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Case Report

Yasser's Migration Sign with Tail Apex Syndrome in Brugada Syndrome, Variable Interlacing Arrhythmias, and Recurrent Axis Deviation-Cardiovascular Discoveries

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Abstract

Introduction: Brugada syndrome (BrS) is a rare genetic or inherited disease in which there is abnormal cardiac channelopathy. It is one of the most common inherited primary arrhythmia syndromes. It often presents with arrhythmic syncope or sudden cardiac death. The cardiac axis represents the sum of all depolarization vectors of the heart. The vector analysis determines the direction of the net flow of current through the heart. The cardiac axis deviations have a large differential diagnosis.

Case presentation: A middle-aged married female housewife, patient, was presented to the ICU with junctional tachycardia, diabetes, non-specific chest pain, Brugada syndrome, and past bronchial asthma. Migratory Brugada syndrome, variable interlacing arrhythmias, ECG axis deviations, and mild chest and urinary tract infections in a diabetic patient are the most probable diagnosis. Electrocardiography, oxygenation, chest CT, brain CT, ABG, and echocardiography are the interventions. There is a dramatic clinical, and electrocardiographic improvement had happened.

Conclusion: "Yasser's migration sign" and "Tail Apex Syndrome" are new cardiovascular discoveries. Variable and changeable topical changes in coving-like ST-segment elevations of Brugada syndrome in the ECG throughout the course of the ICU admission are fantastic, a new description. Genetic migrations of Brugada syndrome may be a suggested theory. Amiodarone is suggested as a cause for these different serial changes in axis deviations in the ECG. Variable documented arrhythmias such as junctional tachycardia, sinusoidal AF, and sinus tachycardia are newly reported in Brugada syndrome.

Keywords: Yasser's migration sign; Tail apex syndrome; Brugada syndrome; sinusoidal fibrillation; junctional tachycardia; axis deviation; and cardiovascular discoveries

Abbreviations

ASD: Atrial septal defect

BrS: Brugada syndrome

ECG: Electrocardiography

ICU: Intensive care unit

JT: Junctional tachycardia

LAD: Left axis deviation

MI: Myocardial infarction

O_2: Oxygen

RAD: Right axis deviation

RBBB: Right bundle branch block

SCD: Sudden cardiac death

VF: Ventricular fibrillation

WPW: Wolff-Parkinson-White

1. Introduction

Brugada syndrome (BrS) is a rare genetic or inherited disease in which there is abnormal cardiac channelopathy [1]. The BrS was initially described as a new ECG condition in 1992 [2] by Pedro and Josep Brugada [3]. It is one of

the most common inherited primary arrhythmia syndromes. It often presents with arrhythmic syncope or sudden cardiac death (SCD) due to polymorphic ventricular tachycardia (PVT) and ventricular fibrillation (VF). Vagotonia or fever is usually a precipitating factor in apparently healthy adults [1]. Ventricular fibrillation and SCD occur in structurally normal hearts. To date, 18 genes have been associated with the disease, with the voltage-gated sodium channel α type V gene (SCN5A) being the most common one to date. However, only 30-35% of diagnosed cases are attributable to pathogenic variants in known genes, emphasizing the need for further genetic studies [4]. The most commonly involved gene is SCN5A, which encodes the cardiac sodium channel [2]. The prevalence of the syndrome (0.01%-0.3%) varies among regions and ethnicities, being the highest in Southeast Asia. BrS is diagnosed by the "coved type" ST-segment elevation ≥2mm followed by a negative T-wave in ≥1 of the right precordial leads V1-V2. The typical electrocardiogram in BrS is often concealed by fluctuations between normal, non-diagnostic, and diagnostic ST-segment patterns in the same patient, thus hindering the diagnosis. Presently, the majority of BrS patients is incidentally diagnosed, and may remain asymptomatic for their lifetime. However, BrS is responsible for 4-12% of all SCDs and for ~20% of SCDs in patients with structurally normal hearts [1]. Sudden cardiac death is linked to the hereditary condition known as Brugada syndrome (BrS), an autosomal dominant heart illness [5]. Identification of an underlying genetic culprit continues to be elusive in the majority of patients, while discord regarding the condition's underlying pathophysiology also persists, with strong lines of evidence present for both the "depolarization" and "repolarization" hypotheses [6]. Arrhythmic risk is the highest in SCD survivors and in patients with spontaneous BrS electrocardiogram and arrhythmic syncope, but risk stratification for SCD in asymptomatic subjects has not yet been fully defined [1]. The most effective approach to unmasking this diagnostic pattern is the use of ajmaline and flecainide tests, and the most effective intervention to reducing the risk of death is the implantation of a cardioverter defibrillator [4]. Recent achievements have expanded our understanding of the genetics and electrophysiological mechanisms underlying BrS, while radiofrequency catheter ablation may be an effective new approach to treat ventricular tachyarrhythmias in BrS patients with arrhythmic storms [1]. The abnormal heart rhythms seen in those with Brugada syndrome often occur at rest [3].It should also be noted that a type 1 Brugada ECG may also be provoked by a variety of clinical insults and conditions, including myocardial ischemia, metabolic abnormalities, and pectus excavatum [6]. The condition is characterized by 'coved' ST-segment elevations in the anterior precordial electrocardiogram leads [3]. Pharmacologic therapy is designed to produce an inward shift in the balance of currents active during the early phases of the right ventricular action potential (AP) and can be used to abort electrical storms or as an adjunct or alternative to device therapy when use of an implantable cardioverter defibrillator is not possible. Isoproterenol, cilostazol, and milrinone boost calcium channel current and drugs like quinidine, bepridil, and the Chinese herb extract Wenxin Keli inhibit the transient outward current, acting to diminish the AP notch and thus to suppress the substrate and trigger for ventricular tachycardia or fibrillation. Radiofrequency ablation of the right ventricular outflow tract epicardium of patients with BrS has recently been shown to reduce arrhythmia vulnerability and the electrocardiographic manifestation of the disease, presumably by destroying the cells with more prominent AP notch [7]. The criteria for diagnosing BrS have evolved since the condition's initial description, and debate continues regarding the need for additional identifiable clinical features beyond the distinctive electrocardiographic pattern, particularly in cases in which a type 1 pattern is only observed during provocative drug challenge. Criteria for concluding a type 1 ECG pattern require J-point elevation ≥ 2 mm in one or more lead among V1 or V2 positioned in the second, third, or fourth intercostal space, in association with a coved ST-segment morphology, whereas the type 2 pattern requires ≥ 2 mm of J-point elevation in similar lead positions in association with a saddleback ST-segment morphology [8]. The most recent Heart Rhythm Society/European Heart Rhythm Association/Asia Pacific Heart Rhythm Society (HRS/EHRA/APHRS) expert consensus statement indicates that a type 1 Brugada ECG pattern, either spontaneous, fever, or drug-induced, is sufficient to satisfy a diagnosis of BrS [9]. Determining the electrical axis of the heart is an essential step for the initial ECG interpretation [10]. The cardiac axis represents the sum of all depolarization vectors of the heart. The vector analysis determines the direction of the net flow of current through the heart [11]. There are five types of axes: 1. Normal axis (between -30° and +90°), 2. Left axis deviation (LAD: between -30° and -90°), 3. Right axis deviation (RAD: between -90° and 180°), 4. Extreme axis deviation (between +90° and 180° or beyond +100°). 5. Indeterminate axis: the QRS complex is isoelectric or equiphasic in all leads with no dominant QRS deflection [12]. Physiological normal variations, age-related changes, left ventricular hypertrophy (LVH), left bundle branch block (LBBB), left anterior fascicular block (LAFB), inferior myocardial infarction (MI), Wolff-Parkinson-White (WPW) syndrome, premature ventricular complexes (PVCs), ventricular tachycardia (VT), primum atrial septal defect (ASD), endocardial cushion defect, hyperkalemia, emphysema mechanical shift, such as with expiration or raised diaphragm (eg, pregnancy, ascites, abdominal tumor, organomegaly), and paced rhythm are implicated causes in left axis deviation [13-16]. Physiological normal variations such as children and young adults, limb lead reversal of left and right arm electrodes, right ventricular (RV) overload syndromes (acute or chronic), RV hypertrophy (RVH), left posterior fascicular block (LPFB), right bundle branch block (RBBB), lateral MI, WPW syndrome, PVCs, VT, secundum ASD, dextrocardia, left pneumothorax, mechanical shift, such as with inspiration or emphysema, RV strain (eg, pulmonary embolism (PE), pulmonary stenosis (PS), pulmonary hypertension (PHT), chronic pulmonary disease, and cor pulmonale are implicated causes in right axis deviation [13-16]. The physician will be worried if the axis suddenly changed from the last ECG, with chest pain or dyspnea, and ST-segment elevation. New RAD deserves more concern than the left. Sodium channel blockers such as amiodarone can cause RAD of the terminal QRS [17]. The presence of the RBBB, RAD, or LAD may be key for the diagnosis of bifascicular heart block [10].

