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Case Report

Unmasking Vasospastic Angina Pectoris Using Dobutamine Stress-Echocardiography

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Abstract

A 50 years old man was presented to our emergency department complaining of persistent retrosternal chest pain in the last 3 hours. He described similar episodes in the past couple of years. His ECG showed negative T- Waves in the precordial leads (V2-V6) with minimal T-wave changes in the peripheral leads. His echocardiographic findings were within normal. High sensitive Troponin was minimally elevated with normal creatinine kinase (CKMB). Angiographically we identified a coronary artery disease with intermediate ostial LAD stenosis with a negative iFR value (0.98). A complementary cardiac MRI excluded any relevant pericardial and myocardial diseases. We carried out stress echocardiography trying to reproduce his subjective symptoms and to re-assess the clinical findings. Directly before the investigation, we observed a complete recovery of the T-wave changes. During the investigation, starting from stage 3, he reported severe chest pain similar to his recurrent experience. In the ECG we observed horizontal precordial ST-segment depression. Echocardiographically we saw apical and anteroseptal hypokinesia. The patient was given sublingual nitroglycerin with complete recovery of his ECG, echocardiographic findings and chest pain. He was sent home on calcium canal blockers and thorough advice on lifestyle modification.

Introduction

In 1959, Dr. Myron Prinzmental was the first to describe a new form of exercise-independent angina and named it a "variant form of angina pectoris", which is now more commonly referred to as vasospastic angina (VSA) [1]. The symptoms are usually associated with reversible ST-segment elevation on the ECG, which relieve promptly under nitroglycerin. Coronary spasm is a heterogeneous phenomenon that can occur in patients with or without coronary atherosclerosis and can be focal or diffuse, sometimes with changing patterns in the same patient [2]. VSA is still not fully understood owing to its complex pathophysiology. Traditionally thought that VSA is completely benign. However, it may be associated with many, though uncommon, clinical scenarios such as stable angina, sudden cardiac death, acute coronary syndrome, arrhythmia or syncope [3,4].

The prevalence of VSA is diffusely heterogeneous and may also vary depending on the eagerness of the practician to investigate VSA, and on the provocative tests, which may differ from one catheterization laboratory to another. Nevertheless, coronary spasm is more frequent than one might think, and may still be underdiagnosed. In a study evaluating the prevalence of pericardial and microvascular coronary spasm in patients with angina symptoms, despite angiographically unobtrusive coronary arteries, the coronary spasm was present in 62% of patients; 45% with pericardial spasm and 55% with microvascular spasm [5]. Using the same design, a multicentre Japanese study evaluated coronary spasm prevalence in patients with non-ST-segment elevation acute coronary syndrome. Out of 1601 patients, 72% had a culprit lesion; among the remaining patients, the acetylcholine provocation test was performed in 221 patients and was positive in 175 (79.2%) patients [6]. It has been reported that the occurrence of cardiovascular events was significantly higher in VSA patients with a multi-vessel spasm

during the ergonovine provocation test than in the single-vessel VSA and non-VSA group [7,8]. It is generally recommended to carry out a provocation test with invasive coronary angiography to identify the location and characteristics of coronary artery spasms. In our case, we applied dobutamine stress-echocardiography as a provocative maneuver instead of intracoronary provocation.

Description

A 50 years old man was presented to our emergency department complaining of persistent retrosternal chest pain in the last 3 hours. He described similar episodes in the past couple of years. His ECG showed negative T- Waves in the precordial leads (V2-V6) with minimal T-wave changes in the peripheral leads. His echocardiographic findings were within normal. High sensitive Troponin was minimally elevated with normal creatinine kinase (CKMB). Angiographically we identified a coronary artery disease with intermediate ostial LAD stenosis with a negative iFR value (0.98). A complementary cardiac MRI excluded any relevant pericardial or myocardial diseases. We carried out stress echocardiography trying to reproduce his subjective symptoms and to re-assess the clinical findings. Directly before the investigation, we observed a complete recovery of the T-wave changes. During the investigation, starting from stage 3, he reported severe chest pain similar to his recurrent experience. In the ECG we observed horizontal precordial ST-segment depression. Echocardiographically we saw apical and anteroseptal hypokinesia. The patient was given sublingual nitroglycerin with complete recovery of his ECG, echocardiographic findings and chest pain. He was sent home on calcium canal blockers and thorough advice on lifestyle modification.

