MATHEWS

Case Report

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Ketoprofen-inducing Sympathetic Crashing Acute Pulmonary Edema (SCAPE), Yasser's AF Variation, Angina, and Kounis-Zafras Syndrome-O2, NTG, and Furosemide Bypassing CPAP/BiPAP and Passing Death

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Background: Drug-induced acute diseases are a very common clinical entity. Clinical cardiovascular medicine is often involved. Ketoprofen is a propionic acid subclass of nonsteroidal anti-inflammatory drugs (NSAIDs) that has antipyretic and analgesic actions. Several cardiovascular and non-cardiovascular causes have been implicated in inducing atrial fibrillation (AF). O2, NTG, and Furosemide are essential cardiorespiratory therapies. Mast cell activation disorders are a keystone for understanding allergic angina with acute coronary syndromes (ACS), which are known as Kounis-Zafras (KZ) syndrome. Nitroglycerine (NTG) and CPAP/BiPAP have a role in the management of SCAPE, serious acute pulmonary edema.

Case Presentation: A middle-aged Egyptian wood polisher patient was admitted to the intensive care unit (ICU) with sympathetic-crashing acute pulmonary edema (SCAPE), angina, and atrial fibrillation after ingestion of a ketoprofen tablet, with implicated Kounis-Zafras syndrome. Electrocardiography, oxygenation, IV nitroglycerine infusion, IV furosemide injection, CXR-PA viewing, and echocardiography were the interventions. A dramatic clinical and electrocardiographic improvement occurred.

Conclusion: Sympathetic-crashing acute pulmonary edema (SCAPE), Yasser's AF variation, angina, and Kounis-Zafras syndrome are probable adverse effects of ketoprofen. Yasser's AF variation is a new phenomenon described as a zigzag line up and down in AF that involves the amplitude and depth of QRS complexes, affecting some leads of ECG and cardiovascular medicine. The disappearance of Yasser's AF variation after management may support the efficacy of oxygenation, IV nitroglycerine infusion, and IV furosemide injection. Kounis-Zafras type II syndrome may be implicated as a theory in the interpretation of ketoprofen-induced severe acute pulmonary edema, angina, and atrial fibrillation. Dramatic clinical and electrocardiographic improvement after early management with oxygenation, IV nitroglycerine infusion, and IV furosemide injection plays a role in hastening recovery from serious acute pulmonary edema without the need for CPAP/BiPAP. It may be attributed to their efficacy in the treatment of fatal SCAPE.

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Keywords: Kounis-Zafras syndrome, Allergic Angina, Ketoprofen, Sympathetic Crashing Acute Pulmonary Edema, SCAPE, Yasser's AF Variation, Oxygen, Nitroglycerine, Furosemide, CPAP/BiPAP, Passing Death

