Prinzmetal angina or coronary spasm related to anaphylactoid reaction?

Prinzmetal anjina veya koroner spazm anafilaktik reaksiyonla ilişkili mi?

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Summary– Prinzmetal's angina is a challenging diagnostic of spontaneous brief episodes of chest pain. Anaphylactoid reactions to radiocontrast media are immediate hypersensitivity responses that can mediate coronary artery spasm. Herein, we report the case of a 61-year-old man who underwent a coronary angiography for angina. The right coronary artery (RCA) was first visualized as normal, but during the left coronary system injections, he developed ST segment elevation and cardiogenic shock. No iatrogenic dissection of the left coronary system, which was initially normal, was displayed, but surprisingly, a retrograde supply to the RCA was visualized. Thus, we re-catheterized the RCA, which indicated a total occlusion of its second segment. Nitrate injections completely relieved the spasm and the clinical condition of the patient normalized. The possible related mechanisms are also discussed.

Özet– Prinzmetal anjina kendiliğinden oluşan kısa süreli göğüs ağrısı ile ortaya çıkan, tanısı oldukça zor bir klinik tablodur. Radyokontrast maddeye karşı koroner arter spazmına aracılık eden akut aşırı duyarlılık reaksiyonu oluşabilir. Bu yazıda, göğüs ağrısı yakınması nedeniyle koroner anjiyografi uygulanmış 61 yaşındaki bir erkek hasta sunuldu. İlk olarak görüntülenen sağ koroner arter (RCA) normal bulundu. Ancak sol koroner sistem enjeksiyonu sırasında EKG'de ST-segment yükselmesi ardından kardiyojenik şok gelişti. İlk incelemede normal olarak görülen sol koroner sisteminde iyatrojenik diseksiyona ait herhangi bir kanıt yoktu. Ancak şaşırtıcı olarak RCA'nın retrograd yolla beslendiği görülüyordu. Bu nedenle RCA anjiyografisi tekrarlandı ve RCA'nın ikinci segmentinde tam tıkanma saptandı. Nitrat enjeksiyonlarıyla RCA spazmı tamamen düzeldi. Hastanın klinik belirtileri normale döndü. Bu olgu nedeniyle ilişkili olası mekanizmalar da tartışıldı.

Prinzmetal's angina is a syndrome characterized by spontaneous episodes of angina that are related to focal spasms of an epicardial coronary artery, which can result in severe myocardial ischemia.[1] The underlying physiopathology of this syndrome remains unclear.[2-5]

Herein we present the case of a patient who presented with recurrent episodes of Prinzmetal's angina that were unmasked during coronary angiography shortly after the injection of radiocontrast media.

CASE REPORT

We report the case of a 61-year-old man who was referred to our hospital for recurrent brief episodes of chest pain. He had hypertension and type 2 diabetes mellitus, but had no history of rhinitis or asthma. No per-critical ECG could be registered, but the inter-critical ECG did not show any ischemic changes. An echocardiography revealed hypertensive cardiomyopathy with no wall motion disorder. His stress test was normal, but since he had recurring symptoms suggestive of angina, we performed a coronary angiography via right trans-radial access with 5 French diagnostic catheters. The right coronary artery (RCA), which we examined first based on our standard practice, was normal and smooth (Fig. 1a). The left coronary artery was also normal (Fig. 1b). However, while we continued recording images in different views, the patient felt a severe angina that was accompanied by ST-segment elevation and a drop in blood pressure. Simultaneously, he presented flushing and pruritis, but had no other signs or symptoms of

Abbreviation:

