

Transient appearance of Brugada-like Type 1 electrocardiogram pattern immediately after biphasic synchronized electrical cardioversion for atrial fibrillation: a case series

Khalid A.S. Abdalla ¹, Antonio Sorgente ^{2*}, and Pedro Brugada^{3,4,5}

¹Academic Affairs, Mafraq Hospital, Bani Yas, PO Box 2951, Abu Dhabi, United Arab Emirates; ²Heart and Vascular Institute, Cleveland Clinic Abu Dhabi, Abu Dhabi Global Market Square, 112412 Abu Dhabi, United Arab Emirates; ³Cardiovascular Division, Free University of Brussels (UZ Brussel) VUB, Avenue du Laerbeek 101, 1090 Jette, Brussels, Belgium; ⁴CEO Medical Centre Prof. Brugada, Brusselse Steenweg 161, 9300 Aalst, Belgium; and ⁵Arrhythmia Unit, Hospiten Estepona, Carretera Nacional 340, Km. 162, 29680 Estepona, Málaga, Spain

Received 22 February 2019; first decision 16 April 2019; accepted 9 October 2019; online publish-ahead-of-print 26 October 2019

Background

ST-segment deviation post-electrical cardioversion is a common finding amongst a significant number of patients. However, the mechanism by which this phenomenon occurs and its clinical implications are not fully understood.

Case summary

Four patients presented to our department with complaints of palpitations. They were found to have atrial fibrillation and were cardioverted using a synchronized direct current shock at 200J. However, their telemetry immediately after the shock showed transiently an ST-segment elevation resembling Brugada Type 1 pattern. All telemetries normalized within 6 s from cardioversion.

Discussion

Through this report, we aim to raise mild concern about a possible safety issue related to synchronized electrical cardioversion with electrodes positioned in an anteroposterior fashion. Also, we try to give a pathophysiological explanation to this finding on the base of the knowledge accumulated through the years on Brugada syndrome.

Keywords

Case series • Brugada syndrome • Na⁺ channel • ST-segment deviation • Electrical cardioversion

Learning points

- Electrical cardioversion through pads positioned in an anteroposterior fashion can cause transient ST-segment deviation which resembles Brugada Type 1 pattern.
- Till more data will be available to support this finding, we recommend caution in the use of defibrillation pads in the anteroposterior fashion in those patients predisposed to ventricular arrhythmias.

Introduction

Direct current (DC) shock has been widely used to cardiovert atrial and ventricular arrhythmias for decades. However, its effect on cardiac myocytes has yet to be completely understood.¹ Direct current shock has been shown to cause transient electrocardiogram (ECG) changes, which include ST-segment deviation.^{1,2} The mechanism by which this phenomenon occurs and its clinical implications are not

* Corresponding author. Tel: +393491512694, Email: sorgente.antonio@gmail.com

Handling Editor: Borislav Dinov

Peer-reviewers: Rami Riziq Yousef Abumualeq and Tor Biering-Sørensen

Compliance Editor: Mark Philip Cassar

Supplementary Material Editor: Peysh A. Patel

© The Author(s) 2019. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

completely understood, and the literature is lacking in this regard.¹ It is estimated that 15–50% of patients undergoing electrical cardioversion will have transient ST-segment deviation.^{1,2}

We are currently unaware of any report of transient ST-segment elevation resembling Brugada Type 1 post-DC shock. We reviewed four cases in which DC shock induced a Brugada Type 1 ECG pattern in patients who do not have an established diagnosis of Brugada syndrome (BS). Through these findings, we aim to raise mild concern about a possible safety issue related to synchronized electrical cardioversion with the electrodes positioned in an anteroposterior fashion. Furthermore, we try to give a pathophysiological explanation to this finding based on of the knowledge accumulated through the years on BS.

We present four cases of attempted electrical cardioversion of persistent atrial fibrillation. Transoesophageal echocardiogram was performed in all cases to rule out the presence of clots in the left atrial appendage and left atrium. All patients received deep sedation via intravenous administration or infusion of fentanyl, midazolam, and propofol under the supervision of a senior anaesthetist. All patients received 300 mg of amiodarone intravenously prior to the procedure in order to increase the likelihood of successful cardioversion. All patients received a synchronized biphasic DC shock at 200 J. The pads were placed in an anteroposterior fashion. All patients had a negative family history for sudden cardiac death and/or inherited channelopathies; none of them though underwent an ajmaline test before or after electrical cardioversion or genetic tests to evidence mutations compatible with BS. ST elevation persisted for a maximum of 6 s after electrical cardioversion. All patients were discharged 2 h after the procedure without complications.

