

## CASE REPORT

# Multiple Organ Damage Due to Boric Acid Toxicity

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**Abstract**

**Background:** Boric acid (BA) is commonly used as pesticide, disinfectant and wood preservative. Its ingestion can cause serious organ damages resulting in death.

**Case report:** A 19 year old man was found dead with two empty packs of BA tablets in vicinity of his body. On autopsy examination, brownish stains along with right angle of mouth and right nostril were found. Stomach was damaged with hemorrhagic gastritis. All internal organs including liver, kidney, spleen and brain were congested. The toxicological analysis of viscera confirmed boron ion toxicity.

**Discussion:** Boric acid is a moderately toxic compound that is a mucosal corrosive agent affecting upper respiratory and gastrointestinal tracts. It can also act as a generalized cellular poison, and thus all end organs including liver, kidneys, brain and gastrointestinal tract are prone to its toxicity. Massive BA poisoning mostly occurs in adults as a result of suicide attempt. Early aggressive hydration and hemodialysis can prevent its mortality.

**Conclusion:** A dead body with erythematous rash, redness of skin, bluish green color of vomitus and fecal matter, corrosive changes in oral and nasal cavity and multiple organ damages can be highly suggestive of BA poisoning.

**Keywords:** Boric Acids; Boron toxicity; Gastritis; Multiple Organ Failure; Suicide

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**INTRODUCTION**

Boric acid (BA) is an odorless compound ( $H_3BO_3$ ; 5.5% Boron) which can be used as pesticide, water softener and disinfectant. It is commercially available in form of borax-based white powders (sodium borate;  $Na_2[B_4O_5(OH)_4] \cdot 8H_2O$ ) (1). Majority of the deaths due to BA poisoning have occurred in infants, though documented cases of its toxicity due to precautionary regulations are rare nowadays (1-4). However, accidental and occupational exposure to BA is not uncommon as it is an active component in wood preservative, household cleaners, cosmetics, pharmaceutical preparations such as dusting powders, mouth washes, eye washes, and personal care products such as toothpastes. It is also used in high concentrations as pesticide (99% boric acid).

Most cases of BA poisoning have been reported to occur accidentally; while chronic occupational BA poisoning was not formerly uncommon in adults (5). Nonetheless, BA ingestion for suicidal purposes has been quite rare. In this paper, a fatal case of toxicity due to BA ingestion for suicidal attempt is reported. The mechanism of toxicity, minimum lethal dose, clinical features and autopsy findings in such cases are discussed.

**CASE REPORT**

A 19 year old man was found dead in the morning of November 2005. Two open packs of BA tablets (each commercial package contains 50 to 100 tablets of 500-1000 mg) were recovered from the vicinity of his body.

According to the history of the case, it was revealed that he had been under psychiatric follow-ups and treatments due to behavioral disorder, and he had a previous suicide attempt.

The body was transferred to the department of forensic medicine of Lady Hardinge Medical College. At first glance, intense cyanosis of the lips and nail beds were evident. On autopsy examination, inflammatory changes in the oral cavity and brownish stains along with right angle of mouth and right nostril were found. Stomach was damaged with hemorrhagic gastritis (Figure 1). All internal organs including liver, kidneys, spleen and brain were congested (Figure 2). The toxicological analysis of viscera confirmed boron ion toxicity.

**DISCUSSION**

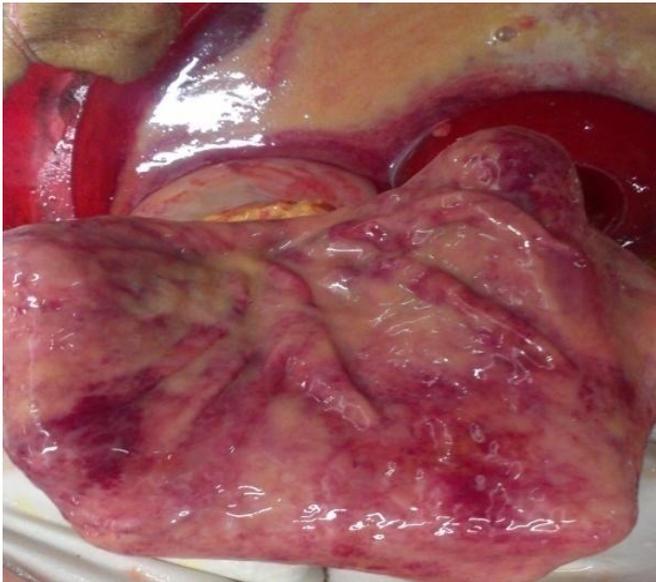
Boric acid is a moderately toxic compound that was first registered in 1948 in the United States as an insecticide for control of cockroaches, termites, fire ants, fleas, silverfish, and many other insects (6). Similar to other acids, it is a mucosal corrosive agent that can affect upper respiratory and gastrointestinal tracts (7,8). Also, it can act as a generalized cellular poison, and thus all end organs including liver, kidneys, brain and gastrointestinal tract are prone to its toxicity (1).

The route of exposure in acute BA poisoning is usually oral which denotes ingestion of insect-killing products (4,5). Acute BA toxicity in young children at home has been quite common which has been mostly due to their curiosity to taste objects (2-4). Hamilton and Wolf reported death of an 18 month old child following the accidental ingestion of a

