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Cocaine and Sudden "Natural" Death

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ABSTRACT: The cardiovascular effects of cocaine may culminate in clinical episodes of angina pectoris, myocardial infarction, arrhythmias, and intracranial hemorrhage. To clarify whether or not cocaine causes fatalities by these mechanisms, we studied 24 cases of sudden, apparently natural deaths as a result of coronary arteriosclerosis (15 cases), hypertensive cardiovascular disease (4 cases), and intracranial hemorrhage (5 cases) associated with cocaine use. In 11 cases, cocaine was found in the blood (average concentration: 0.57 mg/L, range: 0.05 to 1.45 mg/L), whereas in the remainder, cocaine or its major metabolite was found in the urine or other tissues. In the majority of decedents, autopsy disclosed the existence of severe natural disease which could have been exacerbated by the administration of stimulant drugs, including cocaine. These data, and a review of the current medical literature, indicate that cocaine may precipitate the sudden death of an individual with undiagnosed cardiovascular disease. A contributory role of cocaine should be considered in any apparently natural death occurring in a population where cocaine abuse is prevalent.

KEYWORDS: pathology and biology, cocaine, death

The sympathomimetic properties of cocaine lead to elevations in heart rate and blood pressure [*1-4*]. The drug is also thought to be directly cardiotoxic [*5*], and recently it has been suspected of inducing coronary vasospasm [*6,7*]. These pharmacologic effects of cocaine have been clinically associated with angina pectoris, myocardial infarction, cardiac dysrhythmias (including ventricular fibrillation), stroke, and intracranial hemorrhage [*6-16*].

Sudden cardiovascular death has, on occasion, been associated with the recreational use of cocaine [*8,16*]. However, the extent to which cocaine contributed to these deaths has been uncertain. To clarify this issue, 24 cases of sudden, apparently natural, deaths associated with recreational cocaine use were reviewed.

Methods

The case files of the Dade County (Florida) Medical Examiner Department were searched for instances of sudden, apparently natural, deaths associated with toxicologic evidence of cocaine use. Twenty-four cases were identified. These included deaths from coronary arteriosclerosis, spontaneous intracranial hemorrhage, and complications typical of hypertension

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(for example, dissecting aortic aneurysm and intracerebral hemorrhage). Complete autopsies and pertinent toxicological testing had been performed in all cases. The circumstances of death, police reports, medical history, autopsy findings, histologic preparations, and toxicologic reports were all reviewed.

In addition, 39 similar natural deaths occurring in 1984 and 1985 in individuals under 40 years of age were studied. These were compared with the apparently natural deaths of 10 people (also under 40 years of age) who died during this 2-year period in whom cocaine or its metabolite or both was also detected. Since toxicologic testing has not been uniformly undertaken in cases of apparently natural deaths, a true control group could not be established.

Benzoylecgonine in urine or nasal swabs was qualitatively detected by enzyme-multiplied immunoassay testing (EMIT®, Syva). The immunoassays tested for the following in urine: amphetamines, barbiturates, opiates, benzodiazepines, benzoylecgonines, propoxyphene, methadone, methaqualone, and phencyclidine. In addition, colorimetric tests for phenothiazines and salicylate were performed on urine samples. Cocaine in blood or vitreous humor was detected and quantified by gas liquid chromatography utilizing a nitrogen detector [*17,18*]. Of the 24 cases, 23 were assayed for ethanol with gas liquid chromatography. Case 7 was not tested for ethanol.

No cases were encountered where sudden death occurred following the medical administration of cocaine. Hence cocaine, in this presentation, actually refers to the illicit street drug of variable purity and usually diluted ("cut") with mannitol. The preparation may also contain other additives such as procaine, lidocaine, or amphetamine.

Results

Of the 24 cases reviewed, death was attributed to coronary arteriosclerosis in 15 instances, hypertensive cardiovascular disease in 4 others, and intracranial hemorrhage in the 5 remaining cases (Tables 1 and 2). The cases included 1 white female, 13 white males, 5 black females, and 5 black males. They ranged 23 to 71 years of age and averaged 43 years old. The white males had an average age of 48 years and 8 of the 13 were over 40 years old. In contrast, the 10 blacks (male and female) averaged 38 years of age and only 3 were above the age of 40 years.

