

Yasser's Squaring Saddling Syndrome, Partial Modified Yasser's WPW Syndrome, Yasser's Lateral Halo Sign, Neglected Infarction, Bifascicular Block, and Craniofacial Hyperhidrosis Post-Organophosphorus with Interlacing COVID-Pneumonia-Cardiovascular-Radiological Discoveries

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Abstract

Introduction: Hyperhidrosis (HH) is described as overrun sweating that directly influences the quality of life, emotional, social, and professional status of the patient. Silent myocardial ischemia is an ischemic episode without causing noticeable symptoms like chest pain or discomfort, with at least 15% to 30% of acute myocardial infarction have evidence of prior silent myocardial ischemia. Right bundle branch block (RBBB) alternating with either left anterior fascicular block (LAFB) or left posterior fascicular block (LPFB) results in a bifascicular block in the ECG. Organophosphates (OP) are a diverse group of chemical compounds that have cardiovascular, neurological, and respiratory adverse effects. Kounis-Zafra (KZ) syndrome is a newly described syndrome relevant to allergen exposure. COVID-19 infection may have lethal cardiovascular and respiratory complications. The halo sign and the reversed halo sign in CT are associated with patches of ground-glass opacities at the late phase of COVID-19 infection. Case presentation: Elderly married male, a Farmer, a non-smoker, Egyptian, patient, was presented to the POC after over 5 years of follow-up, after two attacks of COVID-19 pneumonia and a neglected and passed myocardial infarction, post-prolonged exposure history of inhalation of organophosphorus. The chest CT, oxygenation, ECG, and echocardiography were the interventions. Conclusion: "Yasser's squaring saddling syndrome", "partial modified Yasser's WPW syndrome", and "Yasser's lateral halo sign" are new cardiovascular and radiological discoveries. Senility, chronic exposure to organophosphates, old myocardial infarction, two attacks of COVID-19 pneumonia, fixed bifascicular heart block, transient trifascicular heart block, dilated cardiomyopathy, bigeminal premature ventricular complexes (PVCs), multiple irregular PVCs, premature ventricular couplets, runs of ventricular tachycardia (VT), and left ventricular (LV) systolic dysfunction with global hypokinesia are serious constellation risk factors. Further wide studies will be recommended.

Keywords: yasser's squaring saddling syndrome; partial modified yasser's wpw syndrome; yasser's lateral halo sign; silent infarction; heart block; craniofacial hyperhidrosis; organophosphorus; covid-pneumonia

Abbreviations

ACS: Acute coronary syndrome

AMI: Acute myocardial infarction

CAS: Coronary artery spasm

CBC: Complete blood count

COVID-19: Coronavirus disease 2019

ECG: Electrocardiography

HH: Hyperhidrosis**IHD:** Ischemic heart disease**ICU:** Intensive care unit**KZ;** Kounis Zafra**LAFB:** Left anterior fascicular block**LPFB:** Left posterior fascicular block**OP:** Organophosphates**PVC:** Premature ventricular complex**RBBB:** Right bundle branch block**VR:** Ventricular rate**VT:** Ventricular tachycardia**WPW syndrome:** Wolff-Parkinson-White syndrome

