Nitroglycerin-Induced Transient Coronary Artery Occlusion

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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,
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 infarc-
tion 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 electro-
cardiogram (with the modified Bruce protocol) for 9 min was negative. A
thallium\textsuperscript{201} 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 tech-
nique, 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 nar-
rowing 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. 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. 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.

Several studies have shown that a transient narrowing of the coronary artery could occur after sublingual NTG. 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

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