Cocaine Detection in Postmortem Samples Following Therapeutic Administration

Kristen M. Bailey*, David J. Clay, Myron A. Gebhardt, Matrina J. Schmidt, Nabila A. Haikal, and James C. Kraner
Office of the Chief Medical Examiner, Charleston, West Virginia

Abstract

Cocaine is one of the most widely abused drugs and one that is frequently encountered in forensic toxicology laboratories. Most often, the detection of cocaine would lead toxicologists and forensic pathologists to believe that the drug was used illicitly; however, cocaine is an effective local anesthetic and vasoconstrictor and is used clinically in surgeries of the eye, ear, nose, and throat. Therefore, it is important to note that the presence of cocaine and its metabolites in forensic samples cannot always be attributed to abuse and that a thorough investigation and review of medical records is warranted before an informed conclusion can be made. In this case report, a 54-year-old male died three days after an altercation in which he suffered multiple injuries. In addition to natural disease and injuries documented at autopsy, cocaine and its metabolites were detected in the decedent's urine, and a review of surgical records showed that earlier on the day of death, he was administered cocaine clinically during a procedure to repair nasal bone fractures. If not for this comprehensive investigation and review of surgical records, the assumption of cocaine abuse might have otherwise been made and the cause and manner of death incorrectly established.

Introduction

Cocaine is a drug that is notorious for its high potential for recreational abuse; however, while generally regarded as an illicit drug, cocaine also has accepted medical use in the U.S. As a topical anesthetic and peripheral vasoconstrictor, cocaine has been employed in surgeries of the eye, ear, nose, and throat for over 100 years (1,2). Cocaine is still widely used as such today, and the persistent popularity of the clinical use of this drug is clearly attributable to its unique ability to simultaneously limit epistaxis and induce local anesthesia (3,4). Reports of the various aspects of cocaine abuse are clear and widespread in the forensic literature, whereas its clinical use can be easily overlooked in the setting of a postmortem investigation. That is, when encountered in a forensic toxicology laboratory, the detection of cocaine and its metabolites may erroneously lead the toxicologist and pathologist to believe that the drug was used illicitly and to incorporate such illicit use in the certification of cause and manner of death.

Case History

A 54-year-old Caucasian male was involved in a physical altercation during which he suffered a broken nose. Three days later, at approximately 9:00 a.m., a surgical procedure involving closed reduction of bilateral nasal bone fractures was performed, and the man was released from the hospital later that day with a prescription for Percocet® (oxycodone 5 mg/acetaminophen 325 mg). Reportedly, he took one Percocet around 4:00 p.m. and went to bed approximately 3 h later. At 8:00 p.m., the man’s wife attempted to wake him and found him breathing but unconscious. She called 911, and EMS responded to the residence to find her performing CPR. The subject was pronounced dead at the scene at 8:40 p.m. Subsequent investigation revealed that the decedent had a history of type II diabetes mellitus, hypertension, and high cholesterol. There was no reported history of drug abuse as ascertained by death investigation, and no illicit drugs or paraphernalia were found at the scene. Given the circumstances leading up to the demise, a full postmortem examination was performed in order to elucidate the contribution of external factors such as physical injury, surgical intervention, and/or drug use to his death. The following pathologic findings were revealed at autopsy: severe atherosclerotic right coronary artery disease with up to 70–80% lumen stenosis, nasal bone and left maxillary sinus fractures (status post closed surgical reduction), and minimal subdural hematoma without associated mass effect. Samples submitted to the toxicology laboratory for testing included blood from the left subclavian vein, gastric contents, urine, and vitreous humor.

Analysis

Subclavian blood was analyzed for volatiles using gas chromatography with flame-ionization detection (direct injection)
and a subclavian blood precipitate was screened for a panel of eleven drugs of abuse by EMIT. A slight elevation in the immunoassay response for benzoylecgonine prompted screening of the urine sample for benzoylecgonine, yielding a positive result. A procedure to confirm the presence of cocaine and its metabolites in blood and urine was performed using solid-phase extraction, trimethylsilyl derivatization, and quantitation by gas chromatography–mass spectrometry, according to the method outlined by SPEware (5).