Timeline

Case series presentation

Patient 1

Patient 1 is a 65-year-old man with persistent atrial fibrillation, and a history of hypertension, hyperlipidaemia, hyperuricaemia, and kidney stones; of transient ischaemic attack, coronary artery disease status post-percutaneous coronary angioplasty of the left anterior descending artery; of catheter ablation of atrial fibrillation; and of two electrical cardioversions after catheter ablation, normal left ventricular function and no valvular abnormality as per his last echocardiogram. He was presented to our outpatient clinic with palpitations and dizziness. Electrocardiogram on admission revealed atrial fibrillation. His height and weight were 160 cm and 79 kg, respectively. His blood pressure was 122/84 mmHg. His electrolytes were as follows: sodium 138 mmol/L, potassium 4.4 mmol/L, and calcium 2.32 mmol/L. His current list of medications included rivaroxaban 20 mg o.d., and rosuvastatin 10 mg o.d.; he was off of antiarrhythmic drugs and/or beta-blockers. He received 300 mg of amiodarone intravenously before cardioversion. *Figure 1A* shows his ECG prior to cardioversion. He received one DC shock at 200 J and was reverted back to sinus rhythm. *Figure 2A* shows his ECG during cardioversion. *Figure 3A* shows his ECG after electrical cardioversion. The patient's clinical status has been unremarkable since electrical cardioversion. He has not experienced any recurrence of atrial fibrillation and is currently on rivaroxaban 20 mg o.d., rosuvastatin 10 mg o.d., and sotalol 80 mg b.i.d.

Patient 2

Patient 2 is a 42-year-old man with a history of rheumatic aortic and mitral valve disease, status post-replacement of both valves with mechanical prostheses and tricuspid annuloplasty, severe tricuspid regurgitation, severe systolic left ventricular dysfunction, and active

Patient 1

A 65-year-old man with persistent atrial fibrillation presented to our outpatient clinic with palpitations and dizziness. Electrocardiogram (ECG) on admission revealed atrial fibrillation. *Figure 1A* shows his ECG prior to cardioversion. He received one direct current (DC) shock at 200 J and was reverted back to sinus rhythm. *Figure 2A* shows his ECG during cardioversion. *Figure 3A* shows his ECG after electrical cardioversion.

Patient 2

A 42-year-old man presented to the emergency room with shortness of breath, paroxysmal nocturnal dyspnoea, orthopnoea, palpitations, and lightheadedness. Electrocardiogram on admission revealed atrial fibrillation. *Figure 1B* shows his ECG prior to cardioversion. He received one DC shock at 200 J and was reverted back to sinus rhythm. *Figure 2B* shows his ECG during cardioversion. *Figure 3B* shows his ECG after electrical cardioversion.

Patient 3

A 66-year-old man with persistent atrial fibrillation presented to the emergency room with palpitations. Electrocardiogram on admission revealed atrial fibrillation. *Figure 1C* shows his ECG prior to cardioversion. He received one DC shock at 200 J and was reverted back to sinus rhythm. *Figure 2C* shows his ECG during cardioversion. *Figure 3C* shows his ECG after electrical cardioversion.

Patient 4

A 56-year-old man presented to the emergency room with palpitations and shortness of breath. Electrocardiogram on admission revealed atrial fibrillation. *Figure 1D* shows his ECG prior to cardioversion. He received a total of three DC shocks and was reverted back to sinus rhythm with early recurrence of atrial fibrillation. *Figure 2D* shows his ECG during cardioversion. *Figure 3D* shows his ECG after last electrical cardioversion.

