

Brugada electrocardiographic pattern due to tricyclic antidepressant overdose

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Abstract

The Brugada syndrome is an arrhythmogenic disease with characteristic coved ST-segment elevation 2 mm or greater in the right precordial leads (type 1 Brugada electrocardiogram [ECG] pattern or “Brugada sign”) and is estimated to be responsible for at least 20% of sudden deaths in patients with structurally normal hearts [*Circulation* 2005;111(5):659–70]. The Brugada sign has been described in asymptomatic patients after exposure to various drugs. As published reports of the drug-induced Brugada sign have become increasingly prevalent, there is growing interest in the mechanisms responsible for this acquired ECG pattern and its clinical significance. We report a case of a patient who developed the type 1 Brugada ECG pattern after intentional overdose of a tricyclic antidepressant agent, review the literature concerning tricyclic antidepressant agent–induced Brugada sign, discuss potential mechanisms, and evaluate the clinical significance of this ECG abnormality.

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Keywords:

Brugada syndrome; ECG; Tricyclic antidepressant agent; Overdose

Case report

A 44-year-old Caucasian man with a history of depression presented to the emergency department 7 hours after ingesting 30 pills of desipramine and 20 pills of clonazepam in a suicide attempt. He denied chest pain, palpitations, syncope, or shortness of breath. His medications included desipramine, clonazepam, and trazodone. He denied alcohol or illicit drug use. There was no family history of syncope or sudden cardiac death.

The patient's temperature was 36.0°C, blood pressure 119/83 mm Hg, heart rate 52 beats per minute, respiratory rate 12 per minute, and oxygen saturation 98% on room air. His cardiac apical impulse was focal and nondisplaced. Jugular venous pressure was normal. Auscultation of the heart revealed bradycardia with a regular rhythm and a normal S1 and S2; there were no murmurs, rubs, or gallops. Hepatomegaly was not present. Peripheral arterial pulse rates were normal. With the exception of flushed skin, dry mucus membranes, and hypoactive bowel sounds, the remainder of the findings from his physical examination was normal.

Routine blood chemistries, cell counts, and chest radiograph were within normal limits. Assay for serum tricyclic

level was not available. The initial electrocardiogram (ECG) showed sinus bradycardia, first-degree atrioventricular (AV) block, PR interval of 228 milliseconds, right bundle branch block morphology, prolonged QRS interval of 132 milliseconds, and coved, downsloping, greater than 2 mm ST-segment elevations in V1–V2 with inverted T waves, consistent with a Brugada type 1 ECG pattern (Fig. 1A). These findings were new compared with his normal ECG 1 year prior (Fig. 1B). Serial troponin-I tests were negative. The patient was treated with intravenous sodium bicarbonate. Electrocardiogram within 5 hours revealed normalization of the right precordial ST-segment elevations and T-wave inversions (Fig. 2). The ECG after therapy showed persistent sinus bradycardia, first-degree AV block, and right bundle branch block morphology. After 48 hours of cardiac monitoring, the patient was transferred to psychiatry for management of his depression. He remained asymptomatic for the duration of his hospitalization.

Discussion

Three electrocardiographic patterns of right precordial ST-segment elevation due to abnormal repolarization are recognized in the Brugada syndrome. Type 1 pattern (“Brugada sign”) has coved ST-segment elevation of 2 mm or greater, followed by an inverted T wave, with little or no isoelectric separation (Fig. 1A). Type 2 pattern also has a high-takeoff ST-segment elevation of 2 mm or

