CASE REPORT

Kurt B. Nolte,[1] *M.D.*

Cocaine, Fetal Loss, and the Role of the Forensic Pathologist

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ABSTRACT: Adverse obstetrical outcomes are often associated with maternal cocaine use. These have included intrauterine growth retardation, abruptio placentae, and an increased incidence of spontaneous abortion and pre-term labor. This report details the case of an 18-week-gestation fetus recovered from a sewage treatment plant. A brain specimen was positive for cocaine. With the present epidemic of cocaine abuse, it is important for the forensic pathologist to seek toxicologic evidence of cocaine in all suspicious fetal and neonatal deaths.

KEYWORDS: pathology and biology, cocaine, neonatal death

Forensic pathologists frequently encounter fetal and neonatal deaths. Oftentimes, these neonates and fetuses are recovered from toilets or from sewage treatment plants, usually as a result of spontaneous delivery into a toilet. Great variation exists from jurisdiction to jursidiction in the extent of medicolegal death investigation of such cases. The usual examination by good medical investigative offices consists of an autopsy and, occasionally, radiographs to determine the gestational age, injuries, and whether it was a livebirth or stillbirth. For neonatal deaths, Mitchell and Davis [*1*] suggest consideration of the maternal postnatal behavior, in addition to investigation of the circumstances of birth and the postmortem findings. Toxicologic studies are usually not done. This paper reports a case of a fetus recovered from a sewage treatment plant, presumably after a spontaneous birth into a toilet. The toxicologic evaluation of a brain specimen demonstrated the presence of cocaine. With the present epidemic of cocaine abuse, there may be a greater incidence of fetal loss because of this drug [*2*]. Cocaine has been associated with an increased incidence of intrauterine growth retardation, abruptio placentae, spontaneous abortion, and premature rupture of placental membranes [*3,4*]. Since there is placental transfer of this drug, it is important that forensic pathologists seek evidence of cocaine in fetal and neonatal deaths.

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[1]Formerly, associate medical examiner, Office of the Chief Medical Examiner, State of Connecticut, Farmington, CT. Currently, medical investigator and assistant professor of pathology, Office of the Medical Investigator, University of New Mexico School of Medicine, Albuquerque, NM.

Case Report

A well-preserved fetus was recovered from the trash separator of a sewage treatment plant (Fig. 1). There were no associated identifying artifacts such as clothing or wrappings. The mother has not been identified. The fetus was examined at a fully equipped state medical examiner's office. The white female fetus had a crown-to-heel length of 23 cm, a crown-to-rump length of 16.5 cm, a foot length of 3.1 cm, and a weight of 170 g, consistent with a gestational age of 18 weeks.

The skin was smooth, thin, and pink-red, without vernix or lanugo. An approximately 15-cm laceration extended from the left side of the head, across the neck, anterior chest, and abdomen, exposing the rib cage and abdominal viscera. Over the posterior scalp, both shoulders, and the back, there was a 15-cm laceration exposing the skull and skeletal muscle. The scalp was almost entirely avulsed from the skull, exposing fractures of both parietal bones and the right frontal bone. There was a slight amount of right retroperitoneal hemorrhage. There were bilateral nonhemorrhagic pulmonary lacerations. A 5-cm-long, 0.8-cm-diameter segment of attached three-vessel umbilical cord was present. No congenital anomalies were observed. The placenta was not recovered.

Complete microscopic examination confirmed that the development was consistent with a gestational age of 18 weeks and that the fetus showed no pathologic abnormality except for a moderate infiltrate of neutrophils and lymphocytes in the umbilical cord (funisitis).

