Folia Cardiologica 2021
vol. 16, no. 4, pages 259–262
DOI: 10.5603/FC.2021.0039

ISSN 2353–7752
e-ISSN 2353–7760

Syncope and electrocardiographic suspicion of acute anterior ST-elevation myocardial infarction — think about Brugada syndrome!

Omdlenie i elektrokardiograficzne podejrzenie zawału serca z uniesieniem odcinka ST nad ścianą przednią — pomyśl o zespole Brugadów!

Małgorzata Wojciechowska[1, 2], Karolina Rybak[1], Maciej Zarębiński[2], Łukasz Pastwa[3], Agnieszka Cudnoch-Jędrzejewska[1]

[1]Department of Experimental and Clinical Physiology, Laboratory of Centre for Preclinical Research, Medical University of Warsaw, Warsaw, Poland
[2]Independent Public Specialist Western Hospital John Paul II, Invasive Cardiology Unit,
[3]Independent Public Specialist Western Hospital John Paul II, Cardiology Unit, Grodzisk Mazowiecki, Poland

Abstract

This is a case of a 52-year-old male after an episode of syncope and with atypical chest pain referred to the hospital with acute anterior ST-elevation myocardial infarction. Urgent coronary angiography showed normal coronary arteries and only an electrocardiogram (ECG) made the next day induced to suspect Brugada syndrome. The patient had implantable cardioverter-defibrillator implanted and because of the high defibrillation threshold, subcutaneous electrode implantation was decided. The present case highlights that ECG changes in Brugada syndrome can mimic ST elevation in the course of the acute coronary syndrome and that subcutaneous electrode implantation may be a useful method of lowering the defibrillation threshold.

Key words: Brugada syndrome, cardioverter-defibrillator implantation, defibrillation threshold, subcutaneous electrode

Folia Cardiologica 2021; 16, 4: 259–262

Introduction

We present a patient after syncope, in whom initial examination and electrocardiogram (ECG) suggested acute coronary syndrome (ACS) with elevated ST-segment but the final diagnosis was Brugada syndrome (BrS).

Case report

A 56-year-old male was admitted to the hospital after syncope, which occurred without any prodromal symptoms, during a walk. His history revealed frequent episodes of palpitation and atypical chest pain, but no previous syncope.

Adress for correspondence: Małgorzata Wojciechowska MD, PhD, Katedra i Zakład Fizjologii Doświadczalnej i Klinicznej, Laboratorium Katedry i Zakładu Fizjologii Doświadczalnej i Klinicznej, Centrum Badań Przedklinicznych, Warszawski Uniwersytet Medyczny, ul. Banacha 1B, 02–097 Warszawa, Poland, phone + 48 22 116 61 13, fax +48 22 116 62 01, e-mail: malgorzata.wojciechowska2@wum.edu.pl

Since childhood, he was treated for asthma with inhaled corticosteroids and beta$_2$ agonists. For a few days before admission to the hospital, he had suffered from an upper respiratory tract infection with fever and he escalated the frequency of inhaled drugs. There was no family history of sudden cardiac death.

On admission he was conscious, Glasgow Coma Scale (GCS) 15 points, vital signs within normal limits. He complained about atypical chest pain. Electrocardiogram (ECG) showed ST elevation in V1–V3 leads. When intracranial bleeding was excluded in the head computed tomography (CT) scan, the patient was qualified for urgent coronary angiography, which showed normal coronary arteries. Echocardiography didn't reveal any abnormalities. Apart from elevated C-reactive protein (CRP), there were no other abnormalities in laboratory tests.

During the next days repeated ECG records revealed changes typical for BrS (Figure 1A–C), so the patient was