1. Introduction

Hyperhidrosis (HH) is described as an overproduction of sweat beyond the physiological necessary amount, which meets the body's thermal regulation. It is mostly regional due to hyperfunctioning of the sweat gland. Unfortunately, it is directly influencing the quality of life, emotional, social, and professional aspects of the patient. HH is either primary or secondary [1]. Primary HH is often bilateral, symmetrical, improves during sleep, and worsens in situations of stress conditions with no detected cause. It may be genetic and familial. Hyperthyroidism, hyperpituitarism, diabetes mellitus, menopause, pregnancy, pheochromocytoma, carcinoid syndrome, acromegaly, Parkinson's disease, spinal cord injury, stroke, Hodgkin's disease, myeloproliferative diseases, tuberculosis, septicemia, fluoxetine, venlafaxine, doxepin, alcoholism, substance abuse, [2] and Frey's syndrome due to parotid surgery [3] are implicated as a secondary HH. Surgical treatment of craniofacial sweating is more likely to be followed by undesirable side effects [4]. Treatment of the underlying cause is the choice for secondary hyperhidrosis [2]. **Organophosphates (OP)** encompass a diverse group of chemical compounds with common applications in pesticides and herbicides that inhibit acetylcholinesterase (AChE) enzyme, resulting in an oversecretion of acetylcholine [5]. According to the WHO, in 1990 estimated that there were 1 million unintentional pesticide poisonings, resulting in approximately 20,000 fatalities [6]. Consequences of hypovolemia or hypoxia, peripheral vasodilatation, and direct myocardial damage may be present with OP poisoning. Muscarinic cardiovascular effects due to muscarinic receptor stimulation result in bradycardia, conduction block, and hypotension by parasympathetic overactivity, whereas nicotinic cardiovascular signs of AChE inhibitor toxicity due to nicotinic receptor stimulation result in hypertension and tachycardia by sympathetic overactivity [5,7]. The electrophysiological abnormalities often include ventricular tachyarrhythmias, torsades de pointes, QT interval prolongation, ST-segment deviations, tall T waves, premature contractions, and AVB [7]. **Silent myocardial ischemia** refers to a condition where myocardial blood flow is reduced without causing noticeable symptoms such as chest pain or discomfort. At least 15% to 30% of patients with acute myocardial infarction have evidence of prior silent myocardial ischemia. Older patients, diabetes mellitus, obstructive sleep apnea, critically ill patients, and perioperative situations are risk factors that increase susceptibility to asymptomatic ischemic episodes [8]. Interruptions in conduction may occur with right bundle branch block (RBBB) alternating with either left anterior fascicular block (LAFB) or left posterior fascicular block (LPFB), producing **bifascicular block** in the ECG [9]. **Wolff-Parkinson-White (WPW)** syndrome is a cardiac

preexcitation syndrome that arises from an abnormal accessory pathway that can result in symptomatic and life-threatening arrhythmias. It hallmarks the ECG of a short PR interval, wide QRS, and delta wave in the presence of sinus rhythm [10]. Allergic acute coronary syndrome or KZ syndrome is hallmarked by the co-association of an ACS with hypersensitivity reactions following an allergenic exposure [11]. KS was initially identified by Kounis and Zavras in 1991 as an "allergic angina syndrome", "allergic angina" or "allergic myocardial infarction" [11,12]. There is ACS-associated mast cell activation from allergic, hypersensitivity, or anaphylactoid reactions [12]. The essential pathogenesis of KS is the inflammatory cytokines mediators released through mast cell activation during a hypersensitivity reaction triggered by food, insect bites, or drugs. There is a subsequent coronary artery spasm (CAS) with possible atheromatous plaque erosion or rupture [12]. The allergic angina commonly starts within one hour of exposure to the offending allergen. Longer onset of ACS has also been reported [13]. Variant presentations of KS have been reported [12]. Three different variants of KS have been described: Type I occurs in structurally normal coronary arteries with no cardiovascular risk factors. The coronary spasm was suggested. With or without an associated acute myocardial infarction (AMI). Type II KS occurs in patients with pre-existing ischemic heart disease (IHD), in whom the acute release of inflammatory mediators induces CAS that may lead to plaque rupture and MI. Type III KS occurs in patients with coronary artery stent-associated thrombosis [11,13-15]. **The halo sign and the reversed halo sign** of CT are associated with patches of ground-glass opacities at the late phase of COVID-19 infection [16]. Both are mostly seen around the nodules [17]. The halo sign is a GGO surrounding a nodule or mass. It is a circumjacent area of hemorrhage around a lesion and might be seen in some patients with COVID-19. The halo sign and reversed halo sign are nonspecific for COVID-19 infection. They may be seen in invasive pulmonary Aspergillosis, Mucormycosis, Cryptococcosis, pulmonary adenocarcinoma, some types of pulmonary metastasis, tuberculosis, with polyangiitis granulomatosis, thromboembolism, and eosinophilic lung diseases [18].

2. Case presentation

A 73-year-old married male, a Farmer, non-smoker Egyptian patient was presented to the POC after over 5 years of follow-up with palpitations, dyspnea, and tachypnea. Dizziness, fatigue, loss of appetite, and bilateral lower limb swellings were associated symptoms. He was a daily worker for spraying pesticides for years. He gave a history of two attacks of COVID-19 pneumonia on the 2020th year. The first attack was on August 02, 2020, and was admitted to the intensive care unit (ICU) in the Frescoer Central Hospital and traditionally managed as a COVID-19 pneumonia (**Figures 1A, 1B, and 1C**). The second attack was on November 07, 2020, and was admitted to the ICU in the same Hospital and was managed as a COVID-19 pneumonia (**Figures 1D and 1E**). Within one month before both attacks of COVID-19 pneumonia, the patient complained of an episode of severe acute angina with profuse sweating. Unfortunately, the patient neglected this attack of chest pain and didn't seek medical care. Upon current general physical examination, the patient shows orthopnea, tachypnea, and distressed respiration. The left forehead and face showed profuse sweating (**Figure 2**). Currently, there is a regular pulse rate (VR of 70), blood pressure (BP) of 100/70 mmHg, respiratory rate of 25 bpm, a temperature of 37.2°C, pulse oximeter of oxygen (O2) saturation of 93%, and GCS of 15/15. Tests for provocative latent tetany were positive. No more relevant clinical data were noted during the clinical examination. The initial ECG tracing was done on the ED on August 02, 2020, showing normal sinus rhythm (NSR), dominant R in V1 with LAD, RBBB, SISIISIII pattern, pathological Q waves in III and aVF leads, sporadic square shape ST-segment elevation in aVR and V1 leads, wide notched

