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Case Report

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Yasser's V1-aVR Rhythm Syndrome and Diagonal Biting Compressive Radiological Yasser's Sign, with Differential Diagnoses of ECG Dextrocardia, Old Anterior Infarction, and Suspected Renal Infarction: Cardiovascular and Radiological Discoveries

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Abstract

Introduction: Anterior myocardial infarction has higher mortality and morbidity than other types. Dextrocardia is a cardiac position that is a mirror image of normal anatomy. In patients with dextrocardia, the standard 12-lead ECG is characteristic. Renal infarction is frequently missed or diagnosed late because patients present with flank pain similar to that in other common conditions.

Case Presentation: A 58-year-old Egyptian male worker was admitted to the intensive care unit (ICU) with CHF, hypotension, hematuria, and uncontrolled diabetes.

Conclusion: "Yasser's V1-aVR Rhythm Syndrome" is a newly described syndrome with a constellation of characteristic ST-segment elevation (STE) preceded by a pathological Q wave and ending with slight shallow inversion in V1; a biphasic P wave with a slight terminal portion in lead V1; a dominant small R wave with transient conversion to QS in lead V1 after management; PR prolongation sparing aVR; pathological Q waves in the anterior and high lateral leads except V5 and V6, with some variations; fixed RAD; four new types of QRS fragmentation; transient QTc prolongation; and a few transient PVCs with and without STE. "Diagonal Biting Compressive Radiological Yasser's Sign" is a newly described sign characterized by an irregular diagonal biting shape in the right middle paramediastinal region of the heart. It is associated with an irregular, diagonally extended, dilated area in the opposite left middle paramediastinal region of the heart. The right PA and lower part of the SVC correspond to the first right diagonal, whereas the PA trunk corresponds to the second left diagonal. The diagonals decreased in size with subsequent management. Future research is recommended.

Keywords

Yasser's V1-aVR Rhythm Syndrome, Diagonal Biting Compressive Radiological Yasser's Sign, PR Prolongation, Dextrocardia, Anterior Infarction, Discoveries

Abbreviations

AF: Atrial fibrillation; AMI: Acute myocardial infarction; AWMI: Anterior wall myocardial infarction; AS: Aortic stenosis; BP: Blood pressure; CKD: Chronic kidney disease; CHF: Congestive heart failure; DM: Diabetes; ECG: Electrocardiogram; fQRS: QRS fragmentation; HTN: Hypertension; ICU: Intensive care unit; IHD: Ischemic heart disease; LAD: Left anterior descending artery; LAD: Left axis deviation; LVD: Left ventricular dysfunction; LVEF: Left ventricular ejection fraction; MI: Myocardial infarction; MS: Mitral stenosis; O2: Oxygen; PA: Pulmonary artery; RA: Renal artery; RAD: Right axis deviation; RI: Renal infarction; STE: ST elevation; STEMI: ST-segment elevation myocardial infarction; SVC: Superior vena cava; VR: Ventricular rate

Introduction

Myocardial infarction (MI) remains a leading cause of mortality globally [1]. Significant progress over the last few decades in the diagnosis and management of patients with acute myocardial infarction (AMI) has contributed to reductions in in-hospital mortality. In the 1960s, the primary target was the treatment of lethal arrhythmias. In the 1980s, major breakthroughs included the development of fibrinolytic therapy to reduce infarct size. Cardiogenic shock is considered the most common cause of mortality in hospitalized patients with AMI [2].

Anterior ST-elevation myocardial infarction (STEMI) is associated with larger infarct sizes, a higher rate of left ventricular dysfunction (LVD) with greater reduction in left ventricular ejection fraction (LVEF), a higher incidence of congestive heart failure (CHF), ventricular arrhythmias, higher morbidity, and higher in-hospital and overall mortality compared with inferior infarctions [1,3]. Anterior wall myocardial infarction (AWMI) results from occlusion of the left anterior descending artery (LAD). Based on the anatomical location of the occlusion in the LAD artery, AWMI can be classified into three different categories: A. Proximal LAD: proximal to the first septal perforator. The ECG includes ST elevation (STE) in leads I, aVL, and V1-V6. A new bundle branch block (BBB) or fascicular block may be present. B. Mid-LAD: distal to the first septal perforator but proximal to the first large diagonal branch. The ECG shows STE in leads I, aVL, and V1-V6. There is no associated BBB. C. Distal LAD: distal to the large diagonal branch or involving the large diagonal branch itself. The ECG shows STE in leads V1-V4, I, aVL, V5, or V6 [1].

Renal infarction (RI) is a medical disorder that results from sudden disruption of renal blood flow (RBF) involving either one of the segmental branches or the major ipsilateral renal artery [4]. Renal infarction is rare [5]. In 1940, a study including 14,411 autopsies reported an incidence of renal infarction of 1.4% [6]. In a later case series conducted in an emergency department over 4 years and involving 250,000 patients, only 17 patients (0.007%) were diagnosed with acute RI [7]. Renal artery (RA) lesions are among the most frequent causes of RI [4]. RI is often missed or diagnosed late because its presentation is similar to nephrolithiasis and pyelonephritis. Depending on severity, RI can lead to renovascular hypertension (HTN), chronic kidney disease (CKD), and end-stage kidney disease (ESRD) [5].

Cardiogenic origins are among the important causes of RI. The primary causes of RI include hypercoagulable conditions, RA damage such as arterial dissection, and cardioembolic disease [7]. Acute RI is mainly caused by two mechanisms: in situ renal artery thrombosis, which is less common, and thromboembolism, which typically originates from a thrombus in the heart or aorta [8]. Thromboemboli or in situ thrombi may cause complete occlusion of the main RA or smaller segmental branch arteries [9,10]. Because atheroemboli are small and typically cause incomplete arterial occlusion of more distal vessels, they often lead to secondary ischemic atrophy rather than RI [5].

Renal infarction is a rare cause of HTN and is usually associated with RA lesions. In cases of RI with acute HTN, blood pressure (BP) outcomes may be favorable following intervention and/or medication, and hypertension may resolve spontaneously [10]. Renal infarction can cause abrupt and severe HTN and, less frequently, ESRD [1]. The most common presentations of RI include acute onset of flank pain, fever, nausea, vomiting, and an abrupt rise in BP due to renin-mediated mechanisms. Frank or microscopic hematuria

is noted in almost all cases. Oliguria may also be present [8]. Atrial fibrillation (AF), history of embolism, mitral stenosis (MS), diabetes mellitus (DM), HTN, and ischemic heart disease (IHD) are common risk factors for RI [11]. Renal angiography remains the gold standard, but CT renal angiography, CT angiography, and dimercaptosuccinic acid (DMSA) radioisotope scanning can also be used to establish the diagnosis. Therapeutic options include open surgery, endovascular therapy, including thrombolysis or thrombectomy with or without angioplasty, and anticoagulation [8].

