EDITORIAL

Cocaine-Associated Chest Pain and Acute Myocardial Infarction

James McCord

Heart & Vascular Institute, Henry Ford Hospital, Detroit, Michigan, United States

The use of cocaine, either intranasal or inhaled, is an increasingly significant public health issue in the United States and Europe. Cocaine is the second most commonly used illicit drug (second to marijuana)[1] in the United States in 2005 there were approximately 450 000 cocaine-related encounters in Emergency Departments.[2] The World Drug Report of 2009 estimates that in individuals between 15 and 64 years of age, 1.4% of the population in Western Europe and 3% of the population in Spain use cocaine.[3] Cocaine us is not only associated with myocardial infarction but also with aortic dissection.[4] In addition, an acute pulmonary condition known as "crack lung" which involves hypoxemia, hemoptysis, respiratory failure, and diffuse pulmonary infiltrates after inhalation of free base cocaine has been described.[5]

In a recent study published in this issue of *Revista Española de Cardiología* Bosch et al[6] report on the large number of patients who presented to an urban University hospital with chest pain. There were 1240 patients age 55 years or less evaluated in the Chest Pain Unit that were questioned concerning the recent use of cocaine. In addition to self-reports the use of cocaine was determined by selective urinary screening when deemed appropriate by the responsible clinician. This is a significant study in that a large number of patients were evaluated which distinguishes it from many other investigations. There were 53 patients that admitted to recent cocaine use, and 10 patients that denied cocaine use but had a positive urine test yielding a total of 63 (5%). If patients older than 55 years were included the frequency of cocaine use was only 2% which is similar to other reports.[7] Since self-reported cocaine use was used, and urinary cocaine screening was only done selectively, the actual frequency of cocaine use is certainly higher than was reported.

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Correspondence: J. McCord, MD,
2799 West Grand Blvd. Detroit. MI 48202. USA
E-mail: jmccord1@hfhs.org

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Patients with recent cocaine use were more likely to be younger, male, and smokers. These findings are consistent with other studies.[8] There were 58 myocardial infarctions in the entire cohort, 10 (17%) of which had recent cocaine use ingestion. This high frequency of cocaine use (17%) in patients with myocardial infarction is at variance with other reported studies of 0.7%-6%.[9,10] The authors cite a more recent study reporting a 14% frequency of myocardial infarction in patients with cocaine use.[11] This higher frequency of myocardial infarction is likely in part related to the newer more sensitive troponin assays that can detect smaller levels of myocardial necrosis in patients that were previously classified as unstable angina. However, these differences may also be due to an increased prevalence of cocaine use. Finally some of the variance between these studies is likely due to different patient populations, such as urban and suburban.

Of particular note in this study[6] is that 60% of the patients with myocardial infarction were treated with beta-blockers. In general beta-blockers are considered contraindicated in patients after cocaine ingestion because of the concern of the unopposed alpha-adrenergic effect leading to increased coronary vasoconstriction. A study of patients undergoing heart catheterization demonstrated that coronary vascular resistance was significantly increased after beta-blockers were administered in patients that had received cocaine.[12] Multiple experimental animal models have shown that after cocaine ingestion the use of beta-blockers leads to decreased coronary blood flow and higher mortality.[13,14] The American Heart Association Scientific Statement of 2008 concerning cocaine-associated chest pain recommends that beta-blockers be avoided even in definite myocardial infarction in the acute setting of cocaine use.[15] Selective use of beta-blockers at discharge is recommended depending on the judgment if the patient is likely to continue using cocaine or not. This recommendation seems particularly prudent in the era of aggressive revascularization for acute coronary syndrome, when more contemporary studies have demonstrated no mortality benefit from the early use of beta-blockers.[16] The early use of beta-blockers in acute coronary syndrome is no longer

included as an American College of Cardiology/American Heart Association quality measure. In the study by Bosch et al[6] there is no mention of adverse events in patients with myocardial infarction that received beta-blockers; reporting of the lack of adverse events would add to the literature in this difficult patient population.