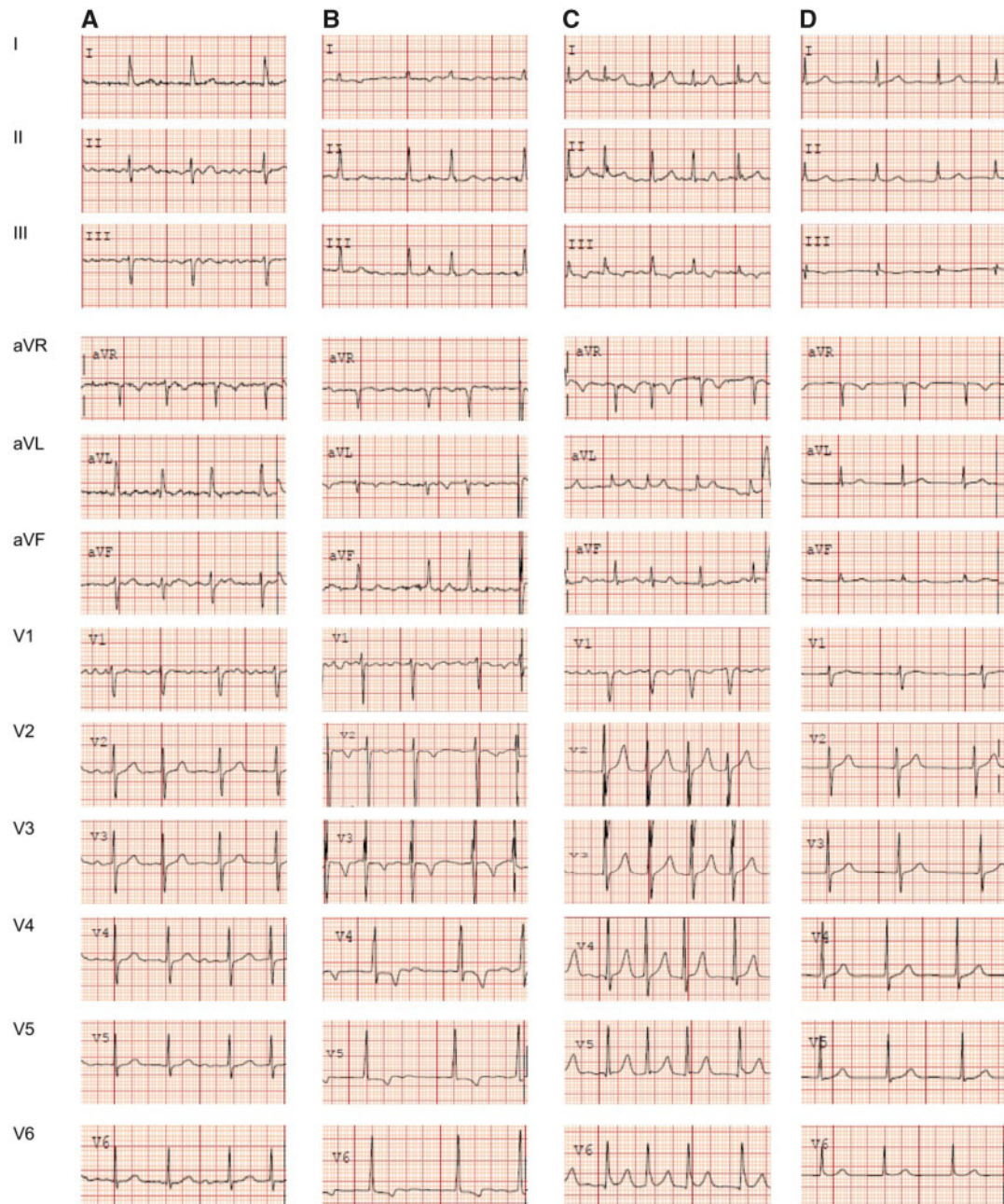


Figure 1 (A–D) Twelve-lead electrocardiograms from the four cases presented to our department.

smoking, but no other cardiovascular risk factors and no previous documentation of atrial fibrillation. He was presented to the emergency room with shortness of breath, paroxysmal nocturnal dyspnoea, orthopnoea, palpitations, and lightheadedness. Electrocardiogram on admission revealed atrial fibrillation. His current list of medications included warfarin in order to have an INR 2–3, furosemide 40 mg b.i.d. i.v., ramipril 1.25 mg o.d., spironolactone 25 mg o.d., digoxin 250 mcg o.d., bisoprolol 2.5 mg o.d., and alprazolam 0.5 mg o.d. His height and weight were 173 cm and 68 kg, respectively. His blood pressure was 85/65 mmHg. His electrolytes were as follows:

sodium 141 mmol/L, potassium 4.2 mmol/L, and calcium 2.31 mmol/L. He received 300 mg of amiodarone intravenously before cardioversion. *Figure 1B* shows his ECG prior to cardioversion. He received one DC shock at 200J and was reverted back to sinus rhythm. *Figure 2B* shows his ECG during cardioversion. *Figure 3B* shows his ECG after electrical cardioversion. The patient had a recurrence of atrial fibrillation a month after the above-mentioned cardioversion; he stopped amiodarone for unknown reasons after electrical cardioversion. Since then, he has been in persistent atrial fibrillation with no improvement in his left ventricular ejection fraction despite being on

