patients. The lack of randomised controlled studies on TN is explained by several reasons[12]: (1) difficult recruitment in a rare disease with exclusively clinical diagnostic criteria; (2) ethical problems of comparing against sham treatment when effective medical and surgical therapies are available; (3) patients' preferences when comparison is against a standard medical treatment, since being assigned to the control group creates a drop-out bias (drop-out or change to the experimental group) or deception bias; (4) lack of equidistance of professionals towards different treatments due to the consequences of ablative surgery vs. MVD; (5) unequal experience with several surgical procedures at a single centre or by the same surgeon; (6) inability to blind to surgical procedures; and (7) measurement of outcomes with extrapolable scales (the Barrow Neurological Institute scale was designed to evaluate the outcomes of radiosurgery, and its correlation with the visual analogue scale is not clear)[13] and treatment objectives, since for pharmacological treatment, a 50% decrease in pain intensity and frequency was required vs. 100% relief with surgery. Given all these limitations, there is a need for new alternatives to perform pragmatic trials on effectiveness. Designing cohort multiple randomised controlled trials may be one such avenue of research.[14]

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https://doi.org/10.1016/j.nrleng.2018.03.016
2173-5808/

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The Brugada pattern in a patient treated with amitriptyline☆

Patrón de Brugada en paciente tratada con amitriptilina

Dear Editor:

Brugada syndrome is an autosomal dominant inherited channelopathy that affects the sodium channels of cardiac cell membranes. It is more frequent in young patients, and diagnosis is based on electrocardiographic (ECG) criteria and clinical history of syncope, or family history of sudden death due to malignant ventricular arrhythmias. Three different Brugada ECG patterns have been described: 1) type I, characterised by a coved-type ST-segment elevation ≥ 2 mm in more than one right precordial lead (V1-V3), followed by negative T wave; this is considered the only diagnostic type of pattern; (2) type II, characterised by an ST-segment elevation ≥ 2 mm in right precordial leads followed by positive or biphasic T wave resulting in a saddle-back configuration; and 3) type III, defined as either of the 2 previous types with ST-segment elevation ≤ 1 mm.[1,2]

Furthermore, several situations and drugs are reported to trigger an ECG pattern of Brugada syndrome.

☆ Please cite this article as: Fernández-Anguita MJ, Martínez-Mateo V, Cejudo Díaz del Campo L, Galindo-Andugar MÁ. Patrón de Brugada en paciente tratada con amitriptilina. Neurología. 2020;35:138—140.

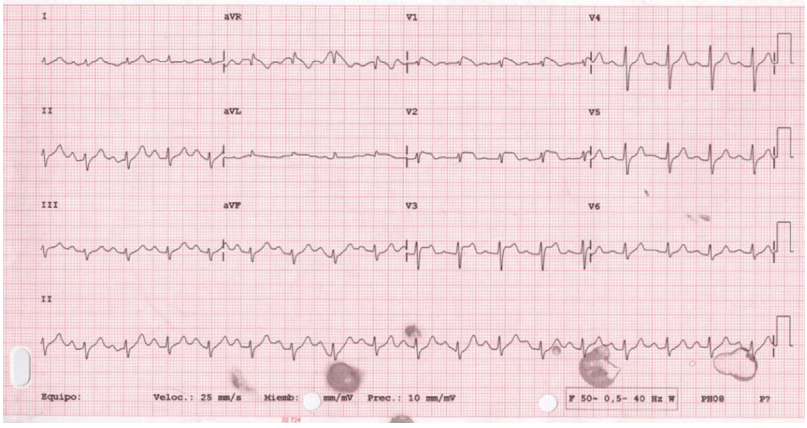

Figure 1 Sinus rhythm with first-degree atrioventricular block with ST-segment elevation of 1 mm in V1-V3 with negative T wave in V1-V2, suggestive of type 1 Brugada pattern.

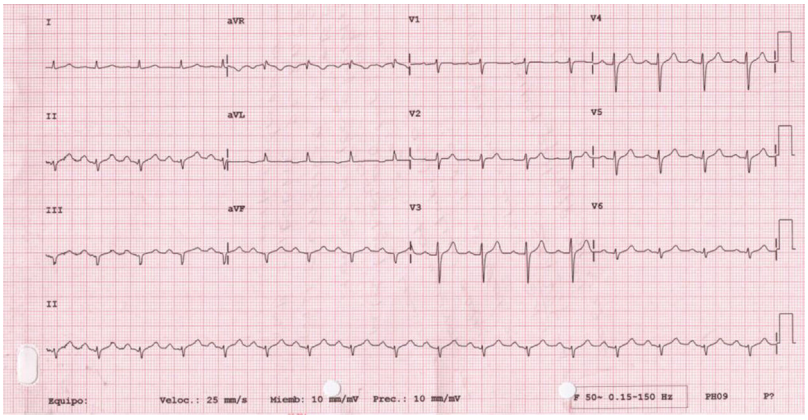

Figure 2 Normalisation of the previous electrocardiographic alterations.