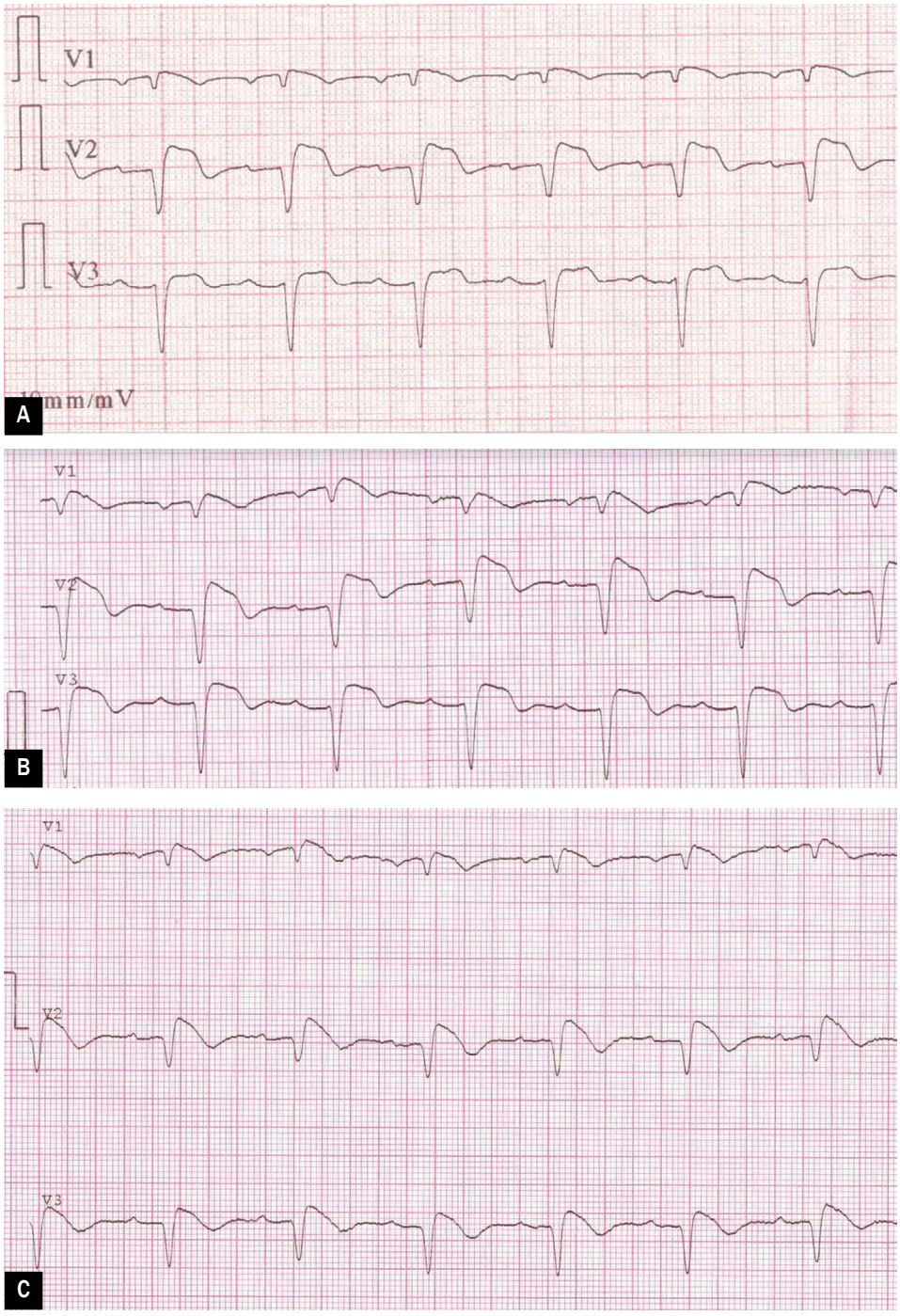

Figure 1A, B. Evolution of electrocardiogram changes in V1–V3 during next days of hospitalization; **C.** Brugada type 1 pattern became evident

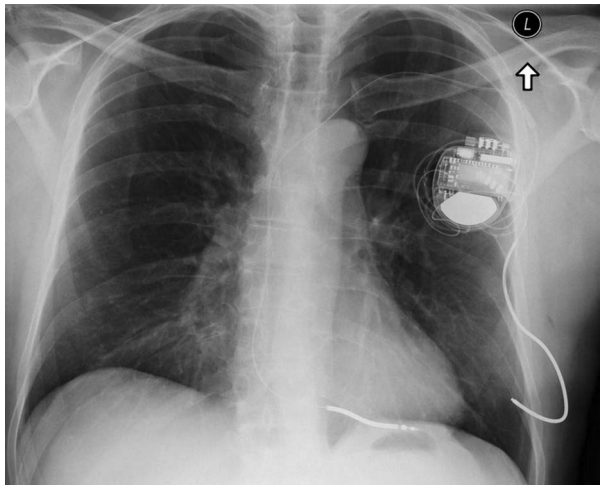

Figure 2. Chest X-ray after implantable cardioverter-defibrillator (ICD) implantation with additional subcutaneous electrode

qualified for implantable cardioverter-defibrillator (ICD) implantation. As the defibrillation test revealed a high defibrillation threshold (DT), the patient underwent subcutaneous electrode implantation (Figure 2) and in the second defibrillation test, DT became acceptable. The patient has been remotely monitored (CareLink) and in two years' follow-up, any arrhythmic episode was recorded.

Discussion

BrS is a rare disorder inherited in an autosomal dominant manner. There is a tendency to severe ventricular arrhythmia and the first sign of the disease may be sudden cardiac death [1]. The diagnosis is based on characteristic ECG features (type 1 pattern, ≥ 2 mm ST-segment elevation in lead V1 and/or V2, Figure 1C), which have high dynamicity, can appear temporarily and change in time [1, 2]. We can diagnose BrS when these ECG changes occur either spontaneously or appear during provocative drug tests with sodium-channel blockers. Lethal arrhythmias usually occur during rest or sleep, which suggests an association with increased parasympathetic tone. Triggers, that unmask ECG type 1 pattern are also some drugs, fever, excessive alcohol intake and large meals [1, 3]. Pathophysiology refers to right ventricular outflow tract (RVOT) and ion channel dysfunction, however, increased fibrosis and decreased expression of gap junction in RVOT has also been hypothesized [1, 4]. The current main therapy is ICD implantation indicated in symptomatic patients (resuscitated sudden cardiac arrest or syncope) [5], but catheter ablation has been recently reported as a new treatment [4]. In patients with spontaneous type 1 pattern but without any symptoms, the electrophysiological test can be used to assess the need for ICD [5].

