EJCRIM
European Journal
of Case Reports
in Internal Medicine

BRUGADA ECG PATTERN IN A CAUCASIAN PATIENT WITH SEVERE HYPOTHYROIDISM

Erminia De Cristofaro, Maria Giulia Canè, Davide Cunzi, Giorgia Prampolini, Silvia Daniela Umbertina Fedeli, Aurelio Negro

Internal Medicine and Hypertension Center, Sant'Anna Hospital Castelnovo ne' monti, Azienda USL-IRCCS di Reggio Emilia, Reggio Emilia, Italy

Corresponding author's e-mail: erminia.decristofaro@ausl.re.it

Received: 22/05/2025
Accepted: 26/05/2025
Published: 18/08/2025

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: The patient's consent was obtained.
This article is licensed under a Commons Attribution Non-Commercial 4.0 License

How to cite this article: De Cristofaro E, Canè MG, Cunzi D, Prampolini G, Fedeli SDU, Negro A. Brugada ECG pattern in a Caucasian patient with severe hypothyroidism. *EJCRIM* 2025;**12**:doi:10.12890/2025_005544

ABSTRACT

Brugada syndrome (BrS) is a disorder leading to potentially life-threatening ventricular arrhythmias in patients with an apparently normal heart. It mostly affects men of Asian descent, and the prevalence varies between ethnicities. Typical ECG abnormalities with no symptoms are referred to as the Brugada pattern, and hypothyroidism is a potential trigger of this. We report on a Caucasian man with severe hypothyroidism and Brugada ECG pattern, which normalised after starting replacement therapy. The association has been previously described in only a few patients.

KEYWORDS

Brugada syndrome, hypothyroidism, ischaemic heart disease

LEARNING POINTS

- Brugada syndrome (BrS) is a potentially life-threatening disease that can be easily recognized on the electrocardiogram (ECG). Typical ECG abnormalities associated with no clinical symptoms is referred to as the Brugada pattern.
- Many studies have underscored the association between hypothyroidism and Brugada ECG pattern, that can be reversed by a prompt initiation of replacement therapy.

INTRODUCTION/BACKGROUND

Brugada syndrome (BrS) is a genetic disorder leading to potentially life-threatening ventricular arrhythmias in patients with an apparently normal heart. It is characterised by a pseudo-right bundle branch block and persistent ST segment elevation in leads V1–V2 on ECG. Based on the features of ST elevation it is possible to differentiate between a type 1 (coved) and type 2 (saddle-back) Brugada ECG pattern. It mostly affects men of Asian descent, but the prevalence varies between ethnicities. There is little evidence about the pathogenesis of BrS: gene mutations of sodium channels have been identified as the principal agent while fever, hypothyroidism and psychotropic drugs are considered as potential triggers of a Brugada ECG pattern[1,2]. We report on a Caucasian man with severe hypothyroidism and concomitant Brugada ECG pattern, which normalised after improvement of thyroid function by replacement therapy. This association has been previously

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

described in only a few patients. To our knowledge, only one had Caucasian origins.

CASE DESCRIPTION

We report on a 75-year-old Caucasian male who was referred to our department because of worsening fatigue, instability, slowing of movements and speech, myalgias, tongue enlargement and weight gain with lower oedema. He was hypertensive and dyslipidaemic; he had no history of syncope or cardiovascular events. He did not have a family history of sudden cardiac death (SCD), syncope or palpitation. On admission, his blood pressure was 128/84 mmHg, pulse rate was 75 beats per minute with a regular rhythm and oxygen level was 96% in ambient air. The laboratory showed severe autoimmune hypothyroidism (thyroid-stimulating hormone 237 µIU/ml (normal value (NV) 0.35–4.5), free T4 1.9 pg/ml (NV 8–18), free T3 0.8 pg/ml (NV 2.3–4.2), antithyroglobulin antibodies >1,000 UI/ml (NV 1.3–4.5), anti-thyroid peroxidase antibodies >13,000 UI/ml (NV 0–60); creatine phosphokinase was 343 U/l (NV 32–294), aspartate aminotransferase 56.0 U/l (NV 2–40), alanine transaminase 66 U/l (NV 4–49) and serum creatinine was 1.23 mg/dl (NV 0.7–1.2). On ECG both type 1 and 2, Brugada waveform in V1–V2 was noted. Transthoracic echocardiography showed left ventricular hypertrophy and mild pericardial effusion. Doppler carotid ultrasound showed no critical stenosis; a brain CT scan was negative. Levothyroxine 25 µg/day was started and progressively increased to 75 µg/day. Serial laboratory tests and ECGs were performed and normalised over two months upon reaching euthyroidism *(Fig. 1 and Table 1)*. A treadmill stress test did not induce any electrocardiographic changes *(Fig. 2)*. However, a Brugada ECG pattern was newly detected during a fever episode a few months after discharge.

	Diagnosis	1 week	3 weeks	8 weeks
Levothyroxine dosage (µg/day)	0	25	50	75
Body weight (kg)	72	70	-	69
TSH (pIU/ml)	237	-	35.6	5.7
fT4 (pg/ml)	1.9	-	1.7	15.2
fT3 (pg/ml)	0.8	-	6.3	-
CPK (U/l)	432	376	-	200
AST (U/l)	56	35	-	24
ALT (U/l)	66	45	-	18
Na (mmol/l)	142	-	-	144
K (mmol/l)	4.8	-	-	4.1
Creatinine (mg/dl)	1.15	-	-	0.95
Coved-type Brugada ECG waveforms (V1–V2 in two intercostal space)	Yes	Yes	No	No

Table 1. Summary of clinical, biochemical and electrocardiographic patient features over time.

