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Yasser's Stressor Test (Fear and Calm Test) and Triphasic Yasser's Stressor Syndrome (Fear, Calm, and Fear Syndrome) - A New Cardiovascular Discoveries and Psychogenic Stress Test with Possible Coronary Artery Spasm

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ABSTRACT

AIM

The study is aimed to clear the cycle effect of fear or anxiety and calm or reassurance on electrocardiographic ST-segment depression in patients present with angina with possible coronary artery spasms.

INTRODUCTION

Anxiety and fear have undoubted hazardous effects on cardiovascular disorders. The effect on the coronary artery may be the most significant. The coronary artery spasm is an established cardiovascular disorder that can affect the ST segment with either depression elevation or both. It can end with vascular occlusion or near-occlusion and possibly coronary heart disease and sudden death.

METHOD

My case study was an observational-retrospective 50 case report series. The study was conducted in Kafr El-Bateekh Central Hospital and physician outpatient clinic. The author reported 50 cases of resting acute angina with reversal and cyclic electrocardiographic ST-segment depression over about 44 months, starting on December 09, 2019, and ending on July 18, 2023.

RESULTS

The mean age in the current study is 50.7 in the range; of 18 years - 82 years, with a female sex predominance (52%). Housewives (34%) and farmers (26%) are the most affected occupations. The main complaint is angina (84%) followed by angina with palpitations (10%). The most common associated risk factors in the study are combined risk factors (56%) followed by single risk factors (44%). The Mean of elapsed time between stage I and

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II/sec (only group I) are 66.65 in a range; of 17 years - 250 years. The response was complete (only group I; 40%), Partial (only group I; 12%), and Absent (only group II; 48%).

CONCLUSION

Yasser's Stressor test is a new cardiovascular discovery and psychogenic stress test targeting the ST-segment passing two stages in the form of fear and calm (Fear-Calm Stressor test). Triphasic Yasser's syndrome is a sequel and constellation of the vicious cycle of Yasser's Stressor test passing three stages in the form of fear, calm, and fear (Fear-Calm-Fear Yasser's syndrome). Coronary artery spasm is a possible pathogenesis for interpretation. Both Yasser's Stressor test and its subsequent Triphasic Yasser's syndrome are easy, simple, cheap, and safe non-invasive exercise tests.

KEYWORDS

Stress test; Coronary artery spasm; Ischemic heart disease; Triphasic Yasser's syndrome; Yasser's stressor test; Fear; Fear-calm stressor test; Fear-calm-fear Yasser's syndrome; Psychiatry

ABBREVIATIONS

ABG: Arterial blood gases AF: Atrial fibrillation AMI: Acute myocardial infarction **BP: Blood pressure** CAS: Coronary artery spasm CBC: Complete blood count CCBs: Ca²⁺ channel blockers CO: Carbon monoxide ECG: Electrocardiography ED: Emergency Department EF: Ejection Fraction HR: Heart Rate HVS: Hyperventilation Syndrome ICU: Intensive Care Unit IHVS; Idiopathic Hyperventilation Syndrome IHD: Ischemic Heart Disease LBBB: Left bundle branch block LOC: Loss of Consciousness **MI: Myocardial Infarctions** NO: Nitric Oxide NSR: Normal Sinus Rhythm O2: Oxygen PG: Prostaglandin POC: Physician Outpatient Clinic **PVCs: 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

ST depression is an ECG finding in which the ST segment is abnormally deviated below the isoelectric line. The ST-segment depression (STD). STD is usually associated with acute coronary syndromes (ACS) [1-3]. Non-specific ST-segment depression are very common ECG finding [4]. It may be detected in any lead of the ECG. The changes may be seen in all or most of the leads (diffuse changes), or they may be present in contiguous specific leads, such as the inferior, lateral, or anterior leads. ST-segment depression is measured from the isoelectric baseline, or when ST-segment depression is present at rest, the amount of additional depression is measured. Anxiety is common in patients with cardiovascular disease, such as coronary artery disease (CAD). Anxiety has been associated with the incidence and in some cases progression, of cardiovascular disease. Reassurance, sitting a calmly, quickly analyzing the problem, and taking an appropriate active decision, one can convert bad stress (distress) into good stress (eustress). The coronary artery spasm (CAS) has a pivotal role in the pathogenesis of coronary heart disease.

PATIENTS AND METHODS

The author had reported 50 cases of acute anginal chest pain with inducible ECG ST-segment depressions. The study was conducted in both Kafr El-Bateekh Central Hospital and the physician outpatient clinic (POC). The author reported 50 cases of angina over about 44 months, starting on December 09, 2019, and ending on July 18, 2023. The study is an observational retrospective case report series (Table 1). There was a different diagnosis for all cases. All selected cases were presented with associated with significant ECG ST-segment depressions. All cases of ECG ST-segment depressions are immediately exposed to the test of fear and calm. The ECG tracings were reported on exposure to the test of fear after and before the calm. The time interval among the ECG tracings was calculated by seconds through ECG timing. The response was recorded according to the appearance and disappearance of ECG ST-segment depressions. Troponin test was done for all cases. The echocardiography was done in 16 cases. ABG, CBC, liver enzymes, ionized calcium, RBS, and renal function tests were done in selected cases. History, clinical data, and the response to fear and calm for all cases were recorded. For more details check (Table 2 - Table 4).

