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Case Report

Atypical Presentation of Raynaud's Disease with Inferior Wall Infarct

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Abstract

Raynaud's disease usually presents as blanching of fingers and toes. However, it can present with atypical symptoms too. Vasospastic angina has been associated with Raynaud's disease and can result in anginal episodes. Here, we present a case of 68- year-old patient who presented with an inferior wall infarct and was also exhibiting signs of Raynaud's disease. We also reviewed the available literature about the pathophysiology of these vasospastic changes in patients with Raynaud's disease.

Introduction

Raynaud Phenomenon (RP) is an exaggerated and episodic vasospasm in response to various stressors such as cold temperature or emotional stress. It presents clinically as sharply demarcated color changes of the skin of the digits and carries a significant burden of pain and hand-related disability. The correlation between Vasospastic Angina and Raynaud's has been explained in several previous observations in the literature. We present a case of an atypical case of Raynaud's with an inferior wall infarct.

Case Report

Mr. A, a 68-year-old physician, presented with a classic anginal episode manifested as central chest pain radiating to both arms for the past 1 hour. It was associated with shortness of breath and diaphoresis. EKG showed ST segment elevation in inferior leads (Figure 1). The universal diagnostic criterion of STEMI was fulfilled (1). His pain partially resolved after administering sublingual nitroglycerin(2), and subsequent EKG showed no ST elevation.

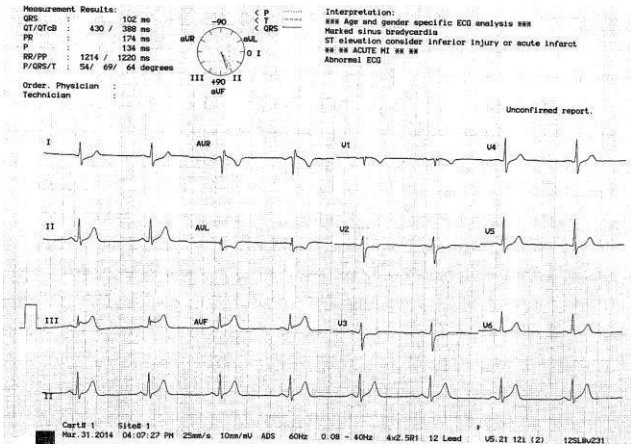

Figure 1: EKG of the patient showing ST segment elevation in leads II, III and AVF.

Over the past ten years, the patient's extremities would show bluish discoloration and blanching when exposed to cold temperatures and while working at his office (Figure 2,3). He also has been having short episodes of chest tightness of moderate severity with no radiation to the arms that were relieved by self-induced cough.

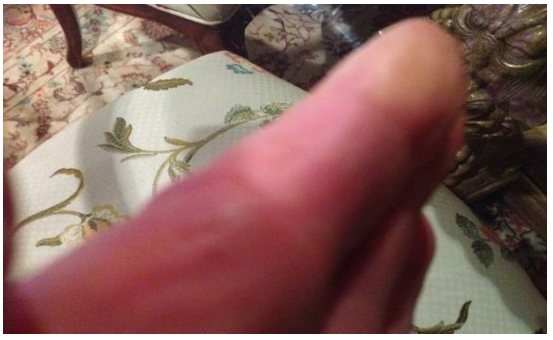

Figure 2: Image shows the discoloration of the patient's fingers in response to cold.

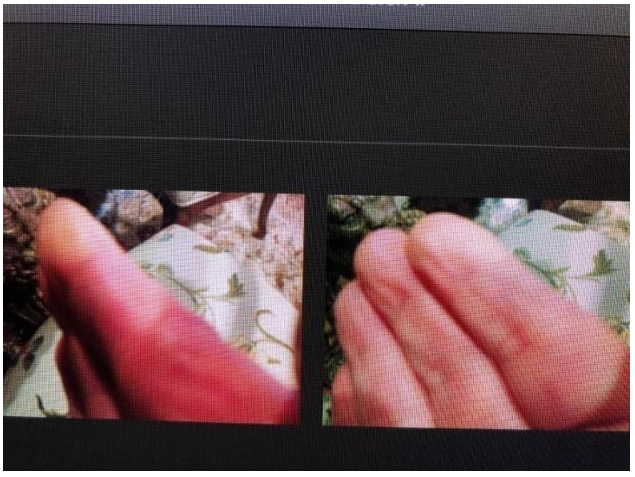

Figure 3: Image shows the blanching of patient's fingers when exposed to colder temperatures.

