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Oxygen Reversal of Coronary Artery Spasm with Modification of International Standards for the Diagnostic Criteria of Coronary Vasomotor Disorders (Yasser's Modification or Oxygen test)

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Abstract

Aim of the study: the study aims to clear the initial effect of non-baric oxygen inhalation on the coronary artery spasm.

Background: Coronary artery spasm (CAS) is a cardiovascular disorder that plays an important role in the pathogenesis of stable angina, unstable angina, myocardial infarction, and sudden cardiac death. Nitrate: calcium channel blockers, and statins are known established medications in the reversal of coronary artery spasms. Oxygen safety versus adverse effects of nitrate, calcium channel blockers, and statins are comparable.

Method of study and patients: My case study was an observational-retrospective seventeen case report series. The study was conducted in Fraskour Central Hospital, Kafr El-Bateekh Central Hospital, and physician outpatient. The author reported the seventeen cases of acute angina with rest chest pain over about 38-months; starting on December 15, 2018, ended on February 7, 2022.

Results: The mean age is 43.2 with the female sex predominance (64.71%). Housewife (29.41%) and students (23.53%) are the most affected occupations. The main complaint is chest pain (64.71%). The most common associated risk factors are female sex (64.71%) and stress (23.53%). Drug-induced (23.53%); hyperventilation syndrome-induced (23.53%); and CO toxicity-induced coronary artery spasm (17.65%) are common diagnoses. The dose of inhaled O_2 dose that achieved the reversal of CAS varied from 5 to 12 liter. A maximal dose (12 minutes) was given for CO toxicity. The duration of inhaled O_2 dose that achieved the reversal CAS varied from 15 to 80 minutes. Maximal duration (80 minutes) was given in CO toxicity. The complete response had happened in 94.12%.

Conclusion: Dramatic clinical reliving and reversal response of electrocardiographic ST-segment depression after oxygen inhalation is an indication for its initial use in coronary artery spasm. Yasser's Modification or Oxygen test for the past "international standards for the diagnostic criteria of coronary vasomotor disorders" improves patient safety and decreases the hazards of nitrate and other medications.

Keywords: Coronary artery spasm; Ischemic heart disease; Oxygen; International standards for the diagnostic criteria of coronary vasomotor disorders; Yasser's modification; Oxygen test

Abbreviations: ABG: Arterial Blood Gases; AMI: Acute Myocardial Infarction; BP: Blood Pressure; CAS: Coronary Artery Spasm; CBC: Complete Blood Count; Ccbs: Ca^{2+} Channel Blockers; CO: Carbon Monoxide; ECA: Epicardial Coronary Artery; ECG: Electrocardiography; ED: Emergency Department; EF: Ejection Fraction; GCS: Glasgow Coma Scale; Hbco: Carboxyhemoglobin; HR: Heart Rate; ICU: Intensive Care Unit; IHD: Ischemic Heart Disease; LOC: Loss of Consciousness; MI: Myocardial Infarctions; NO: Nitric Oxide; NSR: Normal Sinus Rhythm; O_2: Oxygen; PG: Prostaglandin; POC: Physician Outpatient Clinic; PVC_S: Premature Ventricular Contractions; RBBB; Right Bundle Branch Block; RBS: Random Blood Sugar; RR: Respiratory Rate; SCD: Sudden Cardiac Death; STEMI: ST-Elevation Myocardial Infarction; TXA2: Thromboxane A2; UA: Unstable Angina; VD: Vasodilatation; VR: Ventricular Rate

Introduction

The coronary artery spasm (CAS) is a cardiovascular disorder a description of a prompt, severe narrowing of an epicardial coronary artery (ECA) that ends with vessel obstruction or near-obstruction [1]. This abnormal contraction of the ECA is an inducer for myocardial ischemia [2]. CAS has a pivotal role in the pathogenesis of ischemic heart disease (IHD), including stable

angina (SA), unstable angina (UA), myocardial infarction (MI), and sudden cardiac death (SCD) [3]. However, malignant ventricular arrhythmias, acute MI, and SCD are the main remarkable results of obstructive CAS [3]. A spasm is either partial or complete blockage of the blood flow to part of the heart [4]. There is an associated transitory contraction of the muscular wall of one of the arteries causing a decrease or wholly prevention of blood flow4. The exact mechanisms of CAS are still obscure but predominately multifactorial [5]. Coronary artery spasms can be triggered by

a) Tobacco abuse

b) Severe cold

c) non-balance of myocardial oxygen (O_2) supply versus demand [6].

The stress of any type, outrage, and panic are central grounds for the attacks [4,7]. Coronary artery spasms may be "silent" or present with angina, or chest pain. If the CAS persists for a long time, the heart attack cannot be avoided. The main symptom is a type of chest pain called angina. This pain is most often felt under the sternum or left side of the chest. The pain is described as constricting, crushing, pressure, squeezing, and tightness. It is most often severe. The pain may spread to the neck, jaw, shoulder, or arm. The pain of CAS often occurs at rest, it may occur at the same time each day, usually between midnight and 8:00 a.m., lasts from 5 to 30 minutes, and the person may present with a transient loss of consciousness. Unlike angina which is caused by hardening of the coronary arteries, chest pain and dyspnea due to CAS are often not present when you walk or exercise. About 2% of people with angina have CAS [8]. The frequent presentations of IHD due to CAS are chest pain, ECG ST-segment deviations, cold sweat, nausea, vomiting, and syncope in the more intense prolonged event. However, myocardial ischemia due to CAS often occurs without accompanying symptoms [7]. The following abnormalities are the ECG findings during the attack of CAS, ST-segment elevation and/or depression, increase in the amplitude of the T-wave, and negative U-wave at the start or near the end of the attack [9]. CAS is either accompanied by ST-segment depression or ST-segment elevation on electrocardiography (ECG) [10]. It may also occur in angiographically normal coronary arteries as the so-called "variant of the variant10. Coronary angiography is the gold standard for the diagnosis of spastic IHD [11]. Recently, coronary angiography can be done during the anginal attacks with the CAS as the usual cause of variant angina. Coronary angiography also revealed that CAS could occur at the site of stenosis of either mild or severe (focal spasm) or in angiographically normal coronary arteries [1]. However, either mild or critical stenosis CAS at the site of the segment is mostly seen on coronary angiography [1]. The essential diagnosis of CAS mostly involves eliciting the CAS under controlled conditions with ergonovine [12]. The prevention of risk factors is considered the main line of therapy. In nearly 10% to 20% of cases, the spasm is refractory to usual treatment. Moreover, higher doses of calcium channel blockers (CCB) are occasionally essential effective preventive medications for the recurrence of CAS [1]. Treatment of CAS may include medications such as

d) Nitrates are frequently prevent CAS by rapidly relieving chest pain.

e) CCBs relax the arteries and decrease the CAS.

f) Statins are also prophylactic agents CAS [12].

Long-acting nitrates and CCB are the cornerstone drugs of choice (DOC) in preventing future CAS episodes [10]. The sublingual nitrate promptly mitigates the angina attack but long-acting CCBs are very effective for this condition [7].

Patients and Methods

Table 1: Shows remarks on the study method and data.

Issue	Definition
Title	Oxygen reversal of coronary artery spasm with modification of international standards for the diagnostic criteria of coronary vasomotor disorders (Yasser's Modification or Oxygen test)
Estimated Enrollment	17 participants
Study Type	Observational
Observational Model	Case report series
Time	Retrospective
Study Start Date	15-Dec-2018
Estimated Study Completion Date	7-Feb-2022
Analytic method	Comparative using percentage %

The author recorded seventeen cases of acute angina with resting chest pain. The study was conducted in both Fraskour Central Hospital, Kafr El-Bateekh Central Hospital, and the Physician Outpatient Clinic (POC). The author reported seventeen cases of coronary artery spasm over about 38-months, starting on December 15, 2018, ended on February 7, 2022. The study is

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an observational retrospective case report series (Table 1). Most cases were female. There was a different diagnosis for all cases. Eight cases of the current study were previously publicized for the author. All selected cases were associated with significant ECG ST-segment depressions. All cases of ECG ST-segment depressions are immediately exposed to a variable dose of non-baric 100%, oxygen inhalation delivered by nasal cannula. The dose of oxygen inhalation was controlled by the response of either chest pain or symptoms. The ECG tracings before and after oxygenation were reported. Troponin test and echocardiography were requested in all cases. ABG, CBC, liver enzymes, ionized calcium, RBS, and renal function tests were done in selected cases. History, clinical data, O_2 doses, and response or outcome for the cases were recorded. For more details, you can see (Table 2).

Table 2: Summary of the history, clinical, and management data for the study cases.