I report a case of a middle-aged married female housewife, patient, was presented to the ICU with junctional tachycardia, diabetes, non-specific chest pain, migratory Brugada syndrome, and past bronchial asthma with cardiovascular discoveries.

2. Case presentation

A 41-year-old married female housewife, patient, was presented to the intensive care unit (ICU) with palpitations, acute non-specific chest pain, and dizziness. Cough, headaches, and generalized body pain were associated symptoms. The patient has a history of diabetes mellitus 12 years ago, on long-acting insulin. She also has a history of bronchial asthma 20 years ago on intermittent anti-asthmatic medications. Upon general physical examination, the patient had tachypnea and distressed respiration, with a regular pulse rate (junctional tachycardia (JT) with VR of 165), blood pressure (BP) of 110/80 mmHg, respiratory rate of 25 bpm, a temperature of 36°C, and a pulse oximeter of oxygen (O2) saturation of 97%. No more relevant clinical data were noted during the clinical examination. The patient refused the referral for admission to the intensive care unit (ICU). He was initially managed at the ICU, with a junctional tachycardia, diabetes, non-specific chest pain, and Brugada syndrome. Initially, the patient was treated with O2 inhalation via an oxygen system line (100%, using a normal mask, 5L/min). The initial ECG tracing was performed on the initial presentation to the ICU, with one and a half calibration showing junctional tachycardia, normal axis, and coving-like ST-segment elevations in the aVR, V1, and V2

