A Case of Acute Myocardial Infarction Induced by Selective Cyclooxygenase-2 Inhibitor

Kyoung-Woo Seo, Jin-Sun Park, Seung-Jea Tahk, Joon-Han Shin
Department of Cardiology, Ajou University School of Medicine, Suwon 16499, Korea

To the Editor: A 46-year-old male, having a history of coronary artery disease, and coronary artery bypass graft surgery (CABG), was admitted to our clinic with sustained chest pain. He had a left internal mammary artery to the left anterior descending coronary artery, saphenous vein graft (SVG) to diagonal branch, and SVG to obtuse marginal artery. Up to 7 years after CABG, patient had no complaints of chest pain. Two days before admission, medications were administered for back pain: aceclofenac (selective cyclooxygenase-2 [COX-2] inhibitor), orphenadrine (muscle relaxant), and alibendol (digestant). Intermittent chest pain began after taking the drugs, which aggravated and were sustained for 4 h before admission.

The initial electrocardiogram (ECG) showed ST-elevation in lead III, aVF, and V3-6 [Figure 1a]. Laboratory tests revealed elevated levels of troponin T (0.687 ng/ml), creatine kinase (500 U/L), and creatine kinase-MB isoenzyme (42.36 μg/L). ST-elevation myocardial infarction (MI) was diagnosed, and the patient was immediately transferred for primary percutaneous coronary intervention. The immediate coronary angiography showed severe diffuse vasospasm of both native coronary arteries and the graft vessels [Figure 1c]. After intracoronary nitroglycerin infusion, the vasospasm [Figure 1d] as well as ST-elevation on ECG [Figure 1b] was resolved. There was no evidence of newly developed significant