Case No.	Age	Sex	The Main Complaint	BP mg Hg	Pulse bpm	Occupation	RR	O_2 Sat. %	Associated RF	Final Diagnosis	O_2 dose (l/m)/ Duration/min	Outcome
1	14	M	Syncope	100/70	140	Student	12	89	Closed hot bathroom	CO toxicity	12L/m for 20min	Responsive (R)
2	36	F	Chest pain	110/70	100	House-wife	20	94	Ketoprofen	K Z1 syndrome	5L/m for 20min	Responsive
3	11	F	Chest pain	100/60	144	Student	44	87	Hypothyroidism	CO toxicity	12l/m for 30 min	Responsive
4	11	F	Chest pain	110/80	120	Student	24	90	Closed hot bathroom	CO toxicity	12L/m for 30min	Responsive
5	67	M	Chest pain	110/70	120	Farmer	34	89	Asthmatic	Alcohol toxicity	5L/m for 80min	Responsive
6	45	M	Palpiations	150/90	100	Worker-less	18	97	Smoking -football fan	Enthusiasm CAS	5L/m for 20min	Responsive
7	54	M	Chest pain	150/90	104	Farmer	32	96	Smoking -Stress	Tetany CAS	5L/m for 20min	Responsive
8	65	F	Chest pain	110/70	74	House-wife	16	97	Asthmatic -RBBB	Stress CAS	5L/m for 15min	Responsive
9	70	F	Chest pain	140/80	64	House-wife	19	96	Stress	Indomethacin CAS	5L/m for 20min	Partial R
10	16	F	Chest pain	100/80	130	Student	20	98	Stress	Modafinil	5L/m for 20min	Responsive
11	60	F	Chest pain	100/60	112	House-wife	18	95	Obesity	Salbutamol	5L/m for 20min	Responsive
12	50	F	Tachypnea	100/70	80	Gov. officer	26	97	Stress	HVS CAS	5L/m for 20min	Responsive
13	65	M	Chest pain	140/80	120	Worker	20	99	Smoking -Stress	AF CAS	5L/m for 20min	Responsive
14	49	F	Tachypnea	100/80	83	House-wife	22	97	Stress - Obesity	IHVS CAS	5L/m for 20min	Responsive
15	47	F	Tachypnea	100/80	98	Teacher	23	95	Stress	IHVS CAS	5L/m for 15min	Responsive
16	53	M	Chest pain	130/70	83	Security man	25	97	Smoking	Stress CAS	5L/m for 20min	Responsive
17	22	F	Tachypnea	130/70	107	Gov. officer	28	98	Caffeine	IHVS CAS	5L/m for 20min	Responsive

Suggesting hypothesis and research objectives

a) Suggesting hypothesis: The effect of non-baric oxygen inhalation initially can improve the coronary artery spasm.

b) The research objectives: This is to clear the initial effect of non-baric oxygen inhalation on the coronary artery spasm. The dramatic response of coronary artery spasms to oxygen inhalation will be guided to modify international standards for the diagnostic criteria of coronary vasomotor disorders. This document addressed the criteria for vasospastic angina are included the following (A) nitrate-responsive angina, (B) transient ischaemic electrocardiogram changes, and (C) documented coronary artery spasm. This modification with oxygen will be added to the above three criteria (Figure 1)

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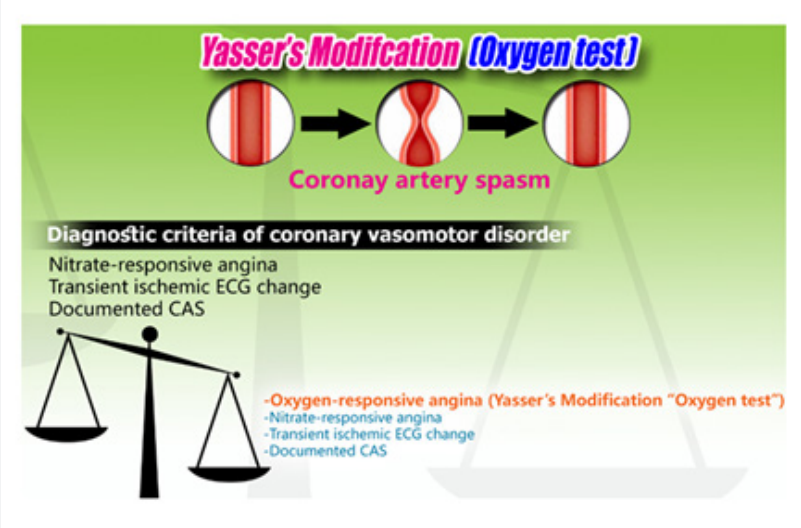

Figure 1: The author's diagrammatic presentation for Yasser's modification or oxygen test.

Eligibility criteria:

a) Inclusion criteria: All cases associated with initial ECG ST-segment depressions.

b) Exclusion criteria: Cardiac chest pain with normal ECG.

Assessment of treatment response was done with the presence of either:

a) Entirely reversal (Complete response) of ECG ST-segment depressions to oxygen inhalation.

b) Incomplete reversal (Partial response) of ECG ST-segment depressions to oxygen inhalation

Case Presentation

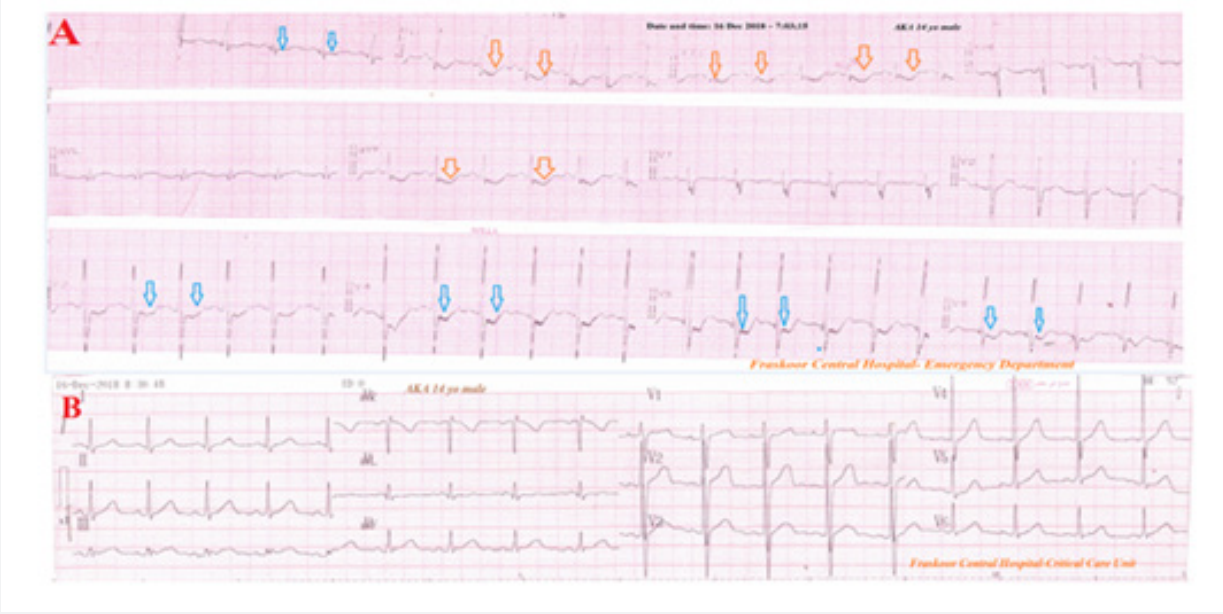

Figure 2: Initial ECG tracing (2A), ECG of (ED) presentation showing marked sinus tachycardia of VR, 140 bpm with ST-segment depressions in leads II, III, aVF (red arrows) and I, V3-6 (blue arrows). The second ECG tracing (2B) was done within 20 minutes of O2 inhalation showing complete normalization of ST-segment depressions and tachycardia.

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Case 1: A 14-year-old Egyptian single male adolescent student patient presented to the emergency department (ED) with a sudden deep loss of consciousness (LOC) in a hot bathroom in a closed space. The flushed face was noted on examination. His Glasgow coma scale (GCS) was 9. The case was managed urgently and only with high concentration O_2 inhalation using the nasal mask (12 L/m an initial emergency ECG showed sinus tachycardia with the ventricular rate (VR, 140 bpm) and ST-segment depressions in leads II, III, aVF, I, and V3-6 (Figure 2A). The second ECG tracing post-oxygenation showed complete normalization of ST-segment depressions with normal sinus rhythm (NSR) (Figure 2B). Troponin-T test was negative. Metabolic acidosis was seen on ABG. The initial suggested diagnosis was Carbon monoxide toxicity-induced CAS. Complete recovery had been achieved and the patient was discharged within 12 hours of ICU admission [13].

Case 2: A 36-year-old, an Egyptian, housewife female patient presented to the physician's outpatient clinic (POC) with anginal chest pain. The patient gave a recent history of toothache. An oral 100 mg ketoprofen tablet was prescribed for the pain. The patient started to complain of acute chest pain within 30 minutes of this oral tablet. On examination, the patient was anxious and irritable. The initial ECG tracing was taken before O_2 therapy showed; NSR with a VR of 96 bpm and straight ST-segment depression in V2-6 leads (Figure 3A). The second ECG tracing showed resolution of ST-segment depression with decreasing VR to 86 bpm after O_2 inhalation 100%, 5 L/m for 20 minutes using a nasal cannula (Figure 3B). The initial suggested diagnosis was Kounis-Zafras type 1 syndrome. Both CPK-MB and troponin levels were normal. Echocardiography showed an absence of hypokinetic abnormalities with a normal ejection fraction (EF: 68%). Dramatic response of clinical chest pain and ECG ST-segment depression improvement post-oxygenation had happened [14].