Cocaine was detected in postmortem heart blood in eleven cases (namely, 1, 5, 6, 8, 9, 11, 12, 13, 14, 16, and 18) in concentrations of 0.05 to 1.45 mg/L (average of 0.57 mg/L). In one instance (Case 7), only the nasal swabs were positive for cocaine. In Case 20, the body had been embalmed before the autopsy, thus precluding a toxicologic evaluation of the blood. Despite the embalming, nasal swabs were positive for cocaine, and the drug was also detected in the ocular fluid. In Case 24, cocaine was detected in an intracerebral hematoma (despite thirteen days of hospitalization before death and subsequent formalin fixation of the specimen). In the remaining ten cases (namely, 2, 3, 4, 10, 15, 17, 19, 21, 22, and 23), benzoylecgonine was detected in urine but no cocaine was detected in the blood. (In Case 21, survival time in the hospital precluded testing for blood cocaine.) In seven of these twenty-four cases (namely, 1, 6, 17, 18, 19, 21, and 23), it was ascertained that the decedent had ingested cocaine within 24 h of death. Alcohol and other drugs were detected in some of these, but there was no evidence of toxicity or that they contributed to the deaths of these individuals (Tables 1 and 2). In Case 12, a blood morphine concentration of 0.92 mg/L was the result of chronic usage in the treatment of lung carcinoma and probably did not reflect toxicity.

In the fifteen deaths attributed to coronary arteriosclerosis (Cases 1 to 15), the stenosis was estimated at 70 to 85% in ten instances and 90% or more in the remaining five. Complete thrombotic occlusion was detected in three (Cases 4, 8, and 9). In another two (Cases 6 and 7), hemorrhage into arteriosclerotic plaques was observed. In eight (Cases 2, 3, 5, 8 to 11, and 13), only one coronary artery was stenotic. Of these, six had stenosis of the left

anterior descending, one had stenosis of the right coronary, and one had stenosis of the first diagonal branch of the left anterior descending (Table 1). Gross or microscopic evidence of myocardial scarring was observed in ten instances, and two (Cases 9 and 14) had acute myocardial infarcts as well.

Most victims were simply found dead, or died shortly after developing various signs/symptoms. In two instances (Cases 10 and 15) death was preceded by "shaking" suggesting generalized seizure activity. In four (Cases 1, 3, 5, and 11), dyspnea or chest pain preceded death. Death occurred shortly after sexual activity in two instances (Cases 1 and 6).

Four individuals died with cardiovascular abnormalities typical for hypertension (Cases 16 to 19). Coronary artery stenosis was absent or only minimal in these cases. Two (Cases 17 and 18) had no history of hypertension. In three (Cases 16, 17, and 19), the major finding at autopsy was a hypertrophied left ventricular myocardium. The hearts weighed between 500 and 840 g. In one instance (Case 17), the coronary arteries were subjectively of small caliber. The fourth individual (Case 18) had a history of hypertension, had a hypertrophied heart, and died from the rupture of a dissecting aortic aneurysm.

Intracranial hemorrhage accounted for five deaths (Table 2). Hypertensive type intracerebral hemorrhage was found in two (Cases 20 and 21), both white males. The rupture of cerebral arteriovenous malformations resulted in the death of two black females (Cases 22 and 24). The fifth (Case 23), a white female, died from the rupture of a saccular aneurysm at the junction of the right anterior and middle cerebral arteries.

Of the 39 comparison cases of sudden natural death in individuals under 40 years of age (1984 to 1985), 17 had adequate toxicologic testing. None had cocaine or benzoylecgonine detected. During this same time period, 10 of the deaths in this series occurred. Thus, 20% of sudden apparently natural deaths in 1984 and 1985, in individuals below 40 years of age, had toxicologic evidence of cocaine use. This number increases to 37% when considering only those in the comparison group who were adequately tested toxicologically. Thus, in this community, it would appear that 20 to 37% of these individuals had evidence of cocaine ingestion.

Discussion

Clinical reports have implicated the sympathomimetic effects of cocaine in precipitating a variety of life threatening or even fatal cardiovascular events [*6–10, 15, 16*]. These include disturbances of cardiac rhythm, myocardial ischemia (in persons with stenotic as well as normal coronary arteries), hypertension, and intracerebral hemorrhage. Many of these reports have established a definite temporal relationship between the ingestion of cocaine and the onset of clinical signs and symptoms. Most reported cases have been below 40 years of age. In this series, about half of the decedents were less than 40 years old. Also, between 20 and 37% of individuals under the age of 40 years had cocaine in their systems at the time of their apparently natural deaths. Consequently, in this community it is now necessary to check all similar cases of natural death for the presence of cocaine and evaluate its possible contribution to the death.

In only 7 cases of this series (namely, 1, 6, 17, 18, 19, 21, and 23) could the ingestion of cocaine be historically implicated in precipitating sudden death by a cardiovascular mechanism. In the remainder, the death was not witnessed or, when present, the witnesses were usually reluctant to disclose any knowledge of illicit drug ingestion. Nonetheless, cocaine was detected in 16 of the 24 victims: in the blood (Cases 1, 5, 6, 8, 9, 11 to 14, 16, and 18), intracerebral hematoma (Case 24), nasal passages (Cases 7, 9, 20, 21, and 23), and ocular fluid (Case 20). The average blood cocaine concentration in this series was 0.57 mg/L, which is quite low when compared to the average of 6.2 mg/L for cocaine overdose victims [*19*].

