

## Case Report

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### Corresponding Author:

**Yasser Mohammed Hassanain Elsayed\***

Damietta Health Affairs - Egyptian Ministry of  
Health, Egypt.

## Test the Vasospastic ST-Segment Depression Reversibility with Nitrates in Non- Obstructive Coronary Disease

### Abstract:

coronary artery spasm is an important common clinical cardiovascular disorder. It is defined as a transient constriction of the muscles in the wall of one or more of the coronary arteries. Myocardial injury and sudden cardiac death are possible remarkable sequels. Despite coronary spasm is a brief and temporary but can be permanent. In other words, it may be associated with or without significant coronary artery disease. However, Coronary spasm is hyperresponsive to the vasodilator effect of nitrates. Patients with different forms of coronary artery disease may respond differently. The quick reversibility response of ST-segment depression after using short-acting nitrates had achieved in my 3-case series. ST-segment depression reversibility with nitrates means that the ST-segment depressions for all cases were transient coronary vasospasm. However, coronary angiography was normal in all 3-cases. This is the principal of our test. Worthwhile, the objective of the test in the current article is to distinguish between the real coronary artery spasm with obstructive coronary artery disease from those with non-obstruction. The emergency differentiation for the cases of coronary artery spasm using nitrates via categorical stratification into obstructive and non-obstructive coronary artery disease is a pivotal step in future management. The test may reduce the need for coronary angiography and its economic impact on the patient.

**Key words:** Vasospastic St-Segment Depression; Reversibility; Coronary Artery Spasm; Nitrates; Non-Obstructive Coronary Disease.

**Abbreviations:** CAS; Coronary artery spasm, ECG: Electrocardiography, ICU; Intensive care unit, IHD; Ischemic heart disease, IVI; Intravenous infusion, MI; myocardial infarction, PCI: Percutaneous coronary intervention, SCD; Sudden cardiac death, UA; Unstable angina

### INTRODUCTION

Coronary artery spasm (CAS) is an abnormal contraction of an epicardial coronary artery resulting in myocardial ischemia [1]. CAS plays an important role in the pathogenesis of IHD, including stable angina, UA, MI, and

SCD [2]. The attacks of coronary spasm are associated with either ST-segment elevation or depression on ECG [2]. Most CAS is associated with ST-segment depression rather than ST-segment elevation [3]. CAS may also occur in angiographically normal coronary arteries as so-called 'variant of the

variant [4]. CAS appears to be a multifactorial disease [5]. The morbidity increases annually in lifestyle-related diseases, such as hypertension, diabetes mellitus, and dyslipidemia [6]. Calcium antagonists are the cornerstone of medical treatment of CAS [5]. Coronary angiography is usually required to establish a definitive diagnosis [5].

Episodes are usually brief and rapidly interrupted by administration of nitrates [7]. Nitrates produce vasodilation with predominant venous effects on large capacitance vessels. They also increase coronary collateral circulation, increase aortic compliance and conductance and blood flow to ischemic areas of the myocardium. In addition, nitrates alleviate anginal symptoms by directly influencing the coronary arteries, coronary collateral circulation, aortic compliance and conductance, and blood flow to ischemic areas of the myocardium [8]. The ECG changes are transient, reversible with vasodilators [9]. As the changes are due to coronary artery spasm rather than acute infarction, they may be completely reversible if treated promptly [10]. It may be impossible to differentiate these two conditions based on ECG alone [9]. Short-acting nitrates are beneficial in acute myocardial ischemia [11]. Although nitroglycerin has been shown to be clinically effective in the therapy of this condition, its exact mechanism of action is still uncertain [12]. Nitroglycerin is the oldest and most commonly prescribed short-acting anti-anginal agent. Despite being in clinical use since 1879, there remains an important need to educate both patients and health care providers on the various benefits of short-acting nitrates [8]. Spasm artery is hyperresponsive to the vasodilator effect of nitroglycerin [13].

- The primary objective for my case series was exploring the presence of ST-segment depression that indicating ischemic heart disease.

- The secondary objective for my case-series was proofing that there was complete normalization of ST-segment depressions after using short-acting nitrates. Normal coronary angiography will confirm the test benefits. The objective of the test in the current article is to distinguish between the real coronary artery spasm with obstructive coronary artery disease from those with non-obstruction. The emergency differentiation for the cases of coronary artery spasm using nitrates via categorical stratification into obstructive and non-obstructive coronary artery disease is a pivotal step in future management. The test may reduce the need for coronary angiography and its economic impact on the patient.