Issue	Definition
Title	Yasser's Stressor Test (Fear and Calm Test) and Triphasic Yasser's Stressor Syndrome (Fear, Calm, and Fear Syndrome)-A New Cardiovascular Discoveries and Psychogenic Stress Test with Possible
	Coronary Artery Spasm; Case Report Series, Retrospective-Observational Study, and Original
	Research
Estimated Enrollment	50 Participants
Study Type	Observational
Observational Model	Case Report Series and Original Research

Time	Retrospective
Study Start Date	December 09, 2019
Estimated Study Completion Date	July 18, 2023
Analytic Method	Comparative using Percentage %

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

C as	Ag e/y	S e	Occu patio	Complaints	Echocardiography	Response	Affected leads	Time interval/
e N		x	'n					sec
0.								
1	54	М	Build er	Angina	Mild LVH with EF 65%	Complete	II, III, aVF, and V4-6	60
2	50	F	House wife	Angina	Normal with EF 75%	Complete	aVF and V3-6	35
3	72	М	Farme r	Angina	LVH, mild AR, MR, TR, DD. with EF 64%	Complete	II and V2-6	30
4	48	F	Teach er	Angina and palpitations	Normal with EF 63%	Complete	II, III, aVF, and V3-6	60
5	62	F	House wife	Angina	DHD and mild MR with EF70%	Complete	I, II, aVF, and V4-6	43
6*	64	F	Farme r	Angina and palpitations	LVH, mild AR, and MR with EF of 62%	Complete	I, aVF, and V4-6	60
7	65	F	Farme r	Angina	Ischemic CMP with EF 42%	Complete	II, III, aVF, and V4-6	60
8	48	F	Teach er	Angina and tachypnea	DD. with EF 68%	Complete	V2-6	60
9	47	F	House wife	Angina	LVH, DD, and mild MVP with EF of 66%	Complete	V4-6	81
10	72	М	Farme r	Angina	Mild MR, DD, and IVS hypokinesia with EF of 50%	Complete	V4-6	60
11	61	М	Drive r	Angina and tachypnea	UA	Partial Still II, aVF, and V6	STE in I, aVL, and STD in II, III, aVF, and V4-6	72
12	62	М	Retire d	Angina	UA	Complete	II, III, aVF, and V4-6	20
13	45	F	House wife	Angina	LAE, RAE, severe MS, moderate AR, moderate TR, with EF 64%	Complete	I, aVL, and V4-6	60
14	64	F	House wife	Angina and palpitations	DD, trivial MR, and mild TR with EF 68%	Partial Still V4-5	I, aVL, and V2-6	30
15	43	F	House wife	Angina	RHD and trivial MR with EF 71%	Partial Still V3-4	V2-6	45
16	52	F	House wife	Angina	DD and trivial MR with EF 59%	Partial Still II and V6	II, III, aVF, and V3-6	20
17	52	F	House wife	Angina	UA	Complete	STD in I, aVL, II, and V1-6 + STE in aVR	90
18	24	F	Gov. office r	Angina	UA	Complete	V3-6	36
19	82	F	House wife	Angina	UA	Complete	II, and V4-6	250
20	18	F	Stude nt	Angina	UA	Complete	V1-6	17
21	72	F	House wife	Angina	UA	Partial Still V4, 5	V2-5	40
22	39	F	House wife	Angina	UA	Complete	V2-6	109
23	53	М	Teach er	Angina	UA	Complete	V4-6	180
24	75	F	House wife	Angina and tachypnea	UA	Complete	II, III, aVF, and V3-6	57
25 *	72	М	Farme	Angina	See case No. 3	Complete	V2-6	120
26 **	62	F	House	Angina	See case No. 5	Partial Still aVF	II, III, aVF, and V3-6	38

Table 2: Summary of the history, clinical, and management data for group I; n:26.

*: recurrent case No. 3; **: recurrent case No. 5

Case No.	Age/y	Sex	Occupation	Complaints
1	50	Μ	Farmer	Angina
2	21	F	Student	Angina
3	21	Μ	Student	Angina
4	33	Μ	Officer	Angina
5	18	F	Student	Angina

6	51	Μ	Worker Angina			
7	60	F	Housewife Angina and palpitat			
8	44	Μ	Farmer	Angina		
9	45	Μ	Carpenter	Angina		
10	47	Μ	Contractor	Angina		
11	72	Μ	Farmer	Angina		
12*	73	Μ	Farmer	Angina		
13	72	F	Housewife	Angina and palpitations		
14	75	Μ	Farmer	Angina		
15	51	Μ	Carpenter	Angina		
16	23	Μ	Student	Angina		
17	34	F	Housewife	Angina		
18	35	Μ	Farmer	Angina		
19	58	F	Farmer	Angina		
20	18	Μ	Student	Angina		
21	26	Μ	Doctor	Angina		
22	43	F	Housewife	Angina		
23	55	Μ	Farmer	Angina		
24	53	Μ	Lawyer	Angina		

Table 3: Summary of the history of data for group II; n:24.

