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Case Report

Intermittent Brugada syndrome in an anorexic adolescent girl

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ABSTRACT

We report an anorexic adolescent girl with an intermittent Brugada syndrome. A 14-year-old anorexic girl with a body mass index (BMI) of 13.15 kg/m^2 was admitted in the acute state of the disease with an ST elevation in V1 and V2, suggestive of Brugada syndrome. After 1 month of re-feeding, a control electrograph (ECG) was normal, but after an 8-month follow-up control with a nearly normal BMI, the ECG was again suggestive of Brugada syndrome. A genetic analysis of the gene SNC5A established a genetic change (p Leu 1582 pro), which provides the final explanation for the Brugada syndrome. Every rhythm problem in the acute state or during the re-feeding procedure deserves a strict follow-up to distinguish iatrogenic from heritable rhythm problems.

<Learning objective: (i) We report the first case of a patient with anorexia nervosa with an intermittent Brugada syndrome. (ii) Moderate hypothermia can decrease the depolarization of pacemaker cells and cause ST-segment changes. (iii) Every rhythm problem in the acute state or during the re-feeding procedure deserves a strict follow-up to distinguish iatrogenic from heritable rhythm problems. (iv) A genetic analysis can make the distinction and is necessary to give advice for the future lifestyle of the patient.>

Introduction

The most common rhythm disorders in the anorexic population are extreme bradycardia <50 bpm with a prolonged QT interval (15–40%) and sudden death in up to 10%. Other electrograph (ECG) abnormalities seen in these patients are low voltage of P waves and QRS complexes, prolonged QTc, rightward QRS axis, non-specific ST-T changes, and the presence of U waves. Less frequent are ectopic atrial foci and second degree AV block Mobitz type I [1].

Minor T wave changes, such as T-wave inversion in V4 and T flattening, are also noticed.

ST depression is observed in patients with anorexia nervosa with extreme weight loss. The explanation of ST disturbances can be found in the early repolarization and acidosis due to hypothermia. Moderate hypothermia can decrease the depolarization of pacemaker cells and cause ST-segment changes.

Case report

A 14-year-old girl with anorexia nervosa was admitted to the department of eating disorders with a body mass index (BMI) of 13.15 kg/m^2 (weight: 35.8 kg and height: 165 cm) and a weight loss of 26.94% in less than 6 months.

In the personal antecedents, she had no episodes of palpitations or suspicious syncopes. She once fainted following a tooth extraction. In childhood she had atopic eczema and there were no surgical procedures or hospitalizations.

The girl has an older brother and a sister age 22 and 20 years, respectively, and both had a syncope during a peripheral venous blood sample. No other suspicious episodes were noticed. Both parents are in good health. The father is an intense sportsman but never had any syncope. In the other family members we recorded neither had syncope nor sudden death.

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During the physical examination, a cachectic girl (BMI: 13.15 kg/m^2, core temperature: 34.9 °C) presented with the typical features of anorexia nervosa namely lanugo hairs, acrocyanosis, hypotension (87/46 mm Hg) and bradycardia 39 bpm. A syncope was not observed, neither chest pain and no medication were taken.

Routine blood chemistry (creatinine, blood urea nitrogen, potassium, sodium, bicarbonate, magnesium and calcium) and thyroid hormones were normal. There was a high ferritin level (258 ng/ml) (*N*: 5.0–122 ng/ml) and a slight elevation of the liver transaminases [aspartate aminotransferase (58 U/L) (*N*: 10–30 U/L), alanine aminotransferase (128 U/L) (*N*: 17–44 U/L)] and troponin I 0.0039 ng/ml (*N*: <0.0034 ng/ml).

Creatinine kinase and lactate dehydrogenase levels were normal. The levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), and insulin-growth factor-1 were low.

In the acute phase, an ECG together with a 2-D-echocardiography with Doppler was obtained.

The ECG showed ST elevation in V1, V2 and less in V3 (suggestive of Brugada syndrome) (Fig. 1). The rhythm was 49 bpm, axis 11°, PR interval: 144 ms, QRS: 114 ms, QT: 422 ms, and QTc 384 ms. Echocardiography also showed a pericardial effusion of 0.9–1.1 cm (apex) and a decreased left ventricular mass/height$^{2.7}$: 17.57 g/m$^{2.7}$

Magnetic resonance imaging of the heart confirmed a moderate pericardial effusion, end diastolic volume of 54.04 ml, end-systolic volume of 26.47 ml, stroke volume of 1654.69 ml, and an ejection fraction of 51% (Fig. 2).

Compatible with the bradyphrenic character of the patient, magnetic resonance imaging of the brain showed "cerebral atrophy" in the frontotemporal and cerebellar region (Fig. 3).

ECG at rest of both parents was normal. After 1 month of slow re-feeding BMI: 13.7 kg/m^2 (to prevent the re-feeding syndrome), there was a normalization of the ECG with no ST elevation in V1 and V2 (Fig. 4). But after 8 months of re-feeding (BMI: 18.5 kg/m^2) (weight: 51 kg and height: 166 cm), a control ECG demonstrated Brugada syndrome again in V1 and V2 (Fig. 5).