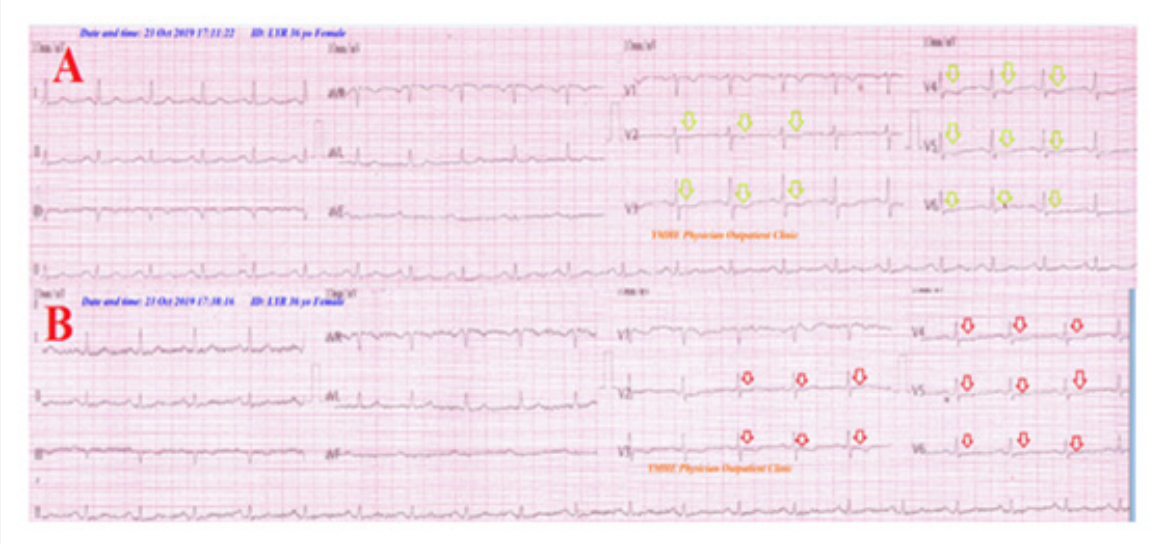

Figure 3: Initial ECG tracing (3A), ECG of ED presentation showing NSR of VR, 96 bpm and straight ST-segment depression in V2-6 ECG leads (lime arrows). The second ECG tracing (3B) was done within 20 minutes of O2 inhalation showing resolution of ST-segment depression with decreasing the VR to 86 bpm after O2 inhalation 100% (red arrows).

Case 3: An 11-year-old Egyptian single female student adolescent patient presented to the ED with sudden acute chest pain, palpitations, and abrupt LOC during the doing in a closed space hot bathroom. The family gave a history of hypothyroidism on a single daily100 mcg dose of levothyroxine sodium tab. The flushed face was noted on examination. Her GCS was: 9. The case was treated urgently only with high concentration O_2 inhalation, 12 L/m, for 30 minutes, using the nasal mask. An initial emergency ECG showed sinus tachycardia of (VR, 144 bpm) with ST-segment depressions in II, III, aVF, I, aVL, and V3-6 leads (Figure 4A). Complete normalization of ST-segment depressions but still showing sinus tachycardia had happened (Figure 4B). Troponin T test was negative. An initial ABG showed partially compensated metabolic acidosis. Later echocardiography was completely normal. The initial suggested diagnosis was Carbon monoxide toxicity-induced CAS. Complete clinical recovery and reversal of ECG changes had achieved. The patient was discharged within 24 hours of ICU admission [15].

Case 4: An 11-year-old Egyptian single female student adolescent patient presented to the ED with sudden acute chest pain, palpitations, and a fall in a hot bathroom in a closed space. The flushed face was noted on examination. Her GCS was: 15. The case was treated urgently only with a high concentration of O_2 inhalation using the nasal mask (12 L/m) An initial emergency

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ECG showed sinus tachycardia (VR, 112 bpm) with ST-segment depressions in V4-6 leads (Figure 5A). Complete normalization of ST-segment depressions had happened within 30 minutes of oxygenation (Figure 5B). The troponin T-test was negative. Metabolic acidosis was seen on ABG. Later echocardiography was completely normal. The initial suggested diagnosis was carbon monoxide toxicity-induced CAS. Complete clinical recovery and reversal of ECG changes had achieved. The patient was discharged within 24 hours of ICU admission [16].

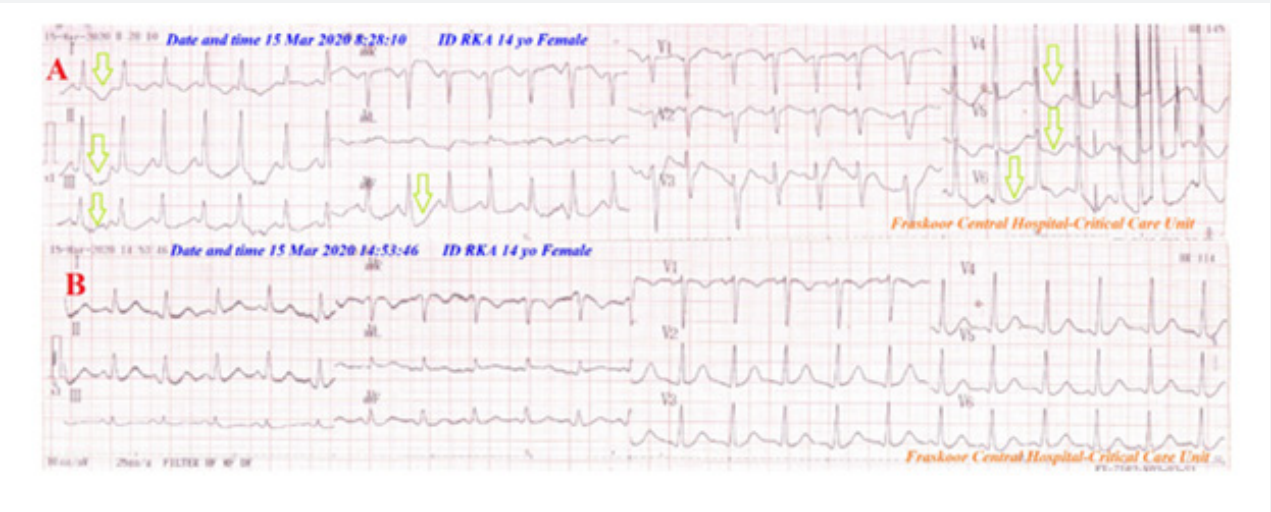

Figure 4: Initial ECG tracing (4A); ECG of (ED) presentation showing sinus tachycardia (VR; 144) with ST-segment depressions in II, III, I, aVL, and V3-6 leads (lime arrows). The second ECG tracing (4B) was done post-oxygenation showing normalization of all the above ST-segment depressions with still showing sinus tachycardia (VR; 116).

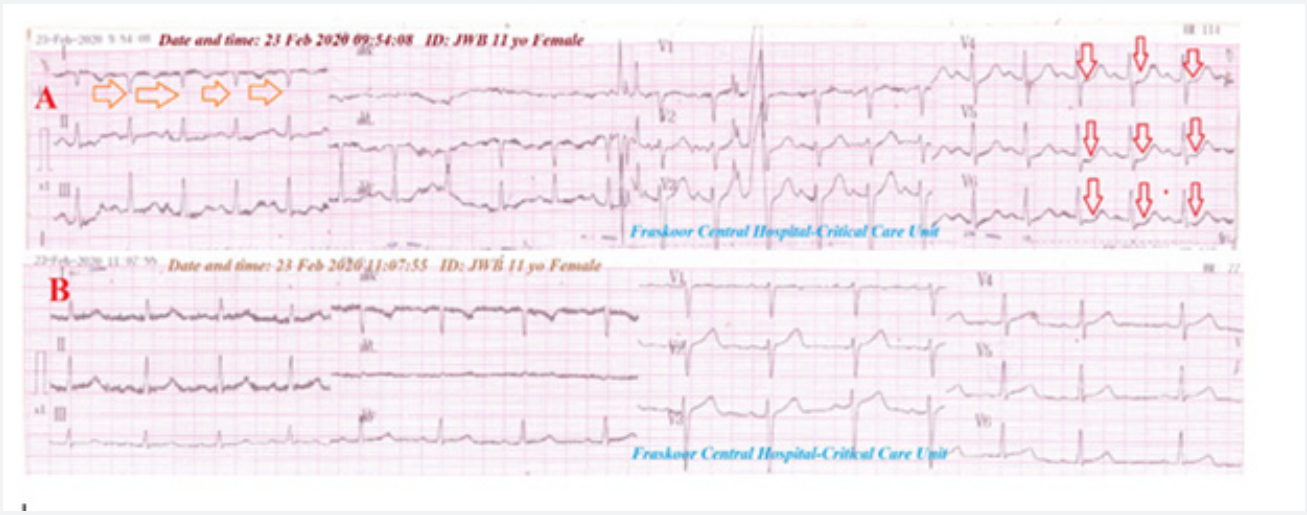

Figure 5: Initial ECG tracing (5A); ECG of (ED) presentation showing sinus tachycardia (VR; 112 bpm) with ST-segment depressions in V4-6 leads (red arrows) with technical LA/RA lead reversal (orange arrows). The second ECG tracing (5B) was done within 30 minutes of oxygenation showing complete normalization of all the above abnormalities.