QRS complexes, normal PR interval, mimic delta waves, a small single wave between P wave and QRS, biphasic T inversion in V2 and V3 leads. There are T-wave inversions in the anterior leads (V1-5 leads) and ST-segment elevation in aVR, with a mimicked morphology of QRS complexes in V1. There is evidence of the Wavy double sign (Yasser's sign) in V5 and V5 leads (**Figure 3A**). The second ECG tracing was taken in the ED on August 02, 2020, within 3 minutes of the above tracing showing NSR (VR of 84), dominant R in V1 with the same above changes, but there is evidence of a Wavy double sign (Yasser's sign) in I and II leads and a Wavy triple sign (Yasser's sign) in V6 lead (**Figure 3B**). The third ECG tracing was done in the ED on November 07, 2020, showing NSR, dominant R in V1 with bigeminal PVCs, LAD, RBBB, SISIISIII pattern, pathological Q waves in III and aVF leads, sporadic square-shaped ST-segment elevation in V1 lead, saddling-shaped ST-segment elevation in V2 lead, and non-specific ST-segment elevation in V2 lead. There is ST-segment depression in V4-6 leads (**Figure 3C**). The fourth ECG tracing was taken in the ED on November 07, 2020, within 1 minute of the above tracing, showing NSR with the same dominant R in V1, LAD, RBBB, S1S2S3, pathological Q waves in III and aVF leads, sporadic square-shaped ST-segment elevation in aVR and V1 leads, wide notched QRS complexes, normal PR interval, mimic delta waves, a small single-wave between P wave and QRS, biphasic T wave inversion in V2 and V3 leads. There is ST-segment depression in V2-6 leads and T-wave inversions in anterior leads (V1-4). There is a disappearance of the above bigeminal PVCs (**Figure 3D**). The initial echocardiography was done on December 14, 2020, showed dilated cardiomyopathy, global hypokinesia, severe mitral regurgitation, severe tricuspid regurgitation, moderate aortic regurgitation, moderate pulmonary hypertension, and LV systolic dysfunction with EF 35%. The fifth ECG tracing was taken in the POC on April 20, 2021, within 6 months of the above tracing, showing NSR, dominant R in V1, with LAD, RBBB, SISIISIII pattern, pathological Q waves in III and aVF leads, sporadic square-shaped ST-segment elevation in aVR and V1 lead, wide QRS complexes, prolonged PR interval, mimic

delta waves, and a small single wave between P wave and QRS (**Figure 3E**). The sixth ECG tracing was taken in the POC on December 15, 2023 within 19 months of the above tracing, showing sinus arrhythmia, dominant R in V1, multiple irregular PVCs, premature ventricular couplets with LAD, RBBB, SISIISIII pattern, pathological Q waves in III and aVF leads, sporadic square-shape ST-segment elevation in aVR and V1 leads, saddle-shape ST-segment elevation in aVL, V2, and V3 leads, non-specific ST-segment elevation in V1 lead, wide QRS complexes, prolonged PR interval, mimic delta waves, a small single wave between P wave and QRS, and small R in V4-6 leads (**Figure 3F**). The seventh ECG tracing was done in the POC on December 15, 2023 within 1 minute of the above tracing, showing sinus arrhythmia, runs of VT, dominant R in V1, multiple irregular PVCs, with LAD, SISIISIII pattern, pathological Q waves in III and aVF leads, sporadic square-shaped ST-segment elevation in aVR lead, saddle-shaped ST-segment elevation in aVL, V2, and V3 leads, non-specific ST-segment elevation in V1 lead, wide QRS complexes, normal PR interval, mimic delta waves, a small single wave between wave P wave and QRS, and small R in V4-6 leads (**Figure 3G**). Lastly, the patient was admitted to the ICU on March 14, 2024, with decompensated chronic heart failure. Unfortunately, the patient died within 3 days of the ICU admission. The last echocardiography was done on the last ICU admission on 14 March 2024, showing LV dilatation and LV systolic dysfunction with EF 47% (**Figure 4**). The initial complete blood count (CBC); Hb was 12.4 g/dl, RBCs; 4.47*103/mm3, WBCs; 6.6*103/mm3 (Neutrophils; 66.3 %, Lymphocytes: 27.2%, Monocytes; 6.5%, Eosinophils; 0% and Basophils 0%), Platelets; 205*103/mm3. The plasma sodium on the last ICU admission was 147.4 mmol/l, and serum potassium was 2.9 mmol/l. The plasma sodium was repeated within 5 days of ICU admission, which was (141mmol/l), and serum potassium was (3.21mmol/l). Silent infarction, bifascicular block, dilated cardiomyopathy, and craniofacial hyperhidrosis post-organophosphorus with interlacing COVID-Pneumonia was **the most probable diagnosis**.