Due to possible significant adverse events, routine DT testing is no longer recommended in all patients at the time of ICD implantation [6]. However, it is reasonable to perform the test, if there are risk factors for high DT, like young age, high body mass index, non-ischemic cardiomyopathy or congenital heart diseases [6, 7]. In the present case, the DT test was performed due to the increased risk of high DT in patients with inherited channelopathies.

Many laboratory, clinical and echocardiographic factors that increase the risk of high DT have been identified, some of them may be reversible and easy to eliminate [7]. Subcutaneous electrode implantation is the method of lowering DT that is used, when (like in this case) the reason for high DT is irreversible or cannot be eliminated [7, 8].

As mentioned before, fever is a strong factor causing changes in the ECG and leading to dangerous arrhythmias in the course of BrS [3]. The patient was instructed about the rapid relief of any fever and for 2 years any arrhythmic episode has been recorded.

Conclusions

The present case highlights that ECG changes in BrS can mimic ST elevation in the course of ACS. Furthermore, a coincidence of ST elevation in V1–V3 and syncope should direct one's thinking towards BrS, even if ECG changes are not typical, as they are very dynamic and may even conceal temporarily [2]. According to a recommendation, this symptomatic patient was qualified for ICD implantation [5] and additionally, he was advised prompt treatment of any fever with antipyretic drugs [3, 5]. Routine DT testing is no longer recommended in all patients with ICD and subcutaneous electrode implantation may be a useful method of lowering DT [6–8].

Conflict of interests

The authors declare that there is no conflict of interest.

Streszczenie

Zaprezentowano opis przypadku 52-letniego mężczyzny, który po epizodzie omdlenia i bólu w klatce piersiowej trafił do szpitala z podejrzeniem ostrego zawału serca z uniesieniem odcinka ST nad ścianą przednią. Pilna koronarografia wykazała prawidłowy obraz tętnic wieńcowych i dopiero zapisy elektrokardiograficzne (EKG) wykonane w kolejnych dobach doprowadziły do rozpoznania zespołu Brugadów. Pacjentowi wszczepiono implantowalny kardiowerter-defibrylator, a ze względu na wysoki próg defibrylacji konieczne było wszczepienie elektrody podskórnej. Przypadek opisanego pacjenta przypomina, że zmiany w EKG w przebiegu zespołu Brugadów mogą naśladować uniesienie odcinka ST w przebiegu ostrego zespołu wieńcowego, a podskórna implantacja elektrody może być skuteczną metodą obniżania progu defibrylacji.

Słowa kluczowe: zespół Brugadów, implantacja kardiowertera-defibrylatora, próg defibrylacji, elektroda podskórna

Folia Cardiologica 2021; 16, 4: 259–262

References

1. Brugada J, Campuzano O, Arbelo E, et al. Present status of Brugada syndrome: JACC state-of-the-art review. J Am Coll Cardiol. 2018; 72(9): 1046–1059, doi: 10.1016/j.jacc.2018.06.037, indexed in Pubmed: 30139433.
2. Dybich P, Bąkowski D, Wożakowska-Kapłon B. [Syncope in male — let us think about Brugada syndrome! Presentation of 3 cases] [Article in Polish]. Kardiol Pol. 2010; 68(12): 1397–400; discussion 1401, indexed in Pubmed: 21174301.
3. Rattanawong P, Vutthikraivit W, Charoensri A, et al. Fever-induced Brugada syndrome is more common than previously suspected: a cross-sectional study from an endemic area. Ann Noninvasive Electrocardiol. 2016; 21(2): 136–141, doi: 10.1111/anec.12288, indexed in Pubmed: 26178440.
4. Nademanee K, Hocini M, Haïssaguerre M. Epicardial substrate ablation for Brugada syndrome. Heart Rhythm. 2017; 14(3): 457–461, doi: 10.1016/j.hrthm.2016.12.001, indexed in Pubmed: 27979714.
5. Priori SG, Blomström-Lundqvist C, Mazzanti A, et al. Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: the Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J. 2015; 36(41): 2793–2867, doi: 10.1093/eurheartj/ehv316, indexed in Pubmed: 26320108.
6. Hayase J, Do DH, Boyle NG. Defibrillation threshold testing: current status. Arrhythm Electrophysiol Rev. 2018; 7(4): 288–293, doi: 10.15420/aer.2018.54.2, indexed in Pubmed: 30588318.
7. Jacob S, Pidlaoan V, Singh J, et al. High defibrillation threshold: the science, signs and solutions. Indian Pacing Electrophysiol J. 2010; 10(1): 21–39, indexed in Pubmed: 20084193.
8. Kempa M, Lubiński A, Wilczek R, et al. Zastosowanie podskórnej elektrody defibrylującej w celu obniżenia progu defibrylacji migotania komór u pacjentów z ICD. Folia Cardiol. 2004; 11(6): 463–470.