*: recurrent case No. 11

Variable	Numbers	%	Range	Means
Age	-	-	18-82	50.7
Sex				
Female				
Group I	19	73.08		
Group II	7	29.17		
Male				
Group I	7	26.92		
Group II	17	70.83		
Occupation				
Housewife	17	34		
Farmer	13	26		
Student	6	12		
Teacher	3	6		
Carpenter	2	4		
Officer	2	4		
Builder	1	2		
Contractor	1	2		
Doctor	1	2		
Worker	1	2		
Lawyer	1	2		
Driver	1	2		
Retired	1	2		
Complaints				
Angina	42	84		
Angina and Palpitations	5	10		
Angina and Tachypnea	3	6		
Elapsed Time between Stage I and II/Sec (Only Group I)	-	-	17-250	66.65
Response				
Complete (Only Group I)	20	40		
Partial (Only Group I)	6	12		
Absent (Only Group II)	24	48		
Risk factors (RF)				
Single RF	28	56		
Multiple RFs	22	44		

Table 4: Statistical summary of age, sex, occupation, complaints, elapsed time between stage I and II/sec and

response; n:50.

Suggesting Hypothesis and Research Objectives

Suggesting hypothesis

The effect of psychogenic stressors and fear initially can induce ECG ST-segment depressions and urgently reversed after stressor removal with the possible coronary artery spasm.

The research objectives

The research objectives are to clear the initial effect of psychogenic stressor and fear initially can induce ECG ST-segment depressions and be urgently reversed after stressor removal with the possible coronary artery spasm. The dramatic response of inducible ECG ST-segment depressions after psychogenic stressors and fear suggests the implication of coronary artery spasms in this process.

Eligibility Criteria

Inclusion criteria

Anginal chest pain with or without normal ECG.

Exclusion criteria

All cases of non-ischemic heart disease chest pain.

Assessment of Treatment Response was Done with

- Entirely reversal (Complete response) of ECG ST-segment depressions post-stressor removal.
- Incomplete reversal (Partial response) of ECG ST-segment depressions to oxygen inhalation.

Description

Yasser's Stressor test is a new cardiovascular discovery and psychogenic stress test targeting the ST-segment passing two stages in the form of fear and calm (Fear-Calm Stressor test) (Figure 1). Triphasic Yasser's stressor syndrome is a sequel and constellation of the vicious cycle of Yasser's Stressor test passing three stages in the form of fear, calm, and fear (Fear-Calm-Fear Yasser's syndrome) (Figure 2). Coronary artery spasm is a possible pathogenesis for interpretation. Both Yasser's Stressor test and its subsequent Triphasic Yasser's syndrome are easy, simple, cheap, and safe non-invasive exercise tests.



Figure 1: Yasser's stressor test (fear and calm test).



Figure 2: Triphasic Yasser's stressor syndrome (fear, calm, and fear syndrome).

Stressors

Active stressor

It is reactive change and inducible ECG ST-segment depressions due to active response after exposure to positive psychogenic stressor and fear.

Forms of active stressor

Stage I

- Remember the patient with questions of fear such as; are you afraid?
- Induction of fear for the patient by thinking format such as; I think you are afraid!

Stage II

• Re-assurance the patient and remind the patient with any sentence indicates such as you are good or there is no problem.

Passive stressor

It is reactive change and passive inducible ECG ST-segment depressions due to passive response after exposure to negative psychogenic stressors and fear.

Forms of passive stressor

Stage I

• The patient will be reactive just by either seeing the doctor or being connected with the electrocardiograph.

Stage II

Re-assurance the patient and remember him by any sentence indicating re-assurance such as You are good or There is no problem be calm, or I hope you will be calm.

Assessment of the Study Cases

With the happening of ECG ST-segment depressions and subsequent normalization after applying either the above active or passive stressors.

The Response was done

Positive test

It indicates the presence of the "ECG ST-segment depressions" after applying either the above active or passive stressors. Positive test either:

Complete associated response

It means complete recovery of the inducible of the "ECG ST-segment depressions" after removal of either the above active or passive stressors.

Incomplete associated response or partial response

It means complete recovery of the inducible of the "ECG ST-segment depressions" after removal of either the above active or passive stressors.

Negative test

There are no "ECG ST-segment depressions" after applying either the above active or passive stressors.

The Limitations of the Study

The limitations of the study were the absence of coronary angiography.

SOME CASES PRESENTATION

Case No. 1

A 54-years-old married male builder heavy smoker Egyptian patient presented to the physician outpatient clinic (POC) with acute anginal chest pain. His vital signs were as follows: Blood pressure (BP) 100/70 mmHg, heart rate (HR) 80 bpm, respiratory rate (RR); 16 bpm, temperature 36.5°C, and oxygen (O₂) sat by pulse oximeter; 99%. The initial emergency ECG showed normal sinus rhythm (NSR) with the ventricular rate (VR; 83 bpm) with ST-segment depressions in leads II, III, aVF, and V4-6 (Figure 3A). The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 78 bpm with complete normalization of the above ST-segment depressions (Figure 3B). The Troponin-I test was negative. The echocardiography showed left ventricular hypertrophy with an ejection fraction (EF) of 65%. The initial suggested diagnosis was angina.



Figure 3: A) The initial emergency ECG showed normal sinus rhythm (NSR) with the ventricular rate (VR; 83 bpm) with ST-segment depressions in II, III, aVF (lime arrows), and V4-6 leads (blue arrows). **B)** The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 78 bpm with complete normalization of the above ST-segment depressions.

Case No. 2

A 50-years-old, an Egyptian, housewife female patient presented to the intensive care unit (ICU) with acute anginal chest pain. Her vital signs were as follows: BP; 130/80 mmHg, HR; 85 bpm, RR; 16 bpm, temperature 37°C, and O₂ sat by pulse oximeter; 98%. The initial emergency ECG showed NSR of VR; 81 bpm with ST-segment depressions in aVF and V3-6 leads (Figure 4A). The second ECG tracing was done within 35 seconds of the first ECG and showed NSR of VR; 83 bpm with complete normalization of the above ST-segment depressions (Figure 4B). The Troponin-I test was negative. The echocardiography showed no abnormalities with an EF of 75%. The initial suggested diagnosis was recurrent angina.



