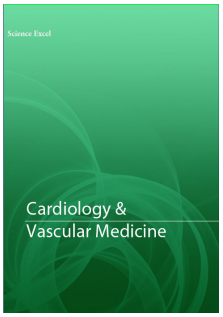
Cardiology and Vascular Medicine

Case Report



Brugada Syndrome in Pulmonary Embolism-Extremely Rare Presentation with Gradual Disappearance despite Later Conversions (Yasser's Conversions) and Yasser's Coving Sign Post-COVID-19 Pneumonia-New Discoveries

Yasser Mohammed Hassanain Elsayed

Critical Care Unit, Kafr El-Bateekh Central Hospital, Egyptian Ministry of Health (MOH), Egypt

Correspondence

Yasser Mohammed Hassanain Elsayed

Critical Care Unit, Kafr El-Bateekh Central Hospital, Egyptian Ministry of Health (MOH), Egypt

- Received Date: 18 Aug 2025
- Accepted Date: 22 Sep 2025
- Publication Date: 24 Oct 2025

Keywords

Brugada syndrome, pneumonia, pulmonary embolism, Yasser's conversion, Yasser's coving radiological sign

Abbreviations

BrP: Brugada phenocopy; BrS: Brugada syndrome; COVID-19: SARS-CoV-2; LAD: Left axis deviation; O2: Oxygen: PE: Pulmonary embolism; RV: Right ventricle; SCD: sudden cardiac death; STE: ST-segment elevation; TWI: T-wave inversion; VF: Ventricular fibrillation; VR: Ventricular rate

Abstract

Introduction: *Brugada syndrome (BrS) is an uncommon genetic disorder accountable for 4-12% SCDs due to ventricular fibrillation. Brugada phenocopy (BrP) is a category of clinically diverse entities causing identical Brugada-like electrocardiographic patterns to actual Brugada syndrome (BrS). Till now, there has been no ECG decision even with application of the new criteria in differentiation between BrS and BrP. Acute pulmonary embolism (PE) is a known serious cardiovascular and respiratory disease. A scarce reports supporting Brugada syndrome post-acute respiratory SARS-CoV-2 (COVID-19) infection have been recorded.* ***Case presentation:*** *A 43-year-old, golden-furniture painter, married male Egyptian heavy smoker patient was admitted to the hospital with pleural chest pain, fever, tachypnea, and tachycardia. He was managed in the ICU for pulmonary embolism, Brugada syndrome, and pneumonia.* ***Conclusion:*** *Acute pulmonary embolism is a new, rare presentation and sequel of Brugada syndrome. Conversions (Yasser's Conversions) from Brugada syndrome type 1 to mixed type with right reversal post-COVID-19 pneumonia and acute pulmonary embolism is an innovative finding. The Coving Radiological Sign (Yasser's Coving Sign) is also a new radiological sign that is parallel to the electrocardiographic coving configuration of Brugada syndrome. Fever is a predisposing factor and an ominous sign for Brugada syndrome. Brugada syndrome and acute pulmonary embolism are a new description of post-COVID-19 pneumonia.*

Introduction

Brugada syndrome is usually seen either spontaneously or after provocative testing with Na+ channel antagonists in questionable cases of BrS. The standard 12-lead ECG can reveal a diagnostic coving pattern (type 1) or suggestive saddle-back pattern (type 2) for BrS. The detectable result of a type 1 ECG pattern is strong evidence for positivity. Lethal arrhythmias may occur with fever. A coving type of ST-segment elevation (STE) ≥2 mm with a T-wave inversion (TWI) ≥1 of V1-V2 leads, spontaneously or provocatively with Na+ channel antagonists, is essential for the identification of BrS. Anyway, genetic testing for the SCN5A gene will be advisable. Undoubtedly, fever-induced coved type 1 of BrS in ECG carries a higher rate of sudden cardiac death (SCD). Treatment options include ICDs, and medications can be used in patients with manifestations. Approximately, Quinidine showed an 80% rate of efficacy in the suppression of vermicular fibrillation (VF). Significantly, education for the patients to avoid precipitating factors of BrS should be applied [1]. Brugada phenocopy (BrP) is a category of clinically diverse entities causing identical Brugada-like ECG patterns of true BrS. It is commonly elicited by some metabolic disorders, mechanical compression, right ventricle (RV) or RVOT pathology, APE, acute myocardial ischemia, myocarditis, pericarditis, ECG modulating factors (such as bradycardia, vagal tone, and fever), LAD artery occlusion, recurrent hypokalemia, RBBB, LVH, pectus excavatum, or ARVC, and exercise [1-8]. Inducible coving type 1 BrS pattern is diverse and variable [2]. A recent study reported that the ECG differentiation between the patterns of BrP and true congenital BrS is very difficult [2-3]. However, lower sensitivity, specificity, and positive and negative predictive values were elicited on using the new ECG criteria in differentiation between BrS and BrP in ECG [3]. So, misdiagnosis challenges, morbidity, and mortality impact may occur on using diagnostic criteria for stratification BrP and BrS. Zhang N, et al [6], reported a case of

Citation: Elsayed YMH. Brugada Syndrome in Pulmonary Embolism-Extremely Rare Presentation with Gradual Disappearance despite Later Conversions (Yasser's Conversions) and Yasser's Coving Sign Post-COVID-19 Pneumonia-New Discoveries. Cardiol Vasc Med. 2025;5(1):1-7.

