Case Reports

Nitroglycerin-Induced Transient Coronary Artery Occlusion

Kan TAKAYANAGI, M.D., Kazuhiro HOSHI, M.D.,
Sachiko KIMURA, M.D., Teruo INOUE, M.D.,
Shigenori MOROOKA, M.D., and Yutaka TAKABATAKE, M.D.

SUMMARY

Nitroglycerin induced a paradoxical disappearance of a stenosed coronary artery in a 57-year-old man with non-Q wave myocardial infarction. On the coronary angiogram, the left anterior descending coronary artery (with a 95% stenosis) became completely invisible 2 min after 0.3 mg sublingual nitroglycerin. Three minutes later, the artery was opacified again. This transient occlusion may have resulted from a passive collapse of the distal portion of the artery, due to insufficient access of nitroglycerin across the stenotic region.

Additional Indexing Words:

Coronary steal Coronary angiogram Coronary artery collapse

ANGIOGRAPHIC studies have shown that nitroglycerin (NTG) can dilate epicardial coronary arteries. In some patients with angina pectoris, however, sublingual NTG causes deterioration, with apparent ST-segment depression on the electrocardiogram.¹⁾ Furthermore, coronary angiographic studies revealed a paradoxical narrowing of the coronary arteries after NTG.²⁾⁻⁵⁾ In this study, sublingual NTG produced a transient occlusion of a stenosed coronary artery in a patient with a subendocardial myocardial infarction. The mechanism of this occlusion is discussed.

CASE REPORT

A 57-year-old male had had chest pain on effort since April, 1989. Since his serum creatinine phosphokinase level was mildly elevated (300 IU/L) and inverted T waves were observed in leads V₂ to V₆ of the electrocardiogram,

From the Department of Cardiology, Koshigaya Hospital, Dokkyo University School of Medicine, Koshigaya, Saitama, Japan.

Address for correspondence: Kan Takayanagi, M.D., Department of Cardiology, Koshigaya Hospital, Dokkyo University School of Medicine, Minami-koshigaya 2-1-50, Koshigaya, Saitama 343, Japan.

Received for publication October 1, 1990.

Accepted January 11, 1991.

Fig. 1.

Fig. 2.

Fig. 1. Effect of sublingual NTG on coronary angiogram (right anterior oblique view with cranial angulation). A: Control. There was a 95% stenosis at the proximal portion of the left anterior descending coronary artery (LAD), as indicated by the open arrow. B: Two minutes after one tablet sublingual NTG, the distal portion of LAD was not opacified. C: After 4 min, the distal portion of LAD appeared again with the 95% stenosis as indicated by the open arrow.

Fig. 2. Effect of intravenous NTG on coronary angiogram (left anterior oblique view). A: Control. LAD was clearly visualized. B: Thirty seconds after 0.3 mg intravenous NTG, opacification of the LAD was delayed (closed arrows) and the septal branches could not be observed. C: One minute after NTG injection, the peripheral portion of the LAD appeared again with moderate dilation.

he was hospitalized for further evaluation of non-Q wave myocardial infarction on June 1. On admission, his blood pressure was 130/80 mmHg, and his heart rate was 48/min and regular. Heart sounds were normal. The chest x ray revealed a cardiothoracic ratio of 51%. The exercise electrocardiogram (with the modified Bruce protocol) for 9 min was negative. A thallium²⁰¹ myocardial scintigram with bicycle ergometry indicated a slight and reversible perfusion defect in the anterior area.

Cardiac catheterization, performed on June 30, was normal with an ejection fraction of 69%. On coronary angiography by the Judkins technique, a 95% stenosis was observed in the proximal portion of the left anterior descending coronary artery (LAD), as indicated by an open arrow in the right anterior oblique view with cranial angulation (Fig. 1-A). The right coronary artery was normal. Two minutes after one tablet of sublingual NTG, the left coronary angiogram with the same view revealed that the distal portion of the LAD was not opacified while the rest of the coronary arteries were dilated (Fig. 1-B). There was no significant change in the 12 lead electrocardiograms. Four minutes later, the stenosed segment of the LAD (Fig. 1-C: open arrow) was clearly opacified with marked dilation of the peripheral portion. The patient did not complain of any chest pain during the NTG administration.