leads. There is a loose lead artifact in the V5 lead (Figure 1A). The second ECG tracing was taken within 3 minutes of the above ECG tracing, with one and a half calibration showing junctional tachycardia, interlacing of premature junctional complexes in V1 and V2 leads, with the same changes as above. But with AC artifacts and Wavy triple sign (Yasser's sign) in V6 lead (Figure 1B). The patient was treated with amiodarone IV bolus (300 mg IV over 20 minutes, then a continuous IVI at a rate of 1 mg/min for 6 hours). The patient was monitored hourly for vital signs and O2 saturation. The third ECG tracing was taken within 22 hours of the above ECG tracing, with one and a half calibration showing sinusoidal AF, normal axis, and disappearance of the above coving-like ST-segment elevations (Figure 1C). Amiodarone IV was given as a maintained dose, Diltiazem tablets (60mg, OD), SC Enoxaparin 40 mg, OD, and Warfarin tablet (5 mg, OD) were added. There is a new mild fever (temp of 38.5°C), tachypnea, dry cough, and frequent micturition. Urine analysis on the second day of ICU admission showed: pus over 50, RBCs: 10-12, and epithelial cells (++). The fourth ECG tracing was taken within 42 hours of the above ECG tracing, showing sinus tachycardia, normal axis, a coving-like ST-segment elevations in the aVR, V1, and V2 leads (Figure 1D). The fifth ECG tracing was taken within 6 hours of the above ECG tracing, showing sinus tachycardia, right axis deviation, with coving-like ST-segment elevations in I and aVL leads. There is a loose lead artifact in V2 lead (Figure 1E). Cefotaxime vials (1 gm IV BID) and paracetamol (500 mg TID as needed) were added. The sixth ECG tracing was taken within 1 minute of the above ECG tracing, showing sinusoidal AF, right axis deviation, with coving-like ST-segment elevations in the I and aVL leads. There is a loose lead artifact in V2 lead (Figure 1F). The seventh ECG tracing was taken within 1 minute of the above ECG tracing, showing sinusoidal AF, right axis deviation, with coving-like ST-segment elevations in I and aVL leads. There is a loose lead artifact in the V2 lead (Figure 1G). The eighth ECG tracing was taken within 24 hours of the above ECG tracing, showing sinusoidal AF, right axis deviation, with coving-like ST-segment elevations in the I and aVL leads. There is sagged ST-segment depression in the V4-6 leads (Figure 1H). The ninth ECG tracing was taken within 7 hours of the above ECG tracing, showing sinusoidal AF with normal axis (Figure 1I). The tenth ECG tracing was taken within 5 days of the above ECG tracing, with one and a half calibrations, showing junctional tachycardia, left axis deviation, with coving-like ST-segment elevations in the III, aVR, and V1 leads. There is a retrograde P wave in V6 lead (Figure 1J). The eleventh ECG tracing was taken within 8 hours of the above ECG tracing, showing sinusoidal AF, right axis deviation, with coving-like ST-segment elevations in I and aVL leads. There is equivocal QRS in the aVR lead (Figure 1K). The chest X-ray film PA view performed on the initial presentation to the ICU shows tail-like outward apical elongation. There are mild right and left consolidation opacities (Figure 2A). The plain film of chest CT performed on the initial presentation to the ICU showed tail-like outward apical elongation with a narrow lower black recess separating the left diaphragmatic surface and lower surface of the right heart (Figure 2B). Serial cuts of chest CT were done on the initial presentation to the ICU, showing no abnormalities (Figure 2C). Serial cuts of brain CT were done on the initial presentation to the ICU, showing no abnormalities (Figure 2D). The echocardiography was done within 4 days after the ICU presentation, showing tachycardia, mild mitral regurgitation, and a good LV systolic function of an EF of 53% (Figure 3). The initial laboratory: complete blood count (CBC); Hb was 11.3 g/dl, RBCs; 5.37*103/mm3, WBCs; 12.0*103/mm3 (Neutrophils; 53.7 %, Lymphocytes: 36.1%, Monocytes; 8.2%, Eosinophils; 0% and Basophils 0%), Platelets; 327*103/mm3. CRP was 6.0. SGPT was (26.7 U/L). Serum albumen was 4.2 gm/dl. Serum creatinine was (0.97 mg/dl). RBS was (254 mg/dl). D-dimer was 0.3ug/ml. ABG was done in the third day; (PH; 7.41, PCO2; 49.6 mmHg, HCO3; 31.8 mmHg, So2; 96%, and PaO2; 78 mmHg). INR was 1.04 with a prothrombin time of 13.08 seconds. The associated electrolytes in the ABG profile: Plasma sodium was 160 mmol/L. Serum potassium was (3.2 mmol/L). Serum ionized calcium was (1.01 mmol/L). The CBC was repeated in the third day; Hb was 11.2 g/dl, RBCs; 5.33*103/mm3, WBCs; 13.7*103/mm3 (Neutrophils; 67.6 %, Lymphocytes: 27.6%, Monocytes; 4.8%, Eosinophils; 0% and Basophils 0%), Platelets; 322*103/mm3. The troponin initial test was negative. On the ninth day; The CBC: Hb was 10.8 g/dl, RBCs; 5.17*103/mm3, WBCs; 13.8*103/mm3 (Neutrophils; 63.7 %, Lymphocytes: 31.4%, Monocytes; 4.9%, Eosinophils; 0% and Basophils 0%), Platelets; 210*103/mm3. CRP was 12.0. SGPT was (31.0 U/L). Serum creatinine was (0.6 mg/dl). Rheumatoid factor was negative. Migratory Brugada syndrome, variable interlacing arrhythmias, ECG axis deviations, and mild chest and urinary tract infections in a diabetic patient are the most probable diagnosis. Within eleven days of the above at-ICU management, the patient finally showed nearly complete clinical and ECG improvement. The patient was continued on Diltiazem tablets (60 mg, OD), Amiodarone tablets (200 mg, OD), Warfarin (5mg, OD), long-acting insulin, oral calcium, and Vitamin-D preparations for 14 days with further recommended cardiac and urological follow-up.