Figure 4: A) The initial ICU ECG tracing showed NSR of VR; 81 bpm with ST-segment depressions in aVF (lime arrow) and V3-6 leads (blue arrows). **B**) The second ECG tracing was done within 35 seconds of the first ECG and showed NSR of VR; 83 bpm with complete normalization of the above ST-segment depressions.

Case No. 3

A 72-years-old married male Farmer Egyptian patient presented to the POC with acute anginal chest pain. His vital signs were as follows: BP; 110/80 mmHg, HR; 60 bpm, RR; 18 bpm, temperature 36°C, and O₂ sat by pulse oximeter; 96%. The initial emergency ECG showed NSR of VR; 60 bpm with ST-segment depressions in II and V2-5 leads and evidence of passed silent inferior myocardial infarction (II and aVF) (Figure 5A). The second ECG tracing was done within 30 seconds of the first ECG and showed NSR of VR; 63 bpm with complete normalization of the above ST-segment depressions (Figure 5B). The Troponin-I test was negative. The echocardiography showed LVH, mild AR, MR, TR, and diastolic dysfunction with an EF of 64%. There is laboratory mild hypocalcemia. The initial suggested diagnosis was ibuprofen-inducing angina in passed MI.



Figure 5: A) The initial emergency ECG showed NSR of VR; 60 bpm with ST-segment depressions in V2-5 leads and evidence of passed silent inferior myocardial infarction (pathological Q waves; II and aVF; red arrows).
B) The second ECG tracing was done within 30 seconds of the first ECG and showed NSR of VR; 63 bpm with complete normalization of the above ST-segment depressions but with still evidence of passed silent inferior myocardial infarction (pathological Q waves; II and aVF; red arrows).

Case No. 4

A 48-years-old Egyptian Teacher married female patient presented to the POC with acute anginal chest pain and peripheral numbness with paresthesia. Her vital signs were as follows: BP; 110/70 mmHg, HR; 70 bpm, RR; 24 bpm, temperature 36°C, and O₂ sat by pulse oximeter; 99%. The tests for latent tetany were inducible. The initial emergency ECG showed NSR of VR; 72 bpm with ST-segment depressions in II, III, aVF, and V3-6 leads (Figure 6A). The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 73 bpm with complete normalization of the above ST-segment depressions (Figure 6B). The Troponin-I test was negative. The echocardiography showed no abnormalities with an EF of 63%. The initial suggested diagnosis was angina post-psychogenic hyperventilation syndrome.



Figure 6: A) The initial ICU ECG tracing showed NSR of VR; 72 bpm with ST-segment depressions in II, III, aVF (green arrow), and V3-6 leads (blue arrows). B) The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 73 bpm with complete normalization of the above ST-segment depressions.

Case No. 5

A 62-years-old Egyptian housewife female patient presented to the ICU with acute anginal chest pain. She has metastatic breast carcinoma, thyroidectomy, liver cell failure, and past COVID-19 pneumonia. Her vital signs were as follows: BP; 100/70 mmHg, HR; 80 bpm, RR; 18 bpm, temperature 36.5°C, and O₂ sat by pulse oximeter; 95%. The initial ICU ECG showed NSR of VR; 80 bpm with ST-segment depressions in I, II, aVF, and V4-6 leads (Figure 7A). The second ECG tracing was done within 43 seconds of the first ECG and showed NSR of VR; 81 bpm with complete normalization of the above ST-segment depressions (Figure 7B). The Troponin-I test was negative. The echocardiography showed degenerative heart disease and mild MR with an EF of 70%. The initial suggested diagnosis was recurrent angina.



Figure 7: A) The initial ICU ECG tracing showed NSR of VR; 80 bpm with ST-segment depressions in I, II, aVF (green arrows) and V4-6 leads (blue arrows). B) The second ECG tracing was done within 43 seconds of the first ECG and showed NSR of VR; 81 bpm with complete normalization of the above ST-segment depressions.

Case No. 6

A 64-years-old Egyptian Farmer married female patient presented to the POC with acute anginal chest pain and palpitations. Her vital signs were as follows: BP; 130/80 mmHg, HR; 74 bpm, RR; 16 bpm, temperature 36°C, and O₂ sat by pulse oximeter; 97%. The initial ICU ECG showed NSR of VR; 75 bpm with ST-segment depressions in I, aVF, and V4-6 leads. There are bigeminal premature ventricular contractions. (PVCs) (Figure 8A). The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 79 bpm with complete normalization of the above ST-segment depressions but with still PVCs (Figure 8B). The Troponin-I test was negative. The echocardiography showed LVH, mild AR, and MR with an EF of 62%. The initial suggested diagnosis was multi-formed bigeminal PVCs inducing angina.



Figure 8: A) The initial ICU ECG tracing showed NSR of VR; 75 bpm with ST-segment depressions in I, aVF (green arrows), and V4-6 leads (blue arrows). There are multi-formed bigeminal premature ventricular contractions (orange arrows). B) The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 79 bpm with complete normalization of the above ST-segment depressions but with still PVCs (orange and lime arrows).

