Passive Inhalation of Cocaine by Infants

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Cocaine use is widespread in our society. By 1986, 15% of the United States population had used cocaine, with 40% of those being between 25 and 30 years of age (1). Cocaine is available as a hydrochloride salt which can be injected intravenously or applied to a mucous membrane, usually intranasally (1,2). It is delivered more rapidly to the brain when smoked in its base form called “crack” (1,3). Crack cocaine more likely leads to addiction because it is usually consumed in doses three to six times higher than cocaine hydrochloride and has a more rapid route of delivery (4). Unfortunately, crack cocaine has recently become more available at a relatively low cost, precipitating an increase in the number of cocaine addicts. The habitual use of cocaine by parents and other caretakers can have profound effects on children.

An increasing number of infants are born to women addicted to cocaine (5,6). The adverse impact on infant morbidity and mortality has been recently documented (5-9). Prenatal use of cocaine is associated with spontaneous abortion, premature labor, abruptio placenta, congenital malformations, low birth weight, cerebral infarction, and seizures (5-9). Neurobehavioral abnormalities have been noted in the neonatal period (5). The long-term effects of cocaine exposure on the developing nervous system are unknown.

In a review of 75 cases of child abuse by poisoning, none of the children had cocaine poisoning (10). However, a case of accidental poisoning followed the application of cocaine to a mother’s nipples before breast feeding (7). It has recently been suggested that cocaine poisoning can occur by passive inhalation of crack vapors (11). We report four children with urine cocaine metabolites present on admission to the hospital. All were unintentionally exposed to cocaine vapors in a room where crack was smoked. The erroneous belief that the detection of cocaine metabolites in the urine proved intentional poisoning had profound social and legal implications.

Case Reports

Case 1
A formula fed, 3-month-old girl presented with status epilepticus following several days of vomiting and diarrhea. After successful anticonvulsant therapy she remained comatose. She appeared dehydrated and had severe diaper dermatitis with full-thickness ulcers. Her serum sodium was 162 mEq/L, and BUN 28 mg/dL. Cranial computed tomography and cerebral angiography revealed a superior sagittal sinus thrombosis with extensive bilateral cerebral infarcts. Despite vigorous supportive care, she progressed to brain death. A urine toxicology screen on admission showed benzoylecgonine (BZ) by both radioimmunoassay and gas chromatography. Gas chromatography was performed on the second urine sample. The baby was noticeably wasted. The baby was treated for “waste” the day after the urine sample was obtained.

Case 2
A 12-month-old infant was admitted with fever. He was tachycardic, tachypneic, and in respiratory distress. He had hypotension, hypotension, hypotension, hypothermia, and hypoglycemia. The infant was admitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome. The infant was readmitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome. The infant was readmitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome. The infant was readmitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome. The infant was readmitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome. The infant was readmitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome. The infant was readmitted to the ICU with a diagnosis of sepsis. Urine and blood cultures were negative. The infant was treated with antibiotics. He was discharged home with a diagnosis of respiratory distress syndrome.

Case 3
A 14-month-old boy was admitted to the hospital with a fever of 39.5°C (103°F). He was tachycardic and tachypneic. He had a heart rate of 120 beats/min, a respiratory rate of 30 breaths/min, and a blood pressure of 90/60 mm Hg. He was hypotensive and hypothermic. He had a rash on his chest and abdomen. The rash was maculopapular and petechial. The baby was treated for sepsis. He was discharged home with a diagnosis of sepsis.

Case 4
A 14-month-old boy was admitted to the hospital with a fever of 39.5°C (103°F). He was tachycardic and tachypneic. He had a heart rate of 120 beats/min, a respiratory rate of 30 breaths/min, and a blood pressure of 90/60 mm Hg. He was hypotensive and hypothermic. He had a rash on his chest and abdomen. The rash was maculopapular and petechial. The baby was treated for sepsis. He was discharged home with a diagnosis of sepsis.

