

Adrenal Insufficiency and a Surprising Electrocardiogram

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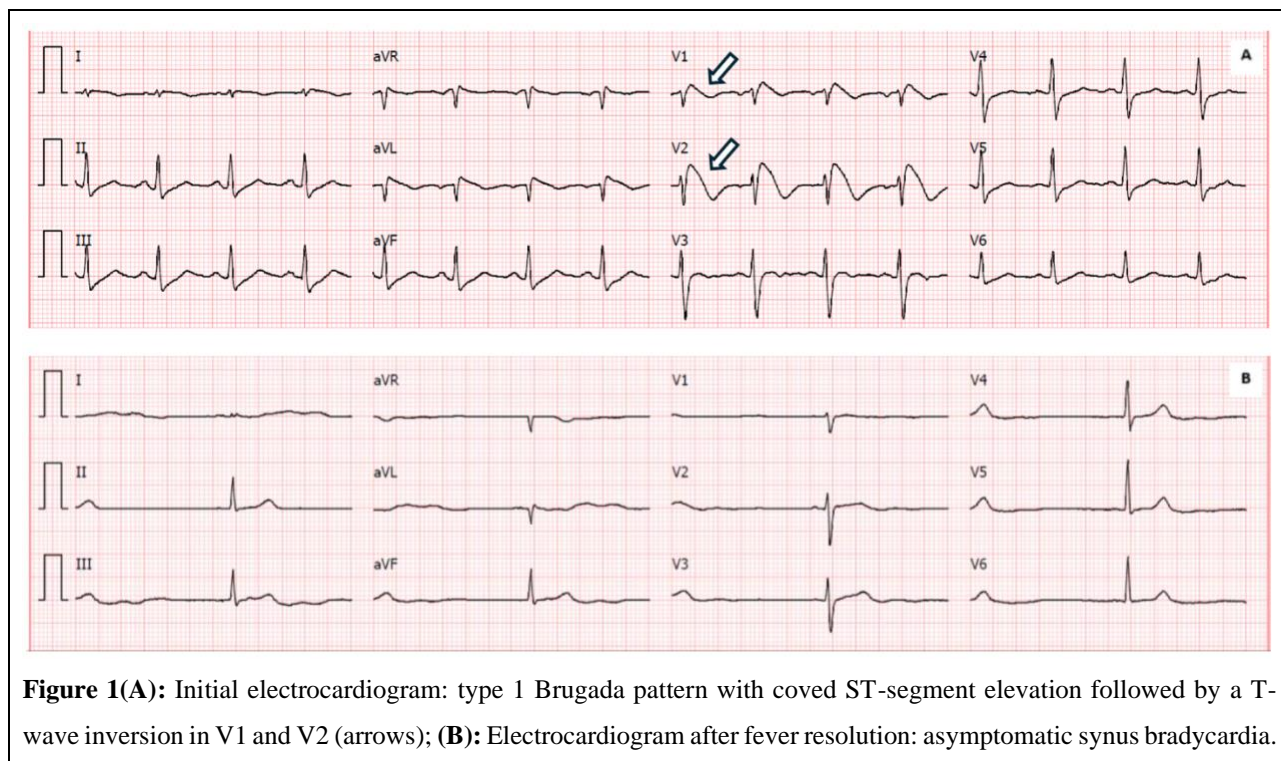


Figure 1(A): Initial electrocardiogram: type 1 Brugada pattern with coved ST-segment elevation followed by a T-wave inversion in V1 and V2 (arrows); **(B):** Electrocardiogram after fever resolution: asymptomatic sinus bradycardia.

Abstract

A 37-year-old male with panhypopituitarism presented with severe hypoglycemia and fever due to a bacterial gastroenteritis. Hospital evaluation revealed a Brugada type 1 electrocardiographic pattern, which resolved following aggressive fever management and treatment with antibiotics and stress-dose hydrocortisone. Brugada syndrome, a hereditary channelopathy associated with ventricular arrhythmias, can be triggered by febrile states. This case underscores the importance of systematic clinical evaluation, precise ECG interpretation, and thorough history-taking in managing life-threatening conditions.

Case Description

A 37-year-old male with a medical history of panhypopituitarism was found unconscious at his residence. The previous day, he had contacted his parents, reporting symptoms of malaise, diarrhea, and episodes of food-related vomiting. Upon assessment by the pre-hospital medical team, the patient was noted to have severe hypoglycemia (29 mg/dL) and fever (39°C). Initial management included intravenous administration of hydrocortisone for suspected adrenal insufficiency, hypertonic glucose, and paracetamol. Upon arrival at the hospital, the electrocardiogram revealed a sinus rhythm with a heart rate of 90 bpm and a Brugada type 1 pattern (Figure 1A).

Blood tests showed elevated levels of C-reactive protein and procalcitonin, indicative of bacterial gastroenteritis. Echocardiographic findings were unremarkable. Empirical antibiotic therapy and stress-dose hydrocortisone were initiated. Following fever management and the restoration of normothermia, resolution of the Brugada pattern was observed (Figure 1B).

Discussion

Brugada syndrome, first described in 1992 by Brugada and Brugada, is a hereditary channelopathy associated with a high risk of ventricular arrhythmias and sudden cardiac death in individuals without structural heart disease, particularly in young to middle-aged males. The electrocardiographic pattern is dynamic, with manifestations that may be spontaneous or concealed and are often unmasked under certain circumstances, such as febrile states.

Conclusion

This case underscores the critical importance of a systematic clinical evaluation, meticulous electrocardiographic interpretation, an understanding of pathological patterns, and a thorough anamnesis to identify and manage life-threatening conditions effectively. Our patient remained clinically and electrically stable under aggressive fever management until the resolution of the infectious complication.

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