We present the case of a 56-year-old woman with history of smoking, diabetes, hypertension, and dyslipidaemia, treated with insulin, metformin, valsartan/hydrochlorothiazide, and atorvastatin. She was also taking topiramate and levetiracetam for epilepsy and amitriptyline for depression. At baseline, she showed significant limitations due to moderate cognitive impairment. No family history of sudden death was reported. She was admitted to our hospital due to vasovagal syncope in a context of common cold with fever not exceeding 38 °C. The first ECG revealed sinus rhythm with first-degree atrioventricular block, with ST-segment elevation of 1 mm in V1-V3 and negative T wave in V1-V2 (Fig. 1). The initial blood analysis showed: creatinine 2.2 mg/dL; sodium 136 mEq/L; potassium 4.7 mEq/L; magnesium 1.2 mEq/L; pH 7.32; and troponin I < 0.01 ng/mL. As acute anteroseptal ST-elevation myocardial infarction was suspected, we consulted the haemodynamics department, who, considering the uncertain diagnosis of acute coronary syndrome and the patient's physical and cognitive impairment, recommended completing a cardiology study and performing an enzyme series, which revealed no abnormalities. An echocardiography revealed left ventricular hypertrophy with normal ejection fraction and no alterations in segmental contractility. The patient progressed favourably, with complete resolution of the infection and normalised kidney function (creatinine at discharge 0.78 mg/dL). An ECG performed 48 hours after admission showed that alterations in the previous study had disappeared (Fig. 2). A Holter ECG study revealed no significant arrhythmic events. The patient, who was taking drugs capable of inducing type 1 Brugada pattern (amitriptyline), was discharged with diagnosis of Brugada phenocopy (BrP) probably secondary to impaired kidney function, with no associated alterations in electrolyte levels. Considering the patient's significant physical and cognitive impairment, a flecainide test was not performed. We discussed the case with the neurology department with a view to adjusting or discontinuing amitriptyline.

BrPs are characterised by ECG patterns identical to type 1 or 2 Brugada patterns despite absence of true congenital Brugada syndrome. Their clinical causes include hyperkalaemia, adrenal insufficiency, hypothermia, mechanical chest compression, myocarditis, pericarditis, or ischaemia. The current diagnostic criteria for BrP are: 1) the ECG pattern has a Brugada type 1 or type 2 morphology; 2) the patient has an underlying condition that is identifiable; 3) the ECG pattern resolves after resolution of the underlying condition; 4) there is a low clinical pretest probability of true Brugada syndrome determined by lack of symptoms, medical history, and family history; 5) the results

of provocative testing with flecainide, procainamide, ajmaline, or other sodium channel blockers are negative; 6) provocative testing is not mandatory if there has been surgical manipulation of the outflow tract in the previous 96 hours; and 7) the results of genetic testing are negative (not a mandatory criterion, because the *SCN5A* mutation is identified in only 20%-30% of probands affected by true Brugada syndrome).[3] Furthermore, several psychoactive drugs, including lithium, amitriptyline, nortriptyline, oxcarbazepine, and clomipramine are contraindicated in patients with Brugada syndrome due to their potential to block sodium channels, which may induce the appearance of malignant arrhythmias, syncope, or sudden cardiac death.[4,5] Such other drugs as topiramate or levetiracetam are not contraindicated for these patients. This article describes a possible BrP in a patient receiving amitriptyline, a probable cause according to the modified Karch and Lasagna algorithm,[6] in the context of transient kidney dysfunction with no evidence of electrolyte imbalance.

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https://doi.org/10.1016/j.nrleng.2017.11.003
2173-5808/
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Compassionate use of human recombinant insulin-like growth factor-1 therapy in Friedreich's ataxia☆

Utilidad del tratamiento como uso compasivo de mecasermina (factor de crecimiento insulínico recombinante humano tipo 1) en ataxia de Friedreich

Dear Editor:

Friedreich ataxia (FA) is the most frequent autosomal recessive ataxia, and the most prevalent of all the hereditary ataxias (2-4 cases per 100 000 population).[1—3] Symptoms include progressive gait/limb ataxia with motor weakness, areflexia in the lower limbs, loss of proprioception, dysarthria, nystagmus, and auditory alterations. Initial symptoms normally manifest during puberty, although onset may occur from early childhood (2-3 years) to adulthood (> 25 years). FA progresses until the patient loses the ability to walk, 10-15 years after symptom onset.[2,4] Cardiac involvement occurs in 90%-100% of patients and is secondary to the infiltrating process; it ranges from minor electrocardiographic (ECG) alterations to arrhythmias and such structural alterations as left ventricular hypertrophy and interstitial fibrosis with significant collagen proliferation and fatty degeneration, a frequent cause of early death in these patients.[1,5—7]

Most of these cases of FA are caused by a large GAA triplet repeat expansion within the first intron of the frataxin (*FXN*) gene, located on chromosome 9q21.11.[1,8] Frataxin is a mitochondrial protein that activates assembly of iron—sulfur (Fe/S) clusters as part of a multiprotein complex. Although its precise physiological function is yet to be established, it is thought to play an essential role in mitochondrial integrity and functioning, and in cellular iron metabolism.[2] Frataxin is also involved, probably indirectly, in the signalling of antioxidant defence systems and pathways controlling cell survival or death.[2,4] Frataxin deficiency is associated with alterations to Fe/S cluster biogenesis and iron homeostasis, as well as increased levels of oxidative stress, ultimately leading to neurodegeneration and heart disease.[2,4]

Despite ongoing research into possible disease-modifying drugs, focusing on compounds increasing frataxin levels or

☆ Please cite this article as: García Ron A, Rodriguez Mesa M. Utilidad del tratamiento como uso compasivo de mecasermina (factor de crecimiento insulínico recombinante humano tipo 1) en ataxia de Friedreich. Neurología. 2020;35:140—143.