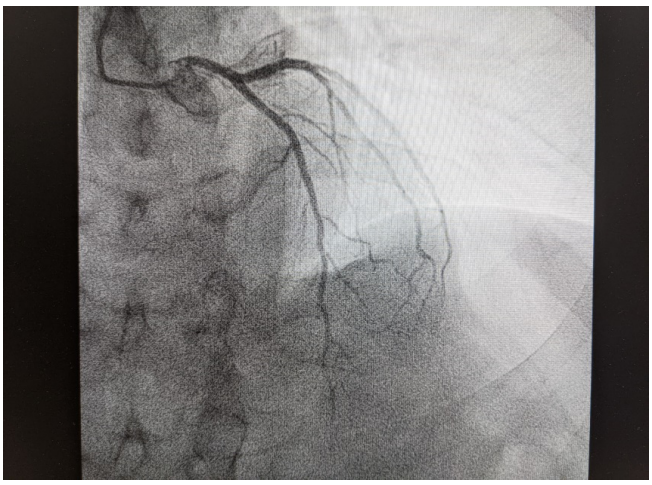

Figure 1: Left Coronary Artery Angiography without Critical Lesion.

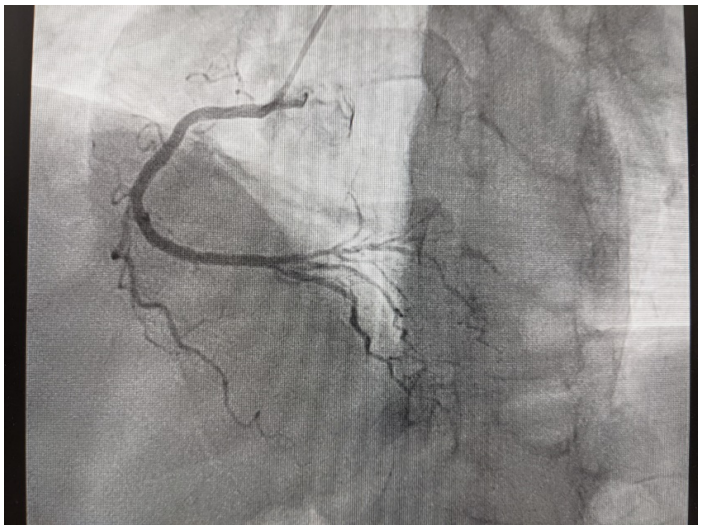

Figure 2: Right Coronary Artery Angiography without Critical Lesion.

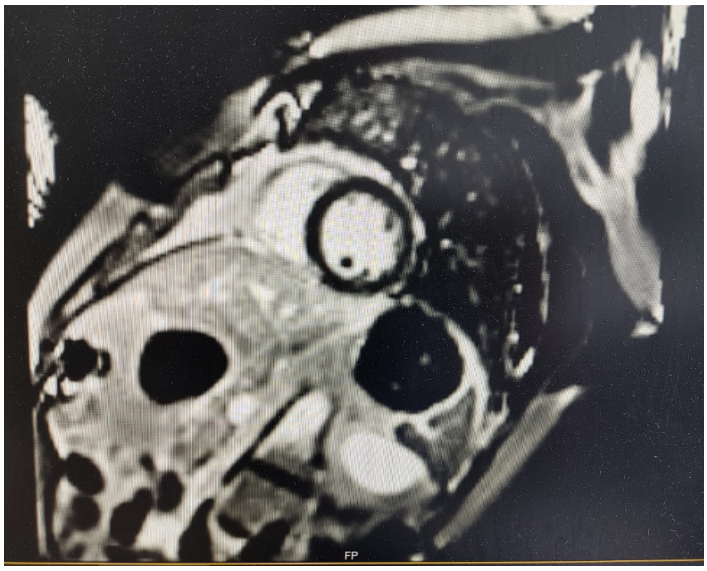

Figure 3: Cardiac MRI (example of a short-axis view phase-sensitive inversion recovery) showed normal left ventricular wall thickness and no late contrast enhancement.