BrP in APE with coving type 1 BrP on the ECG. There are 9 reported cases of BPh after APE that have been recorded. On the contrary, Brugada syndrome is known as an inherited channelopathy. It has either coving type 1 or saddle-back type 2 STE ≥2mm in the V1-3 leads. But BrS carries a serious risk for VF. The APE commonly has poor outcomes and may predispose to SCD [8]. Brugada phenocopy may be a sequel of APE, giving a typical ECG picture of Brugada syndrome. The BrP pattern usually fades after the recovery of the embolism and subsequent negative testing with Na+ channel antagonists [7, 9, 10]. Pulmonary embolism may rarely mimic the ECG pattern of BrS [11]. The BrS ECG pattern is commonly detected in V1–V3 leads as: (A) Coving type-1 BrP is accompanied by coved STE ≥0.2mV rather than inverted T wave. (B) Type-2 saddle-back BrP is accompanied by saddle-back STE and upright T-wave [8]. ECG of the BrP may appear and disappear due to several factors, but it is not related to BrS. ECG of BrP can be dynamic and may be concealed in V1–V3 leads, which is the hallmark of BrS [12]. Unfortunately, there are a little reported cases concerning BrS in SARS-CoV-2 (COVID-19) infection [13-17].

In this article, the author reported a case of Brugada syndrome with acute pulmonary embolism post-COVID-19 pneumonia in a middle-aged male patient with a new electrocardiographic (Yasser's Conversions) and Coving radiological sign (Yasser's Coving Sign) discoveries.

Case presentation

History and clinical examinations

A 43-year-old, furniture painter, Egyptian, married male poliomyelitis heavy smoker patient complains of an increase in breath, chest pain, fever, and palpitations. Chest pain was the pleural and left-sided. He had a history of whole body pains, cough, fatigue, loss of appetite, and smell for 6 days. The recent contact with a COVID-19 patient occurred. The patient had no relevant positive history. On clinical examination, the patient appeared uncomfortable and distressed with a tachycardia (130 bpm), hypotension (90/70 mmHg), tachypnea (26 bpm), fever (40 °C), and hypoxia (O2 sat of 89%. Tachycardia and left pleural rub were heard on chest auscultation.

Investigations and Management

The patient was admitted to the critical care department for suspected acute pulmonary embolism, Brugada syndrome, and pneumonia. Initially, he was treated with oxygen inhalation, cold compresses, and then paracetamol IVI. Heparin sulfate IVB amp (5000 mg), then enoxaparin amp (80 mg, SC, BID), and oral warfarin (daily 5 mg, monitoring with International Normalized Ratio and prothrombin time) were added. Meropenem vial (1000 mg IV, TID) was given. The patient was hourly monitored for vital signs and periodic ECGs. The first ECG tracing was taken on arrival in the ICU, showing sinus tachycardia with coving-like STE in V1-3 leads, TWI in V1-6 leads, and evidence of physiological LAD (Figure 1A). The initial plain chest X-ray film was done on ICU presentation showing left triangular ground-glass pulmonary consolidation with lateral base and apical extension to the mediastinum (Figure 1B). A contrasted chest CT on ICU presentation showing left upper lobe triangular ground-glass pulmonary consolidation with lateral base and apical extension to the mediastinum (Figure 1C-1E). The echocardiography on the presentation showed a trivial TR with a normal ejection fraction (62%) (Figure 2A). The second ECG tracing on the second day of the ICU presentation showed NSR, incomplete RBBB, and very slight mimic coving-like STE in V1 and V2 leads and evidence of physiological LAD (Figure 2B). A chest CT pulmonary angiography (CTPA) on the second day of ICU presentation showed smooth intravascular defects at sub-segmental pulmonary branches with a left upper lobe patchy area of Consolidation. There is a trabeculated and spacious area of the above-ground-glass pulmonary consolidation (Figures 2C and 2D). The initial CBC was; Hb 12.6 g/dl, RBCs; 4.68*103/mm3, WBCs; 22.9*103/mm3 (Neutrophils; 91.4 %, Lymphocytes: 6.8%, Monocytes; 1.1%, Eosinophils; 0.7% and Basophils 0%), and Platelets; 150*103/mm3. D-dimer was 1.14 mg/dl. The troponin test was negative. CRP was 48g/dl. SGPT was 14 U/L and SGOT was 19U/L. Serum albumin was 3.6gm/dl. Total serum bilirubin was 0.8 mg/dl. Serum creatinine was 1.0mg/dl. RBS was 99 mg/dl. Plasma sodium was 121mmol/L. Serum potassium was 3.0mmol/L. Ionized calcium was 0.74 mmol/L. ABG was PH: 7.451, PaCO2: 28.4, HCO3-: 19.8, SaO2: 59, PaO2: 28, BE ecf: - 0.4, showing partially compensated respiratory alkalosis. Screening tests for both HBsAg and HCVAb were negative. ESR: first hour 58 mm/h and first hour over 100 mm/h. The CBC after 5 days was; Hb 13.4 g/dl, RBCs; 5.45*103/mm3, WBCs; 10.4*103/mm3 (Neutrophils; 73.2 %, Lymphocytes: 22.4%, Monocytes; 2.6%, Eosinophils; 1.8% and Basophils 0%), and Platelets; 277*103/mm3. D-dimer after 5 days was 0.50 mg/dl. The third ECG tracing on the fifth day of ICU presentation showed NSR, very slight mimic coving-like STE in only V1 lead, Camel-hump T wave in V4, and wavy triple sign (Yasser's sign) in V5 lead (Figure 3A). The plain chest-XR film, posterior-anterior view, was repeated on the seventh day of hospital presentation and showed normalization of the above triangular ground-glass pulmonary consolidation (Figure 3B). The fourth ECG tracing after 7 days of presentation showed NSR with no abnormalities (Figure 3C). A non-contrasted chest CT was repeated after 7 days of discharge and showed very slight left fine, thready, dull opacities (Figures 3D and 3E). The fifth ECG tracing was done within 36 days of ICU presentation, showing NSR, slight coving-like STE with TWI in only V1 lead and saddle-like STE in only V2 lead (Figure 3F). The sixth ECG tracing with right leads application was done within 36 days of ICU presentation, showing NSR, saddle-like STE in only the V1 lead, and slight coving-like STE with TWI in only the V2 lead (Figure 3G). Brugada syndrome with acute pulmonary embolism post-COVID-19 pneumonia in a middle-aged male patient with a new electrocardiographic (Yasser's Conversions) and Coving radiological sign (Yasser's Coving sign) discoveries was the most probable diagnosis. Within 12 hours of management, nearly dramatic clinical and ECG improvement had happened. Within a week post-stabilization, he was discharged and continued on warfarin tablets (5 mg, OD with advised cardiac and chest follow-up.