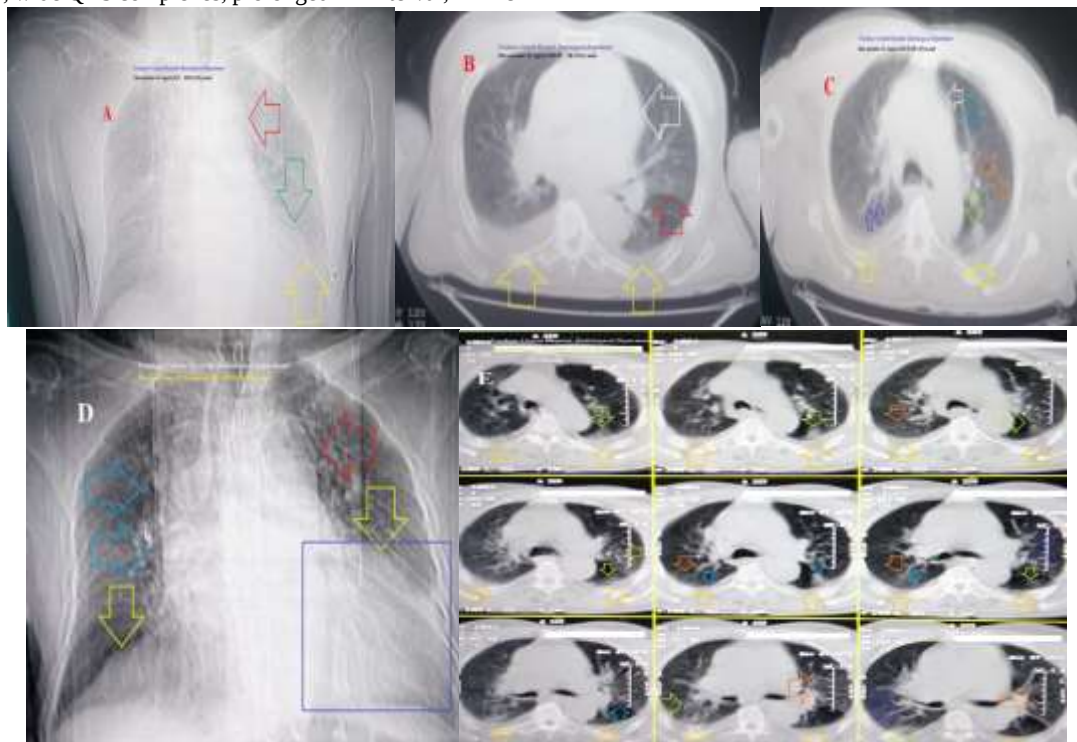


Figure 1: A. Chest X-ray PA view film was done on 02 August 2020, showing cardiomegaly, a wide mediastinum (light blue arrows), aortic dilatation (red arrow), and an obliterated left angle (green and yellow arrows). B. Shortcuts of chest CT scan were done on 02 August 2020, showing cardiomegaly (white arrow), bilateral mild to moderate pleural effusions (yellow arrows), and pulmonary haziness (red arrow). C. Shortcuts of the same chest CT scan showing

the same changes, ground glass consolidations (lime arrow), halo sign (light blue arrow), reversed halo sign (dark blue arrows), and **Yasser's lateral halo sign** (Yasser's sign: orange arrows). **D.** Chest XR PA view film was done on 07 November 2020, showing cardiomegaly, wide mediastinum (light blue arrows), aortic dilatation (red arrow), obliterated left angle (blue rectangular and yellow arrow), and an irregular projected right angle (yellow arrow). **E.** Shortcuts of chest CT scan was done on 07 November 2020, showing ground glass consolidations (lime arrows), bilateral mild to moderate pleural effusions (golden arrows), halo sign (orange arrow), reversed halo sign (light blue arrows), **Yasser's lateral halo sign** (Yasser's sign: dark blue arrows), and pulmonary haziness (rose arrow).



