Acute Myocardial Infarction After Use of Pseudoephedrine for Sinus Congestion

Hector Derreza, MD, Michael D. Fine, MD, and Ara Sadaniantz, MD

Coronary vasospasm has been recognized as an important factor for coronary atherogenesis and the development of symptoms associated with coronary blood flow obstruction that can lead to ischemia and eventually myocardial infarction.[1] Association of drugs and coronary artery vasospasm has been reported for such chemicals as intravenous ephedrine, cocaine, dipyridamole, trimethaphan, and sumatriptan.[2-6] Few cases have been reported in which pseudoephedrine is associated with myocardial infarction. Wiener et al[6] described a healthy 28-year-old man with no cardiac risk factors who had acute myocardial infarction with ST segment elevation after taking 60 mg of pseudoephedrine. Subsequent coronary angiography showed normal coronary arteries in this patient. We describe the association of anginal symptoms and eventual myocardial infarction with ingestion of an over-the-counter remedy containing pseudoephedrine.

Case Report

A 46-year-old man came to the hospital after an acute onset of severe substernal chest pain associated with mild shortness of breath and profuse diaphoresis. His symptoms began 1 hour after taking an over-the-counter medication containing 500 mg of acetaminophen and 15 mg of pseudoephedrine hydrochloride, which he had been taking for 3 months for sinus congestion. He had been taking up to 1 tablet every 6 hours. His cardiac risk factors included male sex and heavy smoking (30 pack-years), but his medical history was not unusual. He had no family history of cardiac disease and denied use of illegal drugs. He was taking no other medication. He worked as a truck driver and walked 3 times per week for up to 1 hour without chest pain. Since beginning the use of the sinus medication, he noted a mild chest pressure lasting from seconds to minutes that began 30 to 60 minutes after ingestion of the sinus medication. He had persistent sinus congestion before admission; however, he was able to work and denied psychosocial stress. The day of admission he took his medication about 30 minutes before developing severe chest pressure similar in quality to previous episodes but greater in intensity.

On admission he was having severe chest pain. His blood pressure was 180/115 mmHg, heart rate 66 beats per minute, and respiratory rate 26/min. He was diaphoretic, his jugular veins were not distended, his heart had a normal S_1 and S_2, no S_3 or S_4, and no murmurs. Findings on an abdominal examination were normal. His extremities had no edema, his pulses were palpable bilaterally, and he had no cyanosis. Laboratory data on admission showed normal findings on a complete blood cell count and automated chemistry screening (sequential multiple analysis-7); his partial thromboplastin time was 25 sec, prothrombin time 11.0 sec, international normalized ratio (INR) 0.9, magnesium 1.3 mEq, and creatine phosphokinase (CPK) 134 IU/L. Findings on his chest radiograph were normal. His electrocardiogram showed a normal sinus rhythm of 75 beats per minute, a normal QRS axis, normal atrioventricular conduction, and 4-mm ST segment elevations in the precordial leads V_1 through V_5 (Figure 1).

He received 4 sublingual nitroglycerin tablets and was given intravenous nitroglycerin. He suddenly developed transient monomorphic ventricular tachycardia with a rate of 101 beats per minute, which resolved spontaneously. His electrocardiogram after this episode showed decreased ST elevations and he became pain-free. It was thought that he spontaneously revascularized. He was admitted to the coronary care unit

Submitted, revised, 4 June 1997.

From the Division of Cardiology, The Miriam Hospital, Brown University School of Medicine, Providence. Address reprint requests to Ara Sadaniantz, MD, Division of Cardiology, The Miriam Hospital, 164 Summit Ave, Providence, RI 02906.