To see the reproducibility of these findings, 0.3 mg NTG was injected intravenously 25 min after the initial sublingual dose. Before this injection, a left coronary angiogram on the left anterior oblique view failed to show the stenosis in the proximal portion of the LAD (Fig. 2-A). Thirty seconds after NTG injection, aortic pressure decreased from 132/72 mmHg to 82/42 mmHg. A coronary angiogram at this moment revealed diffuse narrowing and delayed filling of the LAD, as indicated by closed arrows (Fig. 2-B), with invisible septal branches. The left circumflex artery was dilated. One minute after NTG, the LAD was clearly opacified with marked dilation of its peripheral portions (Fig. 2-C). A final angiogram after 3 min indicated similar findings. In this second study, there were no electrocardiographic changes and no chest pain. An aortocoronary bypass was made to the distal LAD and the patient was free from symptoms for 12 months.

DISCUSSION

In our first study, sublingual NTG induced an apparent occlusion of the highly stenosed LAD and, at the same time, dilated the patent circumflex artery (Fig. 1-B). Although the mechanism of this occlusion was obscure, at least three possibilities could be considered from the two series of angio-

grams. First, there was a possibility that the absorbed NTG could not reach beyond the highly stenosed coronary artery because of insufficient coronary flow. Therefore, the LAD failed to dilate, while the left circumflex artery could fully dilate. Secondly, decreased resistance of the left circumflex coronary artery could shunt blood flow from the stenosed LAD.⁴⁾⁻⁶⁾ These two mechanisms could combine to induce an apparent LAD occlusion. Third, the decreased aortic pressure after NTG administration, both sublingually and intravenously, might reduce the perfusion pressure distal to the marked stenosis and induce passive collapse of the LAD.⁷⁾ However, the contribution of the perfusion pressure to the transient occlusion seemed to be low, since the contrast medium was injected manually with much higher pressure than the aortic pressure. The possibility of a coronary artery spasm overlapping the stenosis could be ruled out because the diffuse narrowing of the LAD was seen after intravenous NTG.^{8),9)}

Several studies have shown that a transient narrowing of the coronary artery could occur after sublingual NTG.²⁾⁻⁵⁾ Most of these findings were observed in the right coronary artery. Therefore, a coronary spasm could not be completely ruled out. In no studies was the reproducibility of this effect checked. We reproduced the paradoxical LAD occlusion in a second trial. The incomplete occlusion after the intravenous NTG might be partly due to the tolerance to NTG and to the different timing of angiography.

Although the duration of the LAD occlusion was short, our findings clearly demonstrate that a marked heterogeneity of coronary artery diameter could occur after NTG in a highly stenotic lesion. In addition to the stenosis, the timing of the contrast medium injection and the route of NTG administration seemed to be important. Further studies on these conditions might clarify the mechanism of the paradoxical NTG action.

REFERENCES

1. Russek HI, Urbach KF, Zohman BL: Paradoxical action of glyceril trinitrate (nitroglycerin) in coronary patients. *JAMA* **158**: 1017, 1955
2. Feldman RL, Pepine CJ, Conti CR: Unusual vasomotor coronary arterial responses after nitroglycerin. *Am J Cardiol* **42**: 517, 1978
3. Curry RC: Coronary vasoconstriction following nitroglycerin. *Cathet Cardiovasc Diagn* **6**: 211, 1980
4. Dalal JJ, McCans JL, Parker JO: Nitroglycerin-induced coronary vasoconstriction. *Cathet Cardiovasc Diagn* **10**: 33, 1984
5. Miller JT, Henry RL, Kern MJ: Potentiation of nitroglycerin-induced ST-segment elevation by beta-adrenergic blockade. *Am Heart J* **112**: 839, 1986
6. Logan SE: On the fluid mechanics of human coronary artery stenosis. *IEEE Trans Biomed Eng* **22**: 327, 1975
7. Santamore WP, Walinsky P: Altered coronary flow responses to vasoactive drugs in the pre-

- sence of coronary arterial stenosis in the dog. *Am J Cardiol* **45**: 276, 1980
8. Factor SF, Kirk ES: Pathophysiology of myocardial ischemia. *in* The Heart, ed by Hurst JW, 6 Ed, McGraw-Hill, New York, p 863, 1986
 9. Becker LC: Conditions for vasodilator-induced coronary steal in experimental myocardial ischemia. *Circulation* **57**: 1103, 1978