These findings underscore the known toxic effects of low doses of cocaine [*4, 20*] and the problems in the detecting and interpretating concentrations of cocaine in the blood [*21*]. In

TABLE 1—*Cardiovascular deaths related to cocaine.*[a]

Case	A	R	S	Cause of Death	Coronary Stenosis (Most Severe)	Heart Weight, g	Blood Cocaine, mg/L	Comments
1	43	B	M	CAD	75%RC, 85% LAD, 85% LC	490	0.40	LVH. Urine salicylates (blood negative).
2	35	W	M	CAD	90% LAD	...	ND	Small caliber, RC. Diazepam (0.6 mg/L) and methadone (0.2 mg/L) in blood. Urine BE.
3	39	B	F	CAD	75% L Circ	340	ND	Small caliber, L Circ. LVH. Acetaminophen (8.8 mg/L) in blood. Urine BE.
4	36	B	F	CAD	80% L Circ, 100% LAD	280	ND	Early organizing coronary thrombus (site unspecified). 0.09% blood alcohol. Urine BE.
5	48	W	M	CAD	70% LAD	460	1.45	LVH.
6	37	W	M	CAD	80% LAD, 80% L Circ, 80% RC	350	0.65	Hemorrhagic plaque, LAD.
7	61	W	M	CAD	90% LAD, 70% L Circ	650	ND	Hemorrhagic plaque (site unspecified). Biventricular hypertrophy. Nasal swabs positive for cocaine.
8	65	W	M	CAD	80% LAD	380	0.43	Recanalized thrombus, RC (60% occluded). 0.09% blood alcohol.
9	55	W	M	CAD	75% RC	500	0.56	Thrombotic occlusion, L Circ. History of "heart condition." Nasal swabs positive for cocaine. Blood lidocaine, 8.4 mg/L.
10	38	B	M	CAD	80% 1st diagonal branch LAD	450	ND	Hypoplastic RC. Myocardial bridges, LAD. LVH. 0.05% blood alcohol. Urine BE.

11	34	W	M	CAD	90% LAD	380	0.24	History of CAD and hypertension. 0.11% blood alcohol.
12	71	W	M	CAD	98% RC, 80% LAD, 75% L Circ	405	0.11	History of hypertension. Abnormal ECG (mild). Morphine (0.92 mg/L) in blood.
13	29	W	M	CAD	80% LAD	330	0.6	LVH.0.13% blood alcohol.
14	53	W	M	CAD	80% LC, 80% L Circ 100% LAD	600	0.4	History of COPD, ASHD, hypertension. LVH. Diminutive RC orifice. 0.06% blood alcohol.
15	65	B	M	CAD	80% LAD, 80% L Circ 90% RC	420	ND	History of "heart condition." Urine BE.
16	52	W	M	HTCVD	minimal	500	1.40	History of diabetes mellitus and "heart condition." LVH.
17	45	B	M	HTCVD	none	840	ND	History of hypertension and IV drug abuse. LVH. Small caliber coronary arteries. Urine BE.
18	38	B	F	Ruptured dissecting aneurysm of aorta	none	700	0.05	Myocardial hypertrophy. History of hypertension.
19	25	B	M	HTCVD	none	500	ND	Sickle cell trait. LVH. trace methaqualone in blood. Urine BE.

"A,R,S = age, race, and sex.
CAD = Coronary arteriosclerotic disease.
RC = Right coronary artery.
LAD = Left anterior descending coronary artery.
LC = Left common coronary artery.
LVH = Left ventricular hypertrophy.
ND = Not detected.
BE = Benzoylecgonine.
L Circ = Left circumflex coronary artery.
COPD = Chronic obstructive pulmonary disease.
ASHD = Arteriosclerotic heart disease.
HTCVD = Hypertensive cardiovascular disease.

TABLE 2—*Fatal intracranial hemorrhage related to cocaine.*[a]

Case	A	R	S	Cause of Death	Cocaine	Heart Weight, g	Other Drugs Detected
20	50	W	M	Hemorrhage of left parietal lobe	0.19 mg/L in vitreous humor. Nasal swabs positive for cocaine.	570	none
21	35	W	M	Hemorrhage of left basal ganglia	Positive nasal swabs. Benzoylecgonine in urine.	...	Acetaminophen (less than 10 mg/L in blood). Ephedrine/ phenylpropanolamine in urine.
22	26	B	F	Ruptured arteriovenous malformation in right parietal lobe of brain	Not detected in blood. Benzoylecgonine in urine.	630	Benzodiazepine in urine.
23	23	W	F	Ruptured saccular aneurysm at junction of right anterior and middle cerebral arteries	Not detected in blood. Benzoylecgonine in urine. Positive nasal swabs for cocaine.	320	none
24	28	B	F	Ruptured arteriovenous malformation, in right temporoparietal region of brain	6.40 mg/L in intracerebral hematoma.	290	Quinine and quinidine in urine.