ABBREVIATIONS

ACS: Acute Coronary Syndrome

AF: Atrial Fibrillation

AMI: Acute Myocardial Infarction

BiPAP: Bilevel Positive Airway Pressure

CAS: Coronary Artery Spasm

CBC: Complete Blood Count

CPAP: Continuous Positive Airway Pressure

DIAF: Drug-induced AF

ECG: Electrocardiography

ICU: Intensive Care Unit

IHD: Ischemic Heart Disease

KZ syndrome: Kounis-Zafras syndrome

LAD: Left Axis Deviation

NSR: Normal Sinus Rhythm

MI: Myocardial Infarction

NSAIDs: Nonsteroidal Anti-inflammatory Drugs

NTG: Nitroglycerine

O2: Oxygen

SCAPE: Sympathetic Crashing Acute Pulmonary Edema

VR: Ventricular Rate

INTRODUCTION

Ketoprofen is a non-selective COX inhibitor and a highly potent propionic acid derivative among nonsteroidal anti-inflammatory drugs (NSAIDs), with antipyretic and analgesic actions. It was synthesized by French Rhône-Poulenc chemists in 1967. Ketoprofen has a short half-life, simple metabolism, wide therapeutic window, a rapid onset of action, flexible dosing, and a reliable tolerance [1]. Its oral efficacy in relieving moderate-to-severe pain was significantly better than that of ibuprofen and/or diclofenac [2] or equivalent to ibuprofen and/or diclofenac [3]. The most common side effects of ketoprofen are nausea and vomiting. Myocardial infarction, stroke, hypertension, heart failure, GIT bleeding, kidney damage, hepatotoxicity, and serious skin reactions such as exfoliative dermatitis, Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN) are considered serious side effects of ketoprofen [4]. Acute pulmonary edema due to sympathetic surge and increased peripheral vascular resistance (PVR) is a critical disorder with marked hypertension, severe dyspnea, and hypoxia. It is defined as sympathetic crashing acute pulmonary edema (SCAPE). SCAPE patients clinically usually present with severe acute respiratory distress syndrome (ARDS) [5]. It is a variant of hypertensive cardiogenic pulmonary edema (PE) and indicates diagnoses and management as early as possible [6]. SCAPE presents with rapid onset, hypertensive crises, sweating, restlessness, severe tachypnea, severe dyspnea, and marked hypoxemia. Diffuse rales are noted on auscultation with a pink, frothy sputum. There is associated sympathetic overactivation: diaphoresis, pallor, severe unwellness, tachycardia, and agitation. A SCAPE may recur [5,7]. SCAPE is a hyperacute sequence of congestive heart failure (CHF) due to an accumulation of fluid in the lung secondary to a sudden increase in hydrostatic pressure causing fluid extravasation from the pulmonary circulation into the interstitium [8]. ACE inhibitors, angiotensin receptor blockers, clonidine, and sympathomimetic intoxication are known acute triggers of SCAPE. SCAPE patients are successfully and rapidly treated with high-dose nitroglycerin (NTG) [5]. High doses of NTG are well tolerated in these patients [5]. Nitrate tolerance may be a result of over-physiological and over-pharmacological NTG doses [9]. Both high-dose diuretics and high-dose IV nitroglycerin are strongly indicated [6]. Atrial fibrillation (AF) is the most common sustained arrhythmia that globally affects millions of people [10]. AF is associated with an increased risk of heart failure, stroke, dementia, and burden on healthcare systems worldwide [11]. Advanced age, alcohol consumption, family history of AF, hypertension, thyroid dysfunction, obstructive sleep apnea, structural heart diseases, and gut microbiota dysbiosis are considered risk factors for AF [12]. Several cardiovascular and non-cardiovascular causes have been implicated as drug-induced AF (DIAF). DIAF includes dobutamine, milrinone, adenosine, anthracycline agents, ibrutinib, trastuzumab, ivabradine, intracoronary acetylcholine, immune checkpoint inhibitors, chimeric antigen receptor T-cell (CAR-T) therapy, diuretics, nicorandil, and acute alcohol consumption [13]. Kounis-Zafras (KZ) syndrome is an extensive mast cell stimulation disorders that are consociated with acute coronary syndromes (ACS). The syndrome was first identified by Kounis and Zavras in 1991 as an "allergic angina syndrome", "allergic angina" or "allergic myocardial infarction" [14-16]. The essential pathogenesis of KZ syndrome includes the

inflammatory cytokine 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 [16]. Allergic angina commonly starts within one hour of exposure to the offending allergen. Prolonged-onset KZ syndrome also has been reported [17]. Variant presentations of the syndrome have been reported [16]. Three different variants of this syndrome have been described: Type I occurs in structurally normal coronary arteries with no cardiovascular risk factors. The coronary spasm was suggested with or without associated acute myocardial infarction (AMI). Type II 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 occurs in patients with coronary artery stent-associated thrombosis [15,17-19].

In this manuscript, I reported a case of a middle-aged Egyptian wood polisher, a heavy smoker, who was presented to the intensive care unit with sympathetic-crashing acute pulmonary edema (SCAPE), angina, and atrial fibrillation after ingestion of a ketoprofen tablet, with implicated Kounis-Zafras syndrome. So, how would you manage this case?