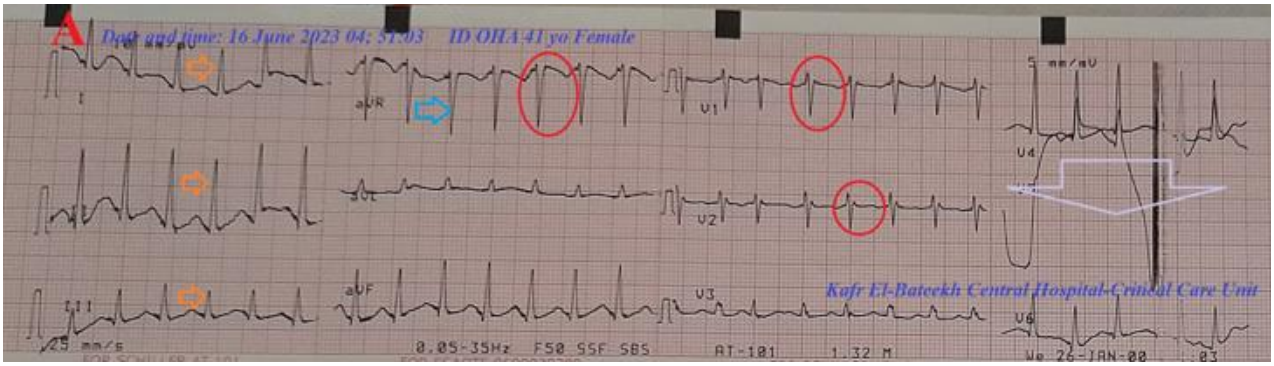

Figure 1: Serial ECG tracings; A. tracing was done on the initial presentation to the ICU with one and a half calibration, showing junctional tachycardia (of VR 165), normal axis (golden arrows), with coving-like ST-segment elevations in the aVR, V1, and V2 leads (red circles). There is a loose lead artifact in V5 lead (large grey arrow).

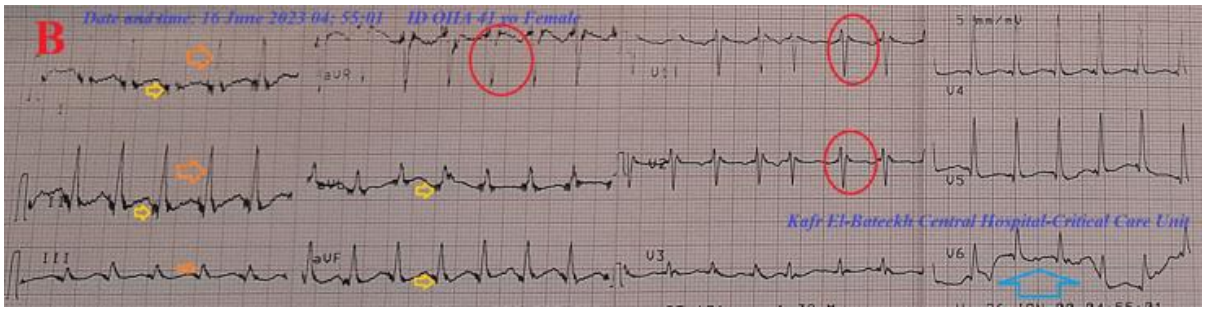

Figure 1B: tracing was taken within 3 minutes of the above ECG tracing with one and a half calibrations, showing junctional tachycardia (of VR 159), interlacing of premature junctional complexes in V1 and V2 leads, with the same changes. But with AC artifacts (orange arrows) and Wavy triple sign (Yasser's sign) in V6 lead (light blue arrow).

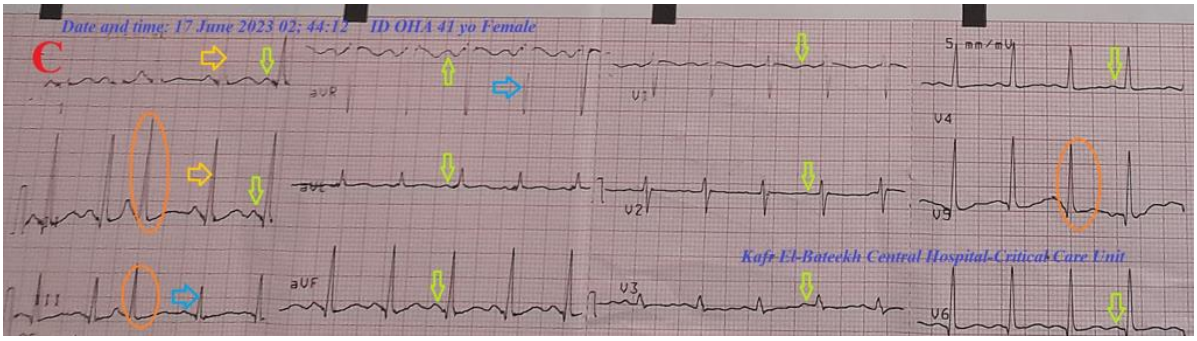

Figure 1C: tracing was taken within 22 hours of the above ECG tracing, with one and a half calibrations, showing sinusoidal AF (of VR 117; lime arrows and golden circles), normal axis (orange and light blue arrows), and disappearance of the above coving-like ST-segment elevations.

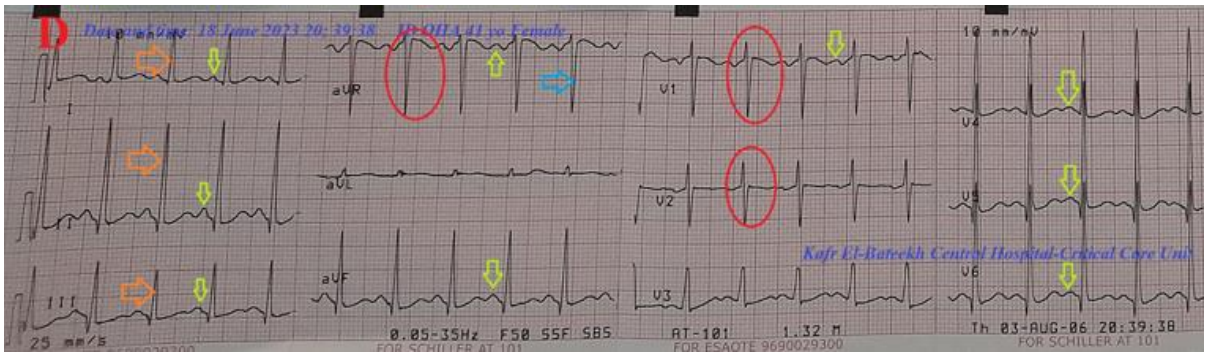

Figure 1D: tracing was taken within 42 hours of the above ECG tracing, showing sinus tachycardia (of VR 123; lime arrows), normal axis (golden arrows and light blue arrows), with coving-like ST-segment elevations in aVR, V1, and V2 leads (red circles).