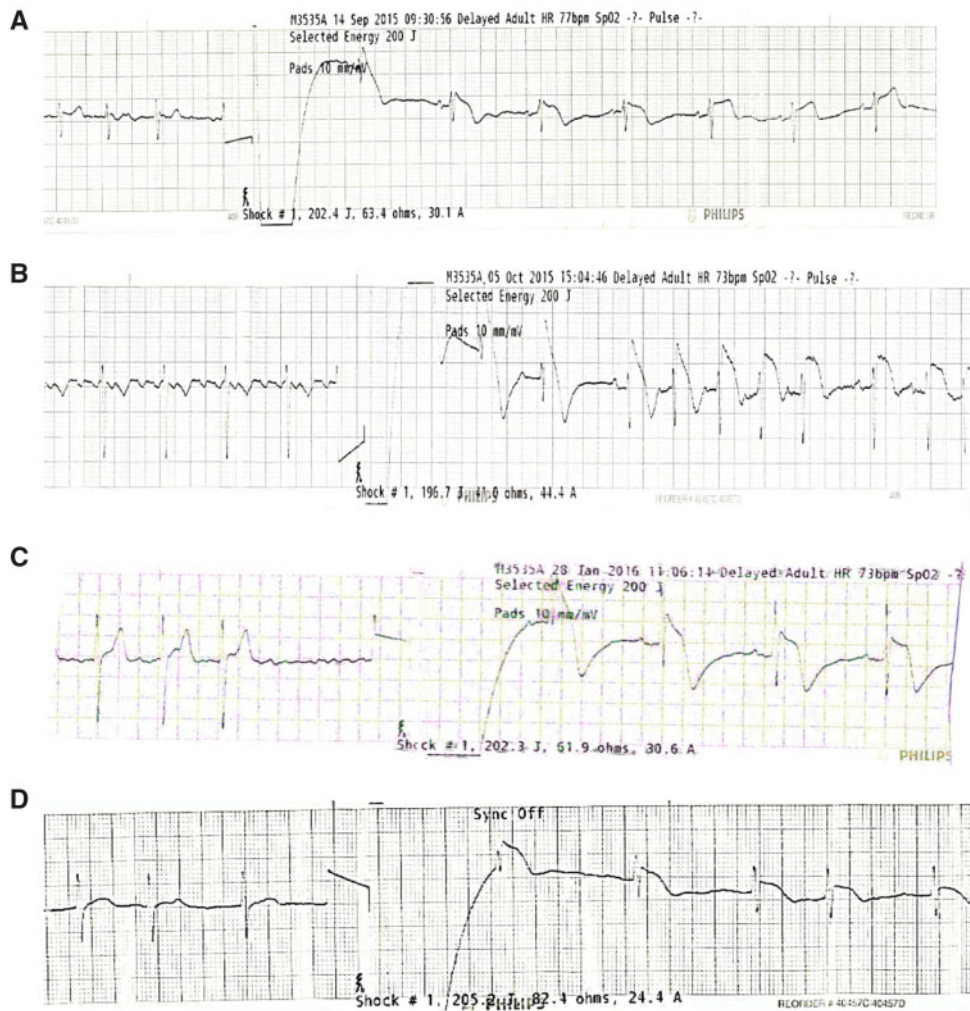


Figure 2 (A–D) Electrocardiogram telemetry at the time of electrical cardioversion in the four cases.

full guideline-directed heart failure medical treatment and has, so far, refused implantation of a defibrillator.

Patient 3

Patient 3 is a 66-year-old man with a history of catheter ablation of atrial fibrillation in January 2016, status post two electrical cardioversions in 2014 and 2015; coronary artery disease with percutaneous coronary angioplasty of the left anterior descending artery in 2008; and hypertension and dyslipidaemia, but normal left ventricular function and no valvular abnormality as per the most recent echocardiogram. He was presented to the emergency room with palpitations. His height and weight were 185 cm and 77 kg, respectively. His blood pressure was 125/77 mmHg. His electrolytes were as follows: sodium 139 mmol/L, potassium 3.6 mmol/L, and calcium 2.06 mmol/l. His list of medications included bisoprolol 2.5 mg o.d., amlodipine 5 mg o.d., pantoprazole 20 mg o.d., ramipril 5 mg o.d., rivaroxaban 20 mg o.d., and atorvastatin 10 mg o.d. Electrocardiogram on admission revealed atrial fibrillation. He received 300 mg of amiodarone intravenously

before cardioversion. *Figure 1C* shows his ECG prior to cardioversion. He received one DC shock at 200J and was reverted back to sinus rhythm. *Figure 2C* shows his ECG during cardioversion. *Figure 3C* shows his ECG after electrical cardioversion. The patient had a recurrence of atrial fibrillation 5 months after cardioversion and a redo catheter ablation for atrial fibrillation afterwards. Since then, no documented recurrence of atrial fibrillation but at least one episode of palpitation lasting 1 h was noted. He is currently on long acting flecainide 200 mg o.d., bisoprolol 2.5 mg o.d., and rivaroxaban 20 mg o.d.

Patient 4

Patient 4 is a 56-year-old man with a history of initial idiopathic dilated cardiomyopathy with a stable left ventricular ejection fraction of 45–50%, pre-diabetes and previous cerebrovascular accidents presented to the emergency room with palpitations and shortness of breath. His height and weight were 168.5 cm and 92 kg, respectively. His blood pressure was 146/92 mmHg. His electrolytes were as