CASE PRESENTATION

A 52-year-old married male wood polisher Egyptian patient was admitted to the intensive care unit (ICU) with dyspnea, tachypnea, angina, palpitations, and profuse sweating. This occurred within 6 hours after ingestion of a single ketoprofen tablet (100mg) for dental pain. Profuse sweating was an associated symptom. There was no history of the same attacks. He is a heavy cigarette smoker (30 cigarettes daily for about 15 years). There is no history of cardiovascular diseases. Informed consent was obtained. Upon general physical examination, the patient appeared well-built and had profuse sweating, showed central cyanosis, and had dyspnea, orthopnea, and tachypnea with an irregular pulse rate (AF with VR of 170), blood pressure (BP) of 270/150 mmHg, respiratory rate of 50 bpm, a temperature of 36°C, and pulse oximeter of oxygen (O2) saturation of 75%. No more relevant clinical data were noted during the clinical examination. The patient was admitted to the ICU with acute pulmonary edema, angina, and rapid AF. The patient was urgently treated in the ICU with high-flow O2 inhalation via an O2 inhalation central system (100% by simple mask, 10L/min), IV furosemide injection (40mg, 3 amp, IVB then maintained at 1 amp IV, 40mg, QID), and IV nitroglycerin infusion (10 mg/50 ml solvent, 10 ug/min and titrated according to BP). Aspirin: 4 oral tablets (75 mg, then OD), clopidogrel: 4 oral tablets (75 mg, then OD), enoxaparin SC (80 mg, BID), and atorvastatin tablets (40 mg, OD) were added after BP control. After controlling the BP and within 48 hours of stablization, SC enoxaparin 80 mg, BID), aspirin tablet (75 mg, OD), clopidogrel tablets (75 mg, OD), captopril tablets (25 mg; BID), and diltiazem tablets (60 mg, OD), warfarin tablet (5 mg, OD), amiodarone tablets (200 mg; BID), furosemide tablets (40 mg fasting, OD), spironolactone tablets (25 mg, fasting, OD) and atorvastatin tablets (20 mg, OD) were added. The patient was monitored hourly for vital signs and O2 saturation. The initial ECG was performed on presentation after ICU admission, showing rapid AF, normal axis deviation, evidence of old inferior and septal MI, wavy triple sign (Yasser's sign), wavy double sign (Yasser's sign), and T-wave inversion (TWI) in the V6 lead. There are variations in the amplitude of the QRS complexes in lead II and in the amplitude and depth of the complexes in lead III (Figure 1A). The second ECG tracing was taken within 43 seconds of the above tracing, showing rapid AF, normal axis deviation, wavy triple sign, wavy double sign, and T wave inversion in the V6 lead. There are variations in the amplitude and depth in the lead III, aVL, and V1 leads (Figure 1B). The third ECG tracing was taken within 11.5 hours of the above tracing, showing atrial flutter with variable block, physiological left axis deviation, and T-wave inversion in I and aVL leads, and ST-segment depression in V4-6 leads (Figure 1C). The fourth ECG tracing was taken within 2 minutes of the above tracing, showing multiple atrial nodal rhythms, physiological left axis deviation, TWI in I and aVL leads, and ST-segment depression in V4-6 leads (Figure 1D). The fifth ECG tracing was taken within 68 seconds of the above tracing, showing atrial flutter with variable block, evidence of U waves in V2 and V3 leads, physiological left axis deviation, TWI in I and aVL leads, and ST-segment depression in V4-6 leads (Figure 1E). The sixth ECG tracing was taken within 29 seconds of the above tracing, showing atrial flutter with variable block, physiological left axis deviation, TWI in I and aVL leads, and ST-segment depression in V4-6 leads (Figure 1F). The seventh ECG tracing was taken within 29 seconds of the above tracing, showing NSR, physiological left axis deviation, and evidence of U waves in V1-V3 leads (Figure 1G). The initial complete blood count (CBC); Hb (17.6g/dl), RBCs (5.38*103/mm3), Hematocrit (47.6%), WBCs (14.1*103/mm3); (Neutrophils; 44.2 %, Lymphocytes: 47.2%, Monocytes; 8.6%, Eosinophils; 0% and Basophils 0%), Platelets; 188*103/mm3. SGPT was 59 U/L,

and SGOT was 80 U/L. S albumen (4.2 gm). Serum creatinine was (1.2mg/dl). RBS was (88 mg/dl). D-dimer was (8.01 mg/dl). Troponin I was 0.09 ng/ml. TSH was (1.15ulU/ml), free T3 was (3.80 pmol/L), and free T4 was (1.36 pmol/L). RBS was (94mg/dl). Plasma sodium was (143.2mmol/L). Serum potassium was (4.14mmol/L). Ionized calcium was (1.09mmol/L). Virology screening for HBV, HCV, and HIV infection was negative. A plain PA CXR was obtained within 7 days of ICU presentation and after clinical stabilization, showing mild cardiomegaly and a healing right perihilar lung shadow (Figure 2). The current echocardiography is obtained within 7 days of ICU presentation and after clinical stabilization, showing moderate to severe LV systolic dysfunction, global hypokinesia, LVH, diastolic dysfunction, mild mitral regurgitation, mild PR, mild TR, and mild pulmonary hypertension, and AF (Figure 3). Ketoprofen-induced sympathetic crashing acute pulmonary edema (SCAPE), Yasser's AF variation, angina, and Kounis-Zafras syndrome were the most probable diagnoses. Nearly complete recovery was achieved within 12 hours. The patient was discharged within 7 days of the above management after happening of dramatic clinical and ECG improvement. Aspirin tablet (75 mg, OD), clopidogrel tablets (75 mg, OD), captopril tablets (25 mg; BID), and diltiazem tablets (60 mg, OD), warfarin tablet (5 mg, OD), amiodarone tablets (200 mg; OD), furosemide tablets (40 mg fasting, OD), spironolactone tablets (25 mg, fasting, OD) and atorvastatin tablets (20 mg, OD) for 30 days were prescribed on discharge with the recommendation for future cardiac and immunological follow-up.

