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ANGINA PECTORIS INDUCED BY SUBLINGUAL NIFEDIPINE

Nobuyuki TAKAHASHI, Yutaka ISHIBASHI, Toshio SHIMADA, Yo MURAKAMI, Toshihiko ASANUMA, Harumi KATOH and Kazuya SANO

The Fourth Department of Internal Medicine, Shimane Medical University, Izumo 693-8501, Japan

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This report describes the case of a 44-year-old man with angina pectoris caused by sublingual nifedipine. He developed chest pain after receiving 5 mg sublingual nifedipine for treatment of hypertensive encephalopathy, and his electrocardiogram showed marked depression of the ST segment in the chest leads. The coronary angiogram did not show critical stenosis, but revealed a 50% stenotic lesion in the left anterior descending coronary artery. The sudden marked decrease in blood pressure induced may have caused an angina attack. The use of sublingual nifedipine for hypertensive emergency should be abandoned in any case.

Key words: nifedipine / hypertensive emergency / angina pectoris

Nifedipine, a dihydropyridine calcium antagonist, is used as an effective antihypertensive agent and has become popular in capsule form for the treatment of hypertensive emergencies (1-7). Serious adverse effects, however, such as cerebrovascular ischemia or myocardial ischemia, have been reported by several investigators (8-14), and it has recently been recommended that the routine use of nifedipine capsules in hypertensive emergencies should be abandoned (15, 16). This warning, however, has not been always followed, especially in primary care. In this report, we present a case of angina pectoris induced by sublingual nifedipine.

Correspondence : Nobuyuki Takahashi, M.D., The Fourth Department of Internal Medicine, Shimane Medical University, 89-1 Enya-cho, Izumo, Shimane 693-8501, Japan. Phone: +81-853-20-2206 Fax: +81-853-20-2201 e-mail address:yuyu@shimane-med.ac.jp

CASE REPORT

A 44-year-old man with a 23-year history of hemodialysis and diabetes mellitus was admitted to the Department of Urology because of the occlusion of his forearm hemodialysis shunt on June 13,1997. He had never experienced chest pain or precordial tightness, and his blood pressure was normal, ranging from 130 to 150 mmHg (systolic) and from 70 to 80 mmHg (diastolic). His electrocardiogram showed complete right bundle branch block on admission. On the next day after admission, he underwent surgery for removal of a thrombus in the occlusive forearm shunt and was doing well after surgery. On June 22, however, he complained of headache, and his blood pressure was elevated to 220/140 mmHg. He did not have abnormal neurological findings such as paresis. A urologist recommended sublingual nifedipine to reduce the blood pressure. Twenty minutes after he was given 5 mg of nifedipine sublingually, his blood pressure decreased to 150/80 mmHg and the headache disap-

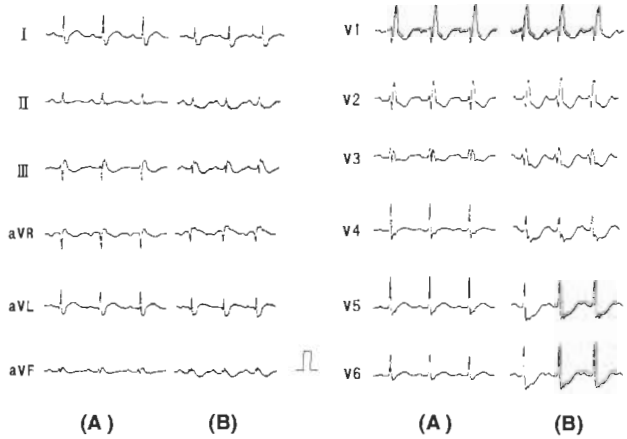

Fig. 1. Electrocardiograms on admission (A) and 20 minutes after nifedipine marked ST depression in the chest leads were demonstrated (B).

peared, but he developed a squeezing substernal chest pain and his electrocardiogram showed marked depressions of the ST segments in the chest V4-6 leads (Fig. 1) that was not improved by intravenous administration of nitroglycerin. Emergency coronary angiography was therefore performed to evaluate the possibility of unstable angina pectoris or myocardial infarction. The coronary angiogram revealed 50% stenosis of the left anterior descending coronary artery (Fig. 2), but no thrombus was found in the stenotic lesion which would indicate unstable angina pectoris. During catheterization, the symptoms and electrocardiographic changes disappeared as the patients blood pressure increased.

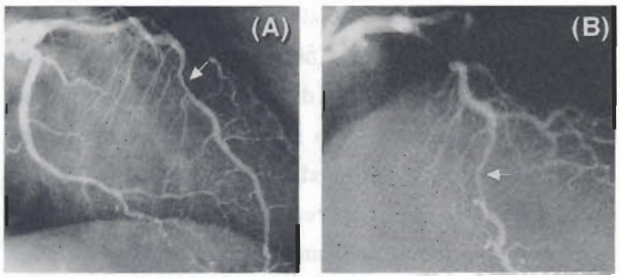

Fig. 2. Coronary angiogram in RAO view (A), and straight cranial view (B).
Fifty% of stenosis was revealed in the left anterior descending coronary artery (arrow).

DISCUSSION

Sublingual nifedipine has been used as an effective antihypertensive agent (1-7), and has become widely popular in capsule form for the treatment of hypertensive emergencies over the past 2 decades, due to its ease of use and its ability to rapidly reduce the blood pressure in hypertensive emergencies. However, despite its widespread use, outcome data has not been available that would allow a critical assessment of the safety or efficacy of this type of intervention. On the contrary, numerous serious adverse effects of sublingual nifedipine have been reported (8-15), and it was recently proposed in the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure that the use of nifedipine for hypertensive emergencies should be abandoned (16).

The mechanism of myocardial ischemia caused by nifedipine is well known. Nifedipine is a potent peripheral arterial dilator that has a rapid onset after sublingual administration, and the resulting marked decrease in blood pressure lowers the coronary artery perfusion pressure. The decrease in coronary perfusion pressure causes subendocardial hypoperfusion in patients whose coronary vasodilatory reserves are impaired by artherosclerosis (17), coronary stenosis or myocardial hypertrophy (18, 19), resulting in myocardial ischemia or infarction. In the present case, the patients long history of hemodialysis may have caused severe artherosclerosis, and the not critical but clearly evident organic stenosis of the left anterior descending coronary artery may also have contributed to this adverse effect. Furthermore, the inter-coronary steal phenomenon (20) between the stenotic and non-stenotic coronary arteries might be also a possible mechanism for the present adverse effect. Although we should consider that the ischemic event in this patient was caused by unstable angina pectoris, the stenotic lesion was not unstable, indicated by the absence of a thrombus. Another mechanism for ischemia is the reflex activation of the sympathetic nerve system, with a resultant increase in heart rate, myocardial contractility and myocardial oxygen consumption. In the present case, the heart rate increased from 84 to 95 beats /min after administration of sublingual nifedipine, but the blood pressure was markedly decreased, suggesting that myocardial oxygen consumption did not increase.

The present case suggests that even if a patient does not have a history of myocardial ischemic attacks or ischemic changes in the electrocardiogram, sublingual nifedipine should not be used in hypertensive emergencies. The adverse effects we encountered may not be limited to nifedipine and could conceivably be caused by other antihypertensive drugs. If patients in hypertensive emergencies have a history of angina pectoris, myocardial infarction or obvious left ventricular hypertrophy, they should receive careful blood pressure and electrocardiographic monitoring during any treatment.

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