Case No. 7

A 65-years-old, Egyptian, Farmer married female patient presented to the POC with acute anginal chest pain. She has chronic heart failure and ischemic cardiomyopathy. Her vital signs were as follows: BP; 140/70 mmHg, HR; 72 bpm, RR; 18bpm, temperature 36°C, and O₂ sat by pulse oximeter; 93%. The initial ICU ECG showed NSR of VR; 75 bpm with ST-segment depressions in II, III, aVF, and V4-6 leads (Figure 9A). The second ECG tracing was done within 60 seconds of the first ECG and showed sinus arrhythmia of VR; 76 bpm with complete normalization of the above ST-segment depressions (Figure 9B). The Troponin-I test was negative. The echocardiography showed ischemic cardiomyopathy (CMP), systolic dysfunction, and global hypokinesia with an EF of 42%. The initial suggested diagnosis was heart failure-associated angina.



Figure 9: A) The initial POC ECG tracing showed NSR of VR; 75 bpm with ST-segment depressions in II, III, aVF (green arrows) and V4-6 leads (blue arrows). B) The second ECG tracing was done within 43 seconds of the first ECG and showed sinus arrhythmia of VR; 76 bpm with complete normalization of the above ST-segment depressions.

A 48-years-old Egyptian Teacher married female patient presented to the POC with acute anginal chest pain, tachypnea, and peripheral numbness with paresthesia. There is a recent history of psycho-familial troubles. Her vital signs were as follows: BP; 100/80 mmHg, HR; 72 bpm, RR; 21 bpm, temperature 36.5°C, and O₂ sat by pulse oximeter; 97%. The tests for latent tetany were inducible. The initial emergency ECG showed NSR of VR; 70 bpm with ST-segment depressions in V2-6 leads (Figure 10A). The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 75 bpm with complete normalization of the above ST-segment depressions (Figure 10B). The Troponin-I test was negative. The echocardiography showed diastolic dysfunction with an EF of 68%. There is ionized hypocalcemia (0.8 mmol/L) The initial suggested diagnosis was angina post-psychogenic hyperventilation syndrome.



Figure 10: A) The initial POC ECG tracing showed NSR of VR; 74 bpm with ST-segment depressions in V2-6 leads (blue arrows). B) The second ECG tracing was done within 60 seconds of the first ECG and showed NSR of VR; 75 bpm with complete normalization of the above ST-segment depressions.

Case No. 9

A 47-years-old Egyptian housewife married female patient presented to the ICU with acute anginal chest pain. She has a hypertensive crisis and right hemiparesis. Her vital signs were as follows: BP; 190/130 mmHg, HR; 80 bpm, RR; 20 bpm, temperature 37.2°C, and O₂ sat by pulse oximeter; 96%. The initial ICU ECG showed NSR of VR; 82 bpm with ST-segment depressions in V4-6 leads (Figure 11A). The second ECG tracing was done within 80 seconds of the first ECG and showed NSR of VR; 81 bpm with complete normalization of the above ST-segment depressions (Figure 11B). The Troponin-I test was negative. There is ionized hypocalcemia (3.8 mg/dl) and hypomagnesemia (1.45 mg/dl) The echocardiography showed LVH, diastolic dysfunction, and mild MVP with EF of 66%. The initial suggested diagnosis was hypertensive crisis-associated angina.



Figure 11: A) The initial POC ECG tracing showed NSR of VR; 82 bpm with ST-segment depressions in V4-6 leads (blue arrows). B) The second ECG tracing was done within 80 seconds of the first ECG and showed NSR of VR; 93 bpm with complete normalization of the above ST-segment depressions.

Case No. 10

A 72-years-old Egyptian Farmer married male patient presented to the POC with acute anginal chest pain. He has chronic obstructive pulmonary disease and liver cirrhosis. His vital signs were as follows: BP; 130/80 mmHg, HR; 50 bpm, RR; 17 bpm, temperature 37.5°C, and O₂ sat by pulse oximeter; 94%. The initial POC ECG showed sinus bradycardia of VR; at 52 bpm with ST-segment depressions in V4-6 leads (Figure 12A). The second ECG tracing was done within 60 seconds of the first ECG and showed sinus bradycardia of VR; at 50 bpm with complete normalization of the above ST-segment depressions (Figure 12B). The Troponin-I test was negative. The echocardiography showed mild MR, diastolic dysfunction, and hypokinesia of IVS with an EF of 50%. The initial suggested diagnosis was hypocalcemia and hypomagnesemia-associated angina.



Figure 12: A) The initial POC ECG tracing showed sinus bradycardia of VR; at 52 bpm with ST-segment depressions in V4-6 leads (blue arrows). **B**) The second ECG tracing was done within 60 seconds of the first ECG and showed sinus bradycardia of VR; at 52 bpm with complete normalization of the above ST-segment depressions.

Case No.11

A 61-years-old Egyptian Driver married male heavy smoker patient presented to the ICU with acute anginal chest pain and dyspnea. He has a recent history of severe psychological stress. His vital signs were as follows: BP; 160/90 mmHg, HR; 72 bpm, RR; 22 bpm, temperature 36°C, and O₂ sat by pulse oximeter; 99%. The initial ICU ECG NSR of VR; was 75 bpm with ST-segment elevations in I, aVL, and ST-segment depressions in II, III, aVF, and V4-6 leads. (Figure 13A). The second ECG tracing was done within 72 seconds of the first ECG and showed NSR of VR; 78 bpm with still ST-segment depressions in II, III, and V5-6 leads (Figure 13B). The Troponin-I test was negative. The initial suggested diagnosis was psychological stress-associated angina.