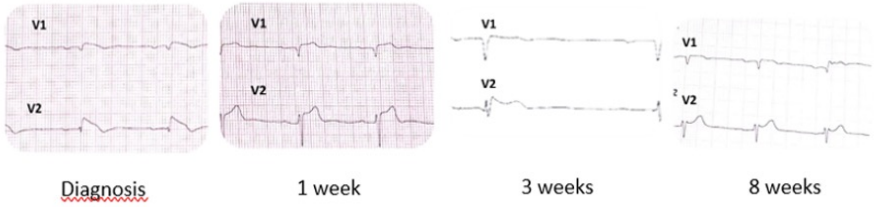


Figure 1. Progressive changes in ECG waveforms in V1–V2.



Figure 2. Progressive changes in ECG waveforms in V1–V2 during treadmill stress test.

DISCUSSION

We present a Caucasian patient with both type 1 and type 2 Brugada ECG patterns triggered by severe hypothyroidism. As reviewed by Bioletto[1] very few cases have been reported, mostly in those of Asian descent. To our knowledge, our case is the second one of Caucasian origin. The BrS is an autosomal dominant genetic disorder with variable expression characterised by abnormal findings on ECG. Its prevalence ranges from 0.02 to 0.1% in Europe, and is apparently higher in Southeast Asia[1,2]. The prevalence and long-term prognosis of individuals with the Brugada ECG pattern in the geriatric population is unknown. Some data report a more indolent and benign clinical course in this population[3]. Based on the features of ST elevation, two different patterns have been identified: type 1 Brugada ECG pattern, referred to as the coved type and type 2 Brugada ECG pattern, referred to as the saddle-back. The first alteration is diagnostic for BrS, while the second one is suggestive for it. The clinical presentation includes arrhythmias (e.g. ventricular fibrillation and polymorphic ventricular tachycardia) that usually occur during sleep and that can result in sudden cardiac arrest or death, syncope and palpitations, usually due to atrial fibrillation. Typical ECG abnormalities associated with no clinical symptoms is referred to as the Brugada pattern. A genetic causative mutation is found in about 30% of cases and usually it involves the *SCN5A* gene, encoding for the cardiac sodium channel, but its prognostic significance remains controversial. There are no clear data in humans able to clarify the pathophysiological mechanism causative for BrS. Animal studies suggest a decrease in sodium inflow currents (I_{Na}), with a relative increase in the transient outward current (I_{to}) regulated by potassium channels. These agents may be influenced and regulated by thyroid hormone levels[1,4,5]. The Brugada pattern may be challenging to diagnose because it is influenced by the autonomic tone, particularly more evident during increased vagal tone periods. A cardiac stress test performed in Brugada patients suggests the possible role of parasympathetic tone in both the incidence of severe arrhythmias during sleep and the ST segment augmentation after exercise. Vagal tone may decrease calcium currents and consequently contribute to ST segment elevation and arrhythmia. The association between SCD and exercise remains controversial[2,3]. It is important to consider that there are some provoking factors able to reveal Brugada ECG pattern in predisposed subjects with a latent ion channel dysfunction such as fever, some medications (sodium channel blockers, propofol, tricyclic antidepressants, fluoxetine, lithium, antihistamines), toxins (cocaine) and metabolic disturbances including severe hypothyroidism[1,3]. There are data suggesting an intermediate risk for SCD in patients with fever-induced type 1 ECG[3]. There is no evidence about the exact pathways by which these conditions can provoke ECG changes.

CONCLUSION

We report another case of Brugada ECG pattern triggered by severe hypothyroidism. The awareness of such association may be helpful to avoid redundant diagnostic tests and overtreatment, considering that the risk stratification in BrS patients is challenging due to the controversial prognostic significance of a possibly positive electrophysiological study.

REFERENCES

1. Bioletto F, Cuboni D, Varaldo E, Bona C, Berton AM, Maccario M, et al. Severe hypothyroidism as a trigger for Brugada-type ECG abnormalities: a case report and literature review. *Arch Endocrinol Metab* 2024;**68**:e230027. doi: 10.20945/2359-4292-2023-0027
2. Gonçalves CM, Vazão A, Carvalho M, Cabral M, Martins A, Amado M, et al. Brugada syndrome and exercise: is it time for a paradigm change? *J. Cardiovasc Dev Dis* 2025; **12**:94. doi: 10.3390/jcdd12030094
3. Brugada J, Campuzano O, Arbelo E, Sarquella-Brugada G, Brugada R. Present status of Brugada syndrome: JACC State-of-the-Art Review. *J Am Coll Cardiol* 2018;**72**:1046– 1059. doi: 10.1016/j.jacc.2018.06.037
4. Taira K, Fujino A, Watanabe T, Ogyu A, Ashikawa K, Shimizu W. Brugada-type electrocardiogram in a patient with hypothyroidism. *J Cardiol Cases* 2010;**2**:e147–150. doi: 10.1016/j.jccase.2010.05.011
5. Kitahara A, Hirai R, Matsui Y, Ikeda Y, Nakamura H. A case of hypothyroidism with Brugada electrocardiographic waveforms. *Endocr J* 2008;**55**:589–594. doi: 10.1507/endocrj.k07e-024