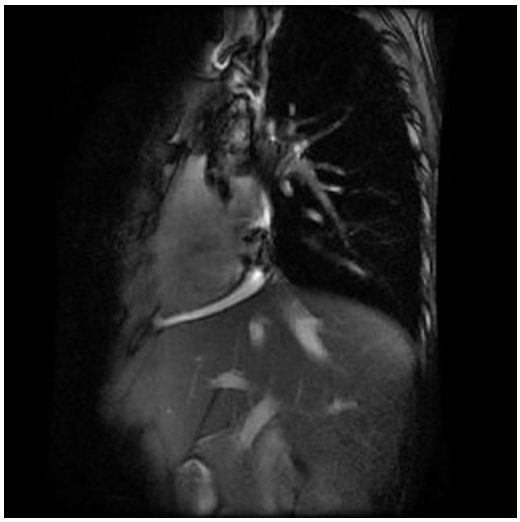

Fig. 2. Magnetic resonance imaging of the heart: moderate pericardial effusion.

The patient did not receive any treatment but education and prevention of arrhythmia via lifestyle awareness (avoidance of certain medications, fever treated with antipyretics) was discussed with the patient and her parents. A genetic analysis of the gene SNC5A established a genetic change (p Leu 1582 pro), which provides an explanation for the Brugada sequence. The mother had a normal genetic analysis and a normal cardiologic examination. The father refused any further cardiologic or genetic analysis.

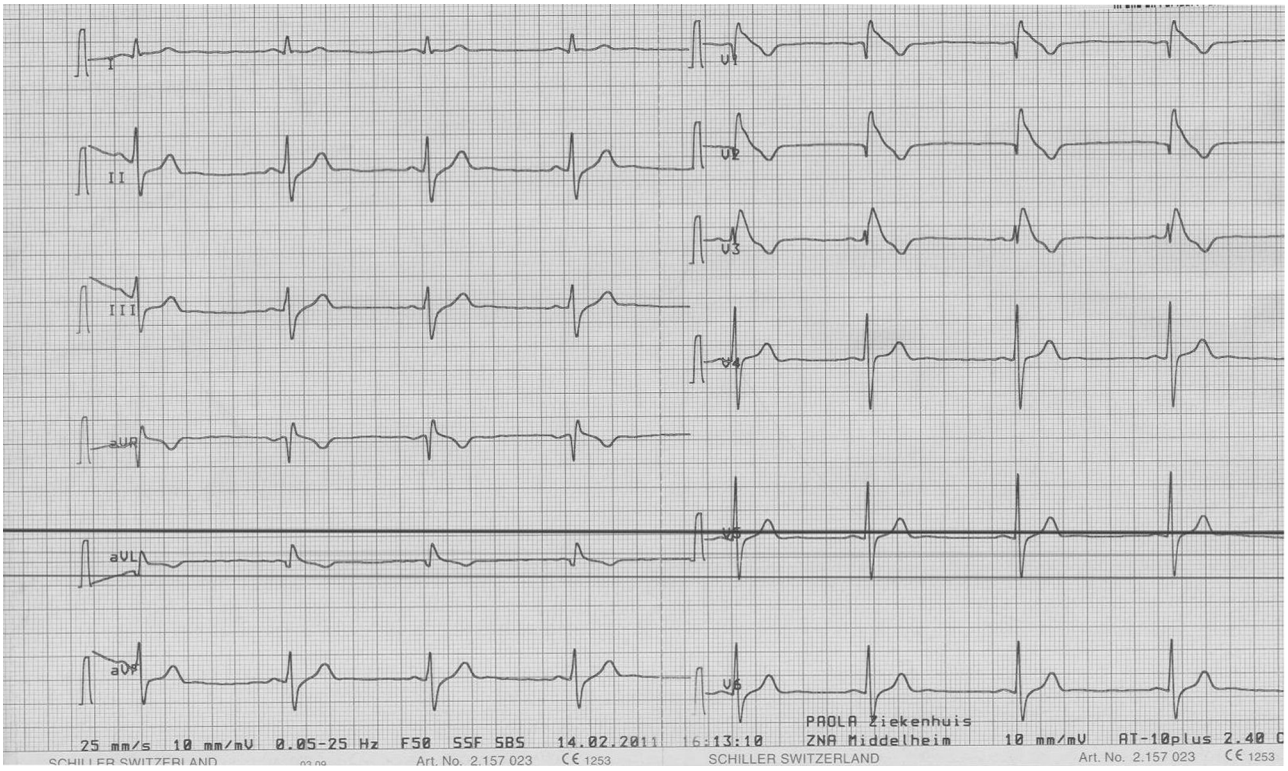

Fig. 1. A 12-lead electrocardiogram – ST elevation in V1, V2 and less in V3. Rhythm: 49 bpm, axis 11°, PR interval: 144 ms, QRS: 114 ms, QT: 422 ms, and QTc 384 ms. Type I electrocardiogram.