Case 5: A 67-year-old married, farmer, male, Egyptian patient presented in the POC with tachypnea, dizziness, and chest pain. Profuse sweating and acute confusion state were the associated symptoms. The patient relatives gave an old history of bronchial asthma and liver cirrhosis. They give a recent asthmatic episode 3 days ago. He was acutely managed by the other POC. The POC

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only prescribed humified O_2 as needed. Within 30 minutes of the presentation, the patient gave a recent history of O_2 inhalation mistakenly using alcohol instead of sterile water in the water cup of the O_2 cylinder. Upon examination, the patient appeared tachypneic, pale, sweaty, and confused. GCS was 12. There is tachycardia during heart auscultation. The initial ECG tracing was done on the alcohol toxicity presentation showing sinus tachycardia with significant ST-segment depressions in both inferior (II, III, and aVF) and anterior leads (V1-6) with VR 102 bpm (Figure 6A). Oxygen inhalation (5 L/min) with an O_2 generator was given. The patient had gradually become calm with clear improvement in the respiratory status. The last ECG tracing was taken within 80 minutes of O_2 inhalation showing NSR with VR of 60 beats/min and the disappearance of above ST-segment depressions (Figure 6B). The troponin test was negative. Chest CT was done within 3 days post-presentation showing no abnormality detected. Later echocardiography was normal with an EF of 61%. Abdominal ultrasound showed severe liver cirrhosis. The initial suggested diagnosis was iatrogenic alcohol inhalation inducing CAS and sinus tachycardia in asthmatic and liver cirrhotic patient. The dramatic response of the above ST-segment depressions to O_2 inhalation occurred [17].

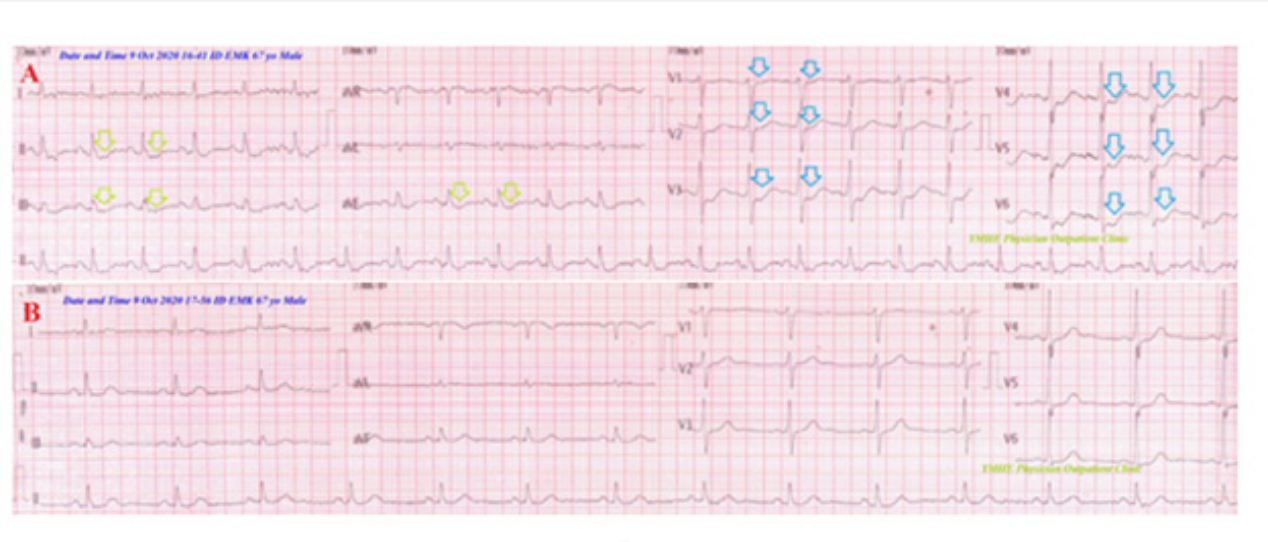

Figure 6: Initial ECG tracing (6A); ECG of (ED) presentation showing sinus tachycardia with significant ST-segment depressions in both inferior (lime arrows; II, III, and aVF) and anterior leads (blue arrows; V1-6) with VR; 102 bpm. The second ECG tracing (6B) was done within 80 minutes of O2 inhalation showing NSR with VR of 60 bpm and the disappearance of above ST-segment depressions.

Case 6: A 45-year-old married male, a football fan workless patient presented to the POC with palpitations, severe ischemic chest pain, and dizziness. Symptoms had happened just after the end of the match and the gain of his team. The patient is a heavy smoker. Upon examination, the patient appeared anxious, sweaty, and irritable. An irregular tachycardia was noted on heart auscultation. The initial ECG tracings showed sinus tachycardia with variable irregular premature ventricular contractions (PVCs) and ST-segment depressions in anterior leads (V2-6) of VR; 104 bpm (Figure 7A &Figure 7B) The patient was initially managed with O_2 inhalation (5 L/min) on an O_2 generator using a nasal cannula for about 20 minutes was given. The ECG tracing was repeated after the set of O_2 inhalation that showed the disappearance of the above abnormalities (Figure 7C). Echocardiography showed grade-I diastolic dysfunction and mild dilatation in the left atrium with an EF of 61%. The initial suggested diagnosis was over-merriment inducing PVCs and CAS [18].

Case 7: A 54-year-old married farmer Egyptian heavy smoker male patient presented to the POC with acute severe chest pain, carpopedal spasm, tachypnea, and palpitations. Numbness and paraesthesia in both extremities and perioral area were associated symptoms. He described the chest pain as a twisting agonizing pain. He gave a recent history of marked psychological stress. Upon general physical examination, generally, the patient was tachypneic and distressed. The patient was treated at the POC with was treated with O_2 inhalation by O_2 cylinder (100%, by nasal cannula, 5L/min). Ionized calcium was mildly low; 0.65 mmol/L. The troponin test had become negative. Serial ECG tracings were done. The initial ECG was done on presentation showing NSR of VR, 96 bpm with ST-segment elevations, pathological Q, and T-wave inversion in high lateral leads (I and aVL). There is a wavy triple sign or Yasser's of hypocalcemia in V1-6 leads. There are also ST-segment depressions in inferior leads (II, III, and aVF, dark blue arrows) (Figure 8A). Mimic HL-STEMI in chest

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tetany with mirror ECG change, Movable phenomenon (Yasser's phenomenon), and CAS was the most probable diagnosis. ECG tracing was taken within 40 minutes of the first ECG tracing and 20 minutes of O_2 inhalation showing normalization of above Wavy triple sign of hypocalcemia and ST-segment depressions. But there is a reversal of pathological Q, in I and aVL leads to be S-waves and normalization of ST-segment elevations in the same leads with NSR of VR 67 (Figure 8B). Within 40 minutes of the above management, the patient finally showed nearly complete clinical and ECG improvement. Then, two-calcium gluconate ampoules (10ml 10%) over IV over 20 minutes was given [19].

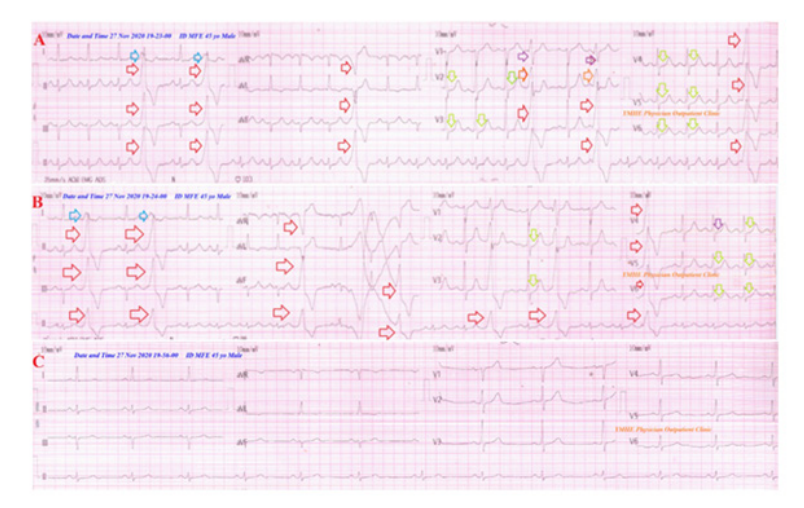

Figure 7: 7A. and 7B. ECG tracings were done upon arrival in the POC showing sinus tachycardia with variable irregular PVCs at VR; 104 (red, orange, and purple arrows) with ST-segment depressions in anterior leads (V2-6) (lime arrows). 7C. ECG tracing was done after the set of O2 inhalation that showed the disappearance of the above abnormalities with slight sinus bradycardia of VR 56 beats/min.

