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CASE REPORT

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Accidental Boric Acid Poisoning Following the Ingestion of Household Pesticide

ABSTRACT: Borate-containing compounds were formerly used as topical antiseptics and were components of many medicinal preparations including skin powders and ointments used for the treatment of burns and diaper rash. These compounds were also used as irrigants for body cavities, including the pleural, vaginal, and rectal cavities. These applications were subsequently discontinued by the medical community when the toxicity and potential lethality of borates were recognized. Although documented cases of borate poisoning are now rare, the chemical is still an active component commonly used in high concentrations in household disinfectants/cleaners, pesticides, and wood preservatives. While the majority of documented borate-related deaths have occurred in infants, the toddler population is currently at risk due to possible exposure to these household products. We present the case of an 18-month-old child who died following the accidental ingestion of a boric acid-containing, commercially available roach pesticide product.

KEYWORDS: forensic science, forensic pathology, borate poisoning, pesticides

Borate has been used in aqueous solutions in household products, in medical therapy, as a form of food preservative, and in the metal, enamel, and glass industries for many years (1,2). Boric acid was first used as a topical antiseptic by Lister in 1872 (3). It has subsequently been used in eyewashes, mouthwashes, skin powders, and ointments. Boric acid has been a component of solutions used as irrigants for wounds, empyema cavities, and for bladder, rectal, and vaginal irrigation (1,4). Cutaneous burns and diaper rash were treated with boric acid ointment and boric acid powder, respectively (5,6).

The earliest cases of boric acid toxicity resulting from its use as an antiseptic and therapy for burns were reported in the 1880s (6). Subsequently, there have been many reports of boric acid toxicity and fatalities resulting from its medicinal use, predominantly in infants and young children (1,5,7–9). Fatalities in a newborn nursery due to the accidental use of boric acid in the preparation of infant formula have also been documented (10,11). Consequently, the medical community has abandoned the use of boric acid solutions as antiseptics and irrigants (12). Borates have also been replaced by less toxic agents as food preservatives (1).

Sodium borate (borax) is currently found in household cleaning products, wood preservatives, and pesticides. Additionally, boric acid at a concentration of 8% or greater is an effective fungicide for vegetable and fruit trees. Borate is used as a powder with a 99% concentration as a household pesticide for the control of cockroaches, ants, and flies (12). Because it is no longer commonly used medicinally, cases of boric acid poisoning are rare and the forensic literature contains little information on the classic signs, symptoms, and pathologic findings of toxicity. Education and awareness of these findings may assist the medical examiner/coroner in recognizing and diagnosing cases of borate toxicity. We present a case of fatal borate toxicity in an 18-month-old child with no significant medical history whose caretakers attempted to conceal the evidence of pesticide ingestion.

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Case Report

A local police department received a 911 emergency call from a young woman who indicated that a radio had fallen on her child and that the child had stopped moving and breathing. Emergency Medical Services responded to the residence and found a young female child lying motionless on the kitchen floor next to a chest freezer. A large portable radio was on the ground, adjacent to the child's feet, with its long axis upward. The child had no detectable pulse or respirations. A police officer at the scene attempted chest compressions. He noted that the child's nostrils appeared to be filled with mucus and that her stomach was abnormally distended. The child's mother indicated that she had been in her bedroom when she heard a loud crash and found her child lying on the floor. The child was transported to a local hospital and was pronounced dead in the emergency room.

The deceased child was an 18-month-old black girl who lived with her biological mother, the mother's paramour, the child's 7-month-old brother, and the maternal grandmother. The family had moved into their current residence 1 week before the child's death. The paramour informed the police that he had been sleeping at the time of the incident and that the grandmother was not at home.

The child had been born prematurely at 26 weeks, weighing 1 pound, 3 ounces, but had no further documented past medical history. The biological mother had a history of neurofibromatosis and the girl's brother had been previously hospitalized with seizures. Subsequent medical studies revealed that the boy had suffered from a viral infection associated with encephalopathic features.