Discussion

Overview

A middle-aged, golden-furniture painter Egyptian heavy smoker patient was admitted and managed in the critical care unit for suspected acute pulmonary embolism, Brugada syndrome, and COVID-19 pneumonia.

A middle-aged, golden-furniture painter Egyptian heavy smoker patient was admitted and managed in the critical care unit for suspected acute pulmonary embolism, Brugada syndrome, and COVID-19 pneumonia.

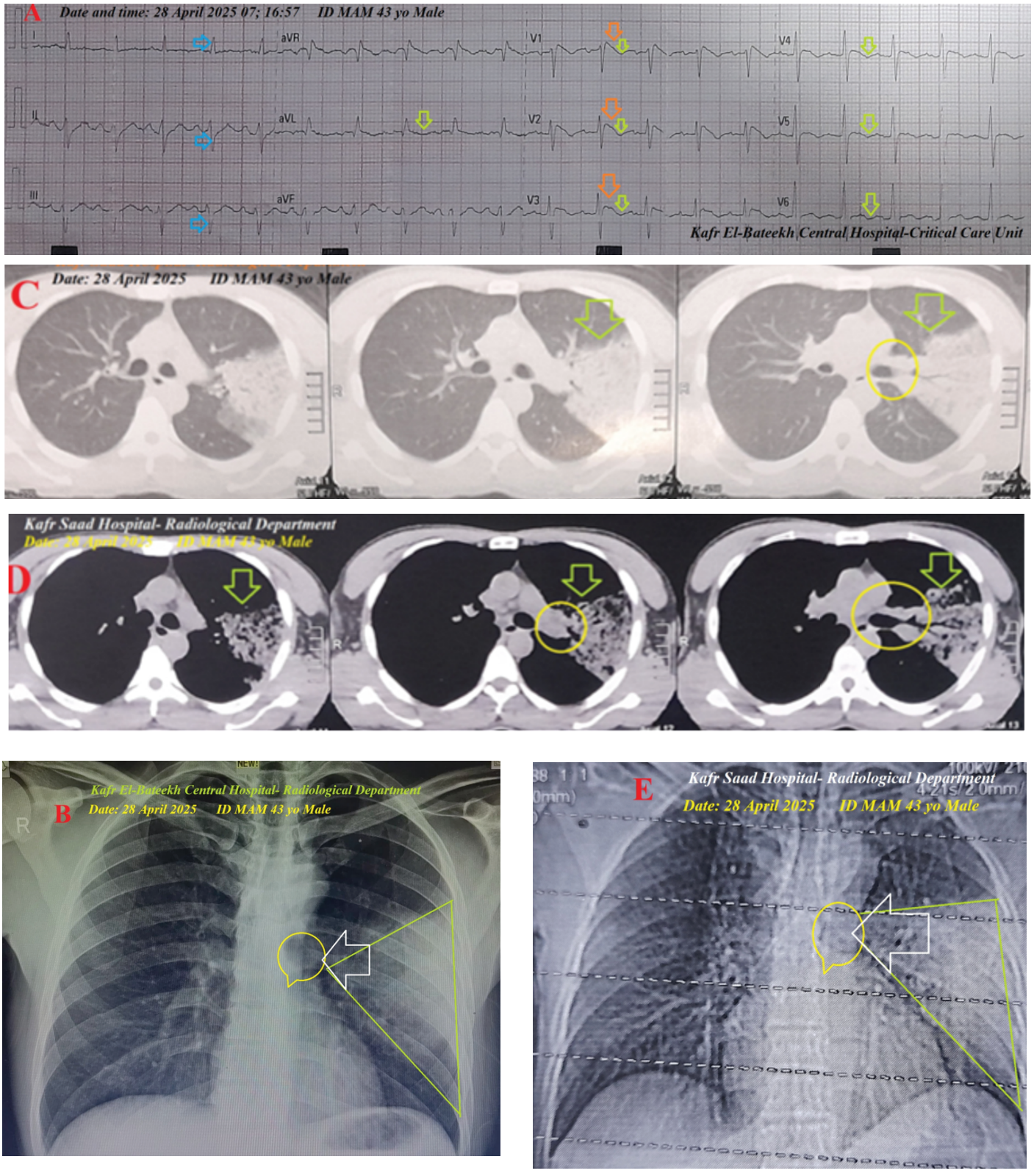


Figure 1: *A. ECG tracing was done on the initial presentation in the ICU, showing sinus tachycardia (of VR: 122) with coving-like ST-segment elevation in V1-3 leads (golden arrows), T-wave inversion in aVL and V1-6 leads (lime arrows), and evidence of pathological left axis deviation (LAD: light blue arrows). B. Plain chest-XR film, posterior-anterior view, was done on ICU admission, showing left triangular ground-glass pulmonary consolidation (lime triangle) with lateral base and apical extension to the mediastinum (white arrow and yellow circle). C. Soft and D. Hard sections of a chest CT without contrast were done on ICU admission, showing left upper lobe triangular ground-glass pulmonary consolidation with lateral base and apical extension to the mediastinum (lime arrow and yellow circle). E. Plain chest-XR film, of a chest CT without contrast was done on ICU admission, showing left triangular ground-glass pulmonary consolidation (lime triangle) with lateral base and apical extension to the mediastinum (white arrow and yellow circle).*