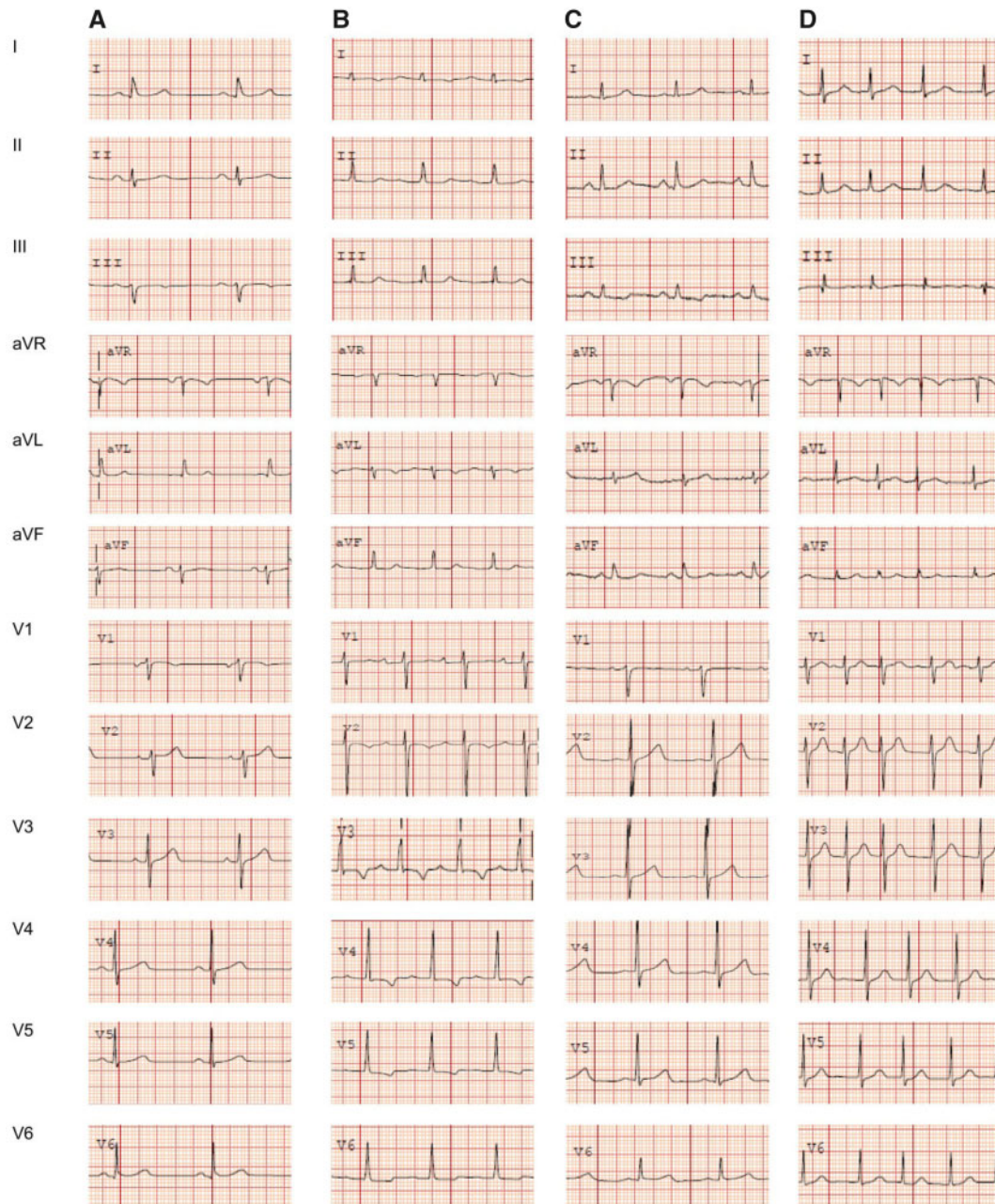


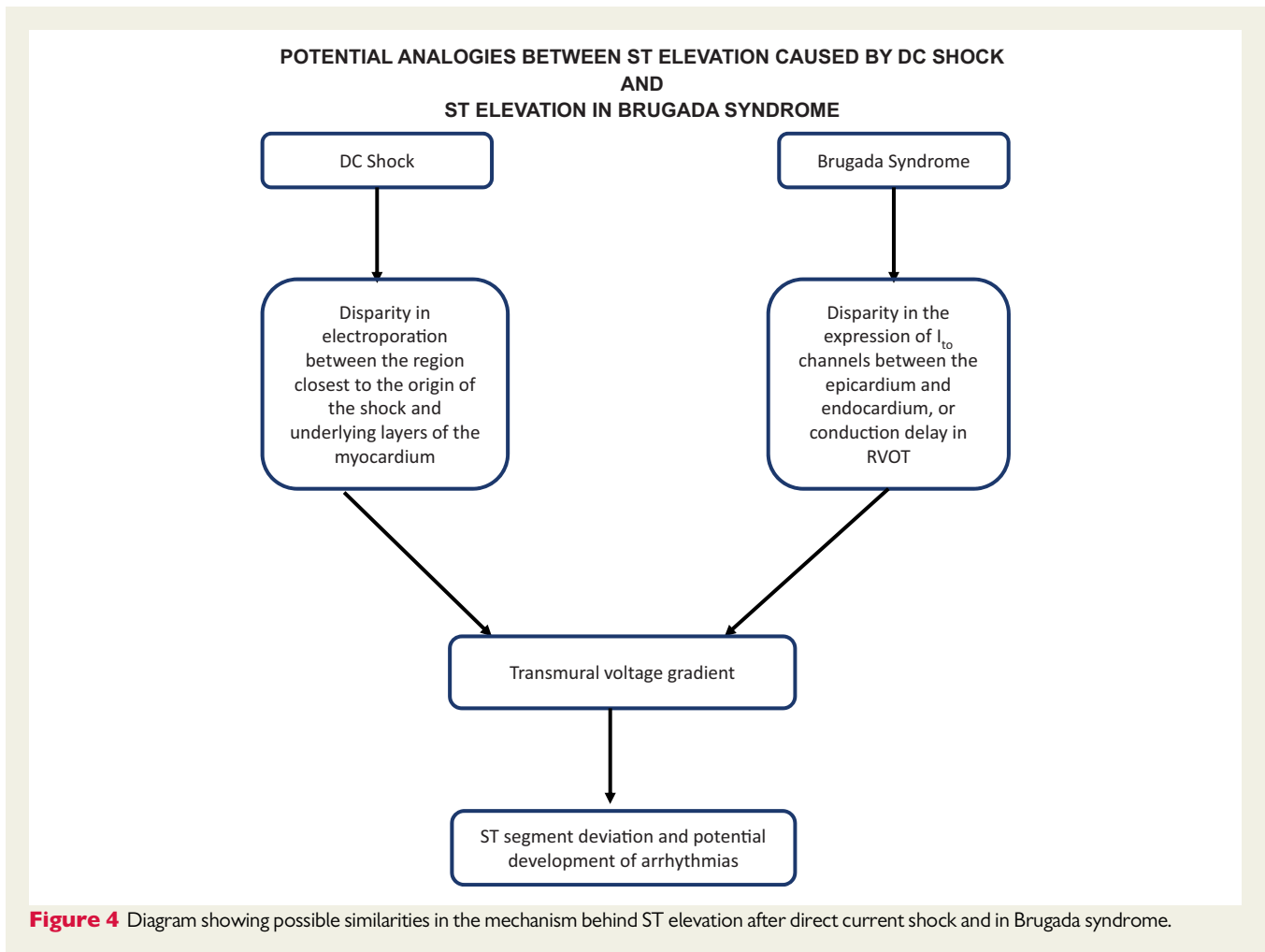
Figure 3 (A–D) Twelve-lead electrocardiograms from the four cases after attempted electrical cardioversion.