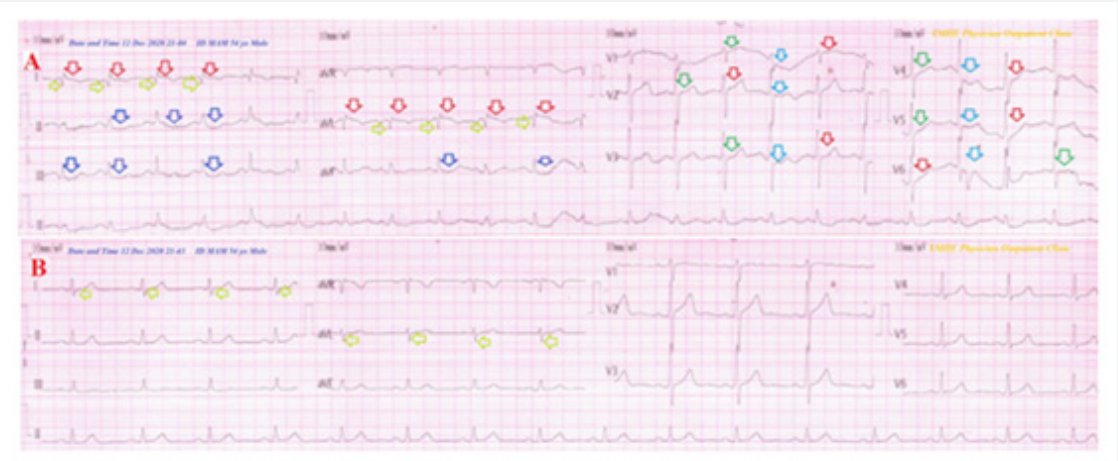

Figure 8: Initial ECG tracing (8A); ECG of POC presentation showing NSR of VR; 96 bpm with ST-segment elevations (red arrows), pathological Q (lime arrows), and T-wave inversion in high lateral leads (I and aVL). There is a wavy triple sign or Yasser's of hypocalcemia in V1-6 leads (red, green, and blue arrows). There are also ST-segment depressions in inferior leads (II, III, and aVF; dark blue arrows). The second ECG tracing (8B) was done within 40 minutes of the first ECG tracing and 20 minutes of O2 inhalation showing normalization of above Wavy triple sign of hypocalcemia and ST-segment depressions. But there is a reversal of pathological Q in I and aVL leads to be S- waves and normalization of ST-segment elevations in the same leads with NSR of VR; 67.

Case 8: A 65-year-old married housewife Egyptian female asthmatic patient presented with her jokey son to the POC for cardiovascular follow-up. Upon general physical examination, generally, the patient was good, not distressed. There were no wheezes on chest examination. The initial ECG tracing was done on the presentation for follow-up showing RBBB with NSR of VR of 70 with movable artifact (in V1 lead) (Figure 9A). During the ECG procedure, her jokey son told her; that ECG may be

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causing electrical shock for her. The mother urgently started to sense severe acute chest pain. The second ECG tracing was done within one minute of the initial ECG and after inducible fear, post-above drama showed NSR of VR, 82 with RBBB and ST-segment depression in both inferior (III and aVF) and anterior (V2-6) leads (Figure 9B). The patient was urgently managed with O_2 inhalation by O_2 cylinder (100%, by nasal cannula, 5L/min) and reassurance. Her son was prevented to be present during doing the third ECG tracing which was done within 15 minutes of O_2 inhalation and after reliving the chest pain showing NSR of VR, 74, normalization of the above ST-segment depression, and still the presence of RBBB (Figure 9C). The troponin test was negative. Echocardiography was normal with an EF of 67%. Fear-inducing CAS in an asthmatic patient with RBBB was the most probable diagnosis [20].

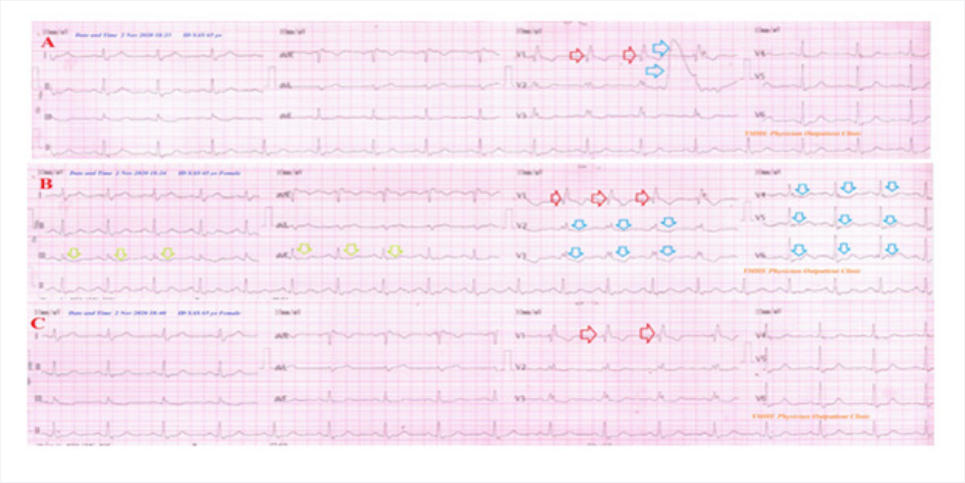

Figure 9: Initial ECG tracing (9A); ECG of POC presentation showing NSR of VR; 70 bpm with RBBB (red arrows). There is a movable artifact in the V1 lead (blue arrows). The second tracing (9B) was done within one minute of the initial ECG and after inducible fear showing NSR of VR; 82 with RBBB (red arrows), and ST-segment depression in both inferior (III and aVF; blue arrows) and anterior (V2-6; lime arrows) leads. The third tracing (9C) was done within 15 minutes of O2 inhalation showing NSR of VR; 74 bpm, normalization of the above ST-segment depression, and still the presence of RBBB (red arrows).

Case 9: A 70-year-old widow housewife Egyptian female patient presented to the POC with acute anginal chest pain after psychological stress. The patient gave a history of osteoarthritis for 15 years ago. She has a recent history of sporadic different doses of indomethacin. Upon general physical examination, generally, the patient was irritable and distressed. She appeared obese. The initial ECG tracing was done on the presentation showing NSR of VR, 62 bpm with ST-segment depression in both inferior (II and aVF) and anterior (V2-6) leads (Figure 10A). The physician had urgently managed the patient with O_2 inhalation by O_2 cylinder (100%, by nasal cannula, 5L/min) and reassurance. The second ECG tracing which was done within 20 minutes of O_2 inhalation showed NSR of VR, 60 bpm and normalization of the above ST-segment depression (Figure 10B). The CBC was within normal. RBS was normal (98 mg/dl). The troponin test was negative. Echocardiography showed an atherosclerotic aortic valve with no significant systolic gradient, trivial mitral regurgitation, and diastolic dysfunction with reversed E/A ratio, with an EF of 56%. Indomethacin-inducing CAS in an elder osteoarthritic patient was the most probable diagnosis.

Case 10: A 16-year-old single student Egyptian girl patient presented to the ED with acute anginal chest pain and palpitations. The patient gave a history of iatrogenic swallowing modafinil tablet (200mg) for 3 hours. Upon general physical examination, generally, the patient was agitated and distressed. The initial ECG tracing was done on the presentation for follow-up showing sinus tachycardia of VR, 129 bpm with ST-segment depression in high lateral (I and aVL) leads (Figure 11A). The physician had urgently managed the patient with O_2 inhalation by O_2 (100%, by nasal cannula, 5L/min). The second ECG tracing which was done within 20 minutes of O_2 inhalation showed NSR of VR, 90 bpm and normalization of the above ST-segment depression with tachycardia (Figure 11B). The troponin test was negative. Echocardiography showed no abnormality with an EF of 65%. Modafinil-inducing CAS in a single student Egyptian girl patient was the most probable diagnosis.

Case 11: A 60-year-old housewife married female Egyptian patient presented to the ED with acute anginal chest pain and palpitations. The patient gave a history of swallowing salbutamol tablet (4mg) 6 hours ago for cough. Upon general physical

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examination, generally, the patient was irritable and distressed. She appeared obese. The initial ECG tracing was done on the presentation for follow-up showing sinus tachycardia of VR, 115 bpm with ST-segment depression in anterior (V2-6) leads. The physician had urgently managed the patient with O_2 inhalation by O_2 cylinder (100%, by nasal cannula, 5L/min) and reassurance. The second ECG tracing which was done within 20 minutes of O_2 inhalation showed NSR of VR, 92 bpm and normalization of the above ST-segment depression with tachycardia. The troponin test was negative. Echocardiography showed no abnormality with an EF of 59%. Salbutamol-inducing CAS in an elder female Egyptian patient was the most probable diagnosis.