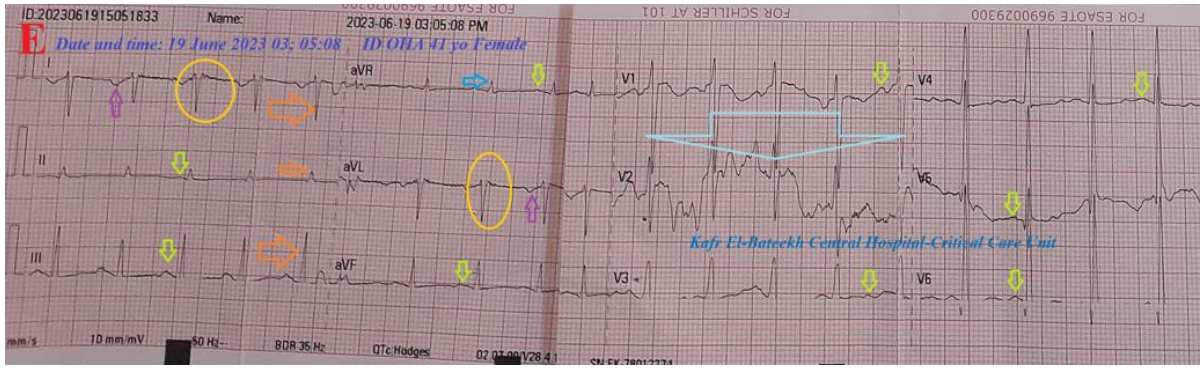

Figure 1E: tracing was taken within 6 hours of the above ECG tracing, showing sinus tachycardia (of VR 114; lime and pink arrows), right axis deviation (golden arrows and light blue arrows), with coving-like ST-segment elevations in I and aVL leads (orange circles). There is a loose lead artifact in V2 lead (large grey arrow).

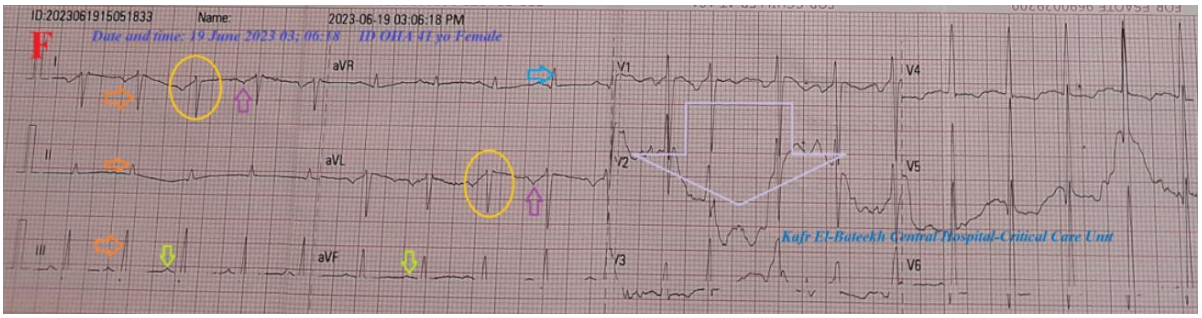

Figure 1F: tracing was taken within 1 minute of the above ECG tracing, showing sinusoidal AF (of VR 119; lime and pink arrows), right axis deviation (golden arrows and light blue arrows), with coving-like ST-segment elevations in I and aVL leads (orange circles). There is a loose lead artifact in V2 lead (large grey arrow).

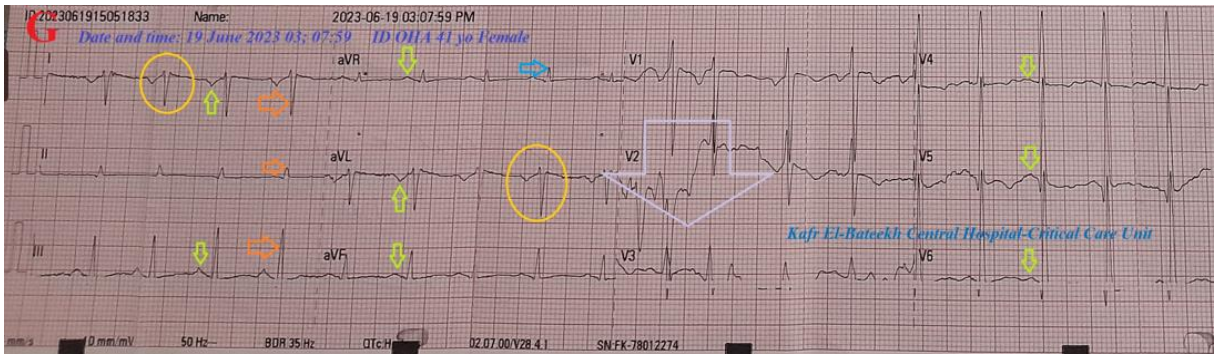

Figure 1G: tracing was taken within 1 minute of the above ECG tracing, showing sinusoidal AF (of VR 111; lime arrows), right axis deviation (golden arrows and light blue arrows), with coving-like ST-segment elevations in I and aVL leads (orange circles). There is a loose lead artifact in V2 lead (large grey arrow).