Figure 2: Photo of the patient from the last presentation to POC showing left-sided craniofacial hyperhidrosis (white arrow) in a smiling patient.

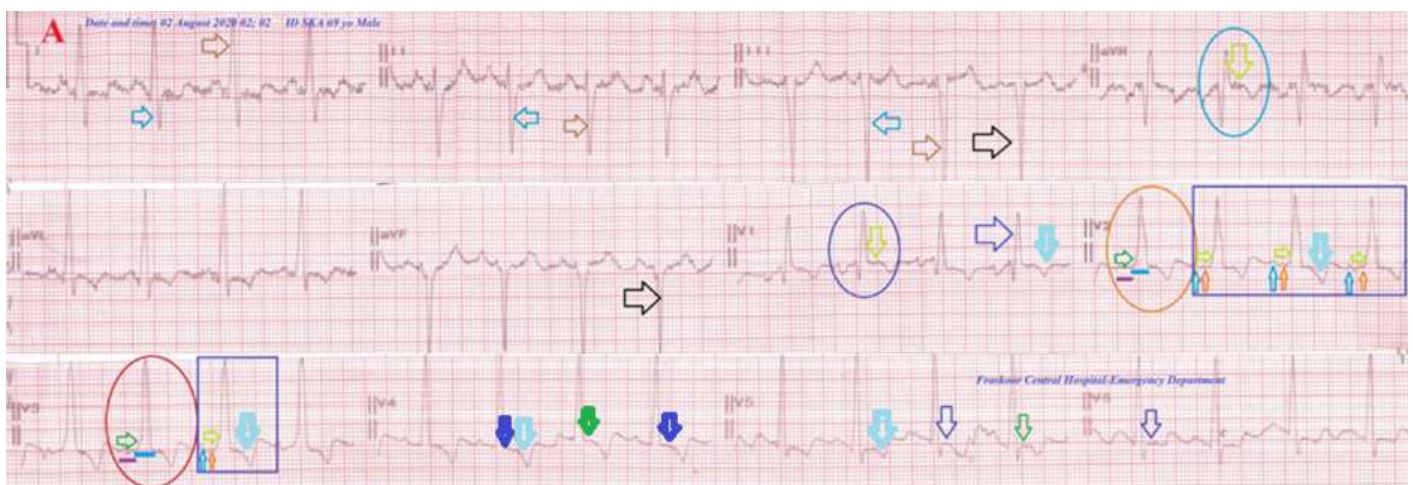


Figure 3: Serial ECG tracings; A. Tracing was done on the ED on August 02, 2020 showing NSR (VR of 85), dominant R in V1 with LAD (brown arrows), RBBB (lime arrow), SISIISIII pattern (light blue arrows), pathological Q waves in III and aVF leads (black arrows), sporadic square-shape ST-segment elevation in aVR and V1 leads ((light blue circle and lime arrow), wide notched QRS complexes (light blue ridge), normal PR interval (pink ridge), mimic delta waves (green and lime arrows), a small single wave between P wave and QRS (golden arrows), biphasic T-wave inversion in V2 and V3 leads. There are T wave inversions in anterior leads (V1-5; light turquoise arrows) and ST-segment elevation in aVR lead (lime arrow) with mimic morphology of QRS complexes in V1. There is evidence of the Wavy double sign (Yasser's sign) in V5 and V5 (dark blue and green arrows).



Figure 3: B. Tracing was done on the ED on August 02, 2020, within 3 minutes of the above tracing showing NSR (VR of 84), dominant R in V1 with the same above changes, but there is evidence of a Wavy double sign (Yasser's sign) in I and II leads (green and dark blue arrows) and a Wavy triple sign (Yasser's sign) in V6 lead (red, green, and dark blue arrows).



Figure 3: C. Tracing was done on the ED on November 07, 2020, showing NSR (VR of 66), dominant R in V1 with bigeminal PVCs (light blue arrows) LAD (green arrows), RBBB (rose arrow), SISIISIII pattern (lime arrows), pathological Q waves in III and aVF leads (red arrows), sporadic square-shaped ST-segment elevation in V1 lead (light blue circle and golden arrows), saddling-shaped ST-segment elevation in V2 lead (pink arrow), and non-specific ST-segment elevation in V2 lead (grey arrow). There is ST-segment depression in V4-6 leads (orange arrows).