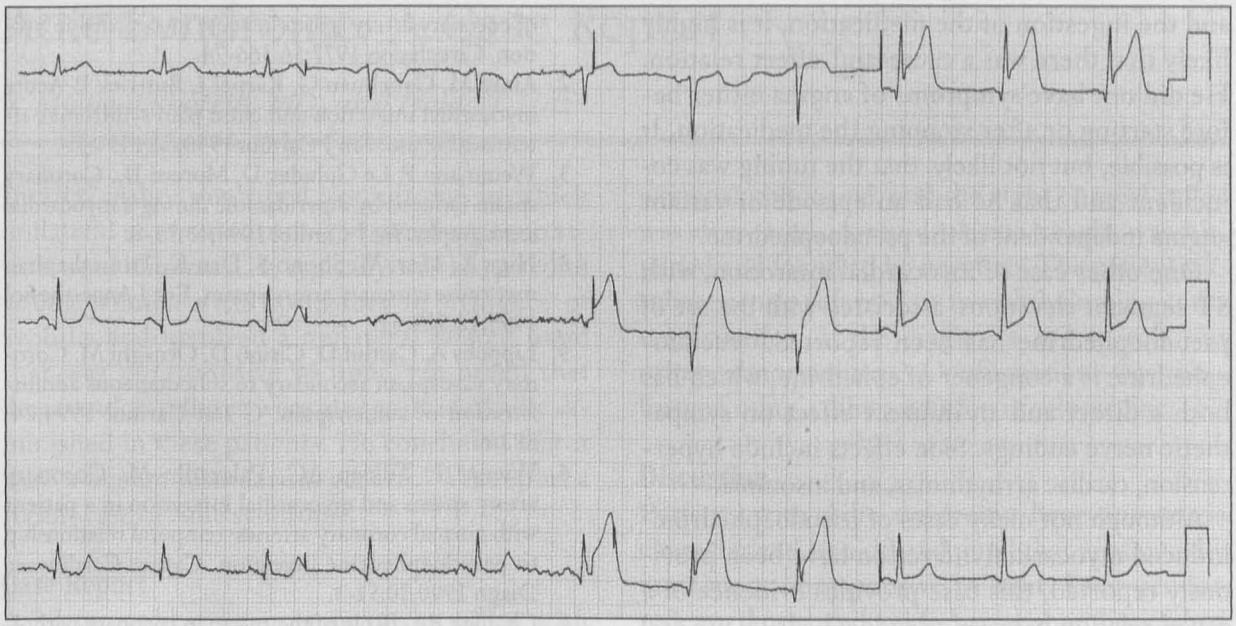

Figure 1. The electrocardiogram showing 4-mm ST segment elevations in the precordial leads V_1 through V_5 consistent with an acute anterior myocardial infarction.

on intravenous heparin and intravenous nitroglycerin. He received 325 mg of chewable aspirin and 25 mg of metoprolol orally. His first CPK was 1701 IU/L with MB fraction of 155 ng/mL and MB index of 9.1 percent (normal value for our laboratory is MB index less than 4 percent). A toxicology screening test of the urine was negative for cocaine. On his lipid profile he had a total cholesterol of 199 mg/dL, high-density lipoprotein 66 mg/dL, low-density lipoprotein 121 mg/dL, and triglyceride level 58 mg/dL.

He underwent cardiac catheterization on hospital day 2. The ventriculogram showed anterolateral and apical hypokinesis with an ejection fraction of 50 percent. On coronary angiography the mid-right coronary artery had 10 percent plaque, and the left anterior descending artery had mild plaques of 30 percent proximally and 20 percent at its mid portion (Figure 2). Nitroglycerin caused unbearable headaches and was discontinued. He was prescribed amlodipine 10 mg/d and was discharged to home on hospital day 6 in good condition. He was advised to avoid the use of any sympathomimetic agent. Nine months after discharge he remains free of anginal symptoms.