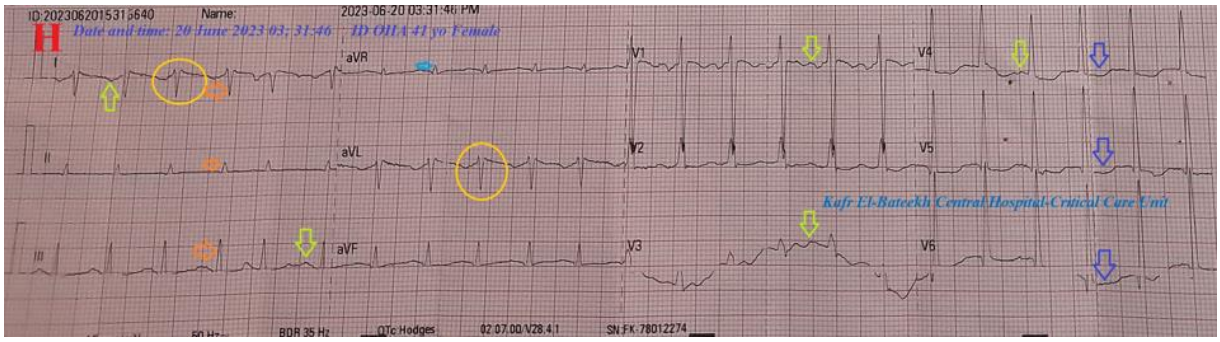

Figure 1H: tracing was taken within 24 hours of the above ECG tracing, showing sinusoidal AF (of VR 137; lime arrows), right axis deviation (golden arrows and light blue arrows), with coving-like ST-segment elevations in I and aVL leads (orange circles). There is sagged ST-segment depression in V4-6 leads (dark blue arrows).

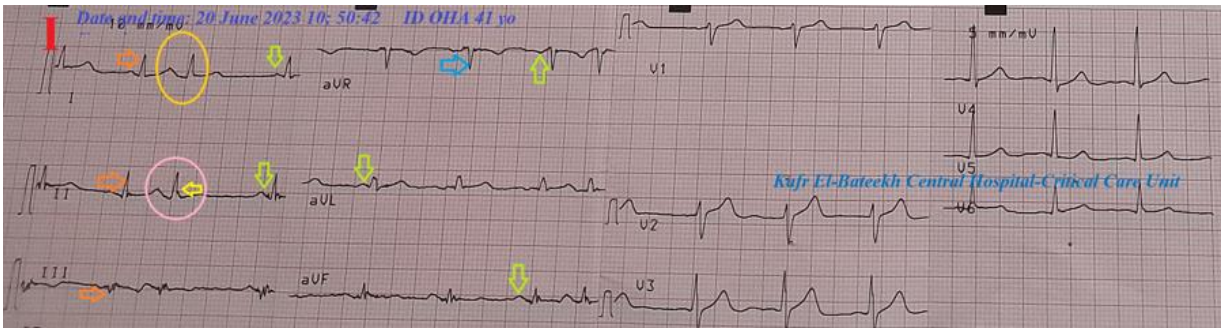

Figure 1I: ECG Tracing was taken within 7 hours of the above ECG tracing, showing sinusoidal AF (of VR 85; lime arrows, golden, and rosy circles) with normal axis (golden arrows and light blue arrows).

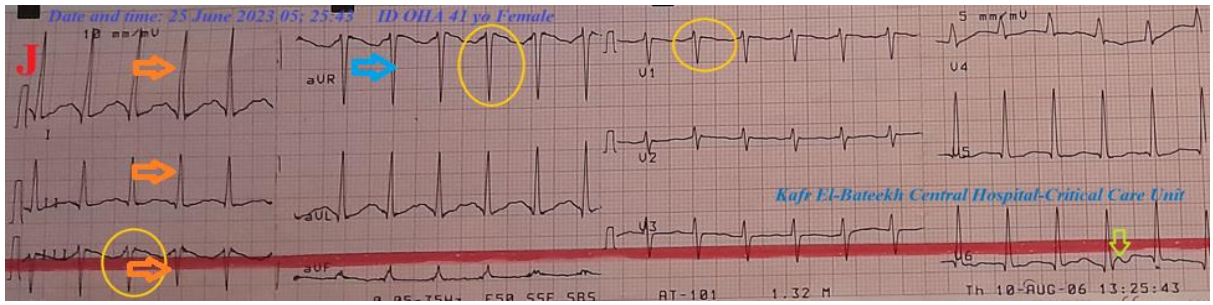

Figure 1J: tracing was taken within 5 days of the above ECG tracing with one and a half calibration, showing junctional tachycardia (of VR 141), left axis deviation (golden arrows and light blue arrows), with coving-like ST-segment elevations in the III, aVR, and V1 leads (orange circles). There is a retrograde P wave in V6 lead (lime arrow).

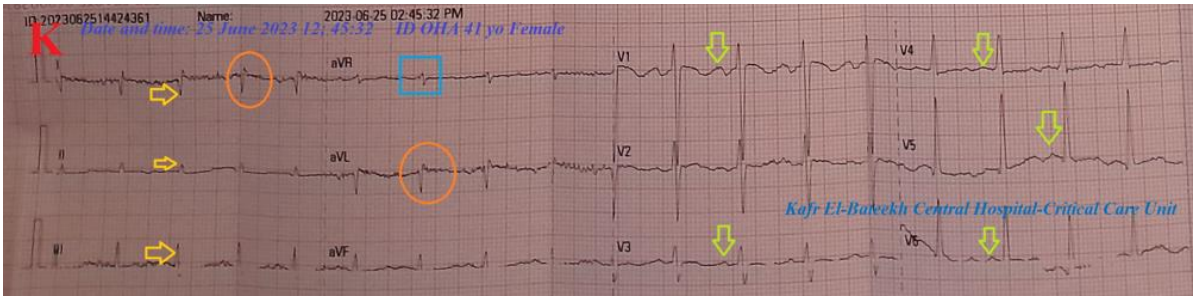

Figure 1K: tracing was taken within 8 hours of the above ECG tracing, showing sinusoidal AF (of VR 108; lime arrows), right axis deviation (orange arrows), with coving-like ST-segment elevations in I and aVL leads (golden circles). There is equivocal QRS in the aVR lead (light Blue Square).