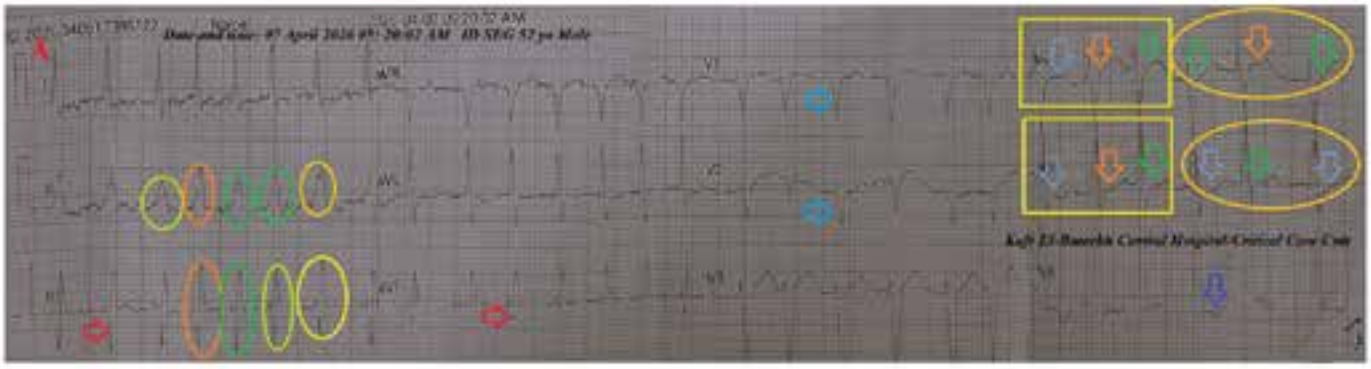

Figure 1A: Serial ECG tracings; A-tracing was done on the initial ECG on presentation after ICU admission showing rapid AF (of VR 165) normal axis deviation, evidence of old inferior (red arrows) and septal MI (light blue arrows), wavy triple sign (Yasser's sign) in lead V4 (gray, golden, and green arrows), wavy double sign (Yasser's sign) in V4 and V5 leads (gray, golden, and green arrows), and T wave inversion in V6 lead (dark blue arrow). There are variations in the amplitude of the QRS complexes in lead II (colored circles) and in the amplitude and depth in lead III (multiple colored circles).

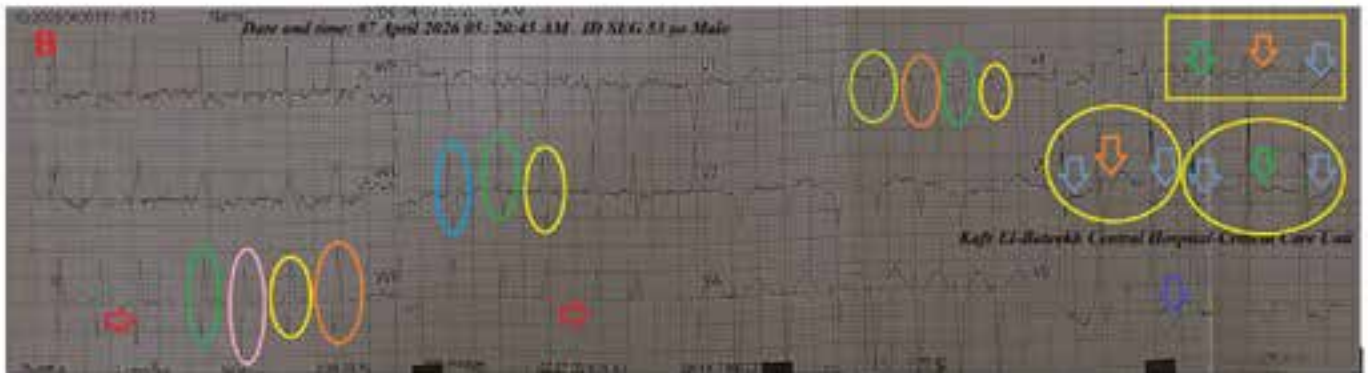

Figure 1B: B-tracing was done on within 43 seconds of the above tracing showing rapid AF (of VR 165) normal axis deviation, wavy triple sign (Yasser's sign) in lead V4 (gray, golden, and green arrows), wavy double sign (Yasser's sign) in V5 lead (gray, golden, and green arrows), and T wave inversion in V6 lead (dark blue arrow). There are variations in the amplitude and depth in the lead III, aVL, and V1 leads (multiple colored circles).

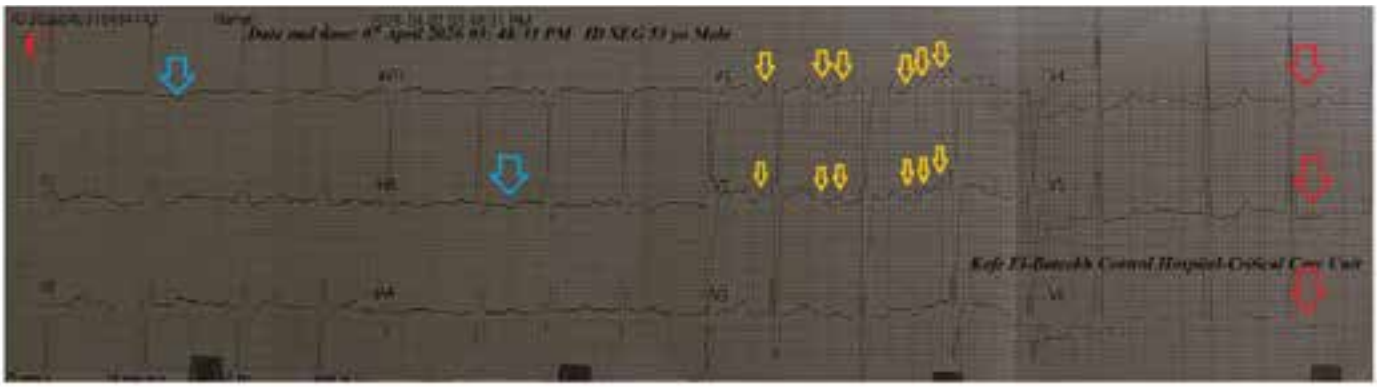

Figure 1C: C-tracing was done within 11.5 hours of the above tracing, showing atrial flutter with variable block (of VR 97, multiple small yellow arrows in V1 and V1 leads), physiological left axis deviation, T wave inversion in I and aVL leads (light blue arrow), and ST-segment depression in V4-6 leads (red arrows).