follows: sodium 139 mmol/L, potassium 4.1 mmol/L, and calcium 2.30 mmol/l. His list of current medications included: bisoprolol 5 mg o.d., sacubitril-valsartan 100 mg b.i.d., digoxin 62.5 mcg o.d., and dabigatran 150 mg b.i.d. Electrocardiogram on admission revealed atrial fibrillation. He received 300 mg of amiodarone intravenously before cardioversion. [Figure 1D](#) shows his ECG prior to cardioversion. He received a total of three DC shocks and was reverted back to sinus rhythm with early recurrence of atrial fibrillation. [Figure 2D](#) shows his ECG during cardioversion. [Figure 3D](#) shows his ECG after the last electrical cardioversion. The patient was left in a state of permanent

atrial fibrillation with rate control medications as listed above; no further clinical events have been reported in the follow-up.

Discussion

This is the first case series showing the transient appearance of Type 1 Brugada pattern on the ECG immediately after electrical cardioversion in patients with diverse clinical backgrounds and persistent atrial fibrillation. Four cases are not enough to make any definitive



conclusions from a scientific perspective. In addition, all the discussion on these cases is only speculative. Nevertheless, we think it could provide some food for thought on the mechanism of the ST elevation in concomitance with electrical cardioversion and could potentially offer some insights into the electrophysiology of BS.

Brugada syndrome is an inherited condition affecting cardiac sodium, potassium, or calcium channels.³ The hallmark of BS is an increased risk for sudden cardiac death in patients with no apparent structural heart defects.^{3,4} Several hypotheses have been proposed to explain the pathophysiology behind BS but the most significant ones are the repolarization and the depolarization hypotheses.

The repolarization hypothesis is based on the presumed disparity of expression of I_{to} channels between the right and left ventricles, and between the epicardium and endocardium. Genetically driven decreased inward sodium current, as is often found in BS, can therefore lead to a reduction of the amplitude of the dome of the action potential in the epicardium of the right ventricular outflow tract (RVOT) or sometimes to its complete suppression. The results of this genetic mutation are a transmural voltage gradient and epicardial

dispersion of repolarization in the RVOT, which causes typical ST-segment changes in right precordial leads and creates a predisposition for phase 2 re-entry tachyarrhythmias.³⁻⁵

Indeed, the depolarization hypothesis states that there is a conduction delay in the RVOT relative to the right ventricle. The delayed activation of the RVOT creates an electrical gradient in this part of the heart similar to that associated with myocardial ischaemia; the typical ST-segment changes seen in BS in the right precordial leads are the electrocardiographic consequences of this delay. The frequent association, in patients with BS, of PQ/QRS prolongation and sinus node dysfunction is indirect clinical proof of the depolarization hypothesis.³⁻⁵

ST-segment deviation post-cardioversion is widely occurring and sparingly studied.¹ Coronary artery spasm is allegedly the culprit behind this phenomenon.^{1,2} However, the fact that it takes at least 45 s after coronary artery spasm for ST-segment changes to occur does not explain the immediate onset of ECG changes post-DC shock.^{1,2} Another proposed mechanism is acute myocyte necrosis.¹ However, in patients undergoing DC-shock cardioversion for atrial fibrillation or flutter, cardiac troponin levels were not elevated in serum