On examination and during one of the episodes, the patient exhibited typical signs of Raynaud's phenomenon, including discoloration and blanching of fingers and toes. However, tibial pulses were intact, and the doppler ultrasound of the lower extremities was normal. His ANA levels showed a titer of 1:160. All other labs including troponin level, electrolytes and renal function tests were normal. Nailfold capillaroscopy was not performed at that time. His cardiovascular risk factors assessment showed no history of smoking, hypertension, hyperlipidemia, or diabetes. However, he had a strong family history of premature coronary artery disease; his father died from an acute myocardial infarction at age 61, and his three brothers died of coronary artery disease in their fifties. The patient had a remote history of H. pylori infection.

When a coronary angiogram was done, it revealed 80% occlusion of the distal Right Coronary Artery (RCA) and a 50% lesion in the mid-Left Anterior Descending artery. The RCA was stented, and patient was placed on dual anti-platelets therapy, high-intensity rosuvastatin, low-dose metoprolol, and amlodipine. On follow-up visit one month later, the patient's condition had improves and he was no longer having any episodes of blanching of his finger and toes.

Discussion

The prevalence of Raynaud's phenomena varies widely between populations. In the US, it is estimated that up to 200,000 people are affected. A higher prevalence among other populations, such as France, is found(3). Raynaud's phenomenon can be either a primary phenomenon or secondary to various underlying medical disorders and drug-related conditions.

Vasospastic angina, previously called Prinzmetal or variant angina, is a form of abnormal vascular response to various stimuli. Prinzmetal et al(4) initially described a clinical syndrome that manifested as rest angina associated with ST-segment elevation that promptly responded to sublingual nitrates. Since this differs from classical angina described by Heberden (effort angina associated with ST depression (5), he referred to it as variant angina.

The correlation between vasospastic angina and Raynaud's has been explained previously in literature(6,7). Miller et al(6) reported a high prevalence of Raynaud's phenomenon in patients with vasospastic angina occurring at a rate of 24% in patients with variant angina as compared to 3% and 5% in controls (p-value<0.01). Hence vasospastic angina in some patients is a manifestation of a generalized vasospastic disorder and could be associated with migraine, Raynaud's phenomenon, and aspirin-induced asthma(8).

Coronary vasospasm can occur in patients with normal coronary arteries as well as in patients with atherosclerosis. However, it is still poorly understood whether coronary vasospasm can promote atherosclerotic alterations or whether the latter may predispose to spasm(9).

The patient described in our case has a typical history of Raynaud's phenomenon diagnosed clinically, along with recurrent attacks of chest pain relieved by nitrates that were thought to be due to vasospastic angina. However, coronary angiography revealed atherosclerotic narrowing with 80% occlusion of the right coronary artery. This finding was similar to an autopsy result in one of the patients described in Prinzmetal et al paper(4), which revealed that the right coronary artery (which presumably had caused resting angina with ST elevation in inferior leads) had an 80% stenosis. This observation led to the conclusion that coronary spasm was associated with atherosclerosis. Later it was found that coronary spasms can also occur in patients with angiographically normal coronary arteries(10).

Recent experimental studies demonstrate that several endothelium-derived factors mediate vascular smooth muscle cell tone and that damaged endothelium may play a major role in the pathogenesis of vasospasm(11). Endothelial damage may occur even in the early stages of atherosclerosis. Zeiher et al (12) indicated that abnormal vasoconstriction was observed in early atherosclerotic sites in patients without variant angina using intra-coronary ultrasound. Yamagishi et al(13) reported that early stages of atherosclerosis were observed by intracoronary ultrasound at the site of spasm in patients with variant angina, even in the absence of an angiographically significant coronary disease. However, these results were challenged by a porcine model of spasm, indicating that endothelial vasodilator function was preserved at the spastic site([14]); it remains to be elucidated.

It was suggested that endothelial dysfunction with decreased nitric oxide release and reduced bioavailability combined with vascular smooth muscle cell hyperreactivity might be important in developing coronary spasm. However, endothelial dysfunction alone might not be sufficient to explain vasospastic angina(15). Therefore, more studies need to be done to understand the pathophysiology associated with the vasospasm associated with Raynaud's disease.

Conclusion:

This case highlights the importance of taking an extensive history of patient with myocardial infarction so that the root cause can be found. Raynaud's phenomenon can present with an infarct due to atherosclerotic and vasospastic changes. This requires additional treatment of Raynaud's disease along with MI so that further episodes can be prevented.

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Disclosures

No relevant conflicts of interest to declare.

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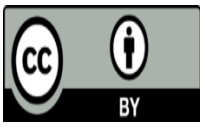

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