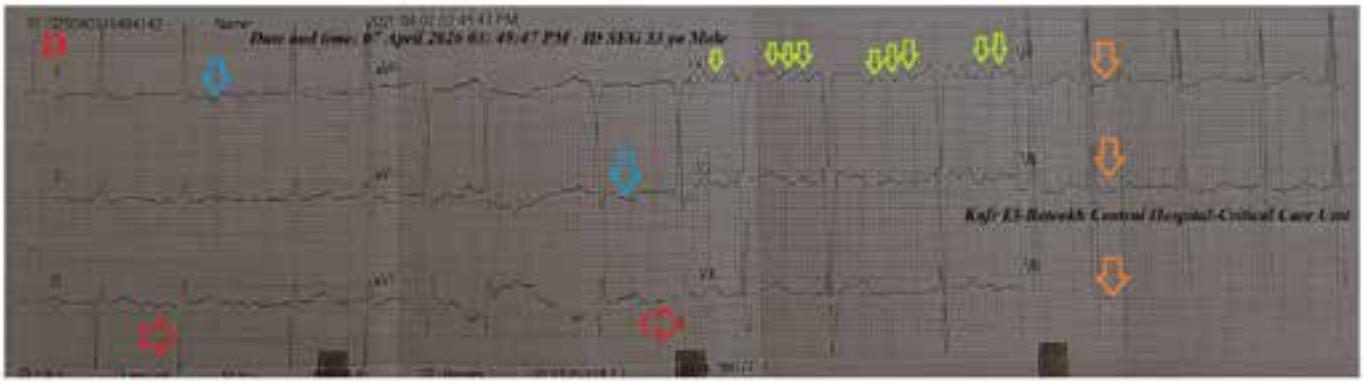

Figure 1D: D-tracing was done within 2 minutes of the above tracing, showing atrial flutter with variable block (of VR 93, multiple small lime arrows in V1 and V1 leads), physiological left axis deviation, T wave inversion in I and aVL leads (light blue arrow), and ST-segment depression in V4-6 leads (golden arrows).

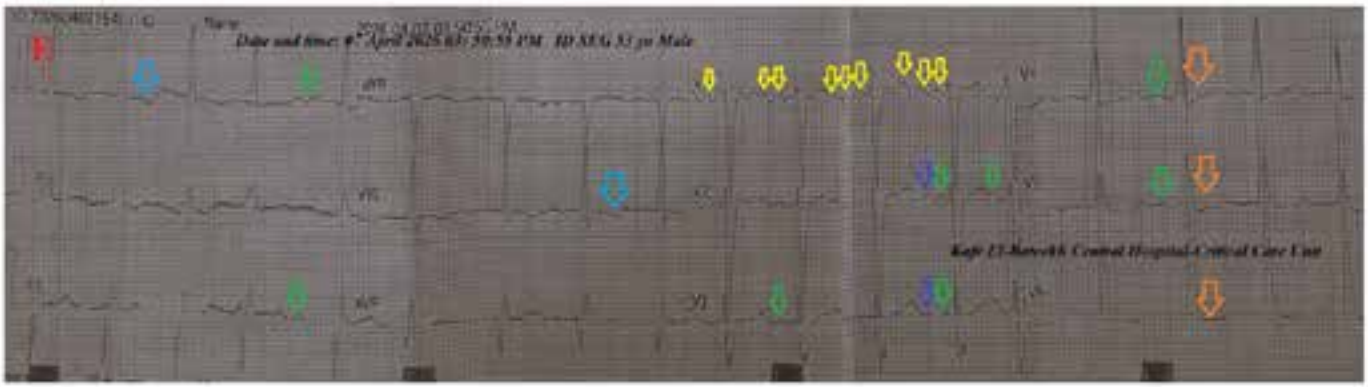

Figure 1E: E-tracing was done within 68 seconds of the above tracing, showing atrial flutter with variable block (of VR 104, multiple small yellow arrows in V1 and V1 leads), P waves (green arrows), evidence of U waves in V2 and V3 leads (dark blue arrows), physiological left axis deviation, T wave inversion in I and aVL leads (light blue arrow), and ST-segment depression in V4-6 leads (golden arrows).