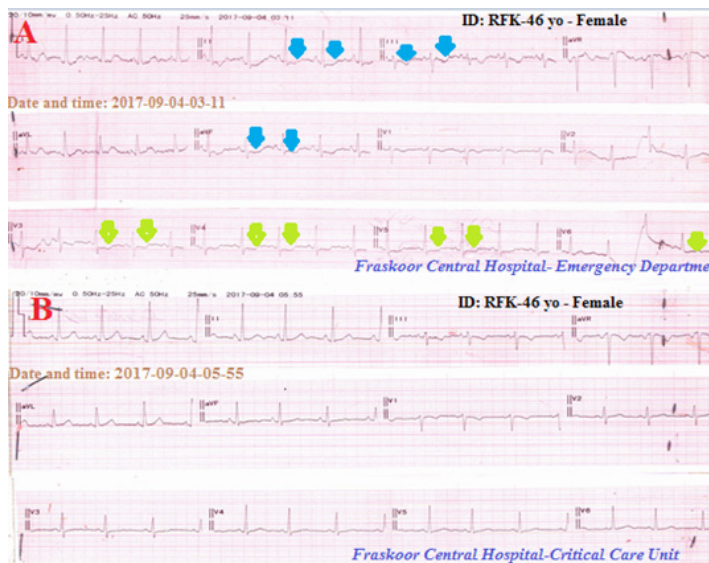
## CASE PRESENTATIONS

- **Case No. 1:** A 46-year-old married, housewife, Egyptian female patient presented with anginal chest pain. She was admitted in ICU as a hypertensive emergency with unstable angina. The patient denied the history of smoking, drugs or any special habits. She was hypertensive patient. Diagnostic PCI for the same attack was done two months ago. No coronary abnormality revealed. Upon physical examination; the patient was in good general condition, with a regular heart rate of 90 bpm, blood pressure of 200/130 mmHg, the temperature of 36.4 °C, and normal pulmonary and heart auscultation. No more relevant clinical data were noted during the clinical examination. The case was managed initially as a hypertensive emergency with angina with nitroglycerin IVI (5 ug/kg/m). Initial ECG tracing showed ST-segment depression in leads II, III, aVF and V3-5 (Figure 1A). The second ECG was done 90 minutes of the first one showed complete normalization of the above ST-segment depressions (Figure 1B). Troponin T test was negative (< 0.001 ng/mL). Echocardiography was completely normal. Coronary angiography was repeated but with no abnormalities detected. The case discharged within 12-hours post-controlling the hypertensive emergency. She continued on oral nitroglycerine (2.5 mg, twice daily) and amlodipine (5 mg, once daily).

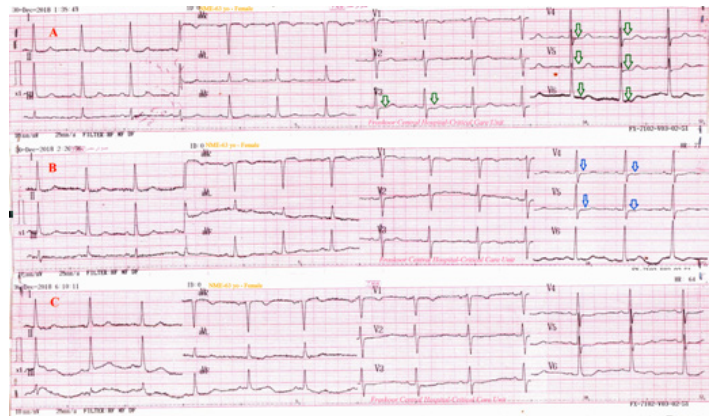
- **Case No. 2:** A 40-year-old married, teacher, Egyptian female patient presented with anginal chest pain. The patient denied the history of smoking, drugs or any special habits. She was hypertensive patient on oral captopril (25 mg twice daily). She refused the hospital referral. Upon physical examination; the patient was in good general condition, with a regular heart rate of 98 bpm, of blood pressure of 160/110 mmHg, and temperature of 37.1 °C. Clinical examination was unremarkable. Initial ECG tracing showed ST-segment depression in V1-5 leads (Figure 2A). The case was only managed with sublingual isosorbide dinitrate tablet (5 mg). The second ECG tracing was taken within 30 minutes of the first ECG. It showed complete normalization of the above ST-segment depressions (Figure 2B). Troponin T test was negative (< 0.001 ng/mL). Echocardiography was completely normal. Within 2 weeks of presentation, the patient had undergone coronary angiography, but there were no detected abnormalities. The case was followed up for 2-hours after relieving of chest pain rather than the normalization of ECG. She continued on oral nitroglycerine (2.5 mg, twice daily) and diltiazem (60 mg, once daily).

