History and electrocardiography as pathway to diagnosis of Brugada syndrome: a case report

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³Zvezdara University Medical Center, Beograd, Serbia KEYWORDS: Brugada syndrome, electrocardiography, hyperthermia.

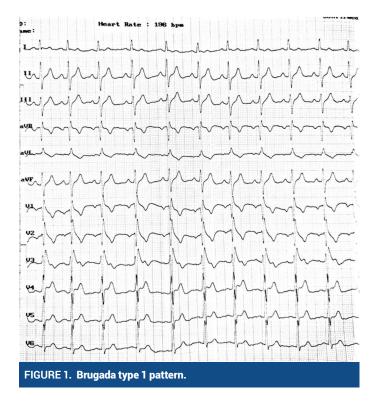
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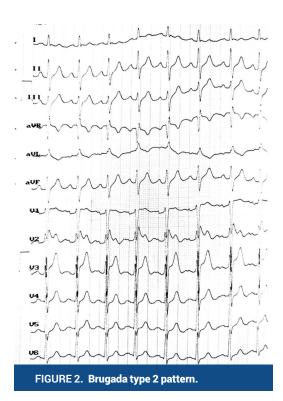
Introduction: The major electrocardiographic feature of Brugada syndrome is a distinct ST-segment elevation in the right precordial leads. Patients with spontaneously emerging Brugada ECG have a high risk of sudden arrhythmic death secondary to ventricular tachycardia/fibrillation. The ECG manifestations of Brugada syndrome are often dynamic. Type 1 pattern is diagnostic of Brugada syndome and is characterized by a coved ST segment elevation ≥2 mm, followed by a negative T wave. 1-3

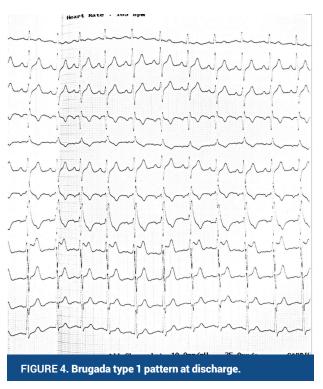
Case report: 23-years-old male has been hospitalized due to piercing pain in the left hemithorax and chills and fever lasting for several hours. He experienced such complaints for the first time in his life. He does not use tobacco, alcohol or psychoactive substances. He plays football for recreation. He reported severe family history: his father died at age of 36, and two paternal uncles died before their age of 25. At admission, he is conscious, oriented, mildly dyspneic, febrile (39.8°C); BP 115/70 mmHg. Laboratory: WBC 13.9 x 10°/L, neutrophils 85%, CRP 87; urine culture showed Escherichia coli >100.000/mL. He has been treated with antipyretic/paracetamol, antibiotic according to antibiogramme, and rehydration therapy. The initial ECG showed type 1 Brugada sign: cove ST elevation in V1-3 with negative T waves; RBBB (Figure 1). After a 12 hours of hospitalization, the ECG showed type 2 Brugada sign: saddle-shaped elevation of ST-segment and J point in V2 (Figure 2). After 24 hours of hospitalization, the ECG showed type 3 Brugada sign (Figure 3). At discharge 7 days later, type 1 Brugada sign develops again — a cove ST elevation in V1-2 and a saddle ST elevation in V3 (Figure 4). Ajmaline test has been performed according to protocol. During administration of a maximum dose of 70 mg, a >2 mm ST elevation was detected in V2-3, making the test positive. Electrophysiological study involved right femo-

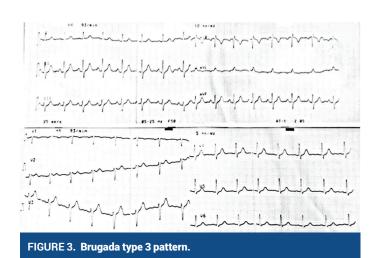


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ral vein access with quadripolar catheter to the right ventricle. Programmed stimulation did not induce ventricular tachycardia/fibrillation. The patient was not indicated for ICD for prevention of sudden cardiac death.

Conclusion: Family history and electrocardiography are the cornerstones of diagnosis of Brugada syndrome even today. Hyperthermia helped damask typical type 1 Brugada sign that showed dynamic changes. Our patient did not meet the criteria for implantation of ICD device.

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