Eleven grams of brain were submitted for toxicological evaluation. A radioimmu-

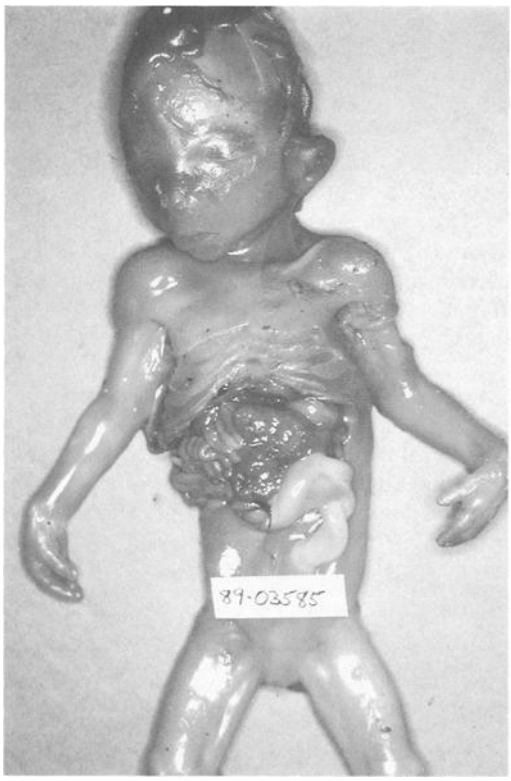

FIG. 1—*Fetus recovered from trash separator with large postmortem lacerations and exposed viscera.*

noassay sensitive for both cocaine and benzoylecgonine was positive. A concentration of 0.08 mg/L of cocaine was demonstrated using gas chromatography. No ethanol, other volatile hydrocarbons, or opiates were detected in the brain.

The cause of death was certified as "maternal chorioamnionitis," with "maternal cocaine use" listed under other significant conditions.

Discussion

The toxicologic finding of cocaine in the brain specimen of a fetus compellingly indicates maternal cocaine use in the immediate antepartum period. This is because the observed half-life of cocaine is short and ranges from 0.8 to 1.25 h, depending on the route of administration [*5*]. The maternal/fetal cocaine concentration ratio in the brain was reported as 6.5:1 in a single case [*6,7*]. In addition, identification of cocaine in neonatal or fetal hair may suggest chronic maternal cocaine abuse [*8*].

As the epidemic of cocaine abuse persists, reports of the various manifestations of cocaine toxicity have increased. Cerebral and cardiac toxicity have been well described [*9,10*]. Adverse obstetrical effects have also been reported. These have included an increased incidence of intrauterine growth retardation, abruptio placentae, spontaneous abortion, and premature rupture of placental membranes [*3,4*]. It has been suggested that cocaine causes fetal genitourinary tract malformations [*11*].

Cocaine acts by blocking the presynaptic reuptake of norepinephrine and dopamine, thereby producing an excess of neurotransmitter at the postsynaptic receptor sites. Activation of the sympathetic nerve terminals by this mechanism results in vasoconstriction, hypertension, and tachycardia [*10*]. Experimental administration of cocaine to pregnant sheep has produced hypertension in the pregnant ewe and fetus, decreased uterine blood flow, and caused fetal hypoxemia [*12,13*]. It has been suggested that fetal hypoxia could explain the intrauterine growth retardation and increased number of cocaine-associated malformations [*14*]. Maternal hypertension could be the source of the increased incidence of placental abruptions [*15*]. Elevated plasma catecholamines have been associated with an increase in uterine contractility in humans [*16*], consistent with the increased incidence of premature labor associated with cocaine.

In this case, the funisitis is consistent with chorioamnionitis and premature rupture of placental membranes. Although chorioamnionitis can and often does occur in the absence of cocaine abuse, maternal cocaine use in this instance logically could have contributed to the premature membrane rupture or premature birth or both because of its pharmacological action [*3*].

Cocaine use among pregnant women has been reported to be as prevalent as 18% in Boston [*17*]. It is therefore important for the forensic pathologist to seek toxicologic evidence of cocaine in the evaluation of fetal and neonatal deaths. Such information is necessary for accurate death certification. In addition, these autopsies provide the opportunity to identify pathologic correlates of the ever-growing body of clinical information about the adverse consequences of cocaine use.

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Address requests for reprints or additional information to
Dr. Kurt B. Nolte
Office of Medical Investigator
University of New Mexico School of Medicine
Albuquerque, NM 87131