- **Case No. 3:** A 62-year-old married, housewife, Egyptian

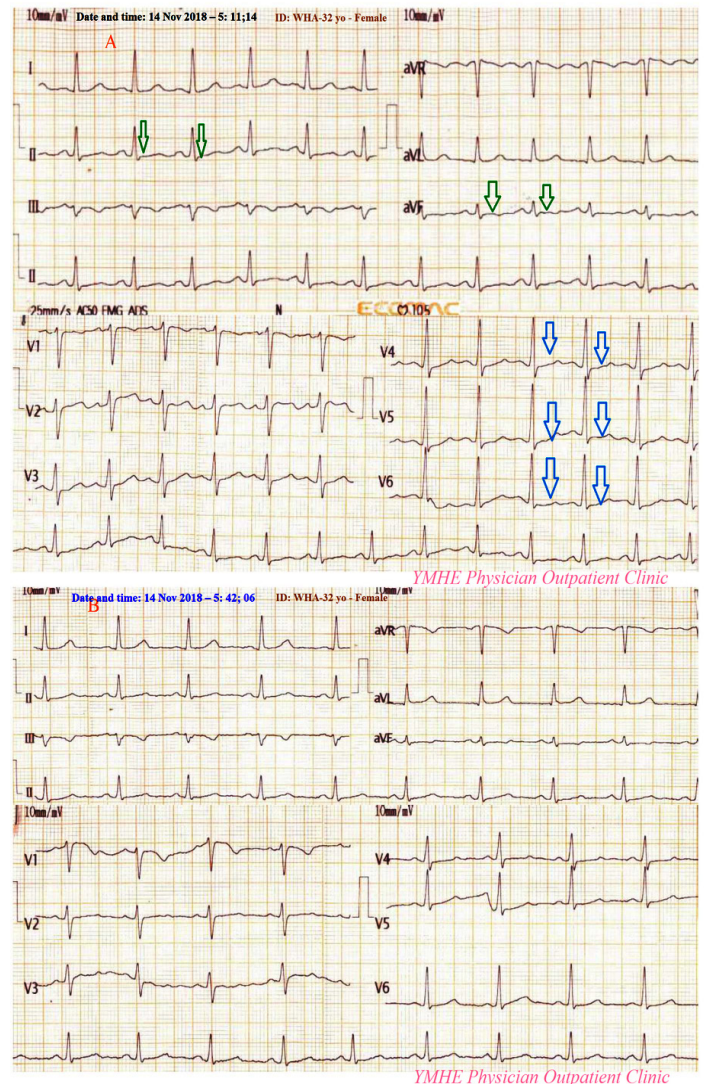
female patient presented with anginal chest pain and dizziness. She was admitted in ICU as unstable angina. The patient denied the history of smoking, drugs or special habits. Upon physical examination; the patient was in good general condition, with a regular heart rate of 84 bpm, blood pressure of 140/80 mmHg, and temperature of 36.9 °C, and normal pulmonary and heart auscultation. Clinical examination was unremarkable. Initial ECG tracing showed ST-segment depressions in ECG leads II, III, aVF (Figure 3A). The case was only managed with sublingual isosorbide dinitrate tablet (5 mg). The second ECG tracing was taken within 60 minutes of the first ECG. It showed complete normalization of above ST-segment depressions (Figure 3B). Troponin T test was negative ( $< 0.001$  ng/mL). Echocardiography was completely normal. Coronary angiography was done but there were no detected abnormalities. The case discharged within 12-hours post-relieving of chest pain rather than the normalization of ECG. She continued on oral nitroglycerine (2.5 mg twice daily) and nifedipine (10 mg once daily).



**Figure 1:** A-ECG tracing on presentation showing ST-segment depressions in Leads: II, III, aVF (blue arrows), and V3-6 (green arrows). B -ECG tracing was taken within 150 minutes after NTG IVI showing normalization of the above ST-segment depressions in Leads: II, III, aVF, and V3-6.



**Figure 2:** A-ECG tracing on presentation showing ST-segment depressions in Leads: V3-6 (green arrows). B-ECG tracing was taken within 50 minutes after NTG IVI showing nearly normalization of previous ST-segment depression in Leads: V3-6 (blue arrows). C-ECG tracing was taken within 5 hours after NTG IVI showing complete normalization of the above ST-segment depressions.



**Figure 3:** A-ECG tracing on presentation showing ST-segment depressions in Leads: II, aVF (green arrows), and V4-6 (blue arrows). B-ECG tracing was taken within 50 minutes after sublingual isosorbide dinitrate tablet (5 mg) showing complete normalization of the above ST-segment depressions.

## DISCUSSION

### Overview:

• All three cases of the current research had presented with angina. Presence of ST-segment depressions in specific ECG leads was characteristic for the cases. Complete normalization of ST-segment depressions after using short-acting nitrate had happened. The ECG changes were transient and reversible indicating the presence of non-obstructive coronary artery spasm. Normal troponin T, echocardiography, and coronary angiography meaning the lesion in affected coronary artery, not a fixed or non-obstructive. The author can't compare the current cases with past similar conditions. There are no similar or known cases were taken the same management. The relevant previously published papers had only signified the response of coronary artery spasm to nitrates. An ignorance of categorizing the underlying lesion into obstructive and non-obstructive coronary artery spasm was the defect in these publications. Nitrates were only given as an effective treatment for episodes of vasospastic angina. Nitrates never prescribed as a differentiated test for both possible conditions [5,14-17].

