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CASE REPORT

Steroid-induced recurrent myocardial ischemia

Ufuk Yildirim*, **Okan Gulel**, **Korhan Soylu**, **Serkan Yuksel**, **Mahmut Sahin**

Ondokuz Mayis University, Faculty of Medicine, Department of Cardiology, Samsun, Turkey

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KEYWORDS
Steroid;
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Abstract We report the case of a female patient under oral prednisolone therapy due to a diagnosis of idiopathic intracranial hypertension with papilledema. Unfortunately, short-term treatment with prednisolone caused an unusual complication in the patient, i.e., recurrent myocardial ischemia. Possible mechanisms leading to this complication were evaluated in the light of current knowledge.

PALAVRAS-CHAVE
Esteróides;
Prednisolona;
Isquemia miocárdica;
Enfarte agudo
do miocárdio;
Fluxo lento coronário

Isquemia do miocárdio recorrente induzida por esteróides

Resumo Descrevemos o caso clínico de um paciente sob terapia prednisolona oral devido a um diagnóstico de hipertensão intracraniana idiopática com papiledema. Infelizmente, o tratamento a curto prazo com prednisolona causou uma complicação incomum no paciente, isto é, isquemia miocárdica recorrente. Os possíveis mecanismos que conduzem a esta complicação foram avaliados em função das literaturas correntes.

Introduction

Glucocorticoids not only promote cardiovascular risk factors such as hypertension, insulin resistance, glucose intolerance, dyslipidemia and obesity, but also have direct effects on the heart and blood vessels, influencing vascular function.[1] Here we describe a female patient under oral prednisolone therapy due to a diagnosis of idiopathic intracranial hypertension with papilledema. Unfortunately, short-term treatment with prednisolone caused an unusual complication in the patient, i.e., recurrent myocardial ischemia.

* Corresponding author.
E-mail address: ufukyildirim2715@yahoo.com.tr (U. Yildirim).

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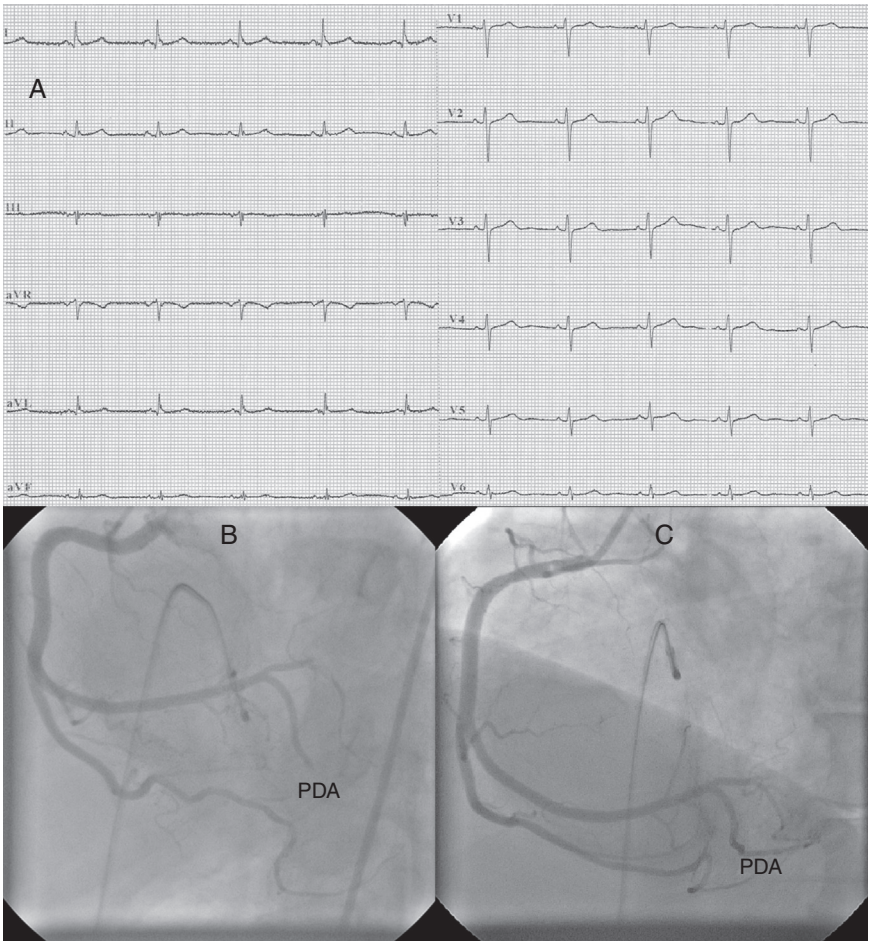

Figure 1 (A) ECG at first admission; (B) right coronary angiography during first myocardial infarction showing total occlusion of the mid portion of the posterior descending artery (PDA); (C) right coronary angiography during first myocardial infarction after percutaneous coronary intervention to the PDA.

Case report

A 64-year-old woman with a history of hyperlipidemia had recurrent episodes of anginal chest pain at rest and with exertion within one month of starting oral prednisolone (40 mg/day). Her chest pain was not resolved by oral nitroglycerine. The resting electrocardiogram (ECG) showed normal sinus rhythm and revealed no ischemic changes (Figure 1A). Diagnostic coronary angiography (CAG) showed no obstructive atherosclerotic lesions, but slow flow was present in the right coronary artery (RCA). She was discharged from the hospital medicated with aspirin (100 mg/day) and diltiazem (90 mg/day).

Two weeks after the first admission, she was again admitted to the emergency department with severe anginal chest pain. The ECG showed nodal rhythm with a heart rate of 40 bpm, ST-segment elevation in leads II, III, aVF and ST-segment depression in leads I and aVL. She was urgently transferred to the cardiac catheterization laboratory. This time, interestingly, CAG showed that the mid portion of the posterior descending artery (PDA) of the RCA was totally occluded (Figure 1B). When a floppy guidewire was passed from the totally occluded PDA, the thrombus moved distally and TIMI III flow was obtained (Figure 1C). At follow-up in the intensive care unit, the ECG returned to sinus rhythm with resolution of the ST segment changes. Pathological Q waves did not develop, but troponin I was elevated at 12 ng/ml (reference: 0–0.2 ng/ml).

Four days after myocardial infarction (MI), the patient again suffered from severe anginal chest pain. The ECG revealed similar abnormalities to those of four days previously (i.e., nodal rhythm, ST-segment elevation in leads II, III and aVF, and ST-segment depression in leads I and aVL) (Figure 2A). With intravenous atropine (1 mg) and isotonic saline infusion, sinus rhythm was restored and blood pressure was normalized. CAG showed that the PDA was open. But this time, there was slow flow in the left anterior descending coronary artery. At follow-up, the ECG showed pathological Q waves and T-wave inversions in leads II, III, and aVF. Additionally, T-wave inversions developed in the anterior precordial leads (Figure 2B). Troponin I was elevated at 41 ng/ml. Transthoracic echocardiography revealed wall motion abnormalities in the left ventricular inferior and posterior walls. There was no thrombus in the cardiac chambers and the interatrial septum was intact, without passage of agitated saline. There was no abnormal mass related to the heart valves and blood tests revealed no coagulation disorder.