The study by Bosch et al[6] in Spain confirms what is being experienced in other parts of the western world. The use of cocaine as a recreational drug is increasing and we are likely to see more of these individuals in Emergency Departments in the United States and Europe. This is important because the recommended treatment of patients with cocaine-associated chest pain is different from the patients who have not used cocaine.[15] Young patients with non-traumatic chest pain should be questioned concerning cocaine use. The Bosch et al study demonstrated in the 63 patients with recent cocaine use, 10 (16%) denied cocaine use but had positive urinary screen for cocaine metabolites. Even if a young patient with chest pain denies cocaine use, the use of cocaine should be considered.

REFERENCES

1. Hughes, A, Sathe, N, Spagnola, K. State Estimates of Substance Use from the 2005-2006 National Surveys on Drug Use and Health. DHHS Publication No. SMA 08-4311, NSDUH Series H-33. Rockville, Md: Substance Abuse and Mental Health Services Administration, Office of Applied Studies; 2008.
2. Substance Abuse and Mental Health Services Administration, Office of Applied Studies. Drug Abuse Warning Network, 2005: National Estimates of Drug-Related Emergency Department Visits. DAWN Series D-29. DHHS Publication No. (SMA) 07-4256. Rockville, Md: Substance Abuse and Mental Health Services Administration. Office of Applied Studies: 2007.
3. World Drug Report 2009. United Nations office for Drugs and Crime. Available from: http//www.unodc.org/unodc/en/data-and-analysis/WDR-2009.html.
4. Hsue PY, Salinas CL, Bolger AF, Benowitz NL, Waters DD. Acute aortic dissection related to crack cocaine. Circulation. 2002;105:1592-5.
5. Forrester JM, Steele AW, Waldron JA, Parsons PE. Crack lung: an acute pulmonary syndrome with a spectrum of clinical and histopathologic findings. Am Rev Respir Dis. 1990;142:462-7.
6. Bosch X, Loma P, Guasch E, Nogué S, Ortiz JT, Sánchez M. Prevalencia, características clínicas y riesgo de infarto de miocardio en pacientes con dolor torácico y consumo de cocaína. Rev Esp Cardiol. 2010;63:1028-34.
7. Bishop CR, Dargan PI, Greene SL, Garnham F, Wood DM. Emergency department presentations with suspected acute coronary syndrome--frequency of self-reported cocaine use. Eur J Emerg Med. 2010;17:164-6.
8. Mittleman MA, Mintzer D, Maclure M, Tofler GH, Sherwood JB, Muller JE. Triggering of myocardial infarction by cocaine. Circulation. 1999;99:2737-41.
9. Hollander JE, Hoffman RS, Gennis P, Fairweather P, DiSano MJ, Schumb DA, et al. Prospective multicenter evaluation of cocaine-associated chest pain. Cocaine Associated Chest Pain (COCHPA) Study Group. Acad Emerg Med. 1994;1:330-9.
10. Feldman J.A., Fish S.S., Beshansky J.R., Griffith J.L.,Woolard R.H., Selker H.P. Acute Cardiac Ischemia in patients with cocaine-associated complaints: results of a multicenter trial. Ann Emerg Med. 2000;36:459-476.
11. Bansal D, Eigenbrodt M, Gupta E, Mehta JL. Traditional risk factors and acute myocardial infarction in patients hospitalized with cocaine-associated chest pain. Clin Cardiol. 2007;30:290-4.
12. Lange RA, Cigarroa RG, Flores ED, McBride W, Kim AS, Wells PJ. Potentiation of cocaine-induced coronary vasoconstriction by beta-adrenergic blockade. Ann Intern Med, 1990;112:897-903.
13. Guinn MM, Bedford JA, Wilson MC. Antagonism of intravenous cocaine lethality in nonhuman primates. Clin Toxicol. 1980;16499-508.
14. Vargas R, Gillis RA, Ramwell PW. Ramwell, Propranolol promotes cocaine-induced spasm of porcine coronary artery. J Pharmacol Exp Ther. 1991;257:644-6.
15. McCord J, Jneid H, Hollander JE, de Lemos JA, Cercek B, Hsue P, et al. Management of cocaine-associated chest pain and myocardial infarction. A scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation. 2008;117:1897-907.
16. Chen ZM, Pan HC, Chen YP, Peto R, Collins R, Jiang LX, et al. Early intravenous then oral metoprolol in 45,852 patients with acute myocardial infarction: randomised placebo-controlled trial. Lancet, 2005;366:1622-32.