• Study question here; How did the test impact in differentiation between obstructive and non-obstructive coronary artery spasm?

• Study method was the observational case-study.

• Limitations of the study:

• There are no known limitations to the study. But, the presumption of possible coronary spasm during the coronary angiography and contraindications of nitrates especially hypotension are possible limitations.

• Recommendations

• It is recommended to widening the research in clearing the effect of nitrates for the differentiation between obstructive and non-obstructive coronary artery spasm.

• Also, it is recommended to extend the researching in the impact of nitrates; on specifically obstructive coronary artery disease.

### CONCLUSIONS

• Nitrates may be used as a test to differentiate between obstructive and non-obstructive coronary artery spasm.

• Nitrates may be used as a test to verify the ST-segment depression of coronary vasospasm from others.

• The average time for response is usually within 30 minutes

of taken nitrates.

• The initial recommended dose for nitrates is; 5 ug/kg/m for IVI nitroglycerin and sublingual 5 mg for isosorbide dinitrate tablet.

• Differentiation for ST-segment depressions in ECG will reduce the indications for coronary angiography and its economic impact on the patient.

• The test using nitrates is easy, cheap, and effective.

• Widening the research in this field using different nitrate preparations will be recommended. Also, in the impact of nitrates; on specifically obstructive coronary artery disease.

### Conflicts of Interest

There are no conflicts of interest.

### ACKNOWLEDGMENT

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### REFERENCES

1. Yasue H, Omote S, Takizawa A, Nagao M. (1983). Coronary arterial spasm in ischemic heart disease and its pathogenesis. A review. *Circ Res* 52( Suppl. I): 147-59.
2. Hirofumi Y, Hitoshi N, Teruhiko I, Eisaku H, et al. (2008). Coronary artery spasm-Clinical features, diagnosis, pathogenesis, and treatment. *Journal of Cardiology*. 51:2-17.
3. Ming JH. (2017). Fluctuations in the amplitude of ST-segment elevation in vasospastic angina. *Medicine (Baltimore)*. 96(11): e6334.
4. Liu PC, chi-Wen C, Ming-jui H, shih-Jen C, et al, (2010). Variant Angina with Angiographically Normal or Near-normal Coronary Arteries: A 10-year Experience. *J Intern Med Taiwan*. 21:79-89.
5. Ming JH, Patrick H and Ming YH. (2014). Coronary Artery Spasm: Review and Update. *Int J Med Sci*. 11(11): 1161-1171.
6. Ishii M, Kaikita K, Sato K, Yamanaga K, et al, (2016). Changes in the risk factors for coronary spasm. *IJC Heart & Vasculature*. 12:85-87.
7. G Coppola, P Carità, E, Corrado E, Borrelli A, et al. (2013). ST segment elevations: Always a marker of acute myocardial infarction?. *Indian Heart J*. 65(4): 412-423.

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8. Boden WE, Padala SK, Cabral KP, Buschmann IR, et al. (2015). Role of short-acting nitroglycerin in the management of ischemic heart disease. *Drug Des Devel Ther.* 19(9): 4793-805.
  9. Burns Ed. (2017). *The ST Segment.*
  10. June E, William JB and Francis M. (2002). Acute myocardial infarction-Part II. ABC of clinical electrocardiography, *BMJ.* 324(7343): 963-966.
  11. Giuseppe C, Paul J and Hans U I. (2015). Use of nitrates in ischemic heart disease. *Expert Opin Pharmacother.* 16(11): 1567-72.
  12. James AM and Richard C. (1978). Topical Nitroglycerin for Ischemic Heart Disease. *JAMA.* 239(20): 2166-2167.
  13. Yasue H and Kugiyama K. (1997). Coronary spasm: clinical features and pathogenesis. *Intern Med.* 36(11):760-5.
  14. Stern S and Bayes de Luna A. (2009). Coronary Artery Spasm A 2009 Update. *Circulation.* 119: 2531-2534.
  15. Lanza GA, Careri G and Crea F. (2011). Mechanisms of Coronary Artery Spasm. *Circulation.* 124: 1774-1782.
  16. Morikawa Y, Mizuno Y, Harada E, Kuboyama O, et al. (2010). Nitrate tolerance as a possible cause of multidrug-resistant coronary artery spasm. *Int Heart J.* 51(3): 211-3.
  17. Davies O, Ajayeoba O and Kurian D. (2014). Coronary artery spasm: An often overlooked diagnosis. *Niger Med J.* 55(4): 356-358.