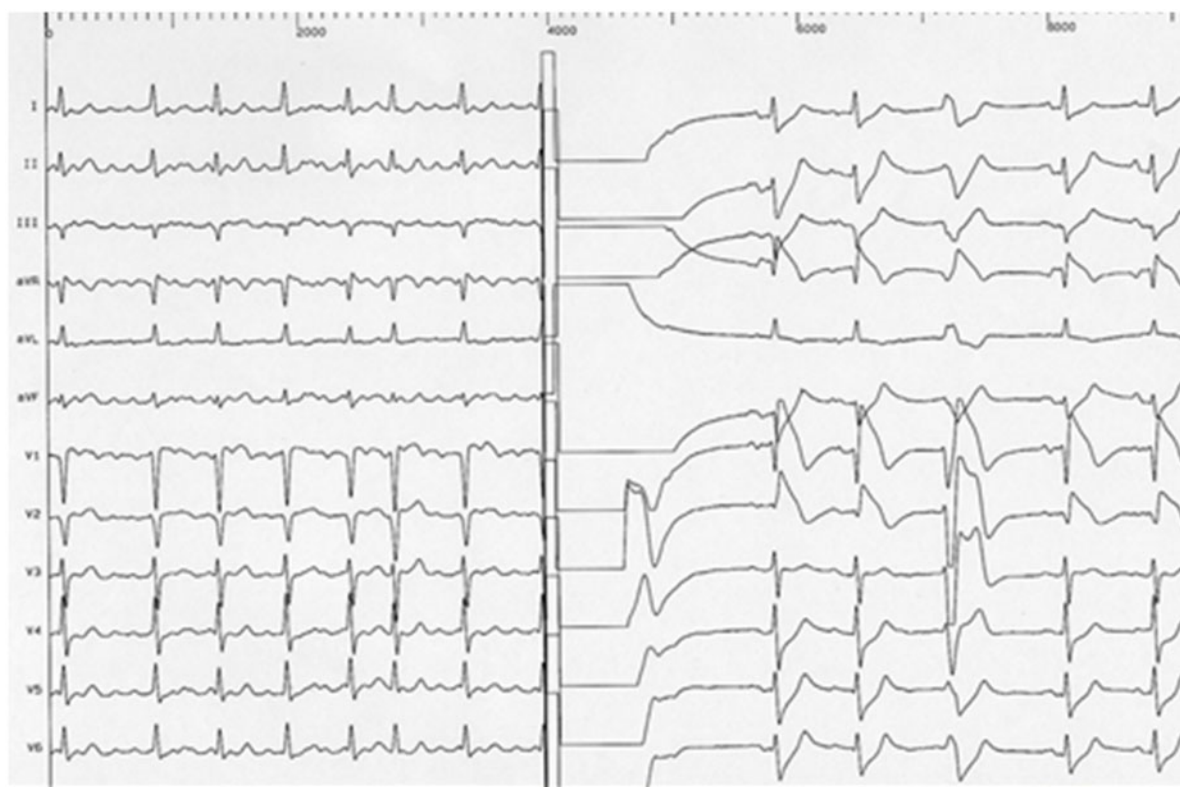


Figure 5 Twelve-lead electrocardiograms before and after synchronized biphasic electrical cardioversion done with paddles positioned in antero-lateral fashion at 200J in a patient with persistent atrial fibrillation, not known to have Brugada syndrome or structural heart disease. Electrocardiogram has been retrieved from a collection of old electrocardiograms from Prof. Pedro Brugada's laboratory. See text for discussion.

post-cardioversion.^{1,2} This indicates that any elevation in serum creatine kinase originated from the skeletal muscle and not from the cardiac muscle.^{1,2} A possible explanation for the observed post-cardioversion ST-segment changes could be derived from the hypotheses proposed to explain the underlying electrophysiology of BS. We believe that the electrical current used to achieve cardioversion produces transient non-transmural depolarization in the region closest to the origin of the shock.^{1,2} This is due to the production of transient microlesions in the sarcolemma, which short circuit the membrane and allow for substantial ionic exchange across the membrane; a process known as electroporation.^{1,6} The potential difference between the depolarized and non-depolarized regions creates the ST-segment deviation observed post-cardioversion.² If this is indeed the mechanism by which ST-segment deviation occurs post-DC shock, it strongly supports the repolarization hypothesis of BS. In our patients, the location of the pads in the anteroposterior position would make the RVOT the region closest to the origin of the shock, where electroporation would be most prevalent, thereby mimicking the affected area in BS and producing similar ECG findings. *Figure 4* shows a diagram which summarizes this hypothesis.

Our finding is confirmed by a case report published in 2017 by Divanji *et al.* in *Journal of Thoracic Disease*⁷: in their case vignette

the authors showed the acute changes in the 12-lead ECGs that have occurred right after electrical cardioversion in a patient with persistent atrial fibrillation. There are many similarities with our case, such as the pads were positioned in an anteroposterior fashion, the patient's list of medications included amiodarone and cardioversion was performed with the delivery of a 200J biphasic shock. The ST elevation occurred in leads V1 and V2 and closely resembled Type 1 Brugada pattern (despite lacking negativization of T waves in the same leads). The patient did not experience any significant complication. The authors reached the same conclusions about the mechanism of this electrocardiographic phenomenon; considering its short duration, they excluded myocardial ischaemia and necrosis^{8,9} and they hypothesized 'non-transmural depolarization of myocardial cell layers leading to a voltage gradient within the ventricular wall, resulting in ST elevation on the surface ECG'.

The ST elevation reported in our case series is possibly higher than that reported in previous studies.¹⁰ In general, monophasic shock is usually associated with higher required energy and subsequently with more significant ST elevation than biphasic shock. The reason behind this possible difference is unknown to us but, we presume, could be linked to the position of the pads.