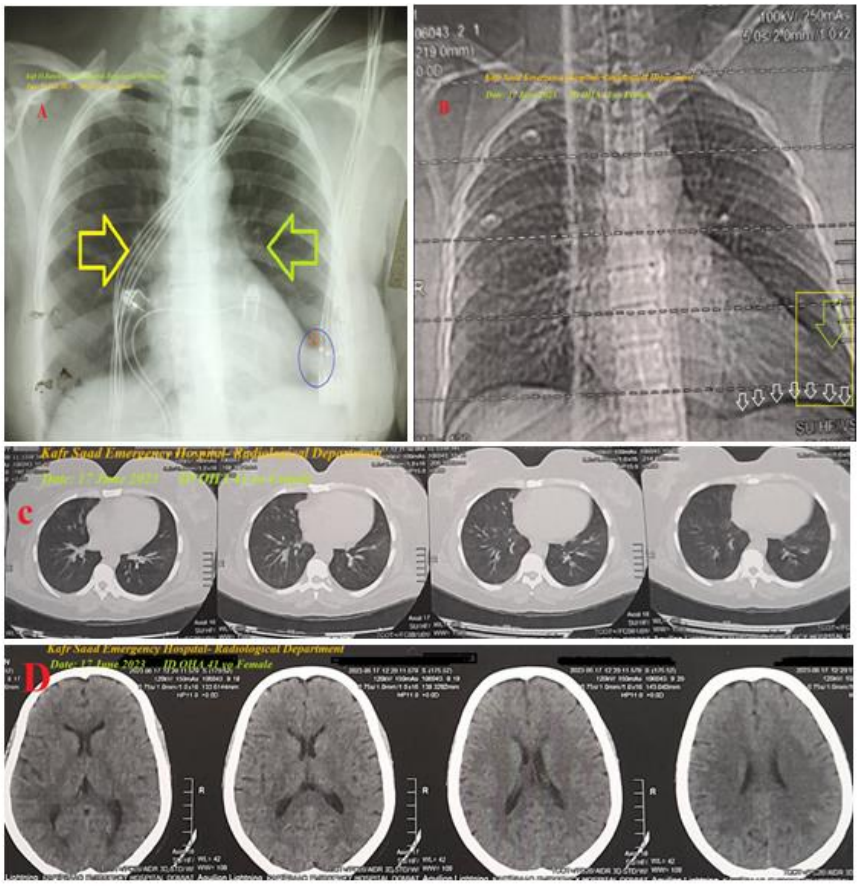

Figure 2A: Plain chest X-ray film, PA view was taken on the initial presentation to the ICU, showing tail-like outward apical elongation (dark blue circle and golden arrow). There are mild right (yellow arrow) and left (lime arrow) consolidation opacities. B. Plain film of chest CT was taken on the initial presentation to the ICU, showing tail-like outward apical elongation (yellow square and lime arrow) with a narrow lower black recess separating the left

diaphragmatic surface and lower surface of the right heart (small white arrow). C. Serial cuts of chest CT, showing no abnormalities. D. Serial cuts of brain CT, showing no abnormalities.

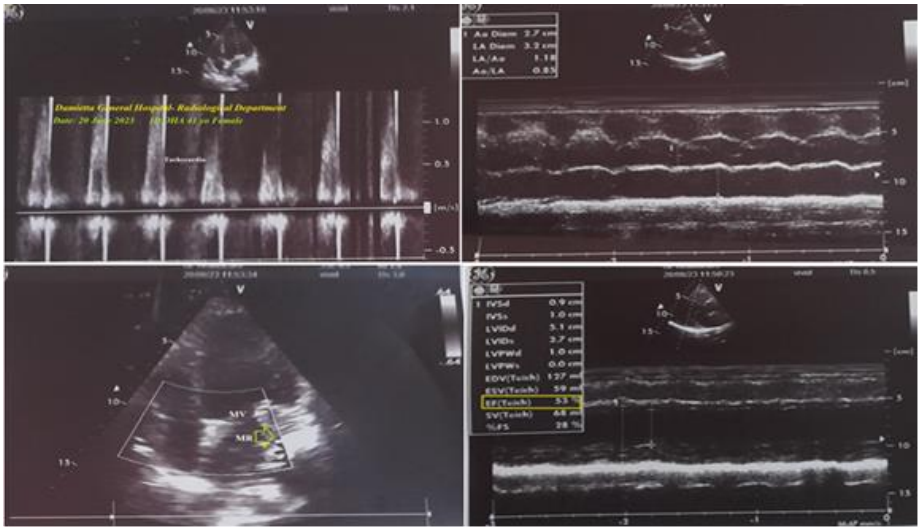

Figure 3: Echocardiography was taken within 4 days after the ICU presentation, showing tachycardia, mild mitral regurgitation (lime arrow), and a good LV systolic function of an EF of 53% (lime-rectangular).

3. Discussion

Overview: A middle-aged married female housewife, patient, was presented to the ICU with junctional tachycardia, diabetes, non-specific chest pain, Brugada syndrome, and past bronchial asthma. The primary objective for my case study was the presence of a middle-aged married female housewife, patient, with junctional tachycardia, diabetes, non-specific chest pain, Brugada syndrome, and past bronchial asthma in the ICU. The secondary objective for my case study was the question of how to manage the case. Mild chest and urinary tract infections were associated with the condition (Figures 2A-2B). There were variable associated arrhythmias throughout the course of the ICU admission. It started with junctional tachycardia (Figures 1A-1B), then sinusoidal AF (Figure 1C), then sinus tachycardia (Figures 1D-1E), then sinusoidal AF (Figures 1F-1I), then junctional tachycardia (Figures 1J-1K). There were also different serial changes in axis deviations in the ECG in the course of the ICU admission. It started with normal axis (Figures 1A-1D), then right axis deviation (Figures 1E-1H), then normal axis (Figure 1I), then left axis deviation (Figure 1J), then right axis deviation (Figure 1K). Indeed, although there is no clear known cause for these different serial changes in axis deviations in the ECG, Amiodarone is suggested as the cause [17]. Naranjo's probability scale was used to assess the probable relationship between these different serial changes in axis deviations and the causative agent, Amiodarone. Naranjo's probability scale in the current case study was +8. It means that there was a probable relationship between the serial changes in axis deviations and the causative Amiodarone (Table 1).