Results

Cocaine and two metabolites were detected and quantitated in the urine: cocaine at 48 ng/mL, benzoylecgonine at 482 ng/mL, and ecgonine methyl ester at 190 ng/mL. No cocaine or cocaine metabolites were detected in the subclavian blood (limit of detection: 5 ng/mL). In addition, oxycodone was present in the blood at a concentration of 73 ng/mL, and diazepam and nordiazepam were quantitated in the blood, both at a concentration of 103 ng/mL. No volatile compounds were detected.

Discussion

Upon detection of cocaine in the urine, it was initially presumed that the decedent used cocaine illicitly. Given such illicit use had occurred, the well-known cardiotoxic effects associated with cocaine could have been considered a contributory factor to death. However, a review of subsequently received operative records revealed that he was administered cocaine topically during surgical reduction of bilateral nasal bone fractures. In a clinical setting, cocaine hydrochloride solutions of 1, 4, or 10% are used to induce local anesthesia and vasoconstriction of the nasal mucosa. In most applications, the 1% or 4% solutions are used in order to lessen the likelihood of toxicity (6). Cotton pledgets measuring approximately 1 cm² are soaked in the solution and wrung out prior to introduction into the nasal cavity with bayonet forceps (3). In this particular case, three cotton pledgets soaked in 4% cocaine were placed in each naris and left in place for 7 min. When utilized in this manner, cocaine is rapidly absorbed through the mucous membranes, and benzoylecgonine can be detected in the urine from 1 to 27 h after application (7). Urinary elimination half-lives of 1.5 h for cocaine, 7.5 h for benzoylecgonine, and 3.6 h for ecgonine methyl ester have been reported (8). In this case study, cocaine was administered approximately 11 h prior to death.

Cocaine abuse can be accompanied by urine benzoylecgonine concentrations that are extremely high, sometimes exceeding 300,000 ng/mL, and studies have illustrated an overlap in urine concentrations of cocaine and its metabolites in instances of therapeutically administered cocaine and in fatalities directly related to or involving cocaine. Urine concentrations of cocaine (412 ng/mL), benzoylecgonine (13,681 ng/mL), and ecgonine methyl ester (5831 ng/mL) have been reported at 5.1, 7.8, and 5 h, respectively, following therapeutic intranasal administration of 32 mg cocaine hydrochloride (9). Benzoylecgonine concentrations ranging from 80 to 386,000 ng/mL and corresponding ecgonine methyl ester concentrations ranging from 60 to 72,000 ng/mL were reported in 93 postmortem urine specimens (10). Jenkins et al. (11) have reported urine cocaine and benzoylecgonine concentrations in 13 cases of cocaine intoxication ranging from 50 to 150,000 ng/mL and 370 to 230,000 ng/mL, respectively. They also reported urine cocaine and benzoylecgonine concentrations in 19 non-drug-related deaths ranging from 38 to 59,000 ng/mL and 880 to 330,000 ng/mL, respectively (11). In light of these published studies, and based solely on urine concentrations of cocaine, benzoylecgonine, and ecgonine methyl ester in this case report, it cannot be unequivocally stated that the decedent did or did not use cocaine illicitly.

After careful consideration of all available case information, including autopsy findings, scene investigation, decedent history, and clinical records documenting therapeutic cocaine administration, the presence of cocaine and its metabolites in the urine was attributed solely to clinical administration. Minimal subdural hematoma, facial fracture reduction surgery, nasal cocaine packing, and detected levels of both oxycodone and diazepam in blood were all judged by the certifying pathologist to be non-contributory to death, and the cause of death was ruled as atherosclerotic coronary artery disease with the manner of death classified as natural.

Conclusions

As is demonstrated by this case, it is important to keep in mind that the presence of cocaine and its metabolites in postmortem samples may not always be attributable to illicit cocaine use. An erroneous presumption of illicit drug use may have significant implications, particularly in the context of a physical altercation with potential legal ramifications (12,13). Additionally, family members of the decedent may suffer undue anguish if false indications of illicit drug use by a loved one are made. Toxicology results, investigative reports, clinical records, and pathologic findings must be taken into consideration concomitantly to ensure accurate interpretation of the presence of cocaine as well as other drugs that may be administered clinically.

References