Figure 3: D. Tracing was done on the ED on November 07, 2020 within 1 minute of the above tracing showing NSR (VR of 82) with the same dominant R in V1, LAD, RBBB, SISIISIII, pathological Q waves in III and aVF leads, sporadic square-shaped ST-segment elevation in aVR and V1 lead (brown circle and golden arrow), wide notched QRS complexes (green ridge and lime arrows), normal PR interval (dark blue ridge), mimic delta waves (rose arrows), a small single wave between P wave and QRS (golden arrows), biphasic T-wave inversion in V2 and V3 leads. There is ST-segment depression in V2-6 leads (orange arrows) and T wave inversions in anterior leads (V1-4; light turquoise arrows). There is a disappearance of the above bigeminal PVCs.



Figure 3: E. Tracing was done on the POC on April 20, 2021, within 6 months of the above tracing, showing NSR (VR of 62), dominant R in V1, with LAD (green arrows), RBBB (brown circle), SISIISIII pattern (lime arrows), pathological Q waves in III and aVF leads (black arrows), sporadic squared-

shaped ST-segment elevation in aVR and V1 leads (light blue circle and golden arrows), wide QRS complexes (green ridge), prolonged PR interval (blue rectangle), mimic delta waves (small red arrows), a small single wave between P wave and QRS (light blue arrows).



Figure 3: F. Tracing was done on the POC on December 15, 2023 within 19 months of the above tracing, showing sinus arrhythmia (VR of 84), dominant R in V1, multiple irregular PVCs (light blue arrows), premature ventricular couplets (rose arrows) with LAD, RBBB (brown circle), SISIISIII pattern (lime arrows), pathological Q waves in III and aVF leads (red arrows), sporadic square-shape ST-segment elevation in aVR and V1 leads (light blue circle and golden arrows), saddle-shape ST-segment elevation in aVL, V2, and V3 leads (light blue circle and pink arrows), non-specific ST-segment elevation in V1 lead (blue rectangle and light turquoise arrows), wide QRS complexes (green ridge), prolonged PR interval (blue rectangle and light turquoise arrows), mimic delta waves (small lime arrows), small single waves between P wave and QRS (light blue arrows), and small R in V4-6 leads (brown arrows).



Figure 4: G. Tracing was done in the POC on December 15, 2023 within 1 minute of the above tracing, showing sinus arrhythmia (VR of 88), runs of VT (light blue arrows), dominant R in V1 (black arrow), multiple irregular PVCs (rose arrows) with LAD, SISIISIII pattern, pathological Q waves in III and aVF leads, sporadic square-shaped ST-segment elevation in aVR lead (light blue circle and golden arrow), saddle-shaped ST-segment elevation in aVL, V2, and V3 leads (light blue circle and pink arrows), non-specific ST-segment elevation in V1 lead (light blue arrow), wide QRS complexes (green ridge), normal PR interval (blue rectangle and light turquoise arrows), mimic delta waves (small lime arrows), a small single wave between P wave and QRS (light blue arrows), and small R in V4-6 leads (brown arrows).

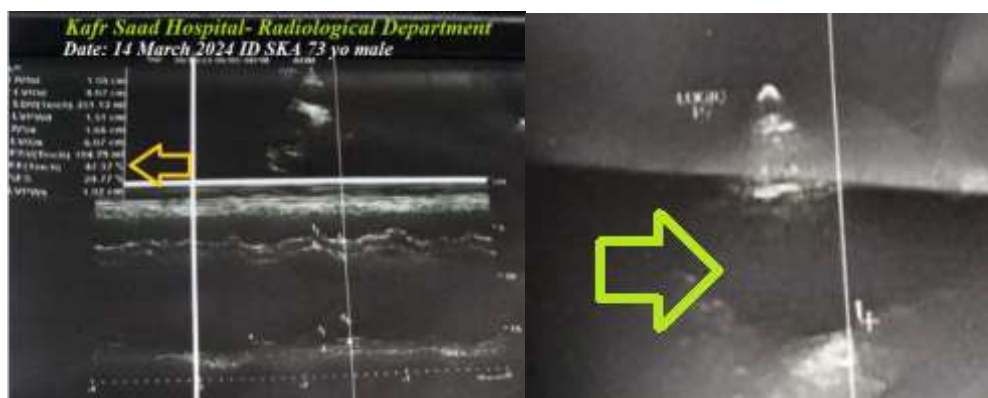


Figure 4: A. The last echocardiography was done on the last ICU admission on 14 March 2024, showing LV dilatation (lime arrow) and LV systolic dysfunction with EF 47% (golden arrow).

