

Prinzmetal's Angina

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This syndrome is due to focal spasm of an epicardial coronary artery, leading to severe myocardial ischemia. Although it is frequently thought that the spasm occurs in arteries without stenosis, many Prinzmetal patients have spasm adjacent to atheromatous plaques. The exact cause of the spasm has not been well defined, but it may be related to the hypercontractility of the vascular smooth muscle due to vasoconstrictor mitogens, leukotrienes, or serotonin. In some patients, it is a manifestation of a vasospastic disorder and it is associated with migraine, Raynaud's phenomenon, or aspirin-induced asthma.

We present a case associated with transient ST-segment depression.

Clinical Case - Back Ground

A 65 year-old, black woman, and history of high blood pressure presented to the emergency department with ongoing oppressive chest pain at rest, intensity 7/10, irradiating to the neck, that appeared after emotional distress. After blood tests and an ECG (Figure 1) were performed, conventional anti-ischemic treatment is started. Troponin I level was 18 U/L. After 24 hours of IV vasodilators, with the patient hemodynamically stable and free of symptoms, a PCA (percutaneous coronary angioplasty) was performed (Figure 2), showing no significant epicardial stenosis. Transthoracic Echocardiogram showed normal EF (70%), normal valvular apparatus and normal left ventricular outflow tract. Based on laboratory, clinical and imaging findings, a diagnosis of Prinzmetal's Angina was attained. The patient was discharged, as she was free of symptoms, after increasing doses of calcium channel blockers. After 3 months of follow-up, she remains free of symptoms.

Discussion

The classic electrocardiographic finding in a patient with Prinzmetal's variant angina is the ST-segment elevation during the ischemic episode. The presence of the ST-segment depression in the ECG during the angina, due to coronary vasospasm, can be attributed to subendocardial ischemia caused by the incomplete occlusion of an epicardial coronary artery and the transitory increase in the coronary flow,

Key Words

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supported by the collateral circulation^{1,2}. According to Tada et al², this collateral circulation contributes with the coronary flow through preexisting vessels towards the ischemic regions during coronary vasospasm, which prevents transmural ischemia, decreasing the degree of ischemia and that is associated with the depression of the ST-segment during the angina episodes^{1,2}. Yamagishi et al³ observed a lower frequency of ST-segment elevation during coronary spasm in patients with an established collateral circulation, confirming these findings³.

Experimental and clinical results suggested that the ST-segment deviation might depend not only on the severity and location of the spasm, but also on the extent of collateral development. Yasue et al⁴ reported that in vasospastic angina, the existence of collateral circulation was associated more frequently with ST depression than with ST elevation^{4,5}. The ST-segment depression during a vasospastic attack may also result from collateral channels through which coronary flow can be established in the presence of pressure gradients created by the spasm^{6,7}. However, such collateral vessels, which may appear transiently, could not be easily demonstrated because of technical difficulties in the simultaneous visualization of the spastic and nonspastic artery donating collateral flow⁴.

The spasms are most commonly focal and can occur simultaneously in more than one site. Even coronary segments that are apparently normal at the coronary angiography, often show evidence of mural atherosclerosis at the intravascular ultrasound. This can result in localized endothelial dysfunction and coronary spasm^{8,9}.

Clinical Picture

Although chest discomfort in the patient with variant angina can be precipitated by exercise, it usually occurs without any preceding increase in myocardial oxygen demand; the majority of patients have normal exercise tolerance and stress testing may be negative^{9,10}. Because the chest discomfort usually occurs at rest without a precipitating cause, it may simulate UA/NSTEMI secondary to coronary atherosclerosis. Episodes of Prinzmetal's angina often occur in clusters, with prolonged asymptomatic periods that can last from weeks to months. Attacks can be precipitated by emotional distress, hyperventilation, exercise, or exposure to cold. A circadian variation in the episodes of angina is very often present, with most attacks occurring in the early morning^{8,10}. Compared with patients presenting chronic stable angina, patients with variant angina are younger and, except for smoking, have fewer coronary risk factors^{6,8}.