RCA *Right coronary artery*

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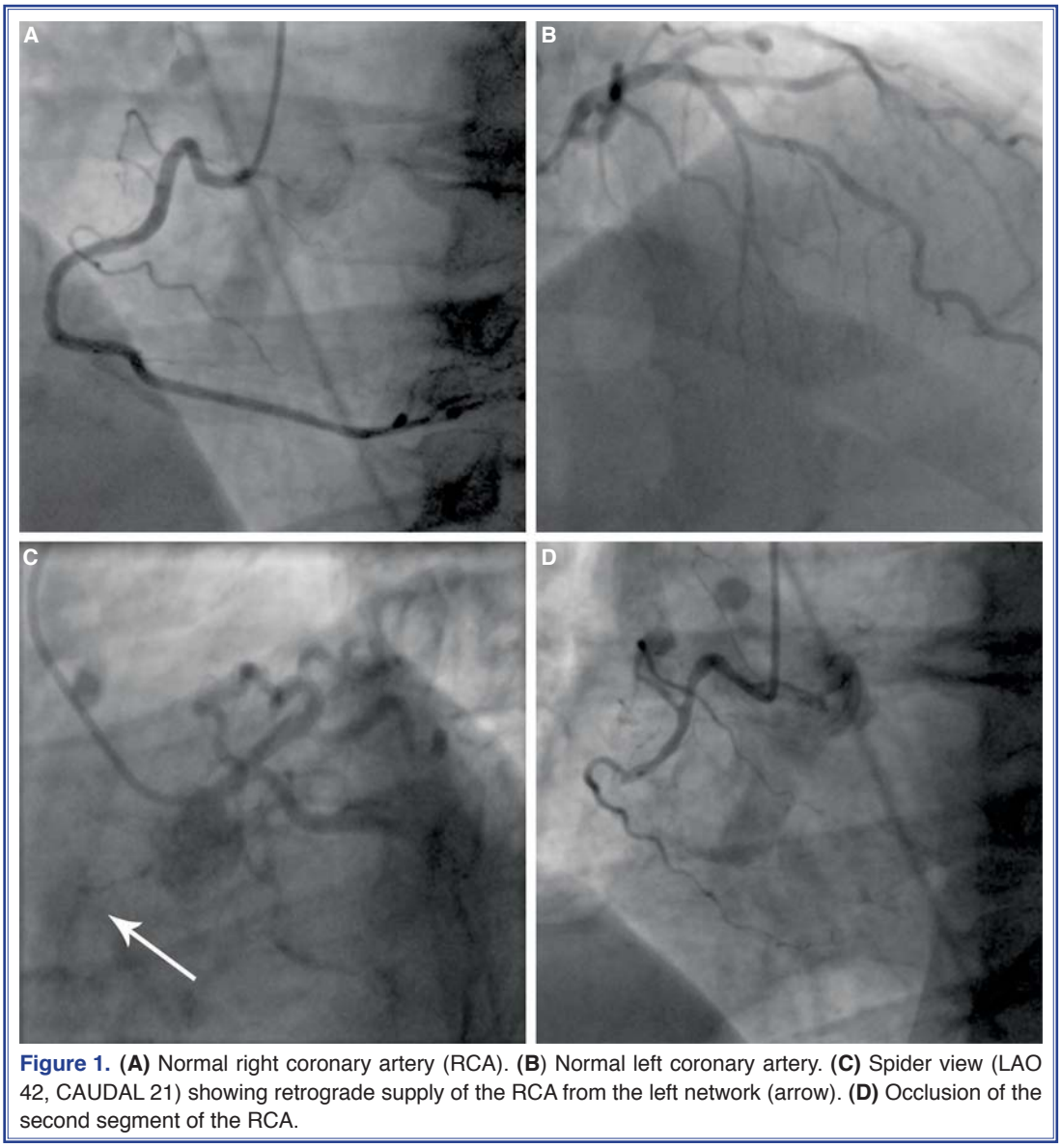

Figure 1. **(A)** Normal right coronary artery (RCA). **(B)** Normal left coronary artery. **(C)** Spider view (LAO 42, CAUDAL 21) showing retrograde supply of the RCA from the left network (arrow). **(D)** Occlusion of the second segment of the RCA.

anaphylaxis (i.e. no erythema, no urticaria, and no angioedema). To rule out iatrogenic dissection or spasm of the left main (LM) coronary artery, we performed a spider view (LAO 42, CAUDAL 21) that showed the patency of the LM. There was no image suggesting dissection, but we did see a retrograde supply to the RCA from the left coronary system (Fig. 1c) that had not been documented in previous views. Without delay, we started a intravascular filling regimen with macromolecules and we turned back to the RCA through a right guiding catheter (Judkins Right 4, Launcher guiding catheter, Medtronic, USA).

The subsequent injection showed a total occlusion of the second segment of the RCA (Fig. 1d). We advanced a guide wire (ChoICETM, Floppy LS, Boston Scientific) through the RCA and injected nitrate selectively several times until the spasm was completely resolved (Fig. 2a, b). There was no need for coronary stenting. The patient's clinical conditions stabilized, and he had complete pain relief, a rise in blood pressure, reversed electrical ischemic changes, and the flush and pruritis disappeared. The left ventriculography highlighted a normal wall motion with severe symmetric hypertrophy (Fig. 2c). The 24-troponin levels remained within the normal ranges. A skin-prick test, which was performed with a complete panel of commercial reagents, was negative. Total IgE serum levels were at normal ranges. The patient was discharged on calcium channel blockers and nitrates.