Dextrocardia is a cardiac position that represents a mirror image of normal anatomy. If there is reversal in the position of both the thoracic and abdominal viscera, this is referred to as dextrocardia with situs inversus (situs inversus totalis) [12]. In patients with dextrocardia, the standard 12-lead ECG shows marked right-axis deviation (RAD) of both the P wave and QRS complex in lead I, with a predominantly negative QRS complex and inverted P and T waves. The QRS complexes in leads aVR and aVL are reversed, while the R wave in lead aVR is positive [13]. There is a reversal of the typical QRS complex progression in the precordial leads, most commonly in V4-V6 [14]. There is a loss of amplitude toward V6, and V1 and V2 are also reversed [15,16].

These precordial lead findings are valuable in differentiating mirror-image dextrocardia from reversed right/left limb-lead placement error, which results in normal R-wave progression from V1 through V6 [13]. In dextrocardia, reversal of the arm leads results in a "normal" P-wave pattern and QRS axis in the frontal-plane limb leads. Right-sided reorientation of the precordial leads (V1R-V6R) results in a typical pattern of septal depolarization and R-wave progression on the 12-lead ECG [14,16].

In this manuscript, I report a new cardiovascular syndrome named "Yasser's V1-aVR Rhythm Syndrome and Diagonal Biting Compressive Radiological Yasser's Sign," with a differential diagnosis including ECG dextrocardia, old anterior infarction, and suspected renal infarction in an elderly male patient.

Case Presentation

History and Physical Examination:

A 58-year-old Egyptian male worker was admitted to the intensive care unit (ICU) with dyspnea, disturbed level of consciousness (DCL), sudden hematuria, right loin pain, vomiting, and dizziness. He gave a history of acute myocardial infarction (AMI) with ICU admission and underwent percutaneous transluminal coronary angioplasty (PTCA) with placement of two drug-eluting stents (DES) after thrombolytic therapy and anti-ischemic measures 3 years earlier (**Fig-1A**). He had stopped smoking cigarettes 10 years earlier. The patient had a history of diabetes mellitus treated with regular mixed insulin, oral torsemide (10 mg once daily), and oral aspirin (75 mg once daily). He also had a history of amputation of two toes of the right foot because of diabetic foot and gangrene 30 days earlier. There was no history of a similar attack. Currently, the patient denied a history of other relevant diseases, medications, or special habits. Written consent was obtained.

On general physical examination, the patient appeared tall and kyphotic, with an ataxic gait, pallor, staring vision (**Fig-1B**), dysarthria, dyspnea, orthopnea, and bilateral pitting lower-limb edema. His pulse rate was regular at 65 bpm, blood pressure was 80/50 mmHg, respiratory rate was 24/min, temperature was 36°C, and oxygen saturation by pulse oximetry was 93%. Chest inspection revealed a barrel-shaped chest, and cardiac auscultation revealed distant heart sounds.

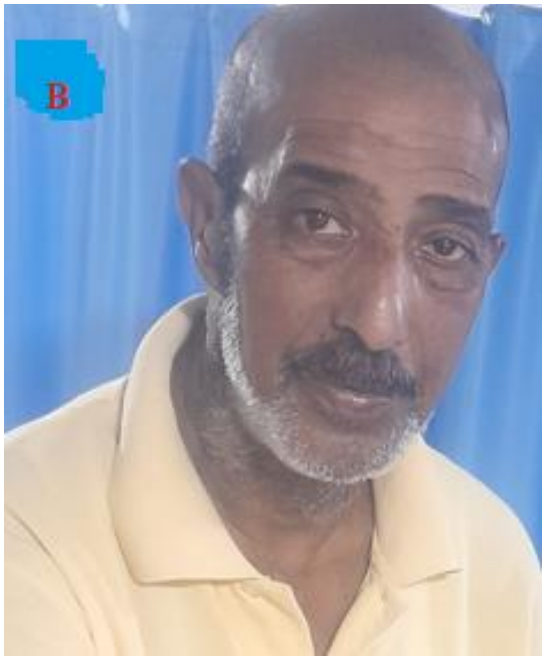


Fig-1B:

Photograph of the patient showing a staring gaze. The image is published with the patient's written informed consent.

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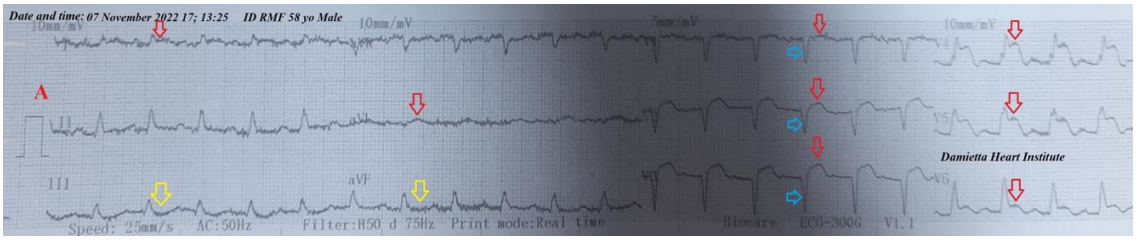


Fig-1A:

ECG tracing obtained three years previously (November 7, 2022) showing acute high lateral and extensive anterior STEMI (red, yellow, and light blue arrows) with sinus tachycardia.

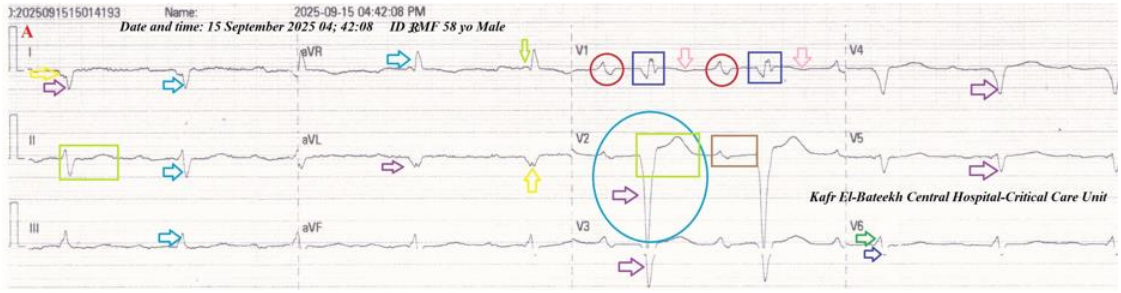


Fig-2A:

ECG tracing was obtained at the initial presentation and ICU admission showing sinus bradycardia (VR: 56), (RAD, light blue arrows) prolonged PR interval (brown rectangle) except aVR lead (lime arrow), pathological Q wave in both high lateral and anterior leads (purple arrow) except V6 (green and dark blue arrows), small equiphasic QRS in V1 (dark blue rectangle), large QS in V2 (light circle), biphasic P wave in V1 (red circle), right QRS fragmentation in lead I (yellow arrow), central QRS fragmentation in aVL lead (yellow arrow), QTc prolongation (lime rectangle), and squaring ST-segment elevation with ending (dark blue rectangle) with superficial small inversion in V1 (rose arrows)

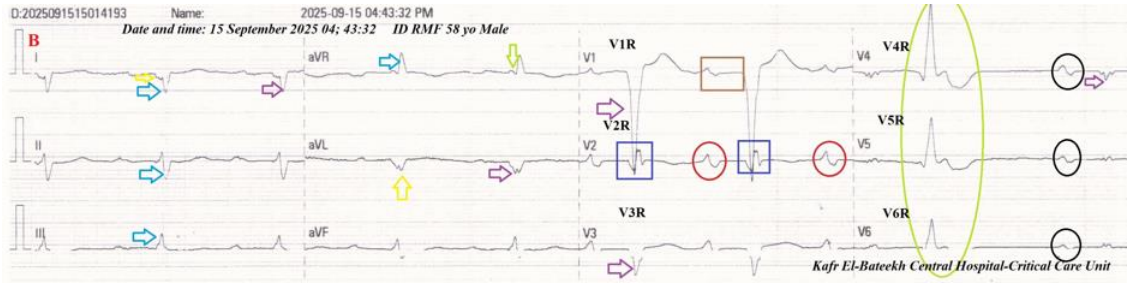


Fig-2B:

The right ECG tracing was obtained within 80 seconds of the preceding tracing, showing the same findings, with 3 PVCs and squaring STE in V4-6 leads (lime oval circle)

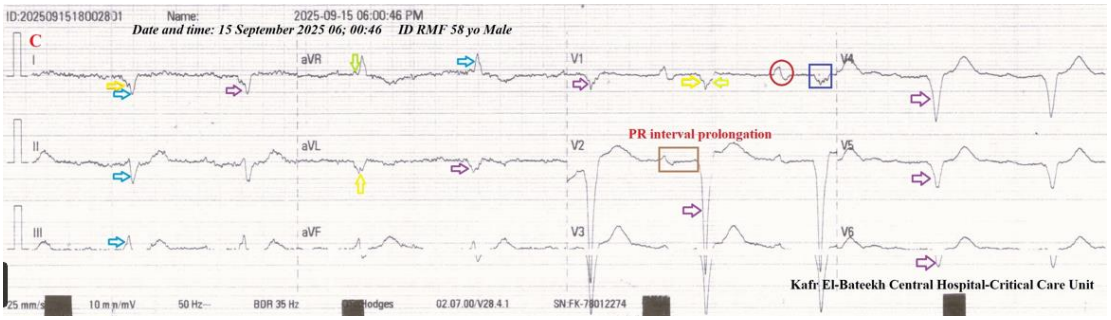


Fig-2C:

ECG tracing was done within 77 minutes of the above tracing, which showed the same findings described above with right and left QRS fragmentation in V1 (yellow arrows), QS in V1 (dark blue rectangle), and pathological Q wave in all high lateral and anterior leads (purple arrow).

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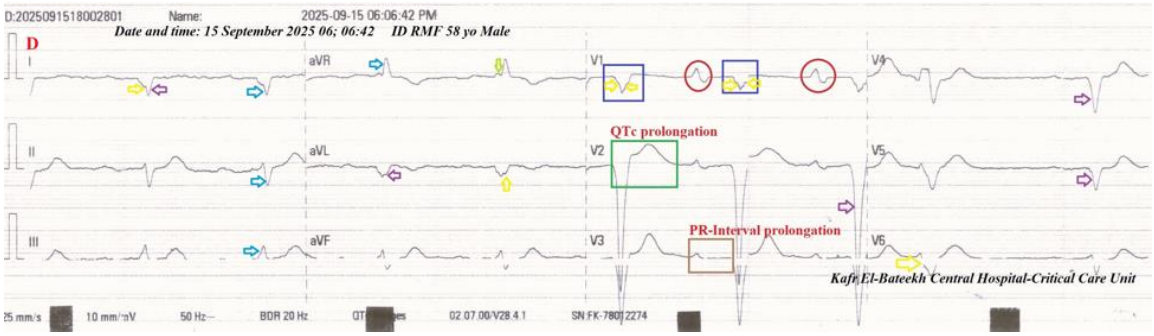


Fig-2D:

ECG tracing was done within 6 minutes of the above tracing, which showed the same findings, with right and left QRS fragmentation in V1 (yellow arrows), right QRS fragmentation in V6 (yellow arrow), and QS in V1 (dark blue rectangle).

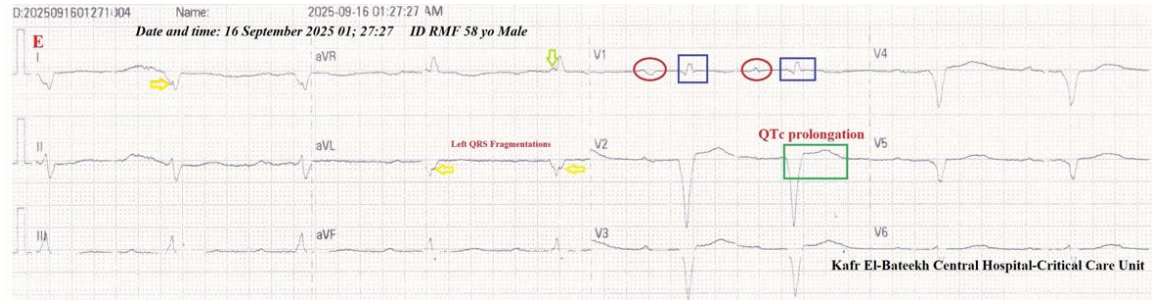


Fig-2E:

ECG tracing was done within 7.5 hours of the above tracing, which showed the same findings described above but with left QRS fragmentation in aVL (yellow arrows), right QRS fragmentation in V6 (yellow arrow), and a small dominant R in V1 (dark blue rectangle).

