eventual occurrence of neurocardiogenic syncope at rest. Prinzmetal's angina also is a syndrome with poorly understood pathophysiologic features consisting of abnormal neurologic control of the coronary vascular tone. The association of abnormal coronary motility, neurocardiogenic syndrome, and bronchogenic carcinoma seems to be new in the literature and may suggest a wider spectrum of the paraneoplastic neuropathies. Possibly, some peculiar clinical features and surely a unique treatment (chemotherapy) seem to characterize the cancer-associated neurocardiogenic syncope.

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Serial Angiography in Cocaine-Induced Myocardial Infarction*

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A young man suffered an acute inferior myocardial infarction following clinical use of cocaine as topical anesthesia. Coronary angiography showed occlusion of both the posterior descending and posterolateral arteries which was resistant to intra coronary administration of nitroglycerin and verapamil, a finding consistent with thrombolic occlusion. A subsequent angiogram 3 months later showed no residual lesions. (CHEST 1997; 111:822-24)

Key words: cocaine; coronary angiography; myocardial infarction

Myocardial infarction following illicit cocaine use is well described, and there are occasional reports of such an occurrence after therapeutic use. The pathophysiology may differ in several ways from myocardial infarction associated with atherosclerotic disease. Vasocostruction, in situ thrombus formation, and platelet aggregation may all be important, but in most cases the exact mechanisms are uncertain. We describe a patient in whom serial angiography provides some insights into the pathophysiology of cocaine-induced myocardial infarction.

CASE REPORT

A 29-year-old man, a nose smoker with no coronary risk factors, underwent septoplasty and reduction of turbinate. Local anesthesia was achieved with injection of 0.5% lidocaine hydrochloride (Xylocaine) and application of a thin film of 12.5% cocaine hydrochloride and 0.1% epinephrine paste to the nasal mucosa. The surgery was uneventful, but 4½ h later he developed chest pain radiating to the left arm and jaw. He first reported symptoms 6 h after surgery when he was found to be sweaty with a pulse of 75 beats per minute and a BP of 120/50 mm Hg. The ECG showed a 3-m V ST segment elevation in the inferior leads consistent with acute myocardial infarction. He was treated with aspirin, 150 mg, repeated doses of sublingually administered nitroglycerin spray, sublinguially administered nifedipine, 20 mg, intravenously administered heparin, 5,000 U, and an intravenous infusion of nitroglycerin. A thrombolytic drug was not given because of recent surgery.

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FIGURE 1. A: right coronary angiogram performed 13 h after intranasal administration of cocaine. Repeated doses of intracoronary nitroglycerin and verapamil have been given. There is occlusion of both the posterior descending and posterolateral branches (arrows) and dilation of the proximal artery. B: angiography performed 3 months later shows the posterior descending and posterolateral arteries are both patent. Vasodilators have not been given.

Because of ongoing chest pain and ST segment elevation, coronary angiography was performed 12 h after application of the cocaine paste. This demonstrated occlusion of the posterior descending and posterolateral branches of the right coronary artery (Fig 1A). The coronary arteries were otherwise normal. Boluses of intracoronary nitroglycerin (4×100 μg) and verapamil (5×100 μg) were administered over a period of 1 h with no evidence of reperfusion. Coronary angioplasty was not performed because of the small territory at risk. The peak creatine kinase level was 797 U/L (normal, <190 U/L). Serial ECGs showed evolution of Q waves in the inferior leads. Echocardiography revealed inferior wall hypokinesis with preservation of left ventricular function. The foramen ovale was not patent. He was discharged on a regimen of aspirin, 150 mg daily. Coronary angiography repeated 3 months later demonstrated patency of both previously occluded arteries (Fig 1B).

DISCUSSION

The occurrence of two occluded arteries seen in this and other cases is consistent with a systemic effect of cocaine, which may act at more than one vascular site. Intranasally administered cocaine is known to cause coronary vasoconstriction, platelet activation, and platelet α-granule release. In addition, intense coronary vasoconstriction may itself damage the endothelium, thereby increasing the likelihood of platelet adhesion and thrombus formation. These factors may explain coronary occlusion at multiple sites due to either vasoconstriction or thrombosis.