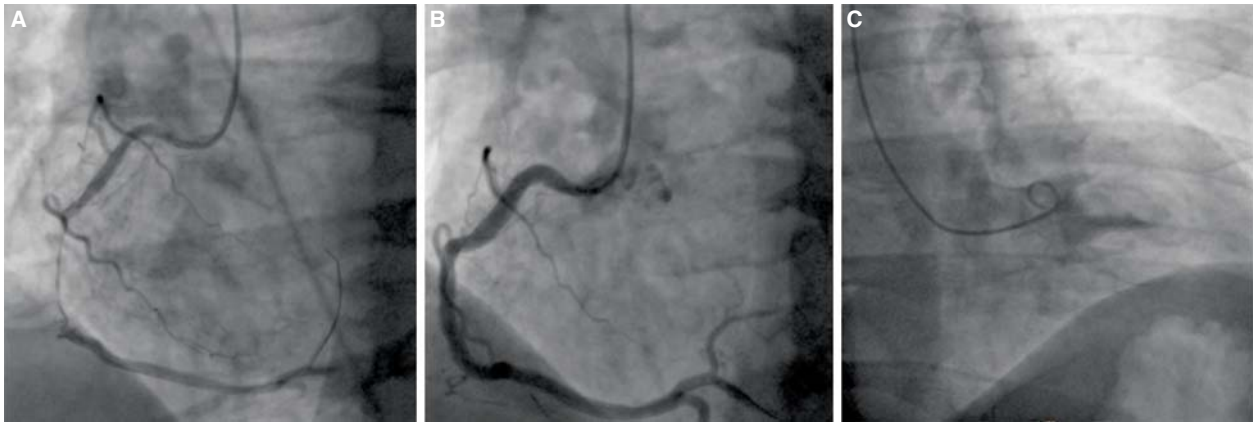

Figure 2. **(A, B)** Guide wire in the RCA before and after relieving the spasm. **(C)** Hypertrophic myocardial wall on left ventriculography.

He has remained symptom-free for 1 year.

DISCUSSION

Prinzmetal's angina, also called variant angina, is a syndrome characterized by spontaneous episodes of angina that are related to focal spasms of an epicardial coronary artery, which can result in severe myocardial ischemia.[1] It is associated with a transient ST-segment elevation on an electrocardiogram with reciprocal depression[1] concomitant with the episodes of chest pain. It generally concerns normal coronary arteries, but sometimes may occur close to atheromatous plaques.[2-4] Ischemic attacks often occur at rest. The ultimate endpoints of coronary artery spasms result in ischemic episodes, but the exact pathophysiology of the mechanism remains unclear.[5] An endothelial dysfunction due to the deficient release of nitric oxide (NO) and inappropriate enhanced vascular smooth muscle contractility have been reported to play the basic roles in the pathogenesis of coronary artery spasms.[6-8] Disruption of the autonomic nervous system and increased oxidative stress have also been implicated as stimulating factors.[9,10] The most accurate way to elucidate variant angina is ergonovine provocation testing combined with a coronary angiogram.[5,11-15] It has been established that cigarette smoking is a major risk factor for coronary spasms.[16] Calcium channel blockers[17,18] and nitrates[5] are the most common symptomatic treatments of coronary spasms.

Anaphylactoid responses to radiocontrast media are immediate hypersensitivity reactions that occur within minutes of administration and are often manifested by erythema, urticaria, and/or angioedema, and occasionally involve the cardiovascular and respiratory systems.[19] The exact pathogenesis remains poorly understood. It is mostly mediated by IgE antibodies, but several other hypotheses have been suggested, such as complement activation, antigen-antibody interactions, and multimediator recruitment. It may also be due to a non-IgE-mediated histamine release via interaction with mast cell and basophil cell membranes, via its hyperosmolarity, or via generation of anaphylatoxins.[20,21] H1 receptors, which are expressed on vascular smooth muscle cells, mediate coronary artery vasoconstriction and increase vascular permeability such that massive histamine release from mast cells during anaphylactic reactions may trigger coronary spasms. Coronary spasms can also be caused by other mast cell-derived vasoactive mediators such as PGD2 and LTs.[19]

In our case, the clinical presentation included flush, pruritis, and coronary vasospasm with a collateral development that was totally reversed with nitrates. There was no need for corticosteroids or epinephrine to control the situation, and only the management of ischemia due to RCA spasm was sufficient to re-establish a stable clinical situation. Bronchospasm, cyanosis, severe hypotension, or local allergic manifestations, such as rhinorrhea, conjunctivitis, or facial edema were not present, so we believe that the coronary spasm was more related to contrast than to an anaphylactoid reaction.

In conclusion, we reported a case of RCA spasm that occurred during the visualization of the left coronary artery after the administration of iodine radio-

contrast during diagnostic coronary angiography. Flushing, pruritis, and coronary vasoconstriction are common features of a diagnosis of anaphylactoid reaction. The occurrence of the coronary spasm during the diagnostic procedure allowed an accurate clinical diagnosis of Prinzmetal's angina and allowed for the most accurate treatment. However, we cannot eliminate the iatrogenic etiology of the coronary vasospasm related to the coronary catheters during the diagnostic procedure.

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Key words: Angina pectoris; coronary angiography; coronary artery disease; Prinzmetal's angina.

Anahtar sözcükler: Anjina pektoris; koroner anjiyografi; koroner arter hastalığı; Prinzmetal anjini.