Figure 13: A) The initial ICU ECG tracing showed NSR of VR; 75 bpm with ST-segment elevations in I, aVL (orange arrows), and ST-segment depressions in II, III, aVF (green arrows), and V4-6 leads (blue arrows). B) The second ECG tracing was done within 72 seconds of the first ECG and showed NSR of VR; 78 bpm with still ST-segment depressions in II, III (green arrows), and V5-6 leads (blue arrows).

For more details for all the study cases (Table 2) mentioned above.

RESULTS

Age

Averages; Range; 18 years - 82 years, Mean; 50.7, Mode; 70, Median; 51.5, Minimal; 18 years, Maximal; 82 years.

Sex

- Female sex; 52% (26 cases), in group I; 73.08% (19 cases), and in group II; 29.17% (7 cases).
- Male sex; 48% (24 cases), in group I; 26.92% (7 cases), and in group II; 70.83% (17 cases).

Occupation

Housewives: 34% (17 cases); Farmers: 26% (13 cases); Students: 12% (6 cases); Teachers: 6% (3 cases); Carpenters: 4% (2 cases); Officers: 4% (2 cases); Builders: 2% (1 case); Contractors: 2% (1 case); Doctors: 2% (1 case); Workers: 52% (1 case); Lawyers: 2% (1 case); Drivers: 2% (1 case); Retired: 2% (1 case) (Figure 14).



Figure 14: Bar chart presentation showing the occupations in the study.

Complaints

Angina: 84% (42 cases); Angina and palpitations: 10% (5 cases); Angina and tachypnea: 6% (3 cases) (Figure 15).



Figure 15: Pie chart presentation showing the percentage of complaints in the study.

Elapsed Time between Stage I and II/Sec

Averages: Range: 17 years - 250 years; Mean: 66.65; Mode: 60; Median: 60; Minimal: 17; Maximal: 250.

The Risk Factors

Single RF: 56% (28 cases); Multiple RFs: 44% (22 cases) (Figure 16) (Table 5).

The risk factors (RF)



Figure 16: Bar chart presentation showing the percentage of the associated risk factors in the study.

Cas	Risk factors	Cas	Risk factors	Cas	Risk factors	Cas	Risk	Cas	Risk
e No		e No		e No		e No	factors	e No	factors
1	Smoker	11	Psychologica l stress- smoker	21	Elder- hypertensive crisis-DM	31	Sinus bradycardi a	41	HTN crises
2	Psychogenic stress	12	Elder-aortic aneurysm	22	Perioperative preparation	32	Sinus bradycardi a	42	Psychogeni c HVS
3	Elder-silent MI- ibuprofen	13	Tight mitral stenosis	23	Psychological stress- hypocalcemia	33	Elder- PSVT	43	Psychologi cal hemiplegia
4	Psychogenic HVS	14	Elder- psychologica l stress	24	Elder-Psychogenic stress	34	Psychologi cal stress	44	Psychologi cal hemiplegia
5	Elder-metastatic carcinoma- COVID-19 pneumonia	15	Perioperative preparation	25	Elder-silent MI- ibuprofen	35	Brugada syndrome	45	HTN crises
6	Elder-bigeminal PVCs	16	Psychologica l stress	26	Elder-metastatic carcinoma- COVID-19 pneumonia	36	Psychologi cal stress	46	Psychogeni c HVS
7	Elder-heart failure- CMP	17	CVA post- accident	27	HTN crises-MI- Brugada syndrome	37	Hypocalce mia	47	Misdiagnos is of angina
8	Psychogenic HVS	18	Pregnancy	28	Psychogenic HVS	38	Elder- PVCs	48	Psychologi cal stress
9	Hypertensive crisis- CVA	19	Elder- hypertensive crisis	29	Familial SCD with LQTS	39	Psychogeni c HVS- PVCs	49	Psychogeni c HVS

10	Elder-hypocalcemia-	20	Sinus	30	Psychological stress	40	Elder-HTN	50	Hiatus
	hypomagnesemia-		tachycardia				crises		hernia
	COPD								

Table 5: The associated risk factors; n:50.

The Response to Stressors

Complete (only group I): 40% (20 cases); Partial (only group I): 12% (6 cases); Absent (only group II): 48% (24 cases) (Figure 17).

The Response to Stressors



Figure 17: Bar chart presentation showing the response to stressors in the study.

DISCUSSION

The mean age in the current study is 50.7 in 18 years - 82 years, with slight female sex predominance (52%). Housewives (34%) and Farmers (26%) are the most affected occupations (Figure 14). The main complaint is angina (84%) followed by angina with palpitations (10%) (Figure 15). The most common associated risk factors in the study are combined risk factors (56%) followed by single risk factors (44%) (Figure 16). The Mean of elapsed time between stage I and II/sec (only group I) are 66.65 in a range; 17 years - 250 years (Table 4). The response was complete (only group I; 40%), Partial (only group I; 12%), and Absent (only group II; 48%) (Figure 17).

Definition

ST depression is an ECG finding in which the ST segment is abnormally deviated below the isoelectric line [1,2].