3. Discussion and "Yasser's squaring saddling syndrome", "partial modified Yasser's WPW syndrome", and "Yasser's lateral halo sign"

• **Overview:** Elderly married male, a Farmer, a non-smoker, Egyptian, patient, was presented to the POC after over 5 years of follow-up, after two attacks of COVID-19 pneumonia and neglected and passed myocardial infarction, post-prolonged exposure history of inhalation of organophosphorus.

• **The primary objective** of my case study was the presence of an elderly married male, a Farmer, non-smoker Egyptian patient, presented with COVID-19 pneumonia, neglected and passed myocardial infarction, and dilated cardiomyopathy in POC.

• **The secondary objective** for my case study was the **question** of: how id you manage the case?

• An episode of severe acute angina with profuse sweating one month before both attacks of COVID-19 pneumonia and later evidence of pathological Q waves in the inferior leads suggestive of inferior myocardial infarction.

• On applying the "Naranjo Algorithm-Adverse Drug Reaction (ADR) Probability Scale", the prolonged exposure history of inhalation of organophosphorus is more definite as a cause for this infarction (**Table 1**).

• Both attacks of COVID-19 pneumonia on the 2020th year were evidenced in the admission history in the ICU and radiological workup (**Figures 1A-D**).

• Radiological cardiomegaly, global hypokinesia, LV systolic dysfunction, dilated cardiac chambers, and multiple valvular disorders will strengthen the diagnosis of dilated cardiomyopathy.

• There are sporadic and randomly beats of square-shaped ST-segment elevations in aVR and V1 leads of various serial ECG tracings (**Figures 3A-G**). The restriction of square-shaped ST-segment elevation to aVR and V1 leads is interesting. There is no clear or interpreted pathogenesis. But both depolarization and repolarization changes may be implicated.

• There are also sporadic and randomly beating saddling-shaped ST-segment elevations in aVL, V2, and V3 leads of various serial ECG tracings (**Figures 3C, 3F, and 3G**). The restriction of square-shaped ST-segment elevation to aVL, V2, and V3 leads is interesting. There is no clear or interpreted pathogenesis. But also, both depolarization and repolarization changes may be implicated.

The combination of squared-shaped ST-segment elevations in aVR and V1 leads (**Figures 3A-G**) and saddling-shaped ST-segment elevations in aVL, V2, and V3 leads (**Figures 3C, 3F, and 3G**) is new described and named as, "Yasser's Squaring Saddling Syndrome" (**Figures 5**).

• There is also a sporadic and randomly wide QRS complex, normal PR interval, mimicking delta waves, and a small single wave between the P wave and QRS complex. This small single wave between P-waves is smaller than the previous P wave and located between the later QRS complex and the preceding P wave (**Figures 3A, 3D, 3E, 3F, and 3G**).

• The preceding PR interval for this included a wide QRS complex is normal in duration.

• This combination of sporadic mimicking delta waves with a wide QRS complex, normal PR interval, and a small single wave between the P wave and QRS complex is newly described and named as "**Partial Modified Yasser's WPW Syndrome**" (**Figures 5**).

• There is a fixed bifasicular heart block in nearly all ECG tracings except tracing 3E and 3F (**Figures 3E, 3F**), showing prolongation of the PR interval, which indicates of transient trifasicular heart block.

• The presence of the right and the left lateral Halo sign is newly described, other than the Halo sign and Reversed Halo sign [16-18], and named as "**Yasser's Lateral Halo Sign**" (**Figures 5**). The author thinks that the necrosis in a small COVID-19 lesion or nodule toward the lateral side may be an interpretative theory.