[a]A, R, S = age, race, and sex.

fact, tachycardia and hypertension (10 to 15% over the resting rate) were reported after administration of as little as 16 to 32 mg of cocaine under experimental conditions [*4*]. Furthermore, patients topically administered up to 3 mL of 5% aqueous cocaine hydrochloride during laryngoscopy attained mean heart rates of 120 and maximum mean systolic blood pressures of 175 mm Hg. Serious cardiac rhythm disturbances including bigeminy and multiple ectopic ventricular contractions were also recorded in these patients [*20*]. Since the average user can be expected to snort several lines (40 to 60 mg per line) of "street cocaine," the potentially toxic effect of cocaine on the cardiovascular system would likely be enhanced. The implication is that these 16 individuals died while under the influence of cocaine and therefore its sympathomimetric effects.

Benzoylecgonine was detected in the urine of the remaining eight cases (namely, 2, 3, 4, 10, 15, 17, 19, and 22). In these, no cocaine was detected in the blood. It is therefore possible that cocaine had no direct contribution to their deaths since benzoylecgonine may persist in the urine for 24 h or more after the ingestion of cocaine [*22*]. However, it must also be pointed out that cocaine is rapidly metabolized [*1*] and continues to deteriorate in the postmortem state and in vitro [*23*]. Future studies may clarify the role of cocaine in such cases by measuring the total amount of ecgonines in the blood or the measurement of cocaine and its metabolites in the brain where it persists at higher levels and for longer periods than in the blood [*21*].

Determination of the cause and manner of death should always take into account all available information. This includes the circumstances of death, environment at the scene, social and medical history, autopsy findings, and toxicologic results. Reliance upon any one factor is fraught with error. Cocaine toxicity as a cause of death must therefore rely on other considerations than toxicology alone, especially in view of the liable nature of this drug in the postmortem state and in vitro. The finding that cocaine blood concentrations overlap in those dying of trauma compared with overdose underscores these statements [*19*]. In instances where there is a clear temporal relationship between cocaine ingestion and the development of the signs and symptoms related to the cardiovascular system, we are of the opinion that cocaine toxicity must be considered a factor in the cause of death. A temporal relationship may be defined as either a witnessed ingestion or evidence of cocaine in the blood of the decedent. A low blood cocaine concentration should not preclude such an opinion. This is underscored by the known toxic effect of even low doses of cocaine and a temporal relationship between ingestion and onset of cardiovascular signs and symptoms, documented over and over again in the medical literature. When prominent natural disease processes (for example, coronary artery disease, hypertensive cardiovascular disease, and cerebrovascular abnormalities) are encountered, the toxic effects of cocaine may quite logically lead to death via its sympathomimetic effects. Hence, acute cocaine toxicity or acute cocainism may be listed as a contributory factor. Alternatively, the anatomic abnormality may be listed as a contributory cause of death and acute cocaine toxicity as the proximate cause. In instances of cardiovascular death where there is no anatomic abnormality, acute cocaine toxicity may be appropriately deemed the cause of death (for example, coronary vasospasm and cardiac dysrhythmia). In cases where there is no cocaine in the blood, the finding of benzoylecgonine in the blood or urine, alone, will not often permit such interpretation unless there is a witnessed ingestion with survival time (that is, metabolism of cocaine to breakdown products).

When cocaine toxicity is the cause of death or a contributory factor, the manner of death in our opinion is accidental: It makes little difference whether the mechanism of death is centrally induced respiratory depression ("overdose"), hypertensive crisis, or cardiac rhythm disturbance. All, within reasonable medical probability, were induced or aggravated by cocaine ingestion.

Conclusion

It is concluded that cocaine may kill via its pharmacologic effects on the cardiovascular system, particularly by aggravating an underlying disease process such as hypertension or

coronary arteriosclerosis. A cardiovascular mechanism of sudden death may also be operative in cases regarded as cocaine overdose but not preceded by generalized seizures [*19*]. Also, cardiovascular toxicity may explain the sudden death associated with cocaine-induced psychotic episodes [*24*]. As the recreational use of cocaine continues to proliferate, its role in precipitating a hypertensive or a cardiovascular crisis must be seriously considered and evaluated by clinicians, coroners, and forensic pathologists alike. Furthermore, future investigations should focus on the possibility that chronic abuse of cocaine may induce coronary artery stenosis [*25*].

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