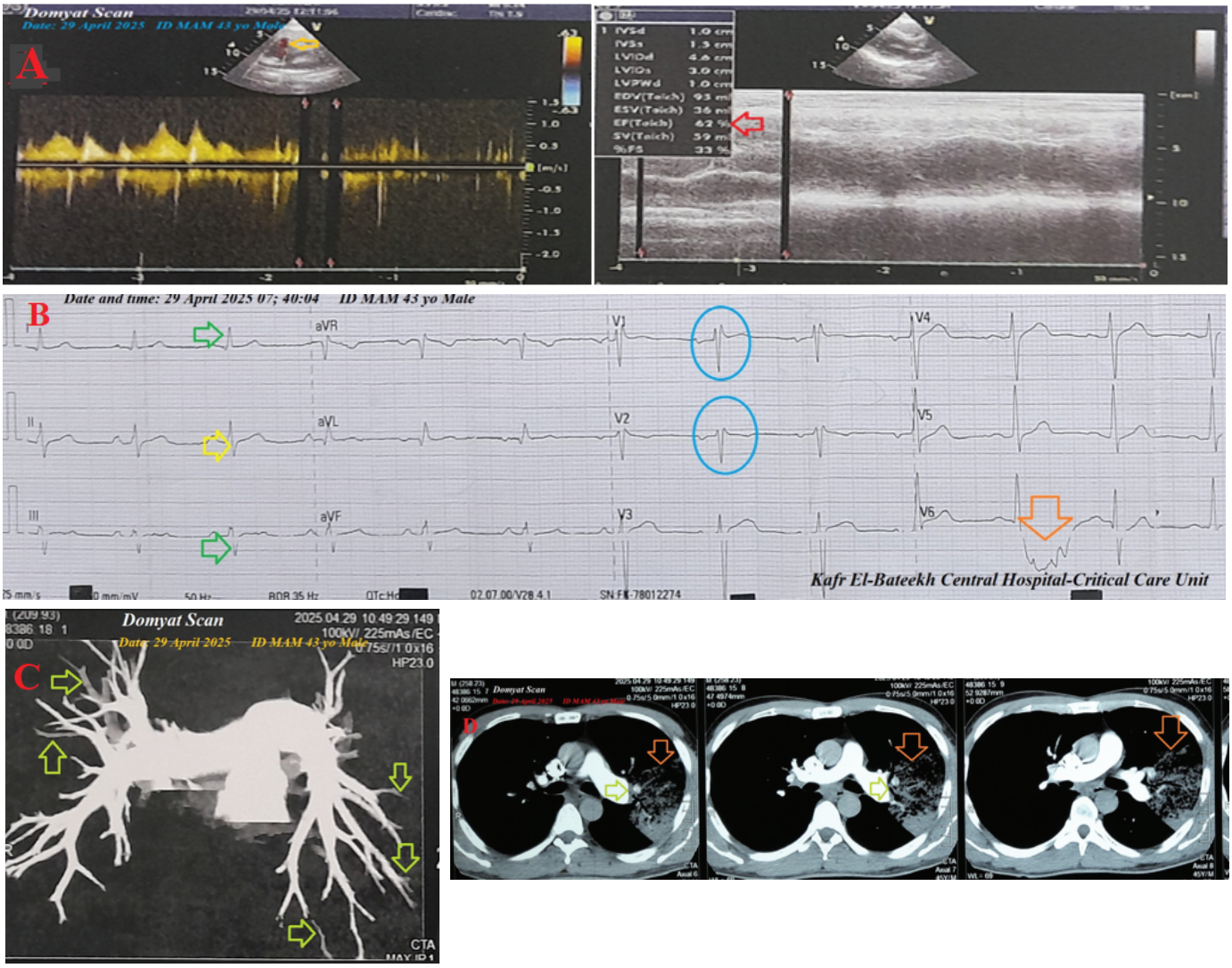
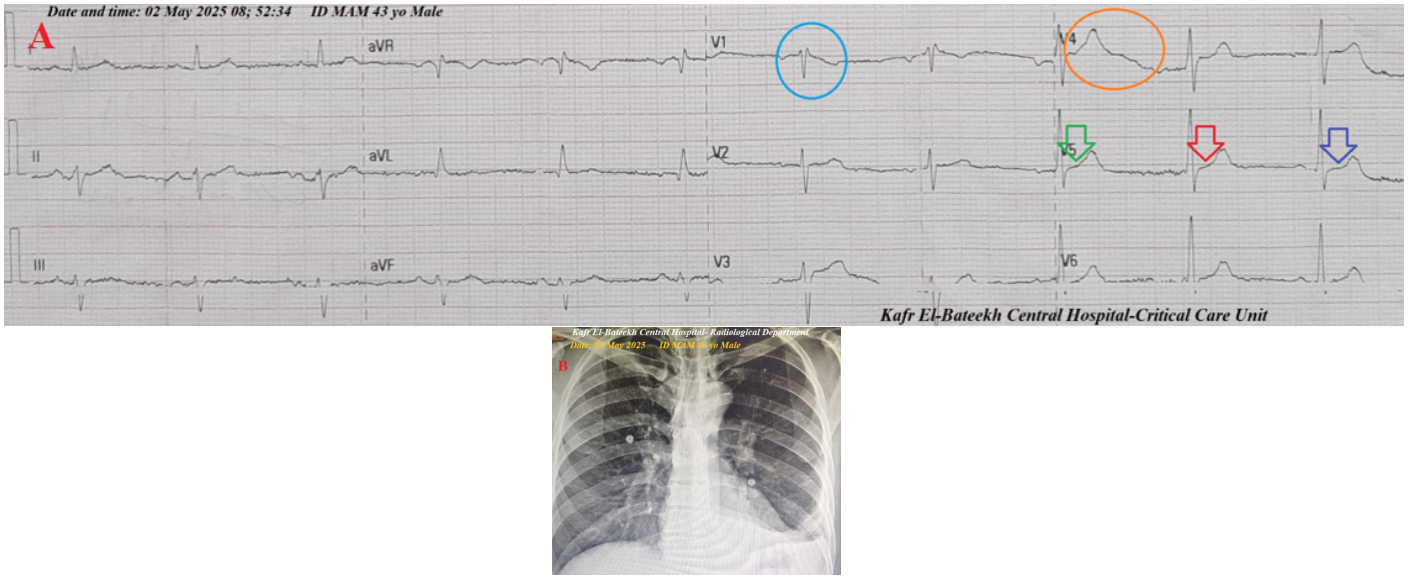


Figure 2 A. *Echocardiography was done on the second day of ICU admission showing trivial tricuspid regurgitation (TR: golden arrows) with normal ejection fraction (EF) of 62% (red arrow). B. ECG tracing was done on the initial presentation in the ICU, showing NSR (of VR: 72) with very slight mimic coving-like ST-segment elevation in V1 and V2 leads (light blue arrows), movable artifact due to loose V4 lead (orange arrow), and evidence of physiological LAD (green and yellow arrows). C. and D, Sections of CTPA were done on the second day of ICU admission showing smooth intravascular defects at sub-segmental pulmonary branches (lime arrows) and left pulmonary artery (yellow arrows). There are trabeculated and spacy of the above-ground-glass pulmonary consolidation (golden arrows).*