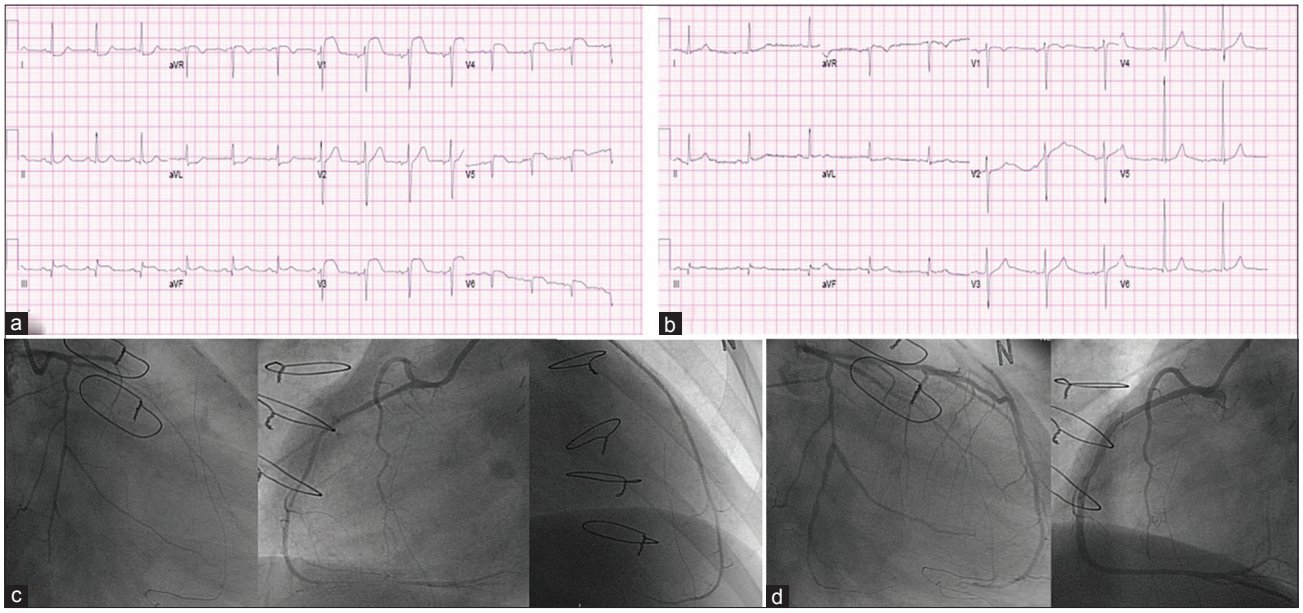

Figure 1: (a) Electrocardiogram showed ST-segment elevation on lead III, aVF, and V3-6. (b) After the administration of intracoronary nitroglycerin, ST-segment elevation was resolved. (c) Baseline coronary angiography showed extensive vasospasm of both native coronary arteries and graft vessels. (d) Spasm was resolved with the administration of intracoronary nitroglycerin.

Address for correspondence: Dr. Jin-Sun Park, Department of Cardiology, Ajou University School of Medicine, 164 Worldcup-ro, Yeongtong-gu, Suwon 16499, Korea
E-Mail: lavioli@hanmail.net

Access this article online

Quick Response Code:

Website: www.cmj.org

DOI: 10.4103/0366-6999.204919

Received: 18-01-2017 **Edited by:** Yi Cui
How to cite this article: Seo KW, Park JS, Tahk SJ, Shin JH. A Case of Acute Myocardial Infarction Induced by Selective Cyclooxygenase-2 Inhibitor. Chin Med J 2017;130:1131-2.

stenosis or graft failure. Severe vasospasm induced by aceclofenac was finally diagnosed.

The patient was discharged on a maximal dose of calcium channel blockers and nitrate. Since the discharge, follow-ups have continued in the outpatient clinic, with no further episodes of chest pain.

Traditional nonsteroidal anti-inflammatory drugs (NSAIDs) play an important role in controlling pain and inflammation by inhibiting COX, composed of two different isoforms, COX-1 and COX-2. COX-1 is constitutively expressed and catalyzes the production of prostaglandins involved in several physiological functions, including thromboxane A_2 (TXA_2) biosynthesis in platelets. Although traditional NSAIDs inhibit both TXA_2 and prostacyclin, selective COX-2 inhibitors do not affect TXA_2 synthesis, due to the lack of COX-2 in platelets. Due to the deficit of inhibiting TXA_2 synthesis, selective COX-2 inhibitors are known to relate with prothrombotic state.[1]

The selective COX-2 inhibitors are known to be associated with an increased risk of death in patients with previous MI.[2] In the present case, the prothrombotic effect of a selective COX-2 inhibitor might not be the main pathophysiology of MI. TXA_2 synthesis is also closely related to vasoconstriction.[3] The impairment of keeping vascular homeostasis, which is regulated through the synthesis and release of vasodilators and vasoconstrictors results in endothelial dysfunction. Although the level of TXA_2 was not assessed in the present case, deficit of inhibiting TXA_2 synthesis was suspected to cause extensive vasospasm, which seemed to be induced by unbalanced COX inhibition.

Selective COX-2 inhibitors could induce severe coronary vasospasm through unbalanced COX inhibition. In addition to the prothrombotic effect, the vasoconstrictive effect of selective COX-2 inhibitor should be considered in patients with cardiovascular disease. Especially, in patients with vasospastic angina or previous MI, the impairment of keeping vascular homeostasis by unbalanced COX inhibition increases the risk of cardiovascular events.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Fitzgerald GA. Coxibs and cardiovascular disease. N Engl J Med 2004;351:1709-11. doi: 10.1056/NEJMp048288.
2. Gislason GH, Jacobsen S, Rasmussen JN, Rasmussen S, Buch P, Friberg J, *et al.* Risk of death or reinfarction associated with the use of selective cyclooxygenase-2 inhibitors and nonselective nonsteroidal antiinflammatory drugs after acute myocardial infarction. Circulation 2006;113:2906-13. doi: 10.1161/CIRCULATIONAHA.106.616219.
3. Gamez-Mendez AM, Vargas-Robles H, Ríos A, Escalante B. Oxidative stress-dependent coronary endothelial dysfunction in obese mice. PLoS One 2015;10:e0138609. doi: 10.1371/journal.pone.0138609.