The electrocardiographic causes of ST segment depression

The ST-segment depression (STD). STD is usually associated with acute coronary syndromes (ACS). This ECG STD may also be present in patients with left ventricular hypertrophy with a strain, therapeutic digitalis levels [3], functional or physiologic variants (e.g., postprandial) [4], hyperventilation syndrome, anemia, tricyclics antidepressant, antiarrhythmic drugs [5], electrolyte imbalance such as (severe hypokalemia and hypomagnesemia), right ventricular hypertrophy with a strain, conduction abnormalities with secondary ST segment changes such as (right bundle branch block, left bundle branch block, and Wolf-Parkinson-White syndrome), some cases of mitral valve prolapse [6], some diseases of the central nervous system such as stroke [7], ventricular paced rhythm, supraventricular tachycardia, reciprocal change in ST-segment MI, and posterior MI [8].

Non-specific and types of ST-segment depression

Non-specific ST-segment depression are very common ECG finding. It may be detected in any lead of the ECG. The changes may be seen in all or most of the leads (diffuse changes), or they may be present in contiguous specific leads, such as the inferior, lateral, or anterior leads. There are three different types of ST-segment depression; horizontal or straightening ST-segment depression, up-slopping ST-segment depression, and down-slopping ST-segment depression [4].

Magnitude prognostic significance of ST segment depression

ST-segment depression is measured from the isoelectric baseline, or when ST-segment depression is present at rest, the amount of additional depression is measured [9]. ST segment depression is considered significant if the ST segment is vertically at least one-half of a small box below baseline with horizontally two boxes after the end of the QRS. In myocardial infarction, the affected leads with ST depression will be reflected in the location of the myocardial ischemia. ST-segment depression is the most specific ECG issue for cardiac ischemia if the ST-segment is sloping down from the J-point. Horizontal or down-sloping ST depression \geq 0.5 mm at the J-point in \geq 2 contiguous leads indicate myocardial ischemia. Horizontal or flat STs are also quite suspicious for ischemia. Up-sloping ST depression is only about 60% accurate for diagnosing ischemia. While ST depression as minimal as 0.5 mV has been demonstrated to reflect myocardial ischemia and unfavourable prognosis, greater magnitudes of depression predict higher short-term mortality [8]. ST-segment depression on initial ECG is strongly associated with unfavourable outcomes in NSTE-ACS and is a major independent prognostic factor in most risk prediction models. Its presence at admission has been shown to significantly correlate with short-term and long-term (\geq 1 year) mortality [10]. Resting ST-segment depression of <1 mm has been shown to increase the sensitivity but to decrease the specificity of exercise testing [11,12].

Considerations in a positive traditional exercise treadmill test

Traditionally, a positive exercise treadmill test (ETT) is defined as the appearance of symptoms (significant dyspnea, chest tightness, fatigue, dizziness, syncope), progressive PVCs >3 beats, new AF, and a blunted BP response (a sustained fall in systolic BP >20 mmHg from the previous stage or a failure to rise from the baseline level) [13]. In a cardiac stress test, an ST depression of at least 1 mm post adenosine administration indicates reversible ischemic heart disease (IHD), while an exercise stress test requires an ST depression of at least 2 mm to significantly indicate reversible IHD [12].

Anxiety and Reactivity

Anxiety is a subjective feeling of unease, discomfort, apprehension, or fearful concern accompanied by a host of autonomic and somatic manifestations [14]. Anxiety is a universal and generally adaptive response to a threat, but in some circumstances, it can become maladaptive [15]. The sympathetic nervous system mediates the symptoms of anxiety [16]. Increased sympathetic activity may be implicated in the pathogenesis of anxiety [17].

Anxiety and cardiovascular disease

Anxiety is common in patients with cardiovascular disease, such as coronary artery disease (CAD). Following an ACS, 20-30% of patients experience elevated levels of anxiety [18,19]. "It's my view and my personal clinical experience that anxiety disorders can play a major role in heart disease," says McCann. "I believe that a really careful look at anxiety would reveal the ways it can severely impact heart disease, both as a contributing factor and as an obstacle in recovery" [20].

Pathogenesis of IHD in anxiety

Interestingly, serotonin has been shown to increase platelet aggregation and may be a method by which anxiety disorders are associated with increased cardiac events, as anxiety disorders have been associated with abnormalities in the serotonin system [21,22]. In general, patients with anxiety and acute stress have greater platelet aggregation, and anxiety disorders, may also be associated with changes in platelet activity [23,24]. Anxiety and its associated disorders are common in patients with cardiovascular disease and may significantly influence cardiac health. Anxiety disorders are associated with the onset and progression of cardiac disease, and in many instances have been linked to adverse cardiovascular outcomes, including mortality. Both physiologic (autonomic dysfunction, inflammation, endothelial dysfunction, changes in platelet aggregation) and health behavior mechanisms may help to explain the relationships between anxiety disorders and cardiovascular disease. Given the associations between anxiety disorders and poor cardiac health, the timely and accurate identification and treatment of these conditions is of the utmost importance. Fortunately, pharmacologic and psychotherapeutic interventions for the management of anxiety disorders are generally safe and effective. Negative psychological states are commonly experienced among patients with cardiovascular disease. The relationship between anxiety and cardiovascular health is complex. Anxiety may be a normal response to a stressful situation, such as an acute cardiac event [25].

Anxiety disorders and ECG

Anxiety disorders may be due to sympathetic overactivity, which plays a pivotal role in ECG changes [26].

Anxiety and studies in cardiovascular disease

Anxiety has been associated with the incidence, and in some cases progression, of cardiovascular disease. In patients without existing cardiac disease, anxiety has been linked to the subsequent development of CAD. In a 2010 meta-analysis including 20 studies and nearly 250,000 patients, **Roest and colleagues** found that anxiety, controlling for other medical variables, when possible, led to a 26% increased risk of incident CAD. Anxiety seemed to be an independent risk factor for incident CHD and cardiac mortality [27].