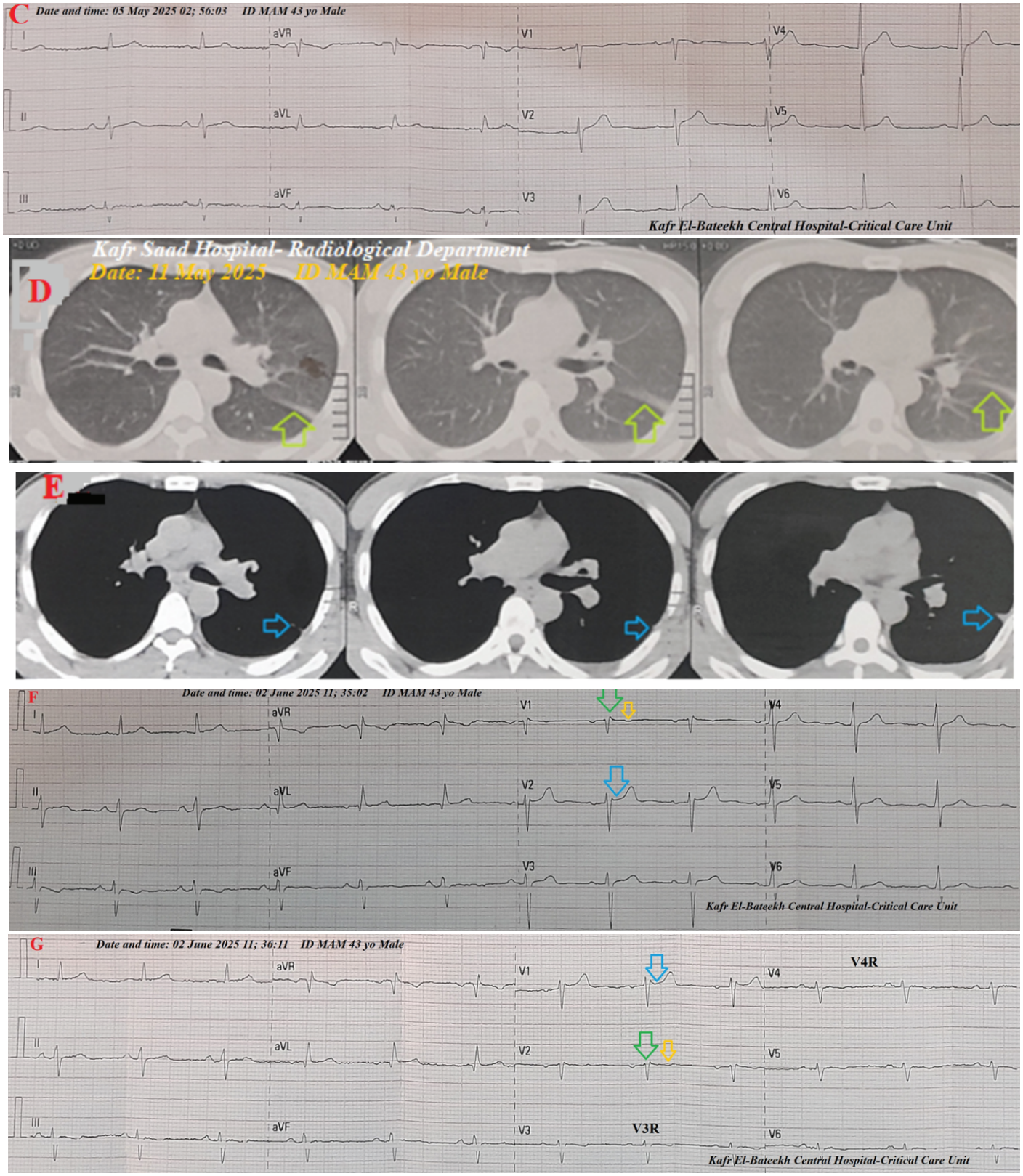


Figure 3: A. ECG tracing was done on the fifth day of ICU admission showing NSR (of VR: 66) with very slight mimic coving-like ST-segment elevation in only V1 lead (light blue arrow), Camel-hump T wave in V4 (orange arrow), and wavy triple sign (Yasser's sign) in V5 lead (green, red, and dark blue arrows). B. Plain chest-XR film, posterior-anterior view, was done on the seventh day of ICU admission showing normalization of the above triangular ground-glass pulmonary consolidation. C. ECG tracing was done within 7 days of the ICU admission showing NSR (of VR: 62) with no abnormalities. D. Soft and E. Hard sections of a chest CT without contrast were done within 7 days after discharge showing Very slight left fine, thready, dull opacities (lime and light blue arrows). F. ECG tracing was done within 36 days of ICU presentation showing NSR (of VR: 72), slight coving-like ST-segment elevation with T wave inversion in only V1 lead (arrow green and golden arrows), and saddle-like ST-segment elevation in only V2 lead (light blue). G. ECG tracing with right leads application was done within 36 days of ICU presentation showing NSR (of VR: 69), saddle-like ST-segment elevation in only V1 lead (light blue arrow), and slight coving-like ST-segment elevation with T wave inversion in only V2 lead (green and golden arrows).

The primary objective for the current case was the existence of a middle-aged, golden-furniture painter married male Egyptian heavy smoker patient who presented with cute pulmonary embolism, Brugada syndrome, and COVID-19 pneumonia in the critical care unit.

The secondary objective for this patient was how you would manage this case in the critical care unit.

A history of contact with a COVID-19 patient and bilateral ground-glass consolidation with laboratory and clinical COVID-19 presentation will intensify the suspicion of COVID-19 infection.

The presence of coving-like STE in V1-3 leads with TWI in V1-6 leads suggests the diagnosis of Brugada syndrome (Figure 1A).