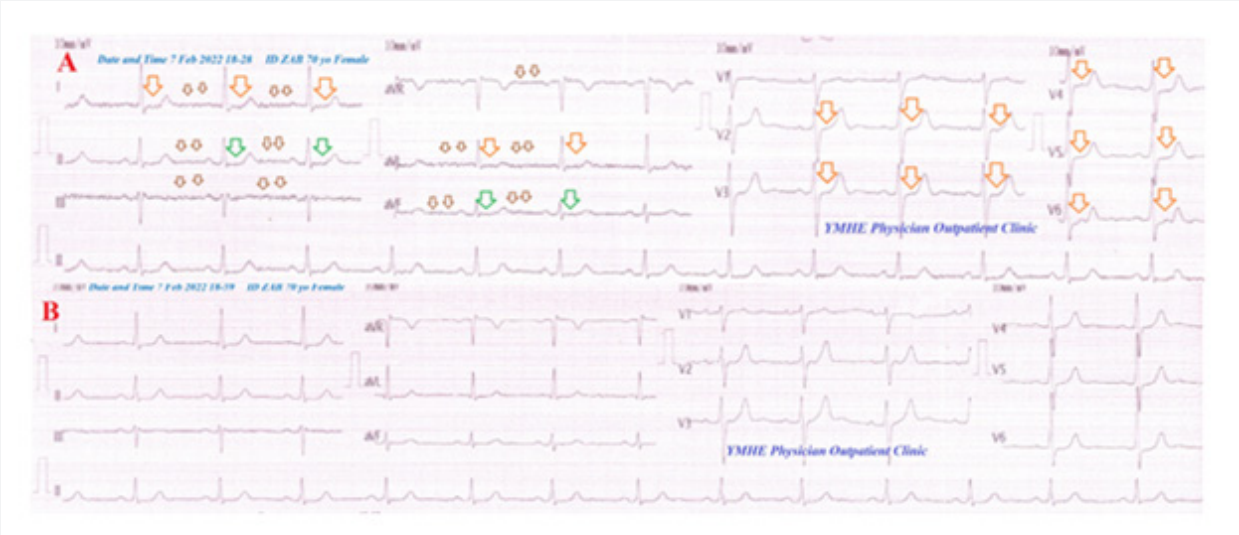

Figure 10: Initial ECG tracing (10A); ECG of (ED) presentation showing NSR of VR; 62 bpm with ST-segment depression in both inferior (II and aVF; green arrows) and anterior (V2-6; orange arrows) leads. There is a tremor artifact in limb leads (brown arrows). The second tracing (10B) was done within 20 minutes of O2 inhalation showing NSR of VR; 60 bpm and normalization of the above ST-segment depression.

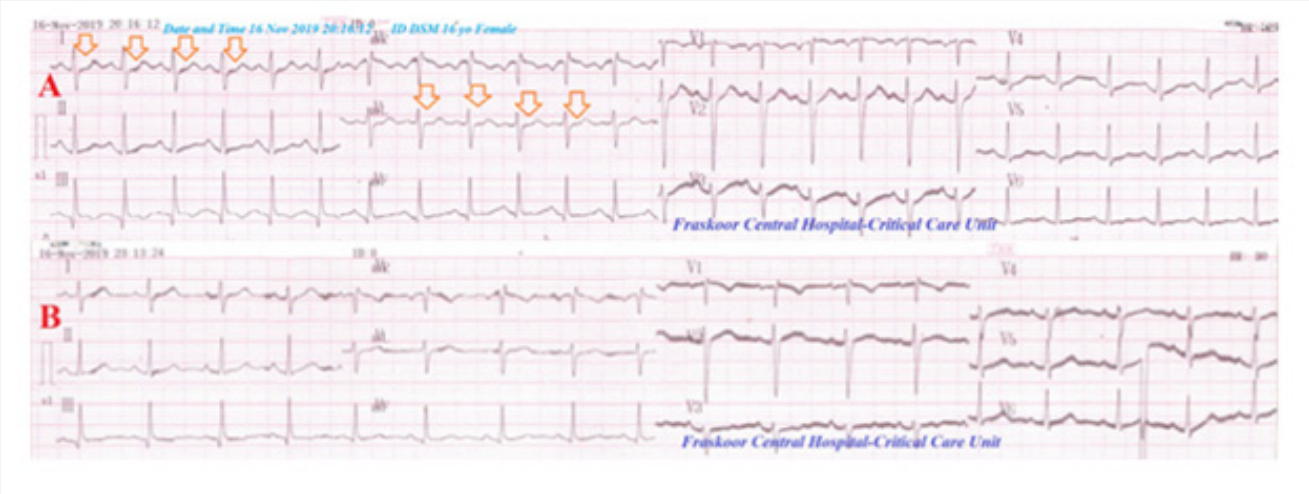

Figure 11: Initial ECG tracing (11A); ECG of ED presentation showing sinus tachycardia of VR; 129 bpm with ST-segment depression in high lateral (I and aVL; orange arrows) leads. The second tracing (11B) was done within 20 minutes of O2 inhalation showing NSR of VR; 90 bpm and normalization of the above ST-segment depression.

Case 12: A 50-year-old Governmental officer married female Egyptian patient presented to the ED with tachypnea, acute non-specific chest tightness, palpitations, peripheral paraesthesia, and numbness. The patient gave a history of recent psycho-familial troubles. She drinks a heavy coffee. Upon general physical examination, generally, the patient was irritable and distressed.

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The initial ECG tracing was done on the presentation for follow-up showing trigeminal PVCs with VR; 80 bpm with ST-segment depression in inferoanterior (II, aVF, and V2-6) leads. The physician had urgently managed the patient in the ICU with O_2 inhalation (100%, by nasal cannula, 5L/min) and reassurance. The second ECG tracing was done within 20 minutes of O_2 inhalation and after reliving the chest pain showed NSR of VR; 82 bpm and normalization of the above ST-segment depression with trigeminal PVCs. The troponin test was negative. Echocardiography showed no abnormality with an EF of 71%. Hyperventilation-inducing CAS in a middle-aged female Egyptian patient was the most probable diagnosis.

Case 13: A 65-year-old married heavy cigarette smoker worker male Egyptian patient was admitted to the ICU with acute angina chest pain and palpitations. Peripheral paraesthesia and numbness were associated symptoms. He is a coffee drinker. The patient gave a history of recent work stress troubles. Upon general physical examination, generally, the patient was irritable and distressed. The initial ECG tracing was done on the presentation for follow-up showing AF with VR; 115 bpm with ST-segment depression in inferolateral (II, III, aVF, and V4-6) leads and wavy triple sign (Yasser's sign, V4-6). The physician had urgently managed the patient in the ICU with O_2 inhalation (100%, by nasal cannula, 5L/min). The second ECG tracing which was done within 20 minutes of O_2 inhalation and after reliving the chest pain showed AF of VR; 98 bpm and normalization of the above ST-segment depression with still wavy triple sign (Yasser's sign; V4). The troponin test was negative. Echocardiography showed no abnormality with an EF of 64%. AF and stress-inducing CAS in an elderly male Egyptian patient was the most probable diagnosis.

Case 14: A 49-year-old married housewife female Egyptian patient presented to the POC with acute non-specific chest pain and tachypnea. Peripheral paraesthesia and numbness were associated symptoms. She is a coffee drinker. Upon general physical examination, generally, the patient was distressed. She appeared obese. The initial ECG tracing was done on the presentation for follow-up showing NSR with VR, 76 bpm with ST-segment depression in inferior and anterolateral (II, III, aVF, I, aVL, and V3-6) leads. The physician had urgently managed the patient in the POC with O_2 inhalation (100%, by nasal cannula, 5L/min). The second ECG tracing was done within 20 minutes of O_2 inhalation and after reliving the chest pain showed NSR of VR; 76 bpm and normalization of the above ST-segment depression. The lipid profile was abnormal. The troponin test was negative. Ionized calcium was low (3.6 mg/dl). Echocardiography showed no abnormality with an EF of 70%. Idiopathic hyperventilation-inducing CAS in a middle-aged patient was the most probable diagnosis.

Case 15: A 47-year-old married teacher female Egyptian patient presented to the POC with acute chest pain and tachypnea. Peripheral paraesthesia and numbness were associated symptoms. The chest pain was anginal. She is a Nescafe drinker. The patient gave a history of recent psycho-familial troubles. Upon general physical examination, generally, the patient was irritable and phobic. The initial ECG tracing was done on the presentation for follow-up showing NSR with VR, 96 bpm with ST-segment depression in anterolateral (V3-6) leads. The physician had urgently managed the patient in the POC with O_2 inhalation (100%, by nasal cannula, 5L/min). The second ECG tracing which was done within 15 minutes of O_2 inhalation and after reliving the chest pain showed NSR of VR; 94 bpm and normalization of the above ST-segment depression. The troponin test was negative. Ionized calcium was low (3.9 mg/dl). Echocardiography showed no abnormality with an EF of 61%. Psychogenic hyperventilation-inducing CAS in a middle-aged patient was the most probable diagnosis.

Case 16: A 53-year-old married security-man heavy smoker Egyptian patient presented to the POC with acute anginal chest pain and tachypnea. Peripheral paraesthesia and numbness were associated symptoms. The patient gave a history of recent economic troubles. Upon general physical examination, generally, the patient was tachypneic. The initial ECG tracing was done on the presentation for follow-up showing NSR with VR; 83 bpm with ST-segment depression in inferior and anterolateral (II, aVF, and V4-6) leads, and wavy triple sign (Yasser's sign; I and III). The physician had urgently managed the patient in the POC with O_2 inhalation (100%, by nasal cannula, 5L/min). The second ECG tracing which was done within 20 minutes of O_2 inhalation and after reliving the chest pain showed NSR of VR; 84 bpm and normalization of the above ST-segment depression. The troponin test was negative. Ionized calcium was low (3.5 mg/dl). Echocardiography showed no abnormality with an EF of 67%. Stress-inducing coronary spasm in a middle-aged patient was the most probable diagnosis.

