Prinzmetal Angina

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Abstract

Variant angina (VA), first described by Prinzmetal in 1959, is caused by transient and recurrent coronary spasm and leads to repetitive episodes of transmural myocardial ischemia. A 59-year-old man with a history of hyperlipidemia and anterior myocardial infarction, which had occurred three months previously and was being treated with fibrinolytics, referred to our hospital with acute substernal chest pain and ST elevation in the anterior leads. Coronary angiography showed coronary spasm, and the patient was relieved after nitrate administration. ***(Iranian Heart Journal 2012; 13 (2):62-64).***

Introduction

Variant angina (VA), first described by Prinzmetal in 1959, is caused by transient and recurrent coronary spasm and gives rise to repetitive episodes of transmural myocardial ischemia. Endothelial dysfunction has been considered an important predisposing condition for this phenomenon. Many different stimuli such as hyperventilation, Ergonovine, Dobutamine or acetylcholine administration, cold pressor test, exercise, and mental stress can trigger coronary vasospasm.

Case Report

A 59-year-old man with a history of hyperlipidemia and anterior myocardial infarction, which had occurred three months previously and was being treated with fibrinolytics, referred to our hospital with acute substernal chest pain and 10 box ST elevation in the anterior leads

He was immediately transferred to the cardiac catheterization laboratory. Coronary angiography showed a significant lesion in the proximal portion of the left anterior descending artery with hazy appearance, in favor of thrombosis, and another significant lesion at the mid portion (Figure 1).

After wiring of the left anterior descending artery and injection of intracoronary Trinitroglycerin, the lesions disappeared. Multiple views were obtained, and they showed two non-obstructing lesions, which suggested nidus for the patient's vasospasm (Figure 2). On the electrocardiogram, ST elevation changed to normal level and the patient's pain subsided. He was transferred to the Coronary Care Unit, where nitrate and calcium channel blocker was started. The patient was discharged in good condition and he has had no complaint since then.

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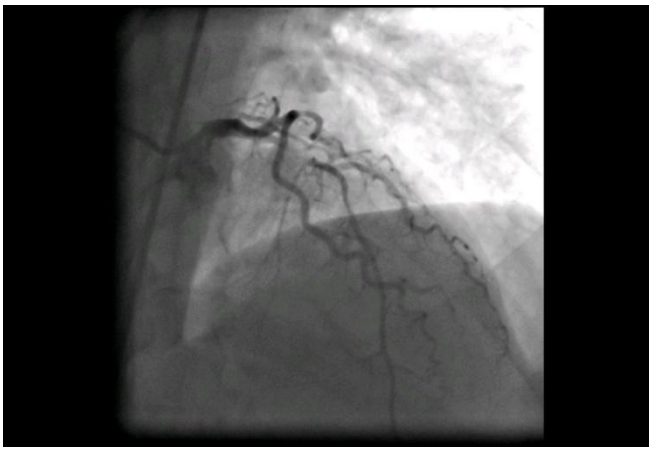

Figure 1: Coronary angiography shows the right anterior oblique view: the LAD is cut at the proximal part and there is a good run-off.

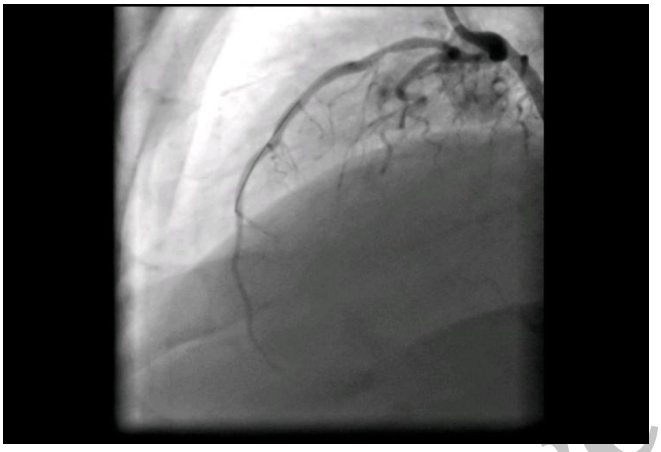

Figure 2: After wiring, the lateral view shows no sign of the previous LAD lesion, confirming the diagnosis of Prinzmetal angina.

Discussion

Prinzmetal angina is a condition that results from increased spasticity at a specific coronary site. The prevalence of coronary spasm is different around the world. It is more common in South Asia probably due to genetic as well as environmental factors. Coronary spasm occurs most often during sleep and is usually not induced by exercise. Typical ECG changes in Prinzmetal angina are ST-segment elevation, negative U wave, and ST-segment depression. Multi-vessel coronary spasm may induce lethal arrhythmia, including advanced AV block, ventricular tachycardia or fibrillation, or even sudden death, and they are often resistant to conventional medical therapy.

The pathophysiology of coronary spasm includes reduced endothelial nitric oxide (NO) activity and elevated markers of oxidative stress. Enhanced thrombogenesis and elevated plasma levels of hsCRP and P-selection are also observed in patients with coronary spasm. Therefore, patients with coronary spasm have endothelial dysfunction and tend to suffer from a low-grade chronic inflammation. Endothelial NO synthase polymorphism, smoking, and low-Grade inflammation are the most important risk factor for coronary spasm.

In coronary spasm, coronary smooth muscle hyper contraction is triggered by an increase of intra cellular Ca2+ in the presence of an increased Ca2+ sensitivity.

Enhanced Ras homolog gene family, member A (RhoA)/ RhoA kinase (ROCK) pathway, has been proved to be involved in increased Ca2+ sensitivity by reduced endothelial NO activity. Accordingly, it is possible that in addition to CCBs, RhoA/ROCK pathway blockers may prove to be useful for the treatment of coronary spasm.

Conclusion

In the setting of acute myocardial infarction, cases with vasospasm are discovered and the treatment should include newer vasodilators and smoking cessation.

References

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