Diagnosis

The key to the diagnosis of variant angina is the documentation of the ST-segment elevation in a patient during transient chest

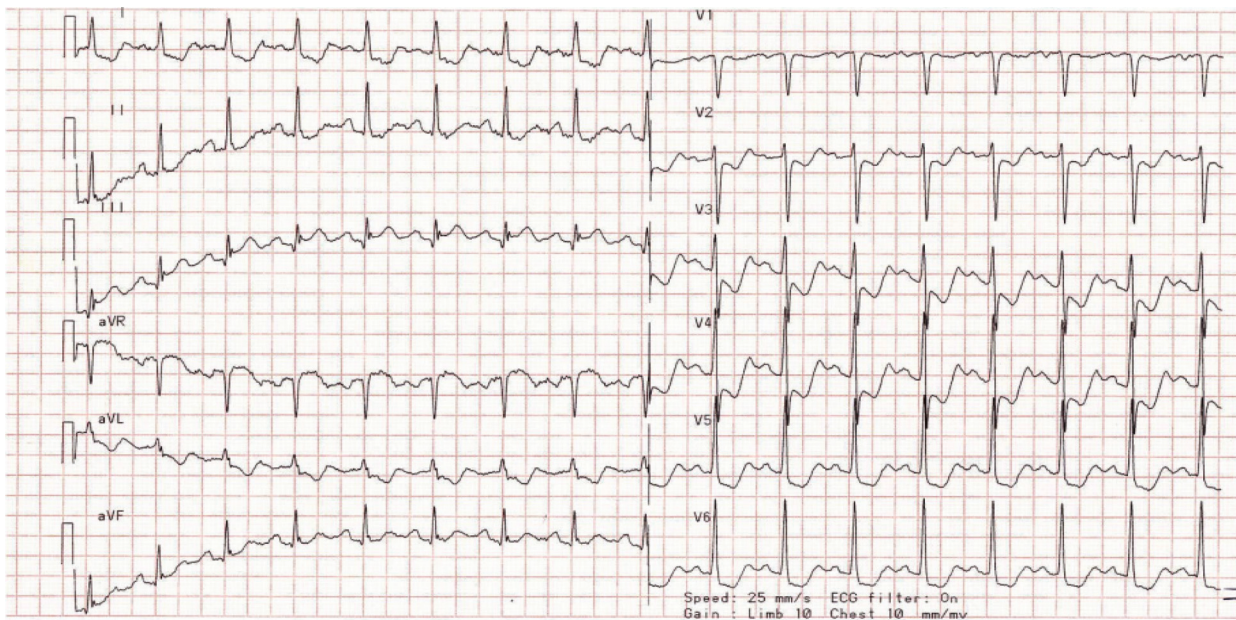


Figure 1 - ST-segment in DI, II, aVL, V2 - V6.



Figure 2 - Normal Left Anterior Descending and Circumflex arteries.

discomfort (which usually occurs at rest, typically in the early morning hours, and is nonreproducible during exercise) and that resolves when the chest discomfort abates^{2,5}. Typically, nitroglycerin (NTG) is especially effective in relieving the spasm. The ST-segment elevation implies in transmural focal ischemia, associated with complete or near-complete coronary occlusion of an epicardial coronary artery in the absence of collateral circulation^{6,9,17}. In variant angina, the dynamic obstruction can be superimposed on severe or nonsevere coronary stenosis or supervene an angiographically normal coronary artery segment. Hence, the coronary angiography is usually part of the workup of these patients and can help to guide the treatment^{4,6}.

Treatment

Nitrates and calcium channel blockers are the main treatments for patients with variant angina. Sublingual or intravenous nitroglycerin often promptly abolishes episodes of variant angina, and long-acting nitrates are useful in preventing recurrence. Calcium antagonists are extremely effective in preventing the coronary artery spasm in variant angina, and maximally tolerated doses should be prescribed. Similar efficacy rates have been observed among the various types of calcium antagonists. Prazosin, a selective α -adrenoreceptor blocker, has also been found to be of value in some patients, while aspirin may actually increase the severity of ischemic episodes. The response to beta-blockers is variable. Coronary revascularization may be helpful in patients with variant angina who also have discrete, proximal fixed obstructive lesions^{1,2,7}.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any post-graduation program.

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