Case 17: A 22-year-old Governmental officer married a female Egyptian patient to the POC with acute anginal chest pain and tachypnea. Peripheral paraesthesia and numbness were associated symptoms. The patient gave a history of recent swallowing two tablets of paracetamol-caffeine (500/65mg). Upon general physical examination, generally, the patient was tachypneic. The initial ECG tracing was done on the presentation for follow-up showing NSR with VR; 107 bpm with ST-segment depression in inferior and anterolateral (II, aVF, and V3-6) leads, and wavy triple sign (Yasser's sign; V5). The physician had urgently managed the patient in the POC with O_2 inhalation (100%, by nasal cannula, 5L/min). The second ECG tracing which was done within 20 minutes of O_2 inhalation and after reliving the chest pain showed NSR of VR; 83 bpm and normalization of the above ST-segment depression. The troponin test was negative. Ionized calcium was low (3.7 mg/dl). Echocardiography showed no abnormality with an EF of 60%. Caffeine-inducing coronary spasm in a young female patient was the most probable diagnosis. For more details for all the study cases (Table 2).

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Results

Averages age: Range: 11-70 years, mean: 43.2, median: 49, Mode: 11, minimal: 11 years, maximal: 70 years. With female sex: 64.71% (11 cases) and male sex: 35.29% (6 cases). Occupation: housewife: 29.41% (5 cases), student: 23.53% (4 cases), farmer: 11.76% (2 cases), gov. officer: 11.76 % (2 cases), worker: 5.88% (1 case), teacher: 5.88% (1 case), security man: 5.88% (1 case), and workerless: 5.88% (1 case) (Figure 12). The main complaint: Chest pain: 64.71% (11 cases), tachypnea: 23.53% (4 cases), palpitations: 5.88% (1 case), and syncope: 5.88% (1 case) (Figure 13).

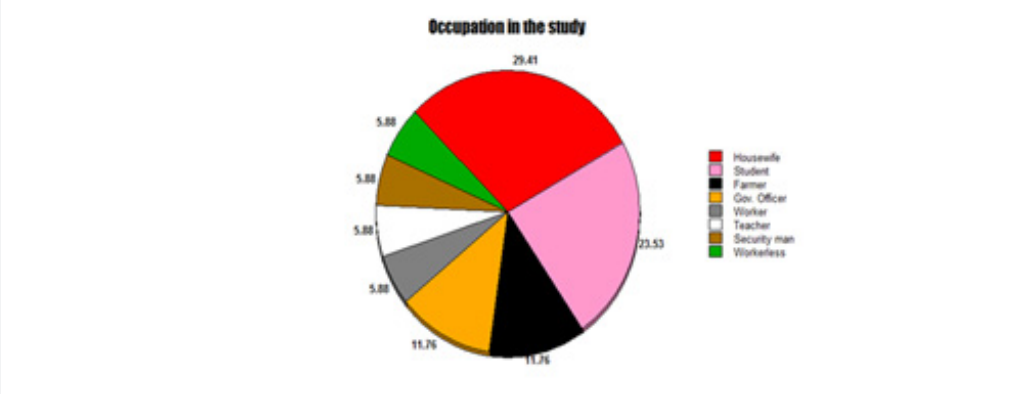

Figure 12: Pie chart presentation showing the percentage of occupation in the study.

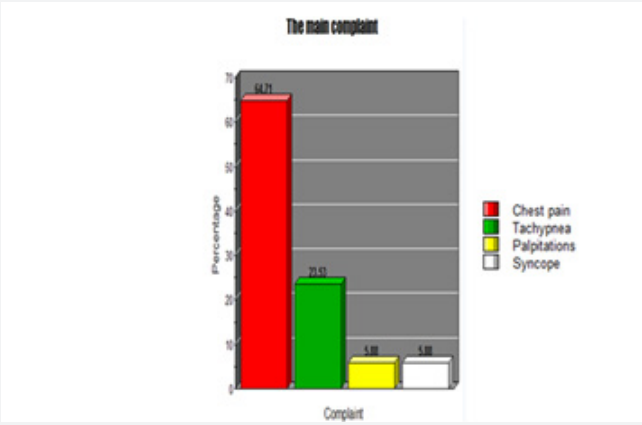

Figure 13: Bar chart presentation showing the main complaint in the study. the set of O2 inhalation that showed the disappearance of the above abnormalities with slight sinus bradycardia of VR 56 beats/min.

The associated risk factors (RF) in the study: Female sex: 64.71% (11 cases), stress: 23.53% (4 cases), CO toxicity: 11.76% (2 cases), smoking: 5.88% (1 case), obesity: 5.88% (1 case), bronchial asthma: 5.88% (1 case), caffeine: 5.88% (1 case), ketoprofen: 5.88% (1 case), combined: smoking with stress: 17.65% (3 cases), stress with obesity: 5.88% (1 case), closed hot bathroom with hyperthyroidism: 5.88% (1 case), and bronchial asthma with RBBB: 5.88% (1 case) (Figure 14).

Averages of inhaled O_2 dose: Duration/min: Mean: 22.77, Median: 20, Mode: 20, Minimal: 15min, Maximal: 80min. Dose/L: Mean: 5.5, Median: 5, Mode: 5, Minimal: 5L, Maximal: 12L. The final diagnosis: Drug-induced (ketoprofen, indomethacin, salbutamol, and modafinil): 23.53% (4 cases), hyperventilation syndrome-induced CAS: 23.53% (4 cases), CO toxicity: 17.65% (3 cases), stress-induced CAS: 17.65% (3 cases), tetany-induced CAS: 5.88% (1 case), alcohol-induced CAS: 5.88% (1 case), and AF-induced CAS: 5.88% (1 case) (Figure 15). Outcome: Responsive (Complete response): 94.12% (16 cases) with partial response: 5.88% % (1 case) (Figure 16).

Discussion

a) The mean age in the current study is: 43.2 with female sex predominance (64.71%). Housewife (29.41%) and students (23.53%) are the most affected occupations (Figure 12). The main complaint is chest pain (64.71%) followed by tachypnea (23.53%) (Figure 13). The most common associated risk factors in the study

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are female sex (64.71%), stress (23.53%), and CO toxicity (11.76%) (Figure 14). Drug-induced CAS (Ketoprofen, indomethacin, salbutamol, and modafinil: 23.53%), hyperventilation syndrome-induced CAS (23.53%), and CO toxicity (17.65%) are common diagnoses (Figure 15).

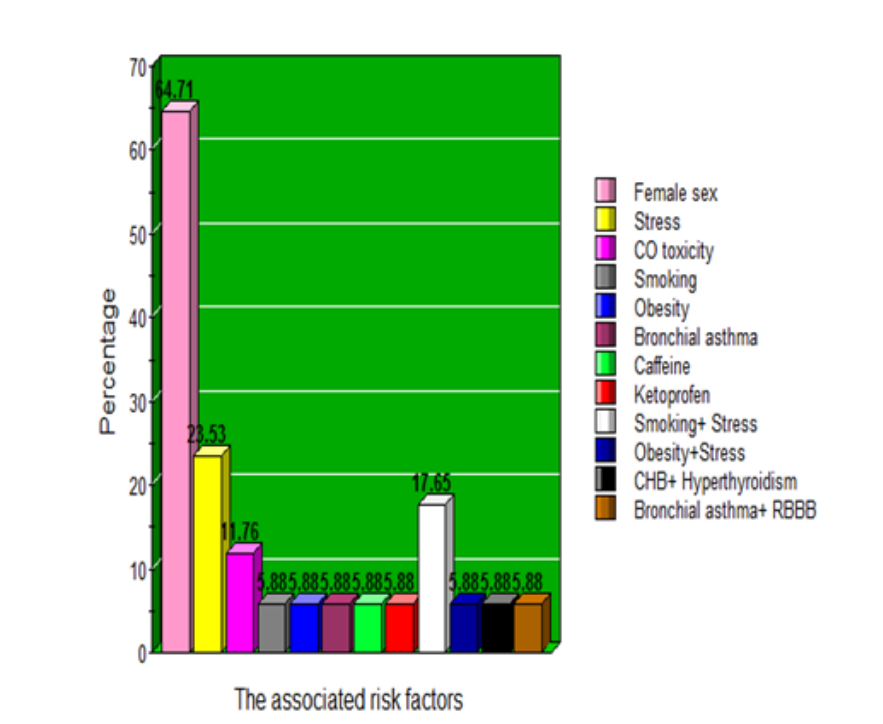

Figure 14: Bar chart presentation showing the percentage of the associated risk factors in the study.

