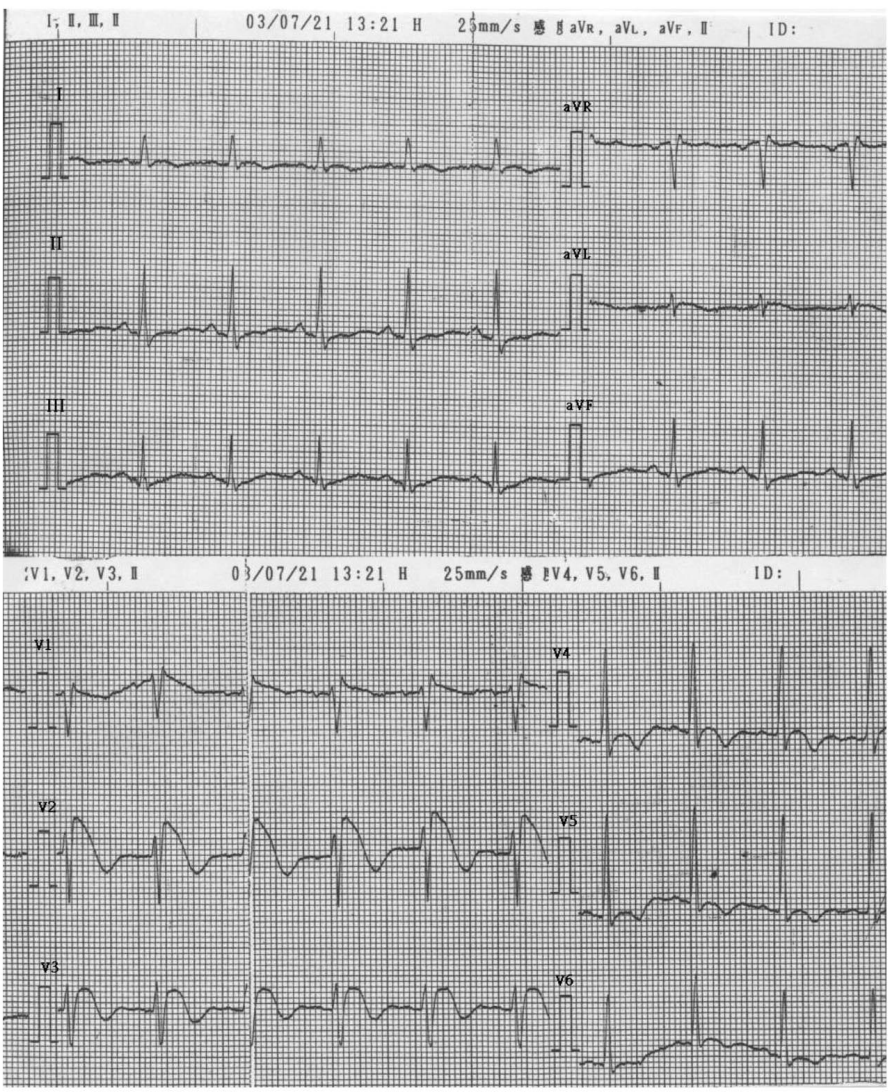

Figure 2. *12-lead electrocardiography showing a pattern of right bundle branch block and ST-segment elevation in leads V1 through V3, the typical characteristics of Brugada syndrome.*