The postmortem examination was performed 28 h after the child's death. The body was that of a well-developed, lean, female child weighing 18 pounds and measuring 30 in. in length. External examination was remarkable for the presence of multiple hyperpigmented macules, consistent with café-au-lait spots. Although the external genitalia were unremarkable, the anal mucosa was irregular, with multiple clefts. Internal examination was remarkable only for pulmonary congestion and edema and cerebral edema. No congenital malformations or evidence of pre-existing natural disease was identified. The stomach contained 80 cc of thick, green-brown liquid and the small and large intestines focally contained a light brown, liquid material. Microscopic examination of the skin from the hyperpigmented macules showed scattered dermal nests of melanocytes. The ano-rectal mucosa showed koilocytotic changes, suggestive of human papilloma virus infection. However, *in situ* hybridization studies using probes for HPV 6/11, 16/18, and 31/33/51 were negative. Examination of the eyes revealed the presence of nodules in the irides composed of a loose stromal accumulation of melanocytes with an overlying surface plaque of spindle-shaped cells, consistent with the Lisch nodules characteristic of neurofibromatosis. The remainder of the postmortem examination was unremarkable and routine toxicologic studies were negative.

Following the postmortem examination, the child's mother, grandmother, and the mother's paramour refused further interviews. A search warrant was obtained for the residence. An open bottle of "Hot Shot Roach Powder," the active ingredient of which is boric acid powder, was found in a bathroom cabinet. A bed comforter bearing dried blue vomitus was located on the back porch. Additional toxicological studies, performed at National Medical Services in Willow Grove, Pennsylvania, revealed a heart blood borate concentration of 8316 mcg/dL (reference range toxic >2000 mcg/dL) and a gastric content borate concentration of 6048 mg/L. The cause of death was attributed to acute borate toxicity and the manner of death was certified as accidental. Subsequent investigation revealed that the child had been brought to the emergency room 2 months before her death following the ingestion of nail polish remover. The younger son was removed from the home and placed in the custody of the biological father.

Discussion

Boric acid is an inorganic acid that is well absorbed through mucus membranes and broken skin surfaces (1). There is negligible absorption through intact, undamaged skin (8,12). Indeed, there are no documented cases of boric acid poisoning resulting from its use on nontraumatized, intact skin (4). Borates are water soluble and widely distribute in body tissues, particularly the brain, liver, and kidney (5,7). They are excreted largely unaltered, predominantly by the kidney (1,7).

The majority of the reported fatal and nonfatal cases of borate toxicity have occurred in infants and young children due to the large amount of borate that would be required to produce a toxic effect in an adult (1). The early symptoms of boric acid intoxication are largely nonspecific and may mimic acute gastroenteritis. Vomiting, diarrhea, lethargy, and in occasional cases a fever have been documented (5,8,10–13). The vomitus may have a blue-green hue if the agent was ingested orally. The loose stools may have a similar color (10). These signs and symptoms are followed by characteristic skin changes, consisting of a diffuse erythema that has been termed a "boiled lobster" appearance before the desquamation of large sheets of skin (8,14). The rash has been described as having similarities to that of scarlet fever (6,7) and may occur whether the ingestion was oral or topical (8). The rash may cover the entire body, including the palms of the hands and the soles of the feet, and may extend to mucus membranes (6,8,12). Some cases of acute borate toxicity lack the skin manifestations (10,12,13). Siegel and Wason (12) reported the presence of skin changes in 76% of cases and described a progression of the rash from the axillae to the inguinal and facial regions before becoming generalized. Alopecia totalis has also been reported following acute and chronic ingestions of borate (12,15,16). Because borates are not caustic, esophageal, and gastric strictures do not develop (12). Damage to the renal tubules can lead to oliguria/anuria (10). Central nervous system symptoms predominate in severe and fatal poisonings and may include convulsions and delirium, as well as circulatory collapse (5,11). Laboratory findings may include hypernatremia, hyperchloremia, and a metabolic acidosis (10). Abnormal liver function tests are uncommon (8,12,14).