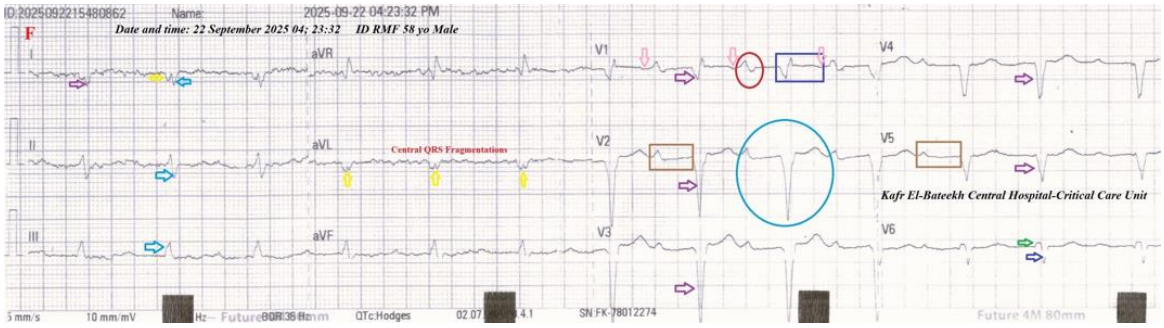


Fig-2F:

ECG tracing was done within 6 days of the above tracing, which showed the same findings described above but with a pathological Q wave in both high lateral and anterior leads (purple arrow) except V6 (green and dark blue arrows), squaring ST-segment elevation with ending (dark blue rectangle), with superficial small inversion in V1 (rose arrows).

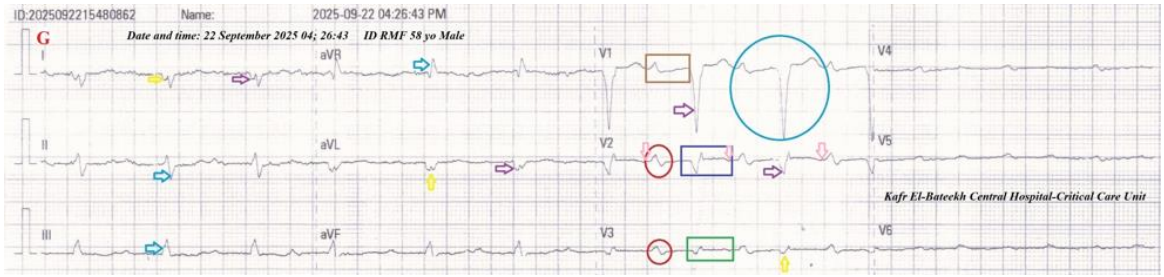


Fig-2G:

The right-sided ECG tracing was obtained within 3.15 minutes of the above tracing, showing reversed findings.

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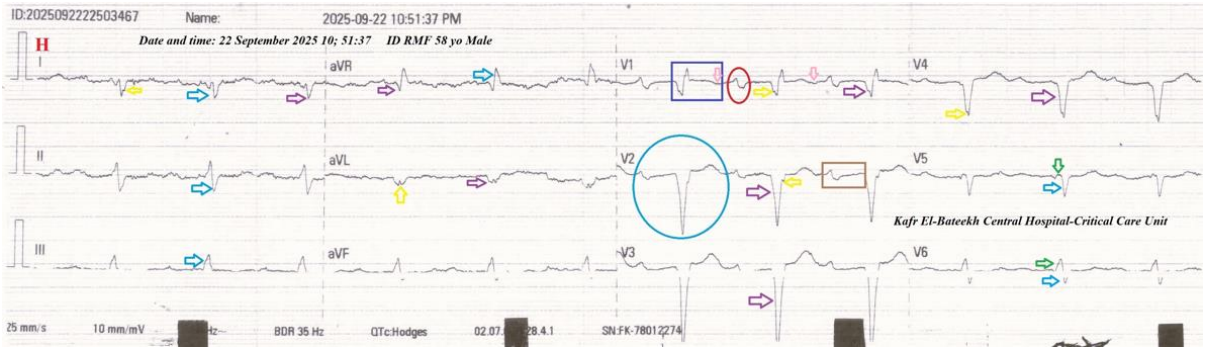


Fig-2H:

ECG tracing was done within 6 hours of the above tracing showing the same findings described for the Fig-2F ECG tracing, but with a stunted R wave in V5 (green and light blue arrows), equiphasic QRS in V6 (green and light blue arrows), right, left, and central fragmentations (yellow arrows).

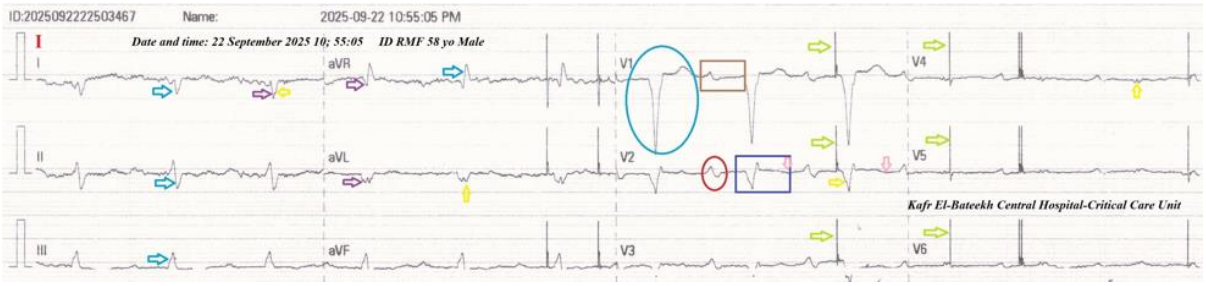


Fig-2I:

The right-sided ECG tracing was done within 4.5 minutes of the above tracing, showing the reversed findings described above, but with spike sign (lime arrows).

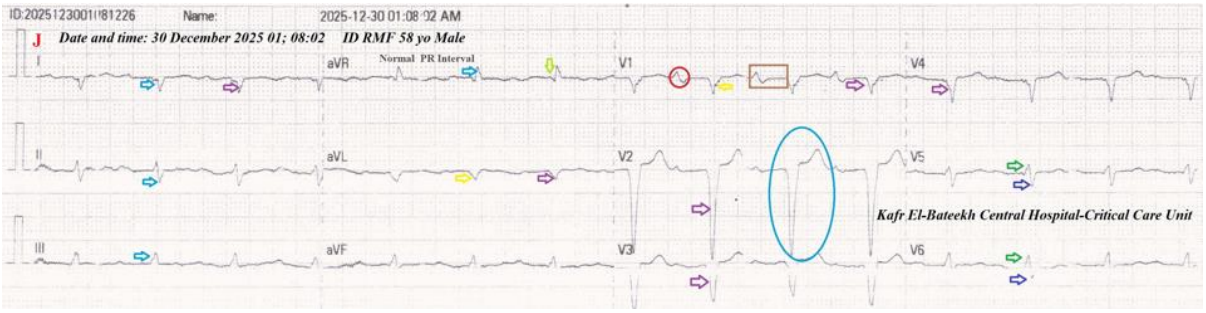


Fig-2J:

ECG tracing was done within 8 days of the above tracing, which showed the same findings but with QS in V1 (dark blue rectangle) and equiphasic QRS in V5 and V6 (green and light blue arrows).