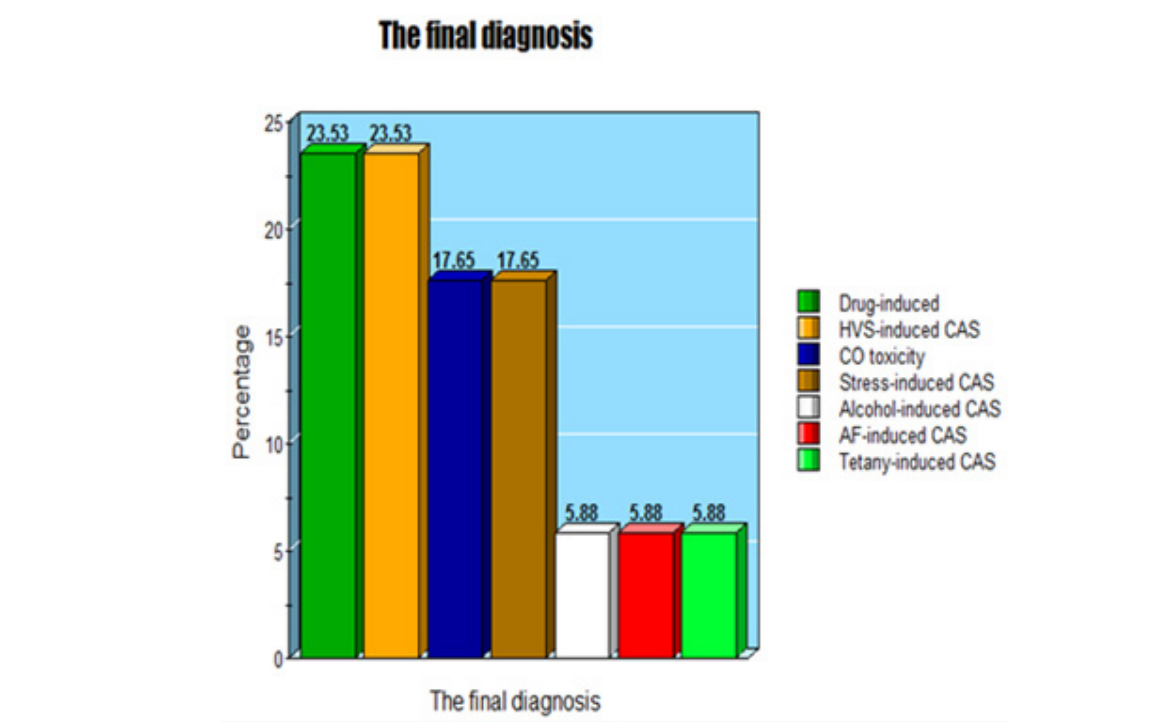

Figure 15: Bar chart presentation showing the final diagnosis in the study.

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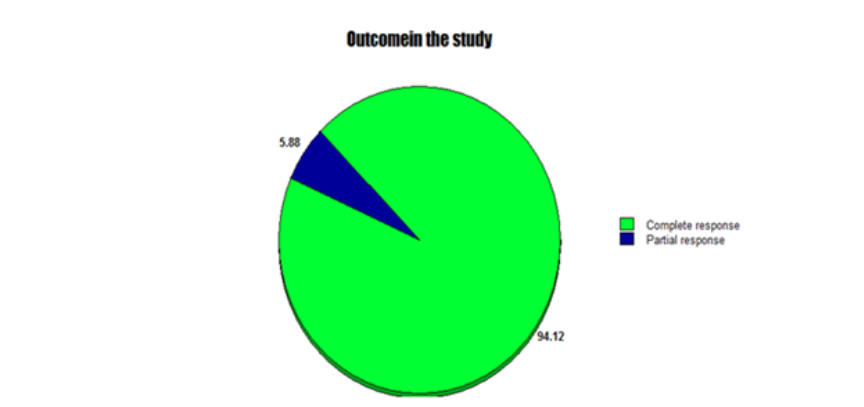

Figure 16: Pie chart presentation showing the outcome post-using oxygen.

b) The dose of inhaled O_2 dose that achieved the CAS in this study varied from 5 to 12 liter. A maximal dose (12 minutes) was given for CO toxicity. The duration of inhaled O_2 dose that achieved the CAS in this study varied from 15 to 80 minutes. Maximal duration (80 minutes) was given in CO toxicity (Table 2). This is due to cellular hypoxia in CO toxicity that is induced by the opposition of O_2 delivery. CO paradoxically binds hemoglobin (Hb) causing proportional practical anemia. Due to higher binding of CO to Hb with 230-270 times more avidly than O_2. Even with small levels of CO, considerable concentrations of HbCO will exist. The CO concentration of 100 ppm yields a HbCO of 16% at enough equilibration to produce a clinical presentation. The interaction of CO to Hb will rise the binding of O_2 molecules at the 3 other different O_2-binding sites causing a leftward shifting in the oxyhemoglobin dissociation curve. The diminishing of the availability of O_2 to the hypoxic tissues will occur [21,22]. O_2 quickens the expulsion of COHb and mitigates tissue hypoxia if it is comparable with air [22]. Using 100 % normobaric O_2 inhalation can hurry the degradation of COHb, with an elimination t 1/2 of about 74 minutes [23].

c) Role of hypoxia in the pathogenesis of CAS is pivotal and unavoidable. The imbalance between myocardial O_2 supply and demand is the key to the development of angina pectoris [6]. The exact mechanism for CAS is still idiopathic. CAS is mostly a disease in middle, elder-aged men, and post-menopausal women. Reactive O_2 species degrade NO and cause vasoconstriction. So, oxidative stress, endothelial dysfunction, and low-grade chronic inflammation play an important role in the pathogenesis of CAS, leading to increased coronary SM Ca_{2+} sensitivity through RhoA/ROCK activation and resultant hypercontraction [24]. A reduced O_2 supply to the heart causes coronary vasodilatation (VD). However, if there is severe and prolonged hypoxia, the VD passes off and CAS results causing a vicious circle with a further decrease of myocardial oxygenation [25]. Coronary VD is caused by a reduced O_2 supply to the heart. If the hypoxia is severe or prolonged, CAS results due to an over-decreasing of myocardial oxygenation. O_2 may stimulate the production of thromboxane A2 (TXA2) and decrease the syntheses of vasoconstrictor prostaglandins (PGs): the opposite effect is achieved with smoking due to the release of carboxyhemoglobin (COHb). O_2 may increase TXA2 production and reduce the formation of vasoconstrictor PGs, while smoking, due to the formation of COHb, may have the reverse effect [12].

d) The decreasing O_2 supply to the heart result in coronary VD. However, severe, or prolonged hypoxia will passes dilatation off and CAS happen. A vicious circle with a moreover decrease in myocardial oxygenation is the consequence. The CAS is associated with increased outflow of prostaglandin (PG)-like material and can be prevented or reversed by inhibitors of PG synthesis such as indomethacin or antagonists of PG action such as chloroquine. The CAS does not appear to be caused by TXA2 since selective inhibitors of TXA2 synthesis enhance the hypoxic spasm and by themselves can cause CAS even in oxygenated hearts. The mechanism may be related to the loss of negative feedback control of the PG pathway by TXA2. O_2 may enhance TXA2 production and reduce the formation of vasoconstrictor PGs. The role of CAS in many cardiac events has been reported. The paper presents a quite different mechanism, overproduction of PGs, as the major factor in producing both the CAS and disorders of the rhythm which often accompany it. Coronary VD is caused by a reduced O_2 supply to the heart. If the hypoxia is severe or prolonged, CAS results, leading to a further reduction of myocardial oxygenation [25].

e) Unfortunately, there are no available relevant studies for alone use of O_2 inhalation in CAS.

f) The Coronary Vasomotion Disorders International Study Group (COVADIS) represented the development of international standards for the diagnostic criteria of coronary vasomotor disorders. The first symposium was held on the 4-5 September 2013 and documented the criteria for coronary artery spasm,

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which included the following (A) nitrate-responsive angina, (C) transient ischaemic electrocardiogram changes, and (C) documented coronary artery spasm [26]. But in my study, these criteria will be modified to add the fourth criterion by the oxygen inhalation in coronary artery spasm as the first initial criterion for the above three one (Yasser's Modification or Oxygen test).

g) Safety of O_2 inhalation in coronary artery spasms obligates us to avoid us to use of hazardous provocation testing in the diagnosis of vasospastic angina. This is especially if the provocative stimulus induced chest pain, transient ECG changes, and a >90 percent constrictor response [27].

h) The complete response of coronary artery spasms to oxygen inhalation in the current study had happened in 94.12%. Although the diagnosis is based on nitrate-responsive angina with associated transient ECG changes [27]. And despite nitrates being the mainstay of medical therapy for CAS [28]. But the side effects can be tolerated. Headache is the most common side effect of nitrates. About 10% of cases are intolerant to nitrates due to disabling headaches or dizziness. Nitrate-induced hypotension is common but often asymptomatic. Nitrates rarely cause coronary steal and IHD. Nitrate rebound may happen with experience of nocturnal anginal episodes during intermittent therapy with nitroglycerin patches. Nitrates are contraindicated with concomitant use of PDE-5 inhibitors used for the treatment of ED, as combination therapy may lead to severe hypotension and even death. Chronic use of nitrates may lead to an increased mortality rate and recurrent MI [29]. However, there are some cases of CAS whose attacks cannot be controlled even with large doses of CCBs and/or their combination with nitrates. A reported case of a 41-year-old man presented with attacks of CAS was resistant to the combined given of nitrates, CCBs, and a statin. The attacks were alleviated and disappeared after the withdrawal of nitrates and recurred after re-administration of the NTG patch. The involvement of nitrate tolerance in the pathogenesis of multidrug-resistant CAS was revealed [30].

i) The study limitations were the absence of coronary catheterization and provocation testing for the diagnosis of vasospastic angina.

Conclusion and Recommendation

a) Dramatic clinical reliving and reversal response of electrocardiographic ST-segment depression after oxygen inhalation is an indication for its initial use in coronary artery spasm.

b) Yasser's Modification or Oxygen test for the past "international standards for the diagnostic criteria of coronary vasomotor disorders" improves patient safety and decreases the hazards of nitrate and other medications.

c) Widening the research for use of the oxygen inhalation in the coronary artery spasm will be recommended

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