There is no specific antidote for borate poisoning (5,17). Patients have been treated with hemodialysis, peritoneal dialysis, and exchange transfusion (7,10).

Documented accidental poisonings and fatalities have resulted from the use of boric acid powder in the treatment of diaper rash (1,5). Additionally, deaths have occurred in newborn nurseries when boric acid, as a 2.5% aqueous solution, was accidentally used in place of sterile water in the preparation of infants' formula (10,11). A boric acid compound has also been used as an antiseptic for rubber bottle nipples and to cleanse the nipples of nursing mothers (11). O'Sullivan and Taylor (9) reported seven cases of boric acid poisoning in infants when their pacifiers were dipped in a proprietary mixture of boric acid and honey, a compound that could be readily purchased over the counter in Dublin, Ireland. The infants developed seizures but remained well after the mixture was withheld.

Because the medical community has abandoned the use of boric acid-containing products for topical administration or as irrigants, there have been few documented reports in the recent literature regarding borate poisonings (12). Currently, such poisonings occur almost exclusively in mobile toddlers due to the accidental ingestion of household poisons (18). However, boric acid poisoning has caused occasional accidental deaths in adults. Valdes-Dapena and Arey (3) described the case of a woman who died after she ate cake that was accidentally baked with boric acid powder instead of baking soda. A series of poisonings was reported from the state of Perak in Malaysia during the 1988 Nine Emperor Gods Festival (19). The 13 deaths occurred in children and were attributed to the presence of a combination of boric acid and aflatoxin impurities in Loh See Fun, noodles shaped like rats' tails. This incident was apparently due to the lack of quality control procedures in the manufacturing process of the noodles.

Boric acid has also been used as an agent of suicide (4,6,20–22). Restuccio et al. (22) reported a fatal suicidal ingestion of two cups of boric acid crystals dissolved in water. Occupational exposure to borax dust has resulted in dermatitis, cough, and shortness of breath (1). Deaths of animals resulting from the accidental ingestion of borates have also been reported in the veterinary medical literature, particularly in cattle exposed to fertilizer (23,24).

Historically, the tumeric acid paper test was used to detect the presence of borate in urine or cerebrospinal fluid specimens. However, this test has proven to be unreliable due to many false-positive reactions (3,8). The diagnosis of borate poisoning in fatal cases relies on the recognition of antemortem signs and symptoms in chronic cases and on toxicologic studies in cases

where a large ingestion is associated with a rapid death or where the history is not available, as in our cited case. Normal human tissues contain borate in trace amounts (1). There are no specific gross or microscopic postmortem findings in cases of borate toxicity (8). Degeneration of the renal tubules has been described in chronic poisonings (6). Other nonspecific changes that have been sporadically reported include cerebral edema and perivascular hemorrhages in the central nervous system, fatty change and/or degeneration of hepatocytes, and edema of the gastrointestinal tract associated with exfoliation of the mucosa (8). Valdes-Dapena and Arey (3) described three fatal cases of boric acid poisoning associated with either acidophilic or basophilic pancreatic acinar cell inclusions.

In contrast to previous years, our case illustrates that the population currently at risk for borate poisoning consists predominantly of mobile young children who may accidentally ingest household pesticides or other household products that contain borates. In some cases, the history and antemortem course may indicate the diagnosis. However, as in our case, the caretakers may be fearful of the authorities and may not seek emergent medical attention for the child. They may also attempt to conceal evidence of the child's exposure to potentially harmful products in the home. In such cases, the diagnosis may be suspected by the blue-green color of the child's vomitus and/or stool and by recognition of the characteristic skin changes if the child survives long enough to develop the rash. Ultimately, however, toxicologic studies are needed to confirm the borate ingestion.

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