Cardiac Arrest in Patients Who Smoke Crack Cocaine

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The aim of the study is to determine the clinical features and outcomes of cocaine users admitted to the hospital after cardiac arrest and compare them with nonusers. Cocaine is associated with cardiovascular complications, including ventricular arrhythmias; however, resuscitated cardiac arrest in relation to cocaine use is not a well-defined clinical entity. We reviewed available hospital charts at San Francisco General Hospital with the International Classification of Diseases, Ninth Revision diagnosis of cardiac arrest and cocaine use from 1994 to 2006. Clinical features and outcomes of cocaine users were compared with those of randomly selected control patients and age-matched controls with resuscitated cardiac arrest without cocaine use. We identified 22 patients with resuscitated cardiac arrest in the setting of cocaine use. Their average age was 42 ± 10 years, >20 years younger than nonusers (68 ± 16 years, p <0.01). After cardiac arrest, 12 of 22 patients (55%) who used cocaine had complete neurologic recovery in contrast to only 3 of 20 unmatched controls (15%, p <0.01) and 7 of 41 age-matched controls (17%, p <0.01). Only 10 of 22 cocaine users (46%) died compared with 15 of 20 unmatched controls (75%, p = 0.05) and 32 of 41 age-matched controls (78%, p <0.01). In a combined analysis of all patients, cocaine use was the only significant predictor of neurologic recovery (p <0.01) and survival (p <0.01).

In conclusion, cocaine use is associated with cardiac arrest. In patients with cardiac arrest, cocaine users are younger than nonusers and more likely to survive with neurologic recovery, even compared with age-matched controls with cardiac arrest.

Methods and Results

We searched the medical record database of San Francisco General Hospital from 1994 to 2006 to identify all patients who were resuscitated after sudden cardiac death (cardiac arrest) who had used cocaine within 24 hours before the cardiac arrest. Patients with electrocardiographic evidence of ST-segment elevation myocardial infarction were excluded. We selected the first available patients with cardiac arrest and no cocaine use as a control group. Because of the large difference in age between cocaine users and controls with cardiac arrest, we selected a second control group that was age matched to the cocaine group. The purpose of this study was to ascertain whether differences seen between users and unmatched controls could be accounted for by age. This study was approved by the Committee on Human Research of the University of California, San Francisco.

The demographics of each patient were recorded in addition to medical history, including hypertension, systolic left ventricular dysfunction, diabetes mellitus, renal disease, and coronary disease. Information about concomitant drug use, alcohol use, and cigarette smoking was also obtained. Left ventricular hypertrophy was determined using standard electrocardiographic (ECG) or echocardiographic mass criteria.\footnote{Urine toxicology test results were positive for cocaine in 20 of 22 cocaine-related cases and not performed in the other 2 patients; only 5 non-cocaine users had urine toxicology screens performed, and all 5 had negative results. Type of arrest (pulseless electrical activity, ventricular tachycardia, ventricular fibrillation, asystole, and other), as well as clinical data, such as ECG data as the QT interval (because cocaine was associated with lengthened QT intervals\textsuperscript{2–3}), cardiac and neurologic studies performed during hospitalization, and laboratory studies were obtained through chart review. Clinical outcomes for each group, including death or neurologic recovery, were compared. The first electrocardiogram after cardiac arrest was obtained, and the QT interval was measured by a cardiologist who was blinded with respect to patient drug status. QT interval was corrected for rate using Bazett’s QT correction formula.\footnote{The Q wave to the end of the T wave was measured; if the T wave was obscured, the T wave was extrapolated from downstroke to baseline.}

Differences between cocaine users and controls were assessed using chi-square tests for binary variables and

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Cooling therapy. Resuscitation times were similar including defibrillation, intubation, therapy with pressors, and cardiac activity and then asystole. And/or ventricular fibrillation followed by pulseless electrical activity. Twenty-two patients with cocaine-related resuscitated cardiac arrests were identified. Clinical features of these patients are compared with those of unmatched and age-matched control groups in Table 1. Mean age of cocaine users was 42 ± 10 years, >20 years younger than the nonuser group (68 ± 16 years, p < 0.01). In cocaine patients, the most common form of the drug used was crack cocaine. The interval between cocaine use and cardiac arrest was unavailable for most patients. Unmatched control patients were more likely to have underlying structural heart disease, have coronary disease, and be treated with antihypertensive medication compared with cocaine users. In both groups, most patients presented with ventricular tachycardia and/or ventricular fibrillation followed by pulseless electrical activity and then asystole.