Gradual disappearance of the typical type 1 Brugada syndrome had happened. It started as typical coving-like STE in V1-3 leads (Figure 1A), then slight coving-like STE in V1 and V2 leads (Figure 2B), then slight coving-like STE in V1 (Figure 3A), and finally completely disappear (Figure 3C) (Figure 4).

An ECG evidence of pathological left axis deviation (LAD) in acute pulmonary embolism is unusual (Figure 1A). There is usually right axis deviation (RAD). So, the presence of LAD may be due to pulling attraction for the triangular consolidation indicating the diagnosis of axial change.

There are escalating changes in the axis deviation of the ECG between pathological and physiological LAD. During the case management (Figure 1A, 2B, 3A, 3F, and 3G).

The apical extension to the mediastinum ground-glass pulmonary consolidation (Figure 1B-3E) may have a role in these changes. This apical extension of the ground-glass pulmonary consolidation to the mediastinum with pulling to the peripheral of the left lung causing cupping or pulling or invagination in the mediastinum may be named as Coving radiological sign (Yasser's coving sign) (Figure 4).

The author thinks that the Coving radiological sign (Yasser's Coving Sign) is parallel to the electrocardiographic Coving configuration of Brugada syndrome.

However, the trabeculated and spacy configuration of the above-ground-glass pulmonary in the CTPA (Figure 2D) in the second day may be due to a response after starting the anticoagulants.

There are conversions (Yasser's Conversions) from Brugada syndrome type 1 to mixed type post-COVID-19 pneumonia with acute pulmonary embolism supporting the diagnosis of a genetic Brugada syndrome, not a Brugada phonocopy or pattern (Figure 3F).

But the presence of the above conversion (Yasser's Conversion) of Brugada syndrome also happened on the reversal of the right leads an interesting (Figure 3G).

There is no known pathogenesis for these conversions. The author suggested that the gradual disappearance of the typical type 1 Brugada syndrome, then the reappearance of the mixed type Brugada syndrome may interpreted with a theory of genetic instability. Unfortunately, there is no supportive evidence.

A new electrocardiographic (Yasser's Conversions) and Coving radiological sign (Yasser's Coving Sign) discoveries were cartoon diagraming in a graphical presentation (Figure 4).

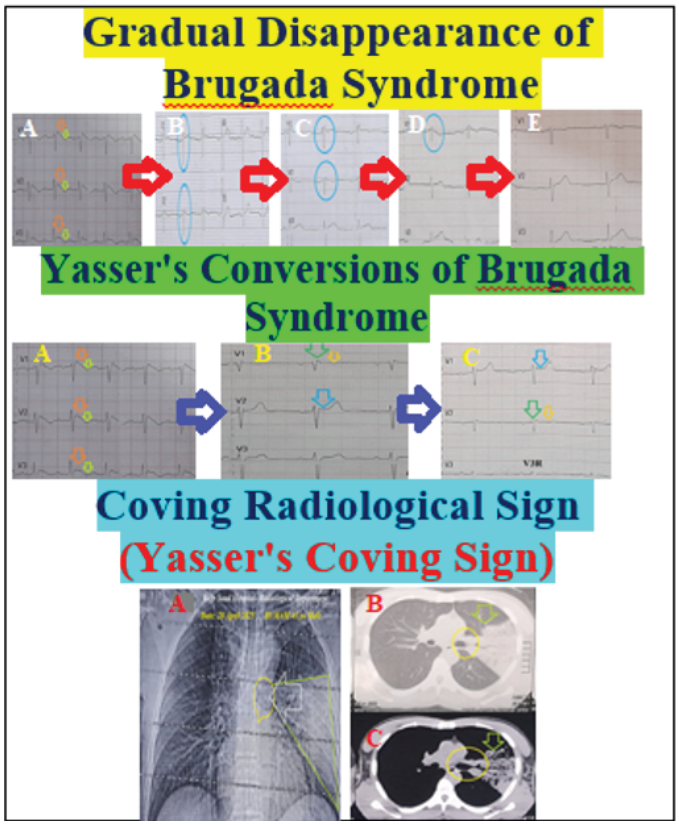


Figure 4- *A graphical presentation of (Yasser's Conversions) and Coving Radiological Sign (Yasser's Coving Sign).*

There is a sporadic 'Camel hump' T-wave noted in the V4 lead ((Figure 3A). It is known as an innovative sign described by Amal Mattu to appoint to T-waves that have a double peak. However, there are two causes implicated in a camel hump T-waves. Prominent U waves fused to the end of the T-wave [18] are interpreted with the associated potassium depletion as was in the current case.

Right bundle branch block with myocardial infarction was the most probable differential diagnosis for the current ECG case study. The ECG criteria with a negative troponin reliably exclude it.

I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison.

The limitation of the current study was the unavailability of genetic phenotyping and testing with sodium channel blockers.

Conclusion and Recommendations

Acute pulmonary embolism is a new, rare presentation and sequel of Brugada syndrome.

Conversions (Yasser's Conversions) from Brugada syndrome type 1 to mixed type with right reversal post-COVID-19 pneumonia and acute pulmonary embolism is an innovative finding.