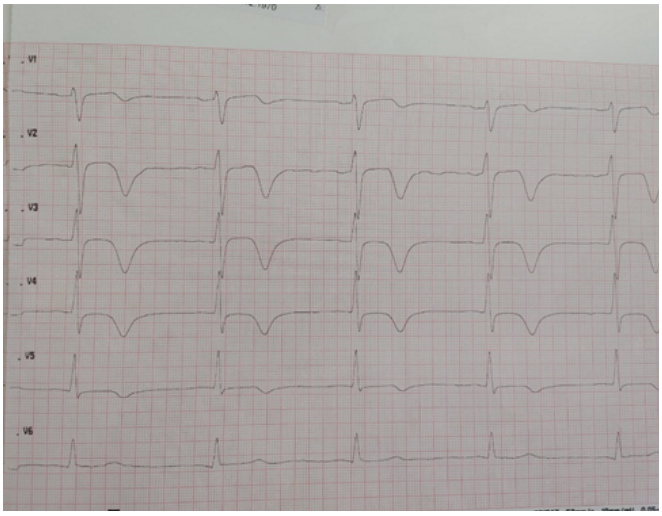

Figure 4: Precordial Leads Registered in the Emergency Department.

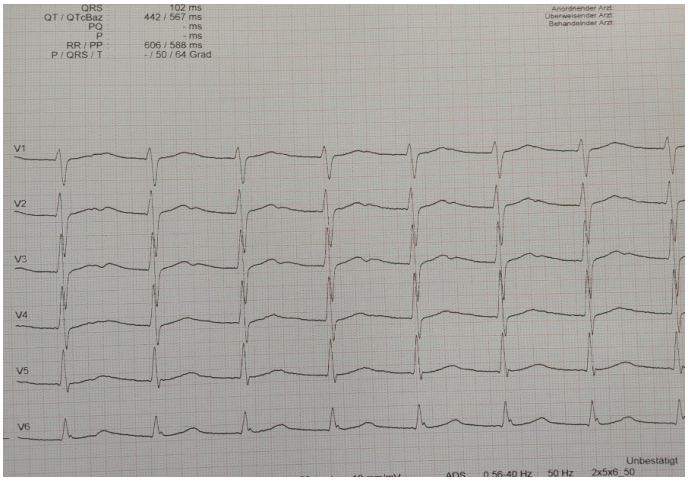

Figure 5: Precordial Leads Registered at Rest.

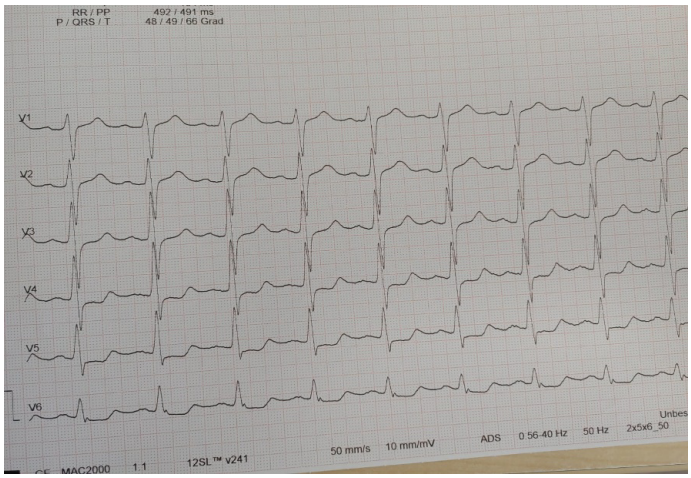

Figure 6: Precordial Leads Registered During Stress Echocardiography, Associated with Severe Chest Pain.

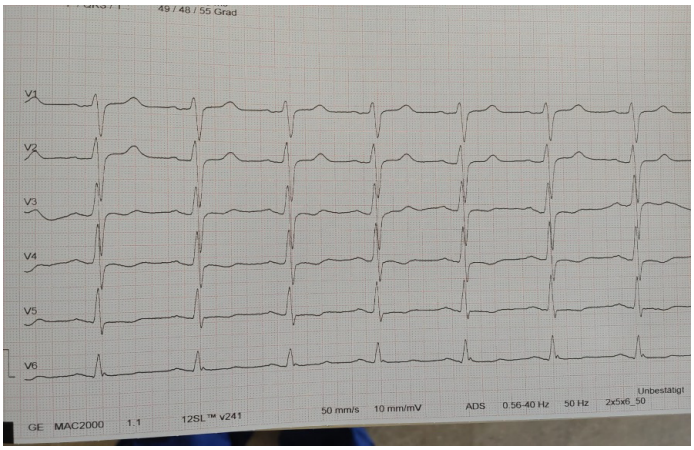

Figure 7: Precordial Leads Registered During Stress Echo Recovery Phase After Sublingual Nitroglycerin.