Each group received similar treatment after arrest, including defibrillation, intubation, therapy with pressors, and cooling therapy. Resuscitation times were similar between cocaine users and nonusers (30 ± 29 vs 25 ± 23 minutes, p = 0.60). There was no significant difference in magnesium values between the 2 groups. Nonmatched controls had lower serum potassium compared with cocaine users, whereas age-matched controls had potassium values similar to cocaine users. One cocaine user had a troponin level >2 ng/ml compared with 5 non-cocaine users (p = 0.06). Comparison of the first electrocardiogram obtained after arrest showed no difference in raw or corrected QT intervals between cocaine users and unmatched controls. Age-matched controls had longer corrected QT intervals compared with cocaine users (482 vs 320 ms, p < 0.01)

More than half the cocaine users (12 of 22) made a full neurologic recovery compared with only 3 of 20 nonusers (p < 0.01). Fewer cocaine users died compared with non-cocaine users (10 vs 15 patients, p = 0.05). Because the comparatively low mortality in cocaine users may have been a consequence of their younger age, we compared their outcome with that of an age-matched group of patients with resuscitated cardiac arrest. Mortality in this group was 32 of 41 (78%), similar to the first control group and higher than in patients who used cocaine (p < 0.01). In logistic regression analysis, cocaine use was the single significant predictor of neurologic recovery (p < 0.01) and survival (p < 0.01). After adjusting for cocaine use, there was no difference in neurologic recovery or survival between cocaine users and controls (unmatched and matched).

**Discussion**

In this study, we found that cocaine users resuscitated from sudden cardiac death were much younger than nonusers with cardiac arrest and less likely to have a history of coronary disease, heart failure, or atrial fibrillation. More than half the cocaine users survived with complete neurologic recovery. Conversely, survival with neurologic recovery was very uncommon in control patients resuscitated after cardiac arrest, even in an age-matched control group. We speculate that cocaine users have better outcomes because they are much less likely to have serious ongoing co-morbid conditions that attenuate their survival. Torsades de pointes, ventricular tachycardia, ventricular fibrillation, and wide-complex arrhythmias were reported after cocaine use in isolated cases. To our knowl-
edge, there are no previously reported series of patients with resuscitated cardiac arrest after cocaine use.

By blocking the reuptake of catecholamines at the synaptic terminals, cocaine acts as a powerful sympathomimetic agent and thereby may increase ventricular irritability and lower the threshold for fibrillation.\(^9\) Cocaine also inhibits sodium transport across membranes during depolarization, thus inhibiting the generation and conduction of action potentials.\(^2,9\) Conversely, at high concentrations, cocaine hastens ventricular repolarization.\(^3\) Although cocaine was associated with a great variety of cardiac arrhythmias, there are few studies of the precise electrophysiologic effects of cocaine in humans.\(^10\) Cocaine increases the risk of acute myocardial infarction by as much as 24-fold in the first hour after use; thus, cardiac arrhythmias may be associated with myocardial ischemia in cocaine users.\(^11\) In addition, cocaine use has been associated with left ventricular hypertrophy\(^12,13\) and dilated cardiomyopathy,\(^14,15\) which may predispose patients to arrhythmias.

Our study is retrospective and information about cocaine use was not obtained uniformly. All patients who reported cocaine use had positive test results for the drug; however, 2 patients were not tested, and urine testing was not performed routinely in patients who experienced cardiac arrest who did not report cocaine use. We did not document Torsades de pointes or QT prolongation in our patients and did not see any other specific arrhythmia that would provide insight into the mechanism of cardiac arrest after cocaine use. Because our hospital is located in an urban setting with a high prevalence of cocaine use, the relation between cocaine use and cardiac arrest might be merely coincidental. This is unlikely for 2 reasons. First, these patients had no other obvious cause of cardiac arrest, and second, life-threatening ventricular arrhythmias have been described after cocaine administration and are consistent with the pharmacologic activity of the drug. Although the short-term survival of cocaine users after arrest was better than that of non-cocaine users, we were not able to follow up patients prospectively and do not know their long-term prognosis.

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