In a recent meta-analysis of 44 studies, anxiety was significantly associated with poor cardiac outcomes, including recurrent cardiac events and mortality, in unadjusted analyses [28].

Calm and fear with reassurance

Re-assurance, sitting in calm, quickly analyzing the problem, and taking an appropriate active decision, one can convert bad stress (distress) into good stress (eustress) [26]. Psychotherapy may reduce stress and manage myocardial ischemia [29,30].

Coronary Artery Spasm

The coronary artery spasm (CAS) is a cardiovascular disorder that describes a sudden, transient, severe contraction and narrowing of an epicardial coronary artery that ends with vascular occlusion or near-occlusion [31,32]. In other words, there is a diminution or entire blockage of blood flow with acute cardiac ischemia [33]. A CAS has a pivotal role in the pathogenesis of coronary heart disease and sudden heart death [34]. The exact mechanisms of CAS are still idiopathic but mostly multifactorial [35]. Coronary artery spasms can be implicated by tobacco smoking, exposure to cold extremes, and an imbalance between myocardial O_2 supply and demand [36]. Fear, anger, and stress (of any type) [33] are primary keys for the attacks [37]. The stress is of any type [33]. Coronary artery spasms may be "silent", or they may result in chest pain or angina. Approximately, 2% of angina have CAS [38]. CAS has mostly ST-segment depression rather than ST-segment elevation on electrocardiography [39]. Coronary angiography is the gold standard for the diagnosis of CAS [40]. The prevention of implicated risk factors is the first line of therapy [31]. Treatment of CAS may include medications such as (1) Nitrates are used to prevent spasms and quickly relieve chest pain. (2) Calcium antagonists relax the arteries and decrease the spasm. (3) Statin also may prevent spasms [41]. Long-acting nitrates and calcium antagonists are the cornerstone drugs of choice in preventing future CAS episodes [39].

Unfortunately, there are no available relevant studies for Yasser's stressor test (fear-calm stressor test) and Triphasic Yasser's syndrome (fear-calm-fear Yasser's syndrome).

Yasser's Stressor test (Fear-Calm Stressor test) and Triphasic Yasser's syndrome (Fear-Calm-Fear Yasser's Syndrome)

Yasser's Stressor test is a new cardiovascular discovery and psychogenic stress test targeting the ST-segment passing two stages in the form of fear and calm (Fear-Calm Stressor test). Triphasic Yasser's syndrome is a sequel and constellation of the vicious cycle of Yasser's Stressor test passing three stages in the form of fear, calm, and fear (Fear-Calm-Fear Yasser's syndrome). Coronary artery spasm is a possible pathogenesis for interpretation. Both Yasser's Stressor test and its subsequent Triphasic Yasser's syndrome are easy, simple, cheap, and safe non-invasive exercise tests.

They are innovative clinical and electrocardiographic tests in cardiovascular science.

These tests are used in cases of acute chest pain.

It is indicated in the cases of angina to explore the presence of ECG ST-segment depressions and subsequent normalization after applying either the above active or passive stressors.

Both Yasser's Stressor test and its subsequent Triphasic Yasser's syndrome are easy, simple, cheap, and safe non-invasive new exercise tests.

Principal of Yasser's Stressor test and Triphasic Yasser's syndrome; this is based on two points;

- Inducible ECG ST-segment depressions if the patient exposed to the above stressors (active or passive) happened
- Normalization of the ECG ST-segment depressions after removal of the above stressors (active or passive) happened
- The elapsed time between stage I and II/Sec (only group I) is calculated from the time on the ECG tracings.
- The response is either complete, partial, or absent (Table 4 and Figure 17).
- or stages of; (inducible ECG ST-segment depressions rormalization of the ECG ST-segment
- depressions again inducible ECG ST-segment depressions) and so on.

• Horizontal, down-sloping, up-slopping ST depression ≥ 0.5 mm at the J-point in ≥ 2 contiguous leads is considered as a positivity for the test in the author's opinion.

The cases were independently managed according to age, associated risk factors, and clinical status.

Interpretation

- Inducible ECG ST-segment depressions after exposure to active or passive stressors mostly due to anxiety and fear.
- Reversible ECG ST-segment depressions after removal of active or passive stressors mostly due to reassurance or calm.
- Activity of sympathetic and parasympathetic nervous systems is probably implicated in both inducible ECG ST-segment depressions after exposure to active or passive stressors.
- Coronary artery spasm is the results of the above suggestions.

Disadvantages

There are no known reported drawbacks to both Yasser's Stressor test and Triphasic Yasser's syndrome.

CONCLUSION AND RECOMMENDATIONS

- Yasser's Stressor test is a new cardiovascular discovery and psychogenic stress test targeting the ST segment passing two stages in the form of fear and calm (Fear-Calm Stressor test).
- Triphasic Yasser's syndrome is a sequel and constellation of the vicious cycle of Yasser's Stressor test passing three stages in the form of fear, calm, and fear (Fear-Calm-Fear Yasser's syndrome).
- Coronary artery spasm is a possible pathogenesis for interpretation.
- Both Yasser's Stressor test and its subsequent Triphasic Yasser's syndrome are easy, simple, cheap, and safe non-invasive exercise tests.
- Widening the research for clarification of both specificity and will be recommended.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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