At present, we do not know if these findings are dependent on the position of the pads on the chest. On the one hand, we can speculate that the anterolateral electrode position would not give rise to the same ST-segment elevation on the ECG. Indeed, the telemetry originating from the pads in an anteroposterior fashion reproduces the same QRS aspect which is visible in V1, one of the leads where Brugada Type 1 is seen, whereas the anterolateral approach usually resembles lead I, which is a lead that rarely shows Brugada Type 1 ST-segment changes. A confirmation of this hypothesis is given in [Figure 5](#). It shows a case of synchronized biphasic electrical cardioversion done with paddles positioned in anterolateral fashion at 200J in a patient with persistent atrial fibrillation, not known to have BS or structural heart disease; here, V1 and V2 clearly show the appearance of a Brugada Type 1 pattern right after the DC shock, whereas lead I shows an ST depression which is a mirror image of the ST elevation in the right precordial leads. On the other hand, this phenomenon could be the proof that the described transient ST elevation could be not dependent on the position of the pads or paddles at the time of cardioversion. A possibility to explain it is that the pressure applied with the paddle on the anterior wall is enough to induce this electrical phenomenon on the right ventricle, regardless of the position of the other paddle, but for sure, further data need to be collected on the topic. Furthermore, we wonder if the ST elevation in V1 and V2 is obtained in the anterolateral fashion when pads instead of paddles are utilized.

A limitation of our case series is that we did not have 12-lead ECGs at the time of the cardioversion, therefore, the appearance of Brugada Type 1 pattern could not be attested completely. Nevertheless, the fact that the pads are positioned in an anteroposterior fashion resembling lead V1 or V2 on the 12-lead ECGs makes this assumption fairly reasonable.

However, we cannot speculate on the possibility that similar electrocardiographic changes can occur with an asynchronous defibrillation or with a different amount of energy given for cardioversion. We also cannot demonstrate that these findings were caused by the concomitant use of amiodarone or anaesthetics. Given the fact that ajmaline test was not performed in any of the patients included in this case series, we cannot claim for sure that these patients did not have an electrophysiological substrate which could have favoured this finding.

Our case series report for the first time in the literature an electrocardiographic change resembling Brugada Type 1 which occurs immediately after synchronized electrical cardioversion. Since this ECG change resembles Brugada Type 1 pattern, which is known to be associated with ventricular arrhythmias, further prospective studies are warranted on the possible arrhythmogenicity of this modality of cardioversion, especially in patients predisposed to ventricular arrhythmias.

Lead author biography



Antonio Sorgente, MD, PhD, is currently working as cardiac electrophysiologist at Cleveland Clinic Abu Dhabi, a facility located in Abu Dhabi (UAE). He is also currently Clinical Assistant Professor of Medicine at Case Western Reserve University, Cleveland, OH, USA. He has the luxury to work with two masters in Electrophysiology, such as Pedro Brugada and Mark Josephson. He first authored 20

papers out of a total of 70. He wrote also a few of chapters for EP related books.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

References

1. Rumeau P, Fourcade J, Duparc A, Hébrard A, Mondoly P, Rollin A, Massabau P, Detis N, Elbaz M, Carrié D, Galinier M, Delay M, Maury P. ST-segment changes after direct current external cardioversion for atrial fibrillation. Incidence, characteristics and predictive factors. *Int J Cardiol* 2011;**148**:341–346.
2. Shan P, Lin J, Xu W, Huang W. ST-segment elevation after direct current shock mimicking acute myocardial infarction: a case report and review of the literature. *Am J Emerg Med* 2014;**32**:1438.e1–1438.e3.
3. Sieira J, Dendramis G, Brugada P. Pathogenesis and management of Brugada syndrome. *Nat Rev Cardiol* 2016;**13**:744–756.
4. Mizusawa Y, Wilde AA. Brugada syndrome. *Circ Arrhythm Electrophysiol* 2012;**5**:606–616.
5. Meregalli PG, Wilde AA, Tan HL. Pathophysiological mechanisms of Brugada syndrome: depolarization disorder, repolarization disorder, or more? *Cardiovasc Res* 2005;**67**:367–378.
6. Jones JL, Jones RE, Balasky G. Microlesion formation in myocardial cells by high-intensity electric field stimulation. *Am J Physiol* 1987;**253**:H480–H486.
7. Divanji P, Badhwar N, Goldschlager N. Post-cardioversion ST-segment elevation: a case-based review of the pathophysiology. *J Thorac Dis* 2017;**9**:5503–5506.
8. Di Diego JM, Antzelevitch C. Acute myocardial ischemia: cellular mechanisms underlying ST segment elevation. *J Electrocardiol* 2014;**47**:486–490.
9. Klabunde R. Cardiac electrophysiology: normal and ischemic ionic currents and the ECG. *Adv Physiol Educ* 2017;**41**:29–37.
10. Ambler JJ, Deakin CD. A randomized controlled trial of efficacy and ST change following use of the Welch-Allyn MRL PIC biphasic waveform versus damped sine monophasic waveform for external DC cardioversion. *Resuscitation* 2006;**71**:146–151.