References
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Cocaine is metabolized to BZ and ecgonine methylester by plasma and liver cholinesterases (2). The half-life of cocaine is 90 minutes, and BZ 6 to 8 hours (12,13). Infants have a longer time administration (12). High dose, chronic cocaine administration has been associated in some cases with the presence of BZ for 10 to 22 hours after the last cocaine ingestion (13). Conclusions concerning cocaine kinetics in terms of the last dose, route of administration, and frequency of use have not been thoroughly studied (12).

Routine urine toxicology screening for cocaine detects BZ by the EMIT method. A positive result is typically 300 ng/mL of BZ or greater (14). False-positive results are not known to occur (15,16). The urine BZ test is very sensitive. Recreational cocaine use can result in a positive test for two to three days following ingestion (13). Clinically, even small amounts of cocaine can produce BZ detection. Ingestion of 50 mg of cocaine is necessary for a pharmacological effect in adults. However, 25 mg of an oral dose of cocaine resulted in a positive test for 48 hours (17). When 12 volunteers received 20 mg of cocaine administered to their corneas, the urine BZ test was positive for 36 hours in the majority (18). The same authors found that ophthalmic instillation of 8 mg of cocaine resulted in a positive urine test for up to 36 hours (unpublished data). Even as little as 2.15 mg of cocaine in a cup of coca tea yielded a urine concentration of BZ greater than 300 ng/mL for 17.5 hours after ingestion (19). Crack is commonly smoked in doses of 300 mg or more. The amount inhaled by a nearby infant would vary depending on the quantity of cocaine released into the air, total time of exposure, and the dimensions of the room. Brief passive smoke exposure could result in cocaine metabolites in the urine.

Cocaine poisoning by passive inhalation of cocaine smoke in infants and toddlers has been suggested in a recent report (11). While these four children had BZ in the urine, it is not clear whether intoxication occurred. One presented with excessive drowsiness and another with a focal seizure during sleep. None were described as having signs of adrenergic overactivity that typifies cocaine intoxication. It seems plausible that at least some of these patients were exposed to subpharmacologic amounts of cocaine, causing positive urine testing. In another report, three infants had unexpected evidence of cocaine ingestion by toxicology testing (20). The proposed routes of administration were intentional poisoning and passive transmission through breast milk. Passive exposure to crack vapors was not mentioned. It is unlikely that any of our patients had cocaine intoxication. While stroke is a reported complication of cocaine abuse (2), our first patient had a severe cerebral venous thrombosis with extensive infarcts apparent immediately after her seizures ceased. This thrombosis is not unexpected with serious dehydration. Our fourth patient had two brief convulsions most consistent with febrile seizures. His mental status and vital signs before and after the seizures and his appetite immediately before the first seizure are not consistent with cocaine poisoning. Also, neither of these infants had detectable BZ 12 hours after the initial urine test. Following pharmacologic doses of cocaine, BZ persists in the urine typically for several days (2,12,13). The absence of BZ 12 hours after the seizures is strong evidence against acute intentional oral intoxication. Following extensive investigation of parents and other caretakers by physicians, nurses, and social and protective service workers, we believe that these four children were not accidentally or intentionally
poisoned but had a positive drug screen from passively inhaling cocaine vapors.

Few would disagree that cocaine addicts generally make poor parents. However, in the city of Detroit, parental use of cocaine alone does not constitute child neglect or abuse. Intentional poisoning of a child would result in legal action. Recently, the forensic medical opinion supplied to the police and child protection services was that cocaine metabolites in the urine are proof of abuse or neglect. In case 1, the mother was charged with murder. Positive drug screens aided in removing the child from the home in cases 2 and 3.

In conclusion, our cases suggest that cocaine metabolites may be present in the urine after passive inhalation of cocaine vapors. Infants who reside in crack houses are chronically exposed to cocaine smoke and are probably at high risk for a positive cocaine urine test. Medical and developmental effects of this situation are unknown. Legal implications are significant. Controlled laboratory studies may be necessary to establish the association of cocaine vapors by passive inhalation and cocaine metabolites in the urine.

References