The Coving Radiological Sign (Yasser's Coving Sign) is also a new radiological sign that is parallel to the electrocardiographic coving configuration of Brugada syndrome.

Fever is a predisposing factor and an ominous sign for Brugada syndrome.

Brugada syndrome and acute pulmonary embolism are new descriptions of post-COVID-19 pneumonia.

Informed consent

Written informed consent was taken from the patient.

Artificial intelligence (AI)

Artificial intelligence (AI) was not used in the manuscript construction.

Conflicts of interest

There are no conflicts of interest.

Acknowledgment

My thanks to the team nurses of the ICU in Kafr El-Bateekh Central Hospital for their support. Also, thanks to my wife for saving time and improving the conditions for me.

References

1. 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(9):1046-1059. doi:10.1016/j.jacc.2018.06.037.
2. Dendramis G. Brugada syndrome and Brugada phenocopy: the importance of a differential diagnosis. Int J Cardiol. 2016;210:25-27. doi:10.1016/j.ijcard.2016.02.097.
3. Gottschalk BH, Garcia-Niebla J, Anselm DD, Jaidka A, de Luna AB, Baranchuk A. New methodologies for measuring Brugada ECG patterns cannot differentiate the ECG pattern of Brugada syndrome from Brugada phenocopy. J Electrocardiol. 2016;49(2):187-191. doi:10.1016/j.jelectrocard.2015.12.011.
4. Enriquez A, Brugada J, Baranchuk A. Exercise-induced Brugada phenocopy. J Cardiovasc Electrophysiol. 2016;27(3):360-361. doi:10.1111/jce.12838.
5. Gottschalk BH, Anselm DD, Brugada J, et al. Expert cardiologists cannot distinguish between Brugada phenocopy and Brugada syndrome electrocardiogram patterns. Europace. 2016;18(7):1095-1100. doi:10.1093/europace/euv278.
6. Zhang N, Liu T, Tse G, et al. Brugada phenocopy in a patient with acute pulmonary embolism presenting with recurrent syncope. Oxf Med Case Reports. 2017;2017(5):omx014. doi:10.1093/omcr/omx014.
7. Anselm DD, Evans JM, Baranchuk A. Brugada phenocopy: a new electrocardiogram phenomenon. World J Cardiol. 2014;6(3):81-86. doi:10.4330/wjc.v6.i3.81.
8. Elikowski W, Łazowski S, Fertała N, et al. Brugada phenocopy in pulmonary embolism: clinicopathological case study and literature review. Pol Merkur Lekarski. 2022;50(300):378-383.
9. Zhan ZQ, Wang CQ, Nikus KC, Pérez-Riera AR, Baranchuk A. Brugada phenocopy in acute pulmonary embolism. Int J Cardiol. 2014;177(3):e153-e155. doi:10.1016/j.ijcard.2014.09.046.
10. Anselm DD, Baranchuk A. Brugada phenocopy in the context of pulmonary embolism. Int J Cardiol. 2013;168(1):560. doi:10.1016/j.ijcard.2013.01.169.
11. Aksu U, Kalkan K, Gulcu O, Topcu S, Tanboga IH. Massive pulmonary embolism mimicking electrocardiographic pattern of Brugada syndrome. Am J Emerg Med. 2016;34(5):933.e1-933.e2. doi:10.1016/j.ajem.2015.09.029.
12. Bayés de Luna A, Brugada J, Baranchuk A, et al. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. J Electrocardiol. 2012;45(5):433-442. doi:10.1016/j.jelectrocard.2012.06.004. PMID: 22920782. Erratum in: J Electrocardiol. 2013;46(1):76.
13. Lemes CIS, Armaganijan LV, Maria AS, et al. Brugada pattern unmasked during COVID-19 infection: a case report. J Cardiol Cases. 2022;25(6):377-380. doi:10.1016/j.jccase.2022.01.003.
14. Shao F, Xu S, Ma X, et al. In-hospital cardiac arrest outcomes among patients with COVID-19 pneumonia in Wuhan, China. Resuscitation. 2020;151:18-23. doi:10.1016/j.resuscitation.2020.04.005.
15. van de Poll SWE, van der Werf C. Two patients with COVID-19 and a fever-induced Brugada-like electrocardiographic pattern. Neth Heart J. 2020;28(7-8):431-436. doi:10.1007/s12471-020-01459-1.
16. Lugenbiel P, Roth L, Seiz M, et al. The arrhythmogenic face of COVID-19: Brugada ECG pattern during acute infection. Eur Heart J Case Rep. 2020;4(FI1):1-2. doi:10.1093/ehjcr/ytaa230.
17. Pasqualetto MC, Corrado A, Secco E, Graceffa F, Rigo F. Transient type 1 Brugada pattern without ongoing fever during COVID-19 pneumonia. Eur J Case Rep Intern Med. 2020;7(7):001808. doi:10.12890/2020_001808.
18. Elsayed YMH. Hypocalcemia-induced camel-hump T-wave, Tee-Pee sign, and bradycardia in a car-painter of a complexed dilemma: a case report. Cardiac. 2020;2(1):1-5. doi:10.35702/card.10007