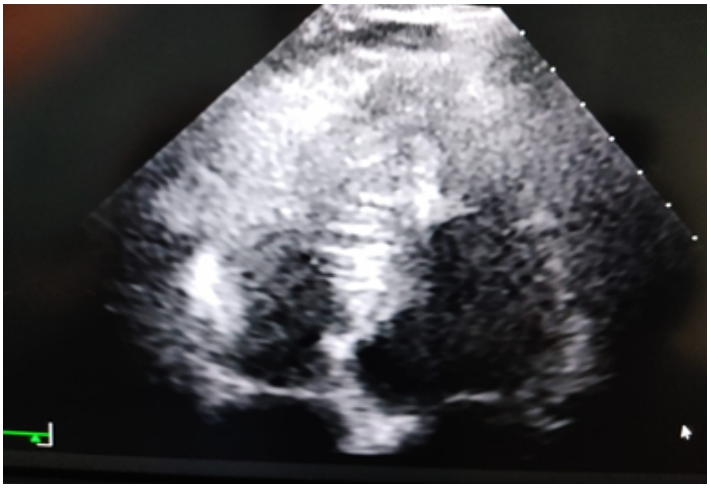

Figure 8: Echocardiography (Apical 4-Chamber View) Directly Before Application of Dobutamine Showing Reserved Apical Contractility.

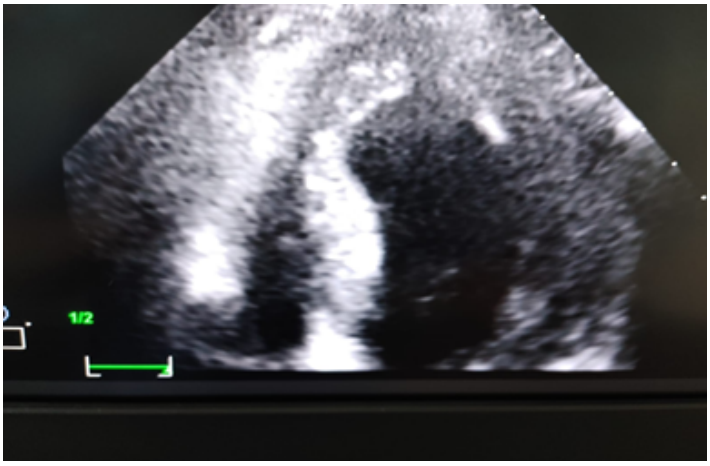

Figure 9: Severe Apical Hypokinesia During Dobutamine Stress Echocardiography, Associated with Relevant ECG Changes.

Discussion

Vasospastic angina is still underdiagnosed, and the patients with intermittent chest pain without relevant angiographic findings will be discharged with an unspecific alternative diagnosis, usually without further investigations. However, in susceptible patients, the diagnosis can be reached through the induction of coronary artery spasms by a variety of pharmacologic agents and even by some physiologic manoeuvres [9]. Many chemicals such as serotonin [10] and histamine [11] can induce coronary vasospasm. Their role as diagnostic agents is currently not well defined. An old study revealed that dopamine can be usefully used as a diagnostic method for uncovering vasospastic angina pectoris [12]. Several invasive methods were applied to diagnose vasospastic angina. In patients with vasospastic angina, changes in coronary flow velocity occur before occlusive spasm. These dynamic changes in coronary flow velocity could be monitored using a Doppler guidewire rather than by monitoring for ischemic electrocardiographic changes [13]. In our case, we used dobutamine stress echocardiography as a non-invasive method not only to diagnose possible vasospastic angina but rather to globally delineate this syndrome under electrocardiographic, echocardiographic, and clinical observation. The ECG changes which we registered during the acute episode were almost absent. During the first two stages, the patient showed no symptoms, the ECG was normal, and the echo findings were within normal. As we stepped up to the 3. At the Stage, he was given 1mg Atropine and the dobutamine doses were increased to 24 microgram/Kg/BSA. After two minutes the patient gave typical chest pain, parallel with deep horizontal ST-Segment depression in the precordial leads. Echocardiographically we noticed severe apical and anteroseptal hypokinesia. The patient was given sublingual nitroglycerin with complete recovery of all ECG and echocardiographic findings, and his pain disappeared. Through a relatively simple Nicht-invasive method we could capture this mysteriOus clinical dilemma and bring it to light under direct clinical and instrumental observation.