Question	Yes	No	Do Not Know	Score
1. Are there previous conclusive reports on this reaction?	+1	0	0	+1
2. Did the adverse event appear after the suspected drug was administered?	+2	-1	0	+2
3. Did the adverse event improve when the drug was discontinued or a specific antagonist was administered?	+1	0	0	+1
4. Did the adverse event reappear when the drug was re-administered?	+2	-1	0	0
5. Are there alternative causes that could on their own have caused the reaction?	-1	+2	0	+2
6. Did the reaction reappear when a placebo was given?	-1	+1	0	0
7. Was the drug detected in blood or other fluids in concentrations known to be toxic?	+1	0	0	0
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	0
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	+1	0	0	+1
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	+1
	Total Score: +8			

Table 1: Naranjo Algorithm-Adverse Drug Reaction (ADR) Probability Scale in the case report.

There were also variable and changeable topical changes in coving-like ST-segment elevations of Brugada syndrome in the ECG in the course of the ICU admission. It started with coving-like ST-segment elevations in aVR, V1, and V2 leads (Figures 1A-1D), then coving-like ST-segment elevations in I and aVL leads (Figures 1E-1H), then the normal ST-segment in all the above leads (Figure 1I), then coving-like ST-segment elevations in III, aVR, and V1 leads (Figure 1J), then coving-like ST-segment elevations in I and aVL leads (Figure 1K). These transitions in coving-like ST-segment elevations from one specific lead to another specific lead are a new description regarding Brugada syndrome. So, it is named as "Yasser's migration sign" (Figure 4).

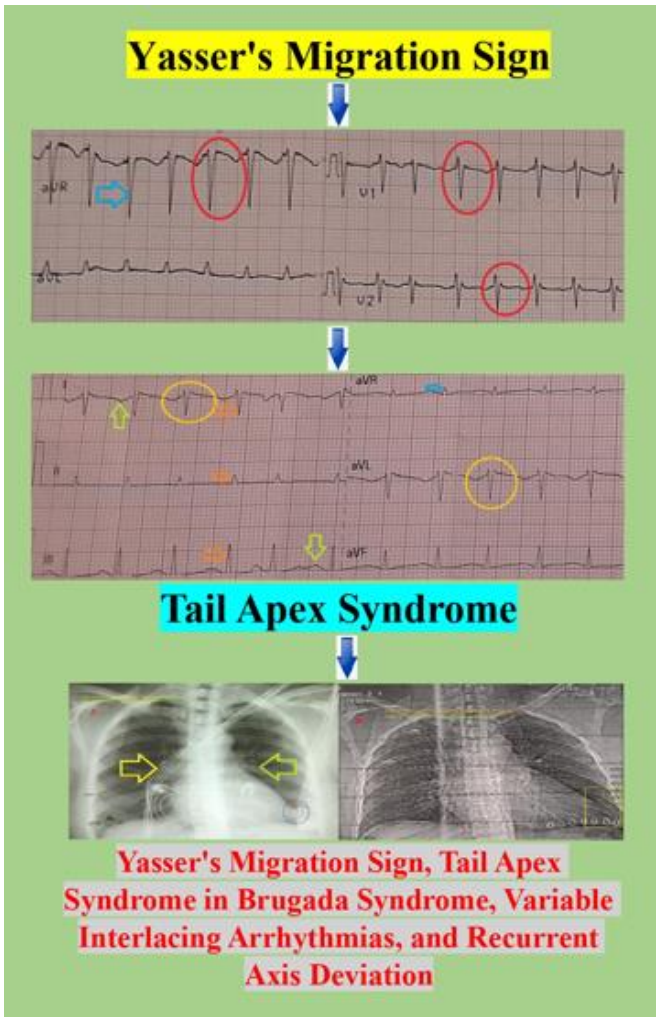

Figure 4: Graphical presentation of Yasser's migration sign and Tail apex syndrome.

There is no known mechanism for these migrations. Genetic migrations of Brugada syndrome may be a suggested theory. Plain chest X-ray film, PA view showing tail-like outward apical elongation with a narrow lower black recess separating the left diaphragmatic surface and lower surface of the right heart. This tail-like outward apical elongation is also newly described. In parallel to these axis deviations and transitions in coving-like ST-segment elevations from one specific lead to another specific ones are new description regarding Brugada syndrome is provided. It may be named as "Tail Apex Syndrome" (Figure 4). Acute pulmonary embolism is the most implicated differential diagnosis. The d-dimer against 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 only limitation of the current study was the unavailability of genetic analysis for Brugada syndrome.

4. Conclusion and Recommendations

"Yasser's migration sign" and "Tail Apex Syndrome" are new cardiovascular discoveries. Variable and changeable topical changes in coving-like ST-segment elevations of Brugada syndrome in the ECG throughout the course of the ICU admission are fantastic, a new description. Genetic migrations of Brugada syndrome may be a suggested theory. Amiodarone is suggested as a cause for these different serial changes in axis deviations in the ECG. Variable documented arrhythmias such as junctional tachycardia, sinusoidal AF, and sinus tachycardia are newly reported in Brugada syndrome.

Conflicts of interest

There are no conflicts of interest.

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