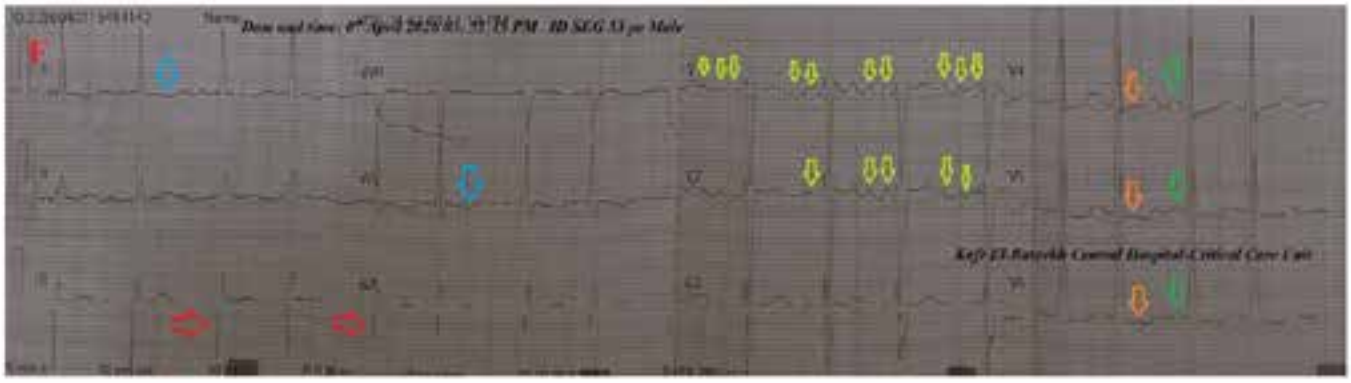

Figure 1F: F-tracing was done within 29 seconds of the above tracing, showing atrial flutter with variable block (of VR 104, multiple small lime arrows in V1 and V1 leads), P waves (green arrows), physiological left axis deviation, T wave inversion in I and aVL leads (light blue arrow), and ST-segment depression in V4-6 leads (golden arrows).

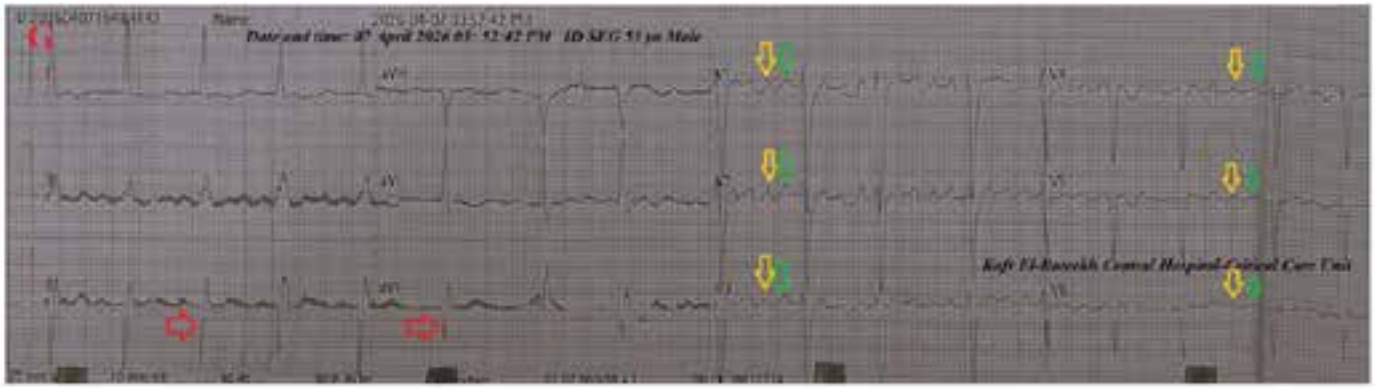

Figure 1G: G-tracing was done within 29 seconds of the above tracing, showing NSR

(of VR 98, green arrows), physiological left axis deviation, and evidence of U waves in V1-V3 leads (yellow arrows).