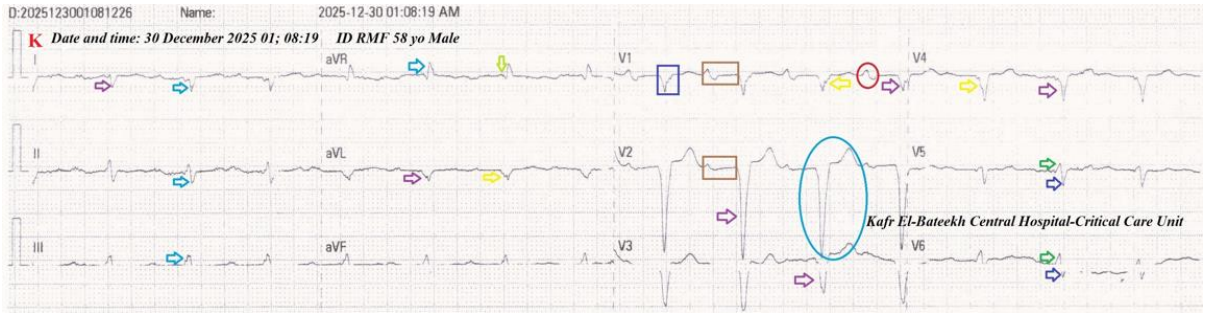


Fig-2K:

ECG tracing was obtained within 17 seconds of the above tracing, which showed the same findings, with right and left QRS fragmentation (yellow arrows), QS in V1 (dark blue rectangle), and equiphasic QRS in V5 and V6 (green and light blue arrows).

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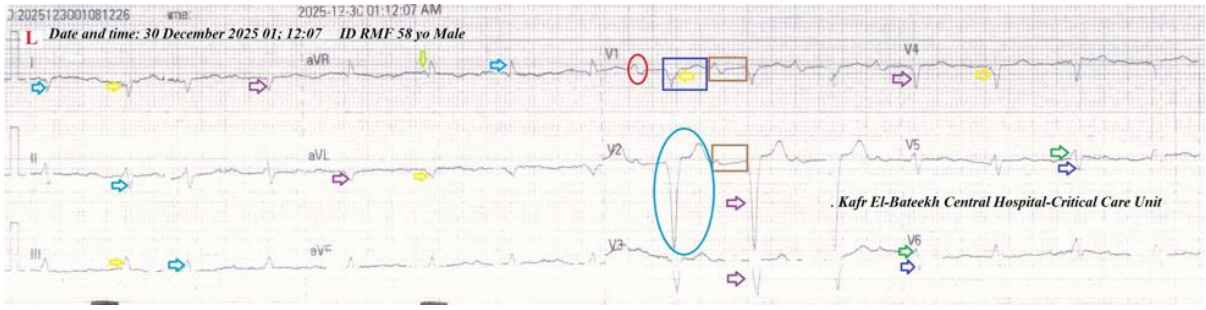


Fig-2L:

ECG tracing was done within 8 minutes of the above tracing, which showed the same findings.

Workup and Management:

At the time of admission, the patient was admitted to the ICU with disturbed level of consciousness (DCL), hypotension, bradycardia, and suspected renal infarction. Initially, the patient was treated in the ICU with oxygen (O_2) inhalation via an oxygen system (100% via a normal mask, 5 L/min). Aspirin, 4 oral tablets (75 mg, then once daily); clopidogrel, 4 oral tablets (75 mg, then once daily); low-dose enoxaparin subcutaneously (80 mg, twice daily); atorvastatin tablets (40 mg, once daily); and mixed long-acting insulin (subcutaneously, four times daily, according to the standard dosing schedule) were added. After improvement in blood pressure, low-dose oral furosemide (40 mg, fasting, once daily) and spironolactone (25 mg, fasting, once daily) were added. The patient was monitored hourly for vital signs, central venous pressure (CVP), and oxygen saturation.

Serial ECG tracings were performed during the initial presentation in the ICU and showed a constellation of findings, including characteristic ST-segment elevation with a preceding pathological Q wave and ending with slight shallow inversion in the V1 lead; a biphasic P wave with a slight terminal portion in the V1 lead; a dominant small R wave with transient conversion to QS in the V1 lead after initial management; prolongation of the PR interval in all ECG leads except the aVR lead, where it was normal; pathological Q waves in all anterior and high lateral leads except V5 and V6, with some variations; fixed right-axis deviation; four new types of QRS fragmentation (right, central, left, and right and left); transient QTc prolongation; and a few transient premature ventricular contractions (PVCs), with and without ST-segment elevation (**Fig-2A** to **Fig-2L**).

A plain chest X-ray performed during the initial presentation and ICU admission showed cardiomegaly, a mid-right parasternal irregular diagonal biting shadow, and a left parasternal irregular diagonal extension shadow. A chest X-ray, posteroanterior view, was repeated at follow-up within 6.5 months of the initial presentation and ICU admission and showed a decrease in the size of both diagonal shadows (**Fig-3A** and **Fig-3B**).

Echocardiography performed within 8 days of the initial presentation and ICU admission showed moderate pulmonary hypertension, moderate mitral regurgitation, mild tricuspid regurgitation, mild pulmonary regurgitation, a normal aortic valve, dilated left atrium (LA), dilated right atrium (RA) and right ventricle (RV), third-degree diastolic dysfunction, moderate reduction in left ventricular (LV) contractility and regional wall motion abnormalities (RWMA), with a low ejection fraction (EF) of 32% (**Fig-3C**).

A brain CT scan performed during the initial presentation and ICU admission showed evidence of chronic lacunar infarctions (**Fig-3D**). A chest CT scan performed during the initial presentation and ICU admission showed cardiomegaly, slight bilateral pleural effusions, multiple nodules in the left lung, a right parasternal irregular diagonal biting shadow, and a left parasternal irregular diagonal extension shadow (**Fig-3E**).

The initial workup on ICU admission showed the following: complete blood count (CBC): hemoglobin (Hb), 9.6 g/dL; red blood cells (RBCs), $4.4 \times 10^3/mm^3$; white blood cells (WBCs), $9.8 \times 10^3/mm^3$ (neutrophils, 76.4%; lymphocytes, 14.9%; monocytes, 8.7%; eosinophils, 0%; and basophils, 0%); and platelets, $259 \times 10^3/mm^3$. C-reactive protein (CRP) was 48 g/dL.