In this case, coronary occlusion was documented 12 h after cocaine administration, at which time there were no systemic effects such as tachycardia, hypertension, or agitation, and tissue levels of cocaine were likely to be low. Systemic and intracoronary vasodilators failed to relieve the occlusion despite significant dilation of the more proximal coronary artery. These findings suggest that cocaine-induced vasoconstriction alone is unlikely to be the cause for the persistent occlusion, and another mechanism, such as thrombosis, with or without vasospasm as the initiating event, is more likely to be the cause. However, angiography alone does not allow a definite diagnosis of the cause of coronary occlusion.

β-Blockers should be avoided in cocaine-induced myocardial infarction because there is clear evidence that they potentiate cocaine-induced coronary vasoconstriction. Nitroglycerin and verapamil have been shown to reverse coronary vasoconstriction induced by cocaine. Aspirin is recommended as an antiplatelet agent. Thrombolytic therapy has been used, but its benefits are uncertain because adequate clinical trials have not been undertaken. Coronary angiography often is normal after cocaine-induced myocardial infarction. This case emphasizes that the findings on angiography depend on its timing, and a normal appearance could be consistent with earlier coronary occlusion due to vasospasm or thrombosis. In this
patient, treatment with vasodilators and antithrombotic drugs was started relatively late in the time course of the myocardial infarction. It is possible that early vigorous therapy could reduce the risk of persistent coronary occlusion which is resistant to treatment.

REFERENCES

Bilateral Functioning Vineberg Grafts*

A 25-Year Follow-up

Michael M. Hughes, MD

This report describes a 25-year follow-up of angiographically proven bilateral functioning Vineberg grafts. This represents the longest angiographic follow-up found in the literature. The relevance of these grafts in present-day revascularization procedures is discussed.

(CHEST 1997; 111:824-26)

Key words: angina pectoris; coronary artery disease; internal thoracic artery implant; Vineberg graft

Abbreviations: LAD=left anterior descending coronary artery; LIMA=left internal mammary artery; RCA=right coronary artery; RIMA=right internal mammary artery

This report describes the results of high-risk percutaneous coronary intervention. The case is unique in that a 25-year follow-up of bilateral functioning Vineberg grafts, angiographically proven, has not been previously reported. The importance of these procedures is discussed.

CASE REPORT

A 76-year-old white man with a past medical history of diabetes mellitus, hypercholesterolemia, peripheral vascular disease, and coronary artery disease initially presented in March 1970 with accelerating angina.

The patient's initial angiogram, March 1970, revealed a severely diseased proximal right coronary artery (RCA) and an ostial left anterior descending (LAD) coronary artery lesion. The patient underwent coronary artery bypass grafting on March 20, 1970, at the Cleveland Clinic Foundation. At the time of surgery, he received a saphenous vein graft to the distal right coronary artery. The right internal mammary artery (RIMA) and left internal mammary artery (LIMA) were mobilized and directly implanted into the anterior and lateral walls, respectively, of the left ventricle.

The patient did quite well and was unavailable for cardiac follow-up until October 1991. At that time, he presented with symptoms of angina, functional class III. A treadmill stress test was markedly positive at 1 min 40 s with marked ST depression and chest pain. The patient became clinically unstable and was admitted to the coronary care unit where his condition was stabilized with medical therapy. He had no rise in enzymes and underwent diagnostic catheterization on October 23, 1991. This revealed an occluded RCA and occluded saphenous vein graft to the RCA. The RCA was totally obstructed in the midsegment and filled via collateral vessels from the LIMA Vineberg graft. The LAD was severely diseased proximally. The circumflex system was dominant with an 80% tandem lesion in the proximal high lateral branch. The left ventricular function was within normal limits. The LIMA Vineberg graft was widely patent and perfused primarily the inferior left ventricular wall. The right internal mammary artery Vineberg graft had a 30% ostial lesion and distally had collateralized the first two diagonal branches and perfused the LAD (Figs 1-3).

The patient underwent a successful percutaneous transluminal coronary angioplasty of his high lateral circumflex artery. Following discharge the patient progressed in his activity level and was essentially a functional class I-II. He continued to receive medical therapy. A thallium stress test was done on December 11, 1991; the patient performed for 4 min 40 s with ST depression and mild chest symptoms. The thallium scan revealed ischemia in the septum, apex, and inferior segments.

The patient again presented with accelerating angina and a non-Q-wave myocardial infarction (creatinine kinase peak of 365) in October 1992. Emergency catheterization was done on presentation. The catheterization revealed the dilated segment of the high lateral circumflex artery, 50 and 40% stenosed at the respective percutaneous transluminal coronary angioplasty sites from October 1991. The patient's LAD was well collateralized.

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