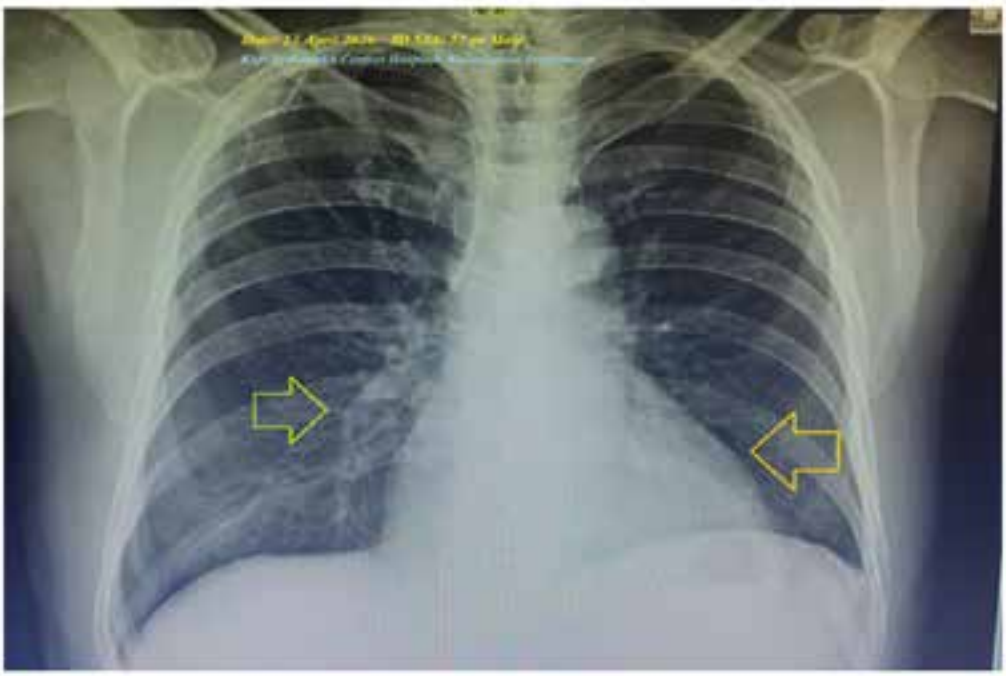

Figure 2: A plain PA CXR was obtained within 7 days of ICU presentation and after clinical stabilization, showing mild cardiomegaly (golden arrow) and a healing right perihilar lung shadow (lime arrow).

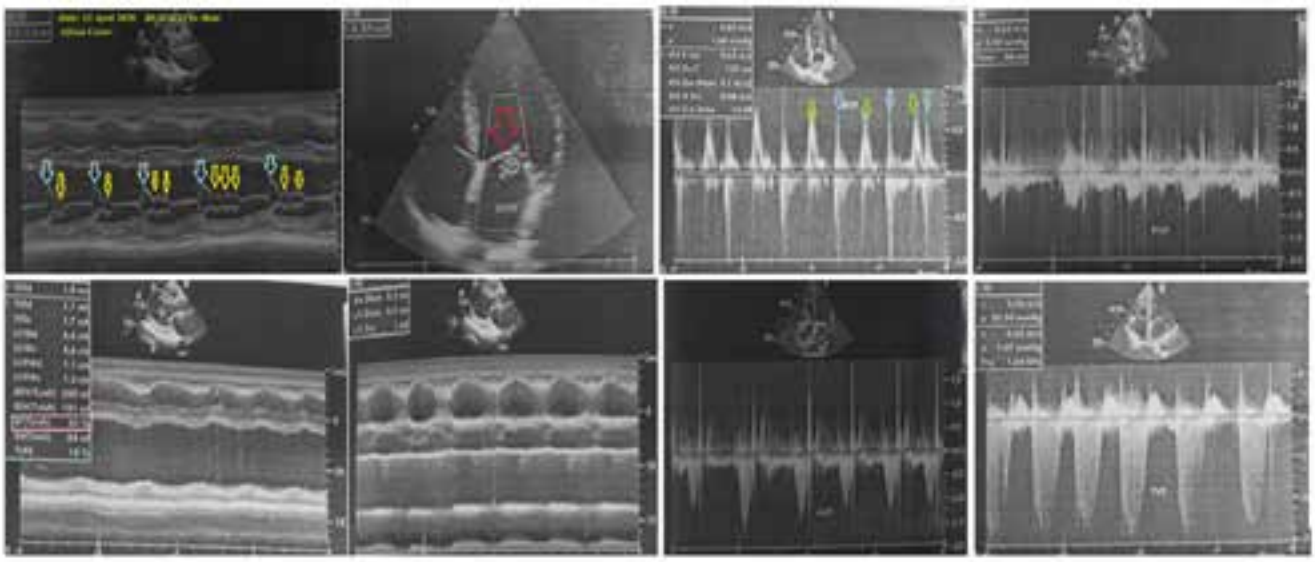

Figure 3: Echocardiography is obtained within 7 days of ICU presentation and after clinical stabilization, showing moderate to severe LV systolic dysfunction, global hypokinesia, LVH, diastolic dysfunction (light blue and lime arrows), mild mitral regurgitation, mild PR, mild TR, and mild pulmonary hypertension, and AF (light blue and lime arrows).

DISCUSSION

Overview

- A middle-aged Egyptian wood polisher, a heavy smoker, who was presented to the intensive care unit with sympathetic-crashing acute pulmonary edema (SCAPE), angina, and atrial fibrillation after ingestion of a ketoprofen tablet, with implicated Kounis-Zafras syndrome.
- **The primary objective** of my case study was the presence of a middle-aged Egyptian wood polisher patient who was presented to the intensive care unit with sympathetic-crashing acute pulmonary edema (SCAPE), angina, and atrial fibrillation after ingestion of a ketoprofen tablet, with implicated Kounis-Zafras syndrome.
- **The secondary objective** for my case study was the **question** of how you would manage the case.
- The presence of rapid-onset acute pulmonary edema with hypertensive crises, severe dyspnea, severe tachypnea, restlessness, profuse sweating, and severe hypoxia strengthens the diagnosis of sympathetic crashing acute pulmonary edema (SCAPE).
- Occurrence of angina after ingestion of a ketoprofen tablet indicates its possible causation. Naranjo's probability scale in the current case study was +7. This means that there was a **Probable** relationship between these adverse drug effects and ketoprofen ingestion **(Table 1)**. Kounis-Zafras syndrome will probably be implicated in the pathogenesis.