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Serum albumin was 3.4 g/dL. Serum creatinine was 1.3 mg/dL. Random blood sugar (RBS) was 346 mg/dL. Plasma sodium was 110 mmol/L. Serum chloride was 69.5 mmol/L. Ionized calcium was 0.95 mmol/L. The troponin test was negative. D-dimer was 0.96 mg/dL. Screening for HCVAb, HBsAg, and HIVAb was negative.

The workup was repeated within 7 days. CBC showed Hb, 10.4 g/dL; RBCs, 3.69 × 10^3/mm^3; WBCs, 6.9 × 10^3/mm^3 (neutrophils, 80.3%; lymphocytes, 12.1%; monocytes, 7.6%; eosinophils, 0%; and basophils, 0%); and platelets, 161 × 10^3/mm^3. CRP was 48 g/dL. Serum albumin was 3.4 g/dL. Serum creatinine was 1.4 mg/dL, and blood urea was 58 mg/dL. RBS was 193 mg/dL. Plasma sodium was 132 mmol/L. Serum potassium was 4.45 mg/dL. Serum chloride was 101 mmol/L. Ionized calcium was 1.16 mmol/L. The troponin test was negative. The international normalized ratio (INR) was 1.95, and prothrombin time (PT) was 16.

A gradual clinical response occurred. Shock, symptomatic bradycardia, congestive heart failure (CHF), old anterior and high lateral myocardial infarction, diabetes, and suspected renal infarction were the most probable diagnoses. Within a few days of starting the medications, the patient showed marked clinical improvement. He was discharged and maintained on aspirin tablets (75 mg, once daily), clopidogrel tablets (75 mg, once daily), atorvastatin tablets (40 mg, once daily), oral furosemide (40 mg, fasting, once daily), and spironolactone (25 mg, fasting, once daily). Future cardiac follow-up was recommended.

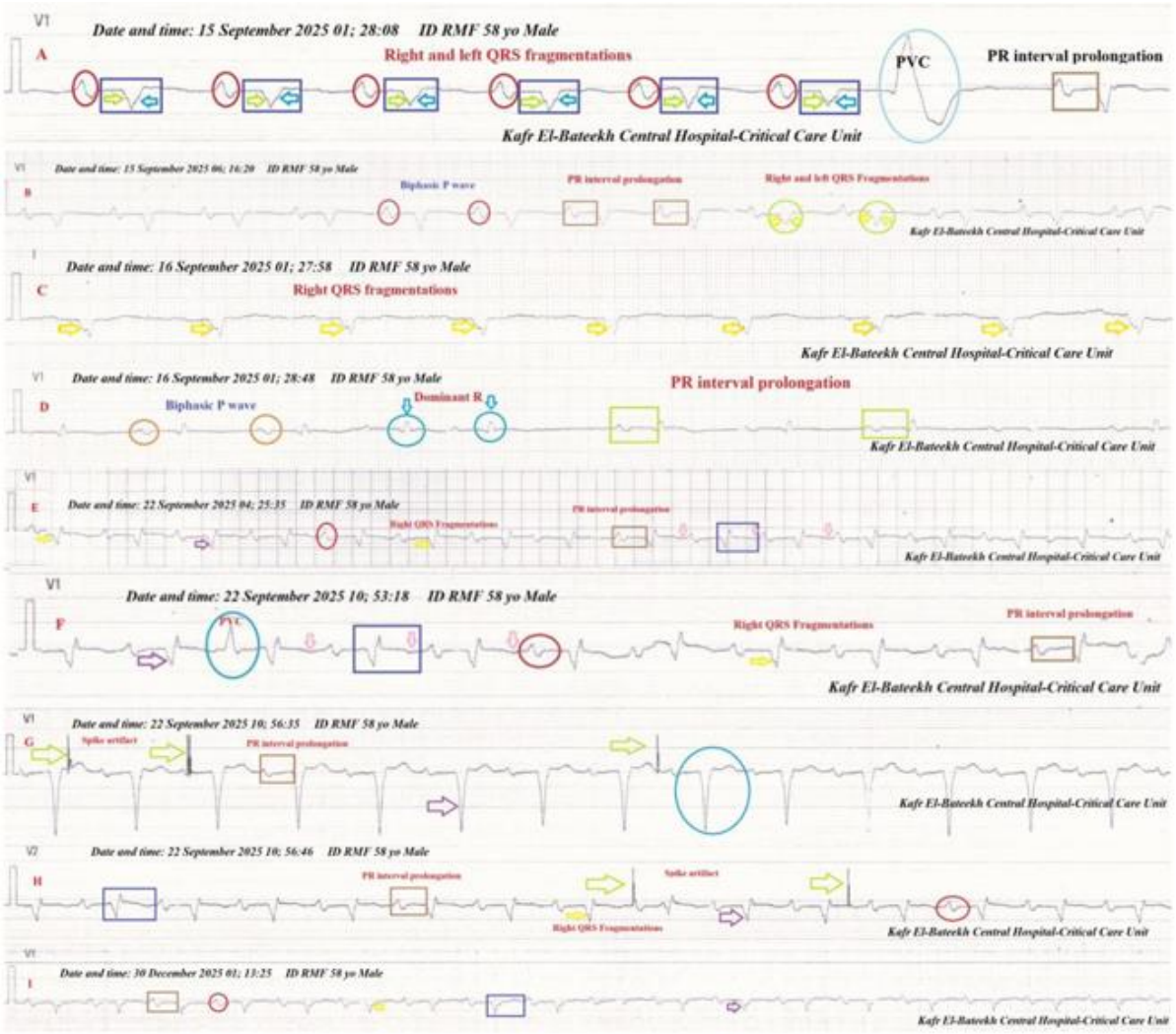


Fig-3A-I:

Serial strips from the ECG tracings showing nearly all the findings described above.

Discussion

Overview:

An elderly Egyptian male worker was admitted to the intensive care unit (ICU) with disturbed level of consciousness (DCL), congestive heart failure (CHF), hypotension, bradycardia, hematuria, and uncontrolled diabetes. The primary objective of this case study was to manage the presence of DCL, CHF, hypotension, bradycardia, suspected renal infarction, and uncontrolled diabetes. The secondary objective of this case study was to determine how this case should be managed in the ICU.

The presence of sudden hematuria, right loin pain, and vomiting, with a normal abdominal ultrasound, strengthens the suspicion of renal infarction. The elevated initial D-dimer, hypotension, the presence of ischemic heart disease (IHD), and associated CHF are possible risk factors for renal infarction.

Hypotension, bradycardia, prolonged PR interval, anemia due to hematuria, hyponatremia, hypokalemia, and hypocalcemia are possible causes of the present DCL.

The presence of pathological Q waves in leads I, aVL, and V1-V6, with variation in V5-V6 throughout the above serial ECGs, indicates evidence of old high lateral and anterior infarction. The initial ECG indicated acute high lateral and anterior STEMI.

There is no clear cause for the current first-degree heart block (prolonged PR interval) in this case. Old MI, CHF, and multiple valvular heart diseases are possible implicated causes.

There is no obvious cause for the presence of multiple nodules in the left lung. Nodular COVID-19 pneumonia may be suggested, especially with associated high CRP, neutrophilia, and lymphocytopenia. Unfortunately, PCR was not performed. Hypersensitivity pneumonitis is also included in the differential diagnosis.

There are pathological Q waves indicating evidence of old high lateral and anterior infarction, right-axis deviation (RAD), a dominant positive aVR, and poor progression of the QRS complex in the chest leads. Therefore, an ECG pattern of dextrocardia is implicated in the differential diagnosis. Chest X-ray findings are against dextrocardia.

Interestingly, the presence of CHF, hypotension, bradycardia, pulmonary hypertension, mitral regurgitation, tricuspid regurgitation, pulmonary regurgitation, reduced LV contractility, regional wall motion abnormalities (RWMA), chronic lacunar infarctions, suspected renal infarction, and uncontrolled diabetes represents a constellation of risk factors.

Description and Principles of "Yasser's V1-aVR Rhythm Syndrome and Diagonal Biting Compressive Radiological Yasser's Sign":

"Yasser's V1-aVR Rhythm Syndrome" is a newly described syndrome with a constellation of the following findings:

A. Characteristic ST-segment elevation with a preceding pathological Q wave and ending with slight shallow inversion in the V1 lead.
B. Biphasic P wave with a slight terminal portion in the V1 lead.
C. A dominant small R wave with transient conversion to QS in the V1 lead after initial management.
D. Prolongation of the PR interval in all ECG leads except the aVR lead, in which it is normal.
E. Pathological Q waves in all anterior and high lateral leads except V5 and V6, with some variations.
F. Fixed right-axis deviation.
G. Four new types of QRS fragmentation: right, central, left, and right and left.
H. Transient QTc prolongation.
I. A few transient premature ventricular contractions (PVCs), with and without ST-segment elevation (**Fig-2A** to **Fig-L** and **Fig-3A** to **Fig-3I**).

"Yasser's V1-aVR Rhythm Syndrome" is associated with the following:
A. Old extensive myocardial infarction.
B. Chronic heart failure.
C. Multiple valvular regurgitations.
D. Pulmonary hypertension.
E. Old lacunar brain infarction.
F. Dysarthria.

G. Staring vision.
H. Mixed personality disorder.

"Diagonal Biting Compressive Radiological Yasser's Sign" is a newly described sign characterized by an irregular diagonal biting-shaped appearance in the right middle paramediastinal region of the heart. This diagonal biting shape is associated with an irregular diagonal extension and dilatation in the opposite left middle paramediastinal region of the heart.

According to anatomical landmarks, the first right diagonal mostly represents the right pulmonary artery and the lower part of the superior vena cava. The second left diagonal primarily represents the pulmonary artery trunk. Both right and left diagonal areas decreased in size following subsequent management (**Fig-4A**, **Fig-4B**, and **Fig-5**).

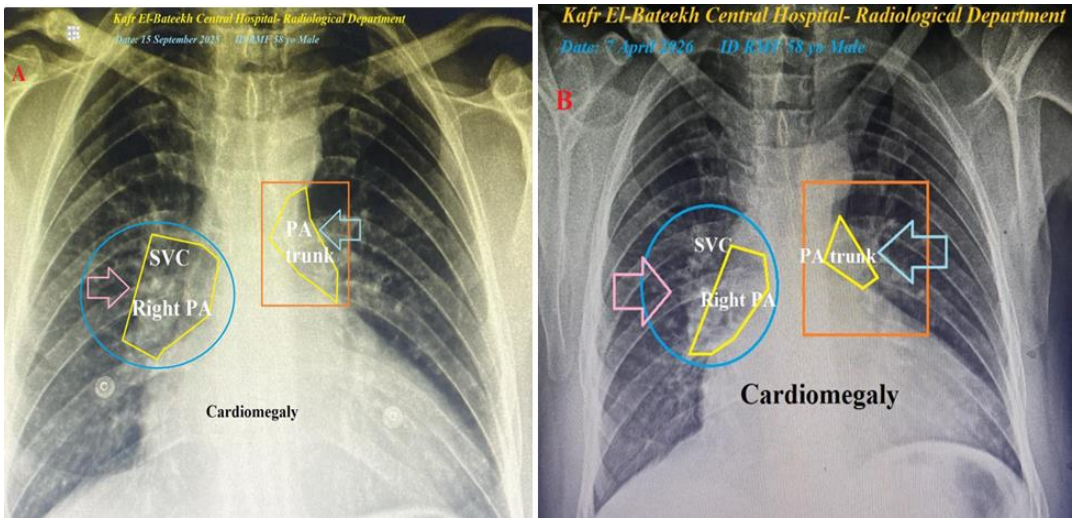


Fig-4A-B:

(A) Posteroanterior chest X-ray obtained at initial presentation and ICU admission showing cardiomegaly, a mid-right parasternal irregular diagonal biting shadow (rose arrow and yellow diagonal), and a left parasternal irregular diagonal extension shadow (light blue arrow and yellow diagonal). (B) Posteroanterior chest X-ray obtained 6.5 months after initial presentation and ICU admission showing a decrease in the size of both diagonals.

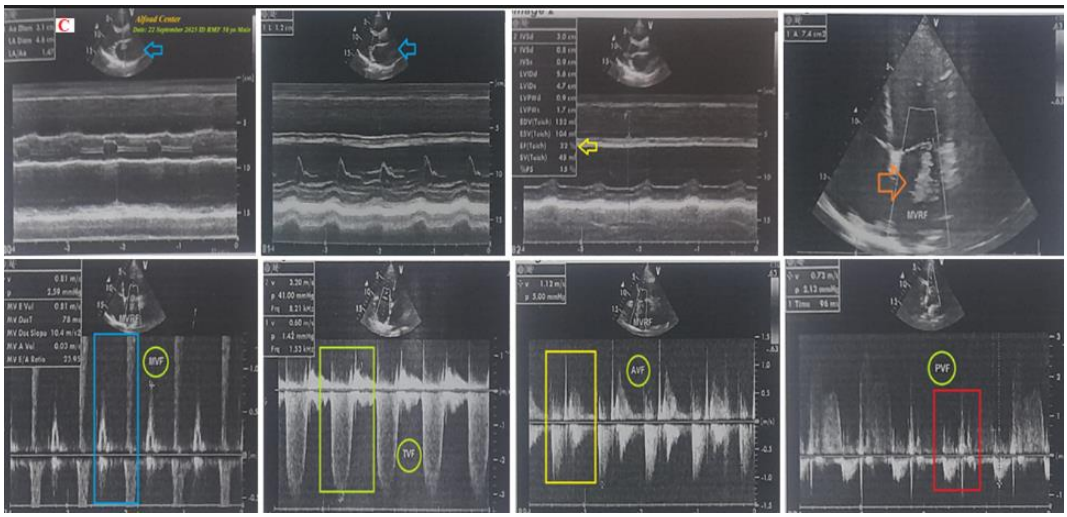


Fig-4C:

Echocardiography performed within eight days of initial presentation and ICU admission showing moderate pulmonary hypertension, moderate mitral regurgitation (yellow rectangle and golden arrow), mild tricuspid regurgitation (lime rectangle), mild pulmonary regurgitation (red rectangle), a normal aortic valve (yellow rectangle), a dilated LA (light blue arrows), dilated RA and RV, third-degree diastolic dysfunction, moderately reduced LV contractility and RWMA, and a low EF of 32% (yellow arrow).

Case Report

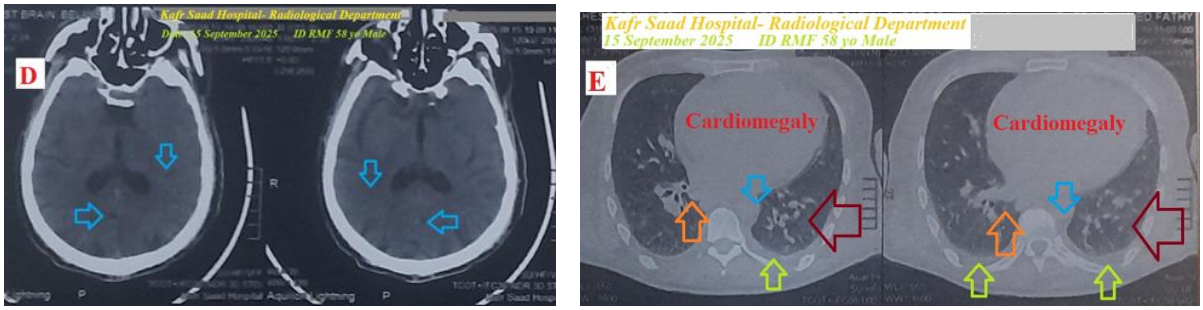


Fig-4D-E:

(D) Brain CT sections obtained at initial presentation and ICU admission showing evidence of chronic lacunar infarctions (light blue arrows). (E) Chest CT sections obtained at initial presentation and ICU admission showing cardiomegaly, slight bilateral pleural effusions (lime arrow), multiple nodules in the left lung, a right parasternal irregular diagonal biting shadow (golden arrow), and a left parasternal irregular diagonal extension shadow (light blue arrow).



Fig-5:

Author-created illustration of "Yasser's V1-aVR Rhythm Syndrome" and "Diagonal Biting Compressive Radiological Yasser's Sign."

There are four new types of fragmented QRS (fQRS), referred to as Yasser's classifications: 1. Right, 2. Central, 3. Left, and 4. Compound right and left. These four types were classified according to the site of fragmentation within the QRS complex. If the fragmentation is present in the initial part of the QRS complex, it is classified as right fragmentation. If it is present in the terminal part of the QRS complex, it is classified as left fragmentation. If it is present in both the initial and terminal parts of the QRS complex, it is classified as compound right and left fragmentation. If it is present in the middle part of the QRS complex, it is classified as central fragmentation (**Fig-2A** to **Fig-2L**, **Fig-3A** to **Fig-3I**, and **Fig-6A** to **Fig-6D**).

Fragmented QRS (fQRS) is an abnormality of myocardial conduction that may serve as a marker of myocardial scar. It is characterized by additional notches within the QRS complex [17]. Both QRS distortion and fQRS provide additional prognostic value compared with the presence of either QRS distortion or fQRS alone for early risk stratification in patients with STEMI treated with primary percutaneous coronary intervention (PPCI) [17]. fQRS can also be used as a useful tool for risk stratification in STEMI [18].

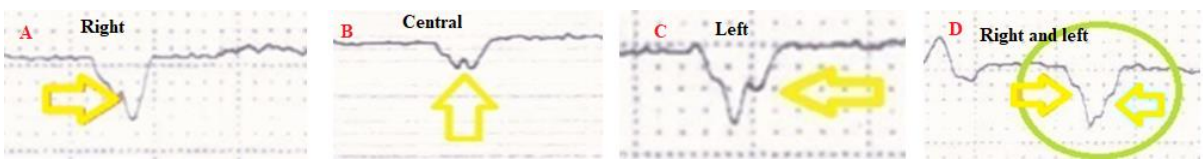


Fig-6A-D:

Yasser's classifications of QRS fragmentation.

The current case could not be compared with similar conditions because no similar or previously reported cases with the same clinical presentation and management were identified for direct comparison. The main limitation of the current study was the unavailability of genetic testing, including karyotype analysis, because of the high cost.

Conclusion and Recommendations

"Yasser's V1-aVR Rhythm Syndrome" is a newly described syndrome characterized by a constellation of findings, including characteristic ST-segment elevation with a preceding pathological Q wave and ending with slight shallow inversion in the V1 lead, a biphasic P wave with a slight terminal portion in the V1 lead, a dominant small R wave with transient conversion to QS in the V1 lead after initial management, prolongation of the PR interval in all ECG leads except the aVR lead, pathological Q waves in all anterior and high lateral leads except V5 and V6 with some variations, fixed right-axis deviation, four new types of QRS fragmentation (right, central, left, and compound right and left), transient QTc prolongation, and a few transient premature ventricular contractions (PVCs), with and without ST-segment elevation.

"Diagonal Biting Compressive Radiological Yasser's Sign" is a newly described sign characterized by an irregular diagonal biting-shaped appearance in the right middle paramediastinal region of the heart. It is associated with an irregular diagonal extension and dilatation in the opposite left middle paramediastinal region of the heart. The first right diagonal is considered to correspond mainly to the right pulmonary artery and the lower part of the superior vena cava, while the second left diagonal primarily corresponds to the pulmonary artery trunk. Both diagonal areas decreased in size following subsequent management.

Further research is recommended to evaluate these newly described electrocardiographic and radiological findings in larger patient populations.

Conflict of Interest

There are no conflicts of interest.

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