Conclusion

Several modalities were tried to diagnose VSA, most of them are invasive and necessitate certain expertise. Dobutamine stress echocardiography, as a simple non-invasive method, besser may can be used safely to uncover vasospastic angina, particularly in young patients with low-risk profiles presented with typical chest pain.

References

1. Prinzmetal M, Kennamer R, Merliss R, Wada T, Bor N (1959) Angina pectoris. I. A variant form of angina pectoris; preliminary report. Am J Med 27: 375-388.
2. Oliva PB, Potts DE, Pluss RG (1973) Coronary arterial spasm in Prinzmetal angina. Documentation by coronary arteriography. N Engl J Med 288: 745-751
3. Nakamura M, Takeshita A, Nose Y (1987) Clinical characteristics associated with myocardial infarction, arrhythmias, and sudden death in patients with vasospastic angina. Circulation 75: 1110-1116.
4. Rodriguez-Manero M, Oloriz T, Waroux JP, Burri H, Kreidieh B, et al. (2018) Long-term prognosis of patients with life-threatening ventricular arrhythmias induced by coronary artery spasm. Europace 20: 851-858.
5. Ong P, Athanasiadis A, Borgulya G, Mahrholdt H, Kaski JC, et al. (2012) High prevalence of a pathological response to acetylcholine testing in patients with stable angina pectoris and unobstructed coronary arteries. The ACOVA Study (Abnormal COronary VAsomotion in patients with stable angina and unobstructed coronary arteries). J Am Coll Cardiol 59: 655-662.

6. Nakayama N, Kaikita K, Fukunaga T, Matsuzawa Y, Sato K, et al. (2014) Clinical features and prognosis of patients with coronary spasm-induced non-ST-segment elevation acute coronary syndrome. J Am Heart Assoc 3: e000795.

7. Kawana A, Takahashi J, Takagi Y, Yasuda S, Sakata Y, et al. (2013) Gender differences in the clinical characteristics and outcomes of patients with vasospastic angina-A report from the Japanese. Coronary Spasm Association. Circ J 77: 1267-1274.

8. Kim H, Jo S, Kim H, Lee M, Seo W,et al. (2020) Sex differences in clinical characteristics and long-term outcomes in patients with vasospastic angina: Results from the VA-Korea registry, a prospective multi-center cohort. Biol Sex Differ 11: 66.

9. Adlam D, Azeem T, Ali T, Gershlick A (2005) Is there a role for provocation testing to diagnose coronary artery spasm? Int J Cardiol 102: 1-7.

10. McFadden EP, Clarke JG, Davies GJ, Kaski JC, Haider AW, et al. (1991) Effect of intracoronary serotonin on coronary vessels in patients with stable angina and patients with variant angina. N Engl J Med 324: 648- 654.

11. Ginsburg R , Bristow MR, Kantrowitz N, Baim DS, Harrison DC (1981) Histamine provocation of clinical coronary artery spasm: implications concerning pathogenesis of variant angina pectoris. Am Heart J 102: 819-822.

12. Crea F, Chierchia S, Kaski JC, Davies GJ, Margonato A, et al. (1986) Provocation of coronary spasm by dopamine in patients with active variant angina pectoris. Circulation 74: 262-269.

13. Yamada T, Okamoto M, Sueda T, Hashimoto M, Matsuura H, et al. (1998) Ergonovine-induced alterations in coronary flow velocity preceding onset of occlusive spasm in patients without significant coronary artery stenoses. Am J Cardiol 81: 688-693.