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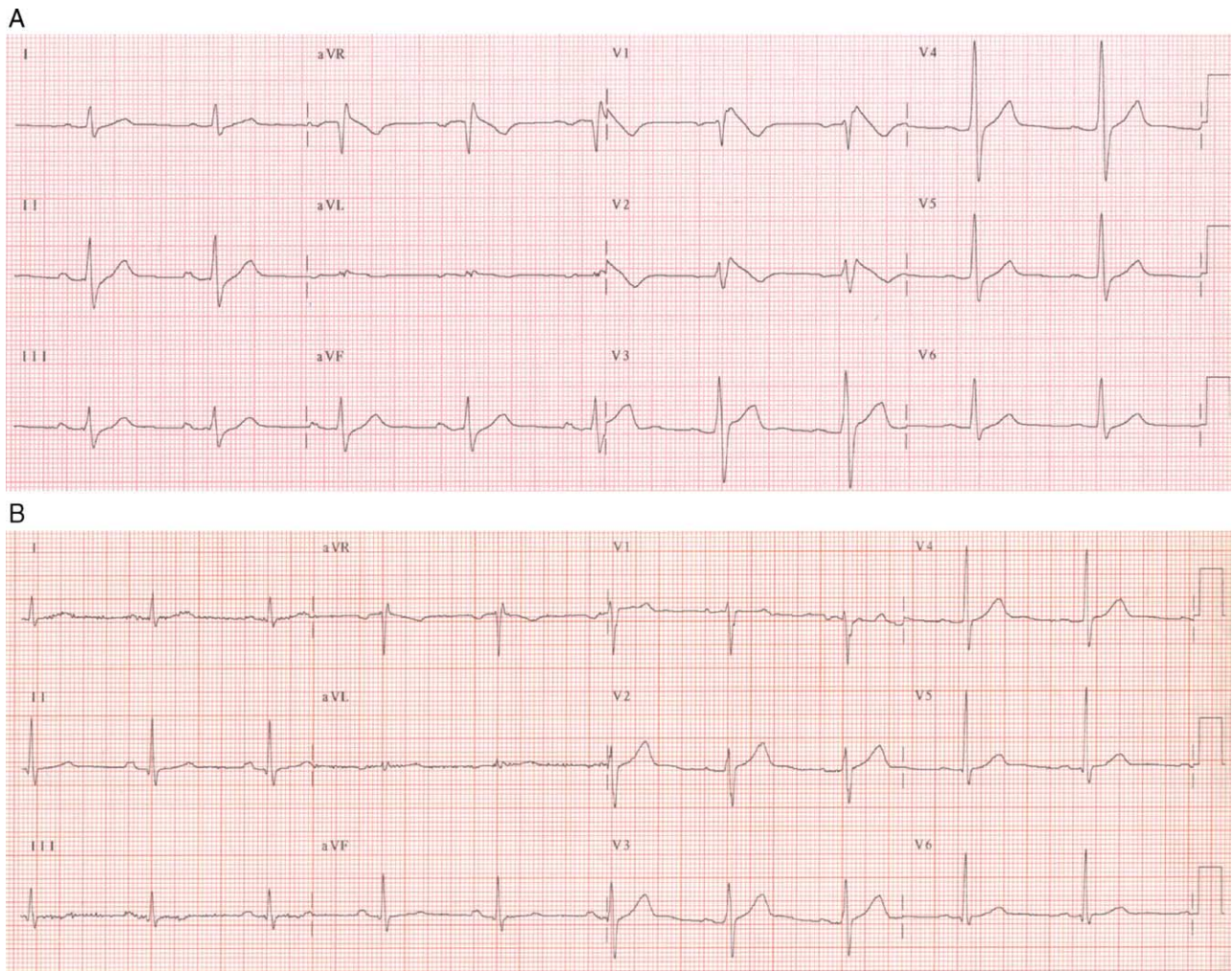


Fig. 1. A, This initial ECG shows sinus bradycardia, first-degree AV block (PR interval 228 milliseconds), right bundle branch block morphology, prolonged QRS interval of 132 milliseconds, and coved, downsloping ST-segment elevations in V1-V2 with inverted T waves, consistent with a Brugada type 1 ECG pattern. Also noted is ST-segment elevation in V3 and upsloping ST-segment depressions in II, III, and aVF. Note the absence of tented T waves that, if present, would suggest the diagnosis of hyperkalemia rather than Brugada pattern. B, Baseline electrocardiogram is normal.

greater with gradually descending ST-segment elevation (remaining ≥ 1 mm above the baseline), followed by a positive or biphasic T wave resulting in a saddleback configuration. Type 3 pattern has either coved or saddleback appearance with right precordial ST-segment elevation of less than 1 mm.¹ Type 1 pattern is diagnostic of the Brugada syndrome, whereas type 2 and 3 patterns require conversion to the type 1 pattern after challenge with a sodium channel blocking agent to be diagnostic.¹

Tricyclic antidepressant agents have been reported to cause both the Brugada sign and Brugada syndrome.^{2–8} The clinical presentation in published case reports has ranged from asymptomatic⁶ to ventricular fibrillation (VF) arrest.^{2,5,7} Various tricyclic antidepressant agents (TCAs), including nortriptyline,^{3,5} amitriptyline,^{7,8} and desipramine^{2,6} have been implicated. The Brugada sign has been observed in cases of TCA overdose^{3,4} as well as during routinely used dosages of TCAs.^{2,6} In one study evaluating the incidence of the Brugada sign in the setting of TCA overdose, 15.3% of 98 cases were retrospectively found to have ECG criteria consistent with type 1 Brugada pattern.⁴ This unexpectedly high incidence may represent an overes-

timate, however, because the ECG diagnosis of Brugada sign was equivocal in 3 cases, and a single patient who presented with the Brugada sign during multiple hospitalizations was included as 3 cases. Two additional retrospective studies examining the incidence of the Brugada sign in TCA overdose report lower incidences of 1.3% (1 of 79 cases)⁹ and 3.2% (8 of 251 cases).¹⁰

The cardiotoxicity of TCAs is well established. TCAs have been shown to block the fast sodium channels in the His-Purkinje system as well as in atrial and ventricular myocardial cells.¹¹ Common electrocardiographic manifestations of TCA overdose include widening of the PR, QRS, and QT intervals; conduction delay and block within the His-Purkinje system; and right bundle branch block. In canine cardiac Purkinje fibers, the mechanism of action of TCAs has been shown to be similar to the effects of class I antiarrhythmic agents, by decreasing the maximum rate of rise of phase 0 depolarization and causing early action potential repolarization.¹² TCAs have been associated with a dose-related risk of sudden cardiac death, with a 2.5-fold greater risk at maximal daily doses of amitriptyline, compared with control subjects.¹³

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