Discussion

Coronary artery spasm plays an important role in acute ischemic events, including acute myocardial infarction and exertional angina.[1,7,8] Smoking is the most important risk factor for coronary artery spasm because of its sympathomimetic action.[9] Age, sex, hypertension, diabetes, and serum levels of cholesterol and uric acid have no notable predictive value for coronary vasospasm.[10] Dipyridamole, trimethaphan, cocaine, ephedrine, and sumatriptan have also been associated with coronary vasospasm.[2-5]

We report a case in which the use of pseudoephedrine was associated with symptoms suggestive of angina pectoris. Eventually the patient came to the emergency department with an acute myocardial infarction after taking pseudoephedrine and was found to have angiographically minimal coronary artery disease. Because of the predictable association of developing symptoms

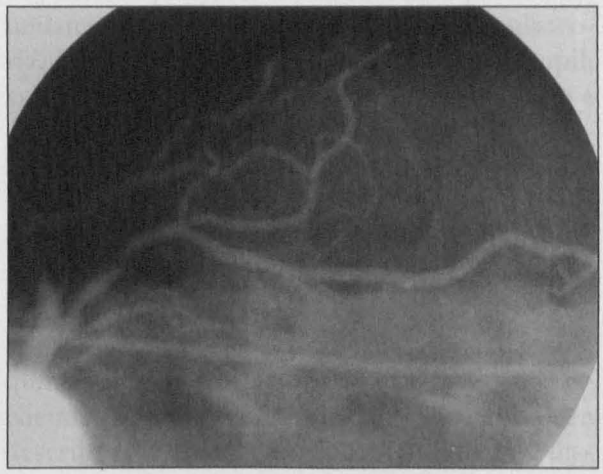

Figure 2. Coronary angiography of the left coronary system with mild plaques.

and the ingestion of the medication, it is highly likely that there was a cause-and-effect relation. He did not have symptoms of angina either before starting or after stopping the medication. It is possible, but not likely, that the timing was coincident and that he had an episode of variant angina independent of the pseudoephedrine.

One other case of myocardial infarction, with ST segment elevations, associated with the use of pseudoephedrine has been reported.[6] Pseudoephedrine is a congener of ephedrine, which has both a direct and an indirect effect on sympathetic nerve endings. Side effects include hypertension, cardiac arrhythmias, and insomnia.

Although not many cases of pseudoephedrine-induced myocardial infarction have been previously reported, this case provides evidence of a causal relation between pseudoephedrine use and acute myocardial infarction in one susceptible patient. We question the safety of this medication in over-the-counter preparations in susceptible patients and suggest awareness of this uncommon but potentially fatal side effect. Further research might be directed to recognition of persons susceptible to myocardial ischemia with this type of medication.

References

1. Oliva PB, Breckinridge JC. Arteriographic evidence of coronary artery spasm in acute myocardial infarction. Circulation 1977;56:366-74.
2. Amin M, Gabelman G, Karpel J, Buttrick P. Acute myocardial infarction and chest pain syndromes after cocaine use. Am J Cardiol 1990;66:1434-7.
3. Weinmann P, Le Guludec D, Moretti JL. Coronary spasm induced by dipyridamole during a myocardial scintigraphy. Int J Cardiol 1994;43:75-8.
4. Higa K, Mori M, Shono S, Dan K. Trimethaphan may cause coronary artery spasm. Eur J Anaesthesiol 1995;12:529-32.
5. Lippolis A, Castini D, Cirino D, Ornaghi M. Coronary vasospasm secondary to subcutaneous administration of sumatriptan. G Ital Cardiol 1994;24:883-6.
6. Wiener I, Tilkian AG, Palazzolo M. Coronary artery spasm and myocardial infarction in a patient with normal coronary arteries: temporal relationship to pseudoephedrine ingestion. Cathet Cardiovasc Diagn 1990;20:51-3.
7. Chahine RA. [Role of the spasm in coronary pathology.] Ann Cardiol Angeiol (Paris) 1982;31:563-70.
8. Maseri A, Chierchia S, L'Abbate A, Biagini A, Distante A, Parodi O, et al. [The role of spasm in angina pectoris, myocardial infarction and sudden death. Indications for future research and treatment.] Arch Mal Coeur Vaiss 1982;75:701-16.
9. Ball K, Turner R. Smoking and the heart: the basis for action. Lancet 1974;2:822-6.
10. Nobuyoshi M, Abe M, Nosaka H, Kimura T, Yokoi H, Hamasaki N, et al. Statistical analysis of clinical risk factors for coronary artery spasm: identification of the most important determinant. Am Heart J 1992;124:32-8.