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	0
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	0
5. Are there alternative causes that could on their own have caused the reaction?	-1	+2	0	+2

Question	Yes	No	Do Not Know	Score
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	0
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	+1
	Total Score: +7			

- The initial and serial ECG tracings show inferior pathological Q waves with negative troponin, indicating the presence of an old silent or passing inferior MI.
- The current angina after oral ingestion of ketoprofen with pre-existing evidence of IHD or inferior M, indicates the presence of type II K-S syndrome. The coronary artery spasm was the suggested interpretation [15,17-19].
- Within 12 hours of forced management, there was a conversion from rapid AF (Figures 1A and 1B) to controlled atrial flutter with variable block (Figures 1C, 1D, 1E, and 1F).
- A heavy cigarette smoker, old MI, rapid AF, and HTN crises are strong risk factors for acute pulmonary edema.
- The dramatic clinical improvement and ECG normalization (Figure 1G) after oxygenation, IV nitroglycerine infusion, and IV furosemide injection without using CPAP/BiPAP may be attributed to their efficacy in the treatment of a fatal SCAPE.
- The author noticed that there are variations in the amplitude and depth of QRS complexes in some ECG leads. These up-and-down variations in QRS complexes in the initial ECG tracings (Figures 1A and 1B) take a zigzag line in description. There are variations disappear in the next serial ECG tracings (Figures 1C, 1D, 1E, and 1F) and after management with oxygenation, IV nitroglycerine infusion, and IV furosemide injection. This phenomenon with appearance and disappearance may be described as a "Yasser's AF variation phenomenon". The disappearance of Yasser's AF variation after management may support the efficacy of oxygenation, IV nitroglycerine infusion, and IV furosemide injection. Indeed, there is no known pathogenesis for this new phenomenon. The variation in myocardial contractility and subsequent stroke volume and cardiac output during rapid AF may be a suggested interpretation (Figure 4).

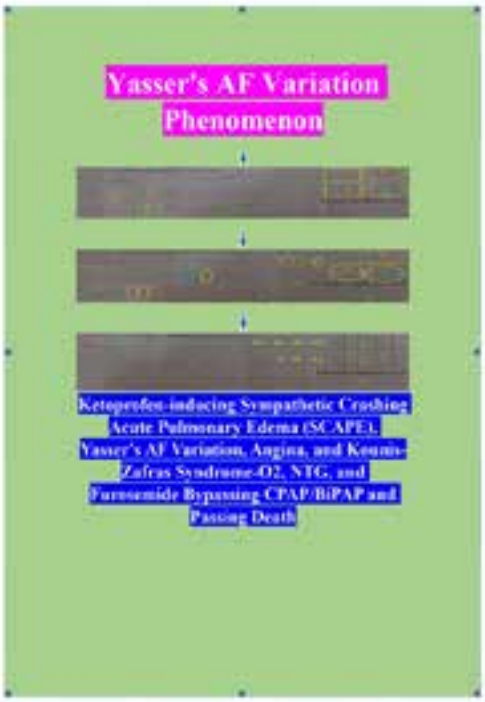

Figure 4: Graphical representation of Yasser's AF variation

- Multiple atrial tachycardia and multifocal atrial rhythm are the most implicated in ECG differential diagnosis. The ECG is against them.
- I can't **compare** the current case with similar conditions. There are no identical or known cases with the same management for a near comparison.
- The strength points in this case report are the triple therapy, oxygenation, IV nitroglycerine infusion, and IV furosemide injection, which are hastening recovery and preventing fatality from serious acute pulmonary edema. Also, the above triple therapy can be used without the need for CPAP/BiPAP. Moreover, the newly described **"Yasser's AF variation".**
- The only limitation of the current study was the unavailability of coronary angiography and CTPA due to the poor financial status of the patient.

CONCLUSION AND RECOMMENDATIONS

Sympathetic-crashing acute pulmonary edema (SCAPE), Yasser's AF variation, angina, and Kounis-Zafras syndrome are probable adverse effects of ketoprofen.

• Yasser's AF variation is a new phenomenon described as a zigzag line up and down in AF that involves the amplitude and depth of QRS complexes, affecting some leads of ECG. The disappearance of Yasser's AF variation after management may support the efficacy of oxygenation, IV nitroglycerine infusion, and IV furosemide injection.

• Kounis-Zafras type II syndrome may be implicated as a theory in the interpretation of ketoprofen-induced severe acute pulmonary edema, angina, and atrial fibrillation.

• Dramatic clinical and electrocardiographic improvement after early management with oxygenation, IV nitroglycerine infusion, and IV furosemide injection plays a role in hastening recovery from serious acute pulmonary edema without the need for CPAP/BiPAP. It may be attributed to their efficacy in the treatment of fatal SCAPE.

STATEMENTS

Conflicts of interest

There are no conflicts of interest.

Acknowledgment

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

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