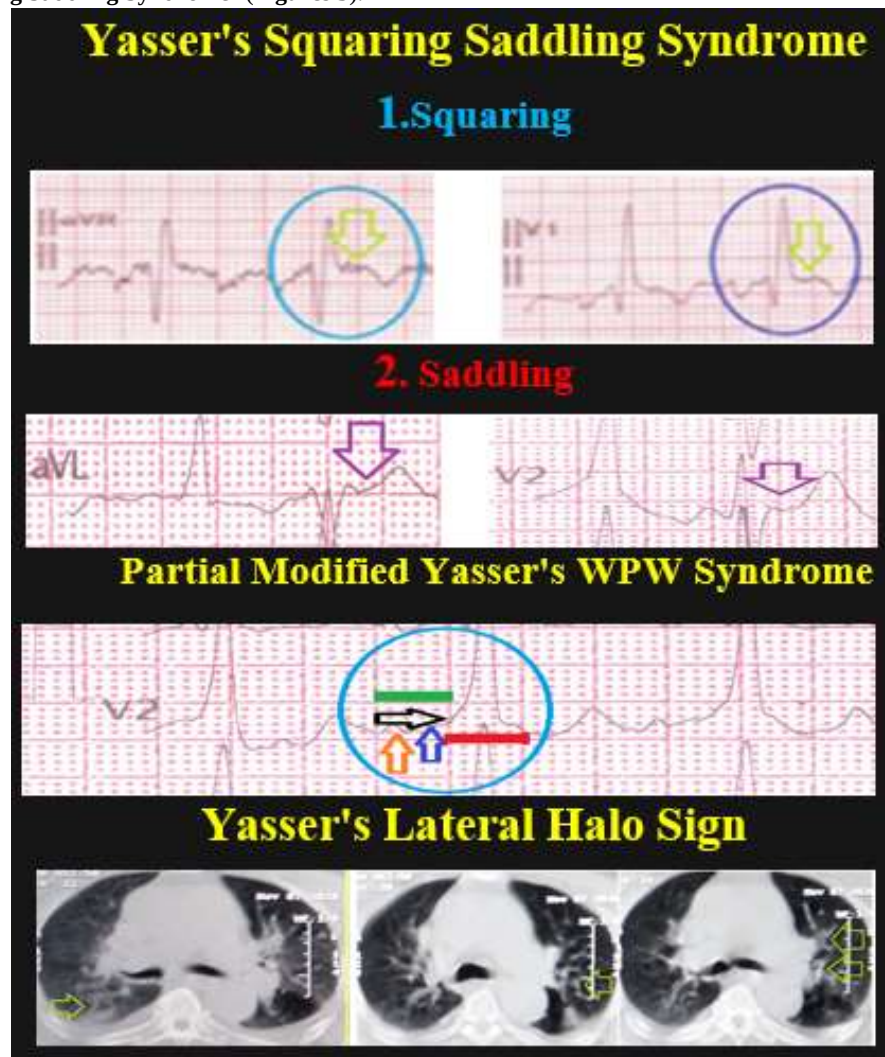


Figure 5: Graphical presentation of "Yasser's squaring saddling syndrome", "partial modified Yasser's WPW syndrome", and "Yasser's lateral halo sign".

• Occurrence of angina as an indication of acute myocardial infarction after inhalation of organophosphorus suggested its definite possible causation. Naranjo's probability scale [19] in the current case study was +11. This means that there was a definite relationship between these adverse drug effects and organophosphorus inhalation (Table 1). Kounis-Zafra syndrome type III is probably implicated in pathogenesis [20].

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

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 readministered?	+2	-1	0	+2
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	+1
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: +11				

• This patient had a strong voice character, stiffness, but mostly smiley on a talk.

• The presence of senility, old myocardial infarction, two attacks of COVID-19 pneumonia, fixed bifasicular heart block, transient trifasicular heart block, dilated cardiomyopathy, bigeminal PVCs, multiple irregular PVCs, premature ventricular couplets, runs of VT, and LV systolic dysfunction with global hypokinesia are considered risk factors.

• The most **differential diagnoses** for the serial ECG are Brugada syndrome, WPW syndrome, and acute myocardial infarction. But all ECG criteria are against them.

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

• The only limitation of the current study was the unavailability of an electrophysiological study and coronary angiography.

4. Conclusion and Recommendations

• "Yasser's squaring saddling syndrome", "partial modified Yasser's WPW syndrome", and "Yasser's lateral halo sign" are new cardiovascular and radiological discoveries.

• Senility, chronic exposure to organophosphates, old myocardial infarction, two attacks of COVID-19 pneumonia, fixed bifasicular heart block, transient trifasicular heart block, dilated cardiomyopathy, bigeminal premature ventricular complexes (PVCs), multiple irregular PVCs, premature ventricular couplets, runs of ventricular tachycardia (VT), and left ventricular (LV) systolic dysfunction with global hypokinesia are serious constellation risk factors.

• Further wide studies will be recommended.

Conflicts of interest

• There are no conflicts of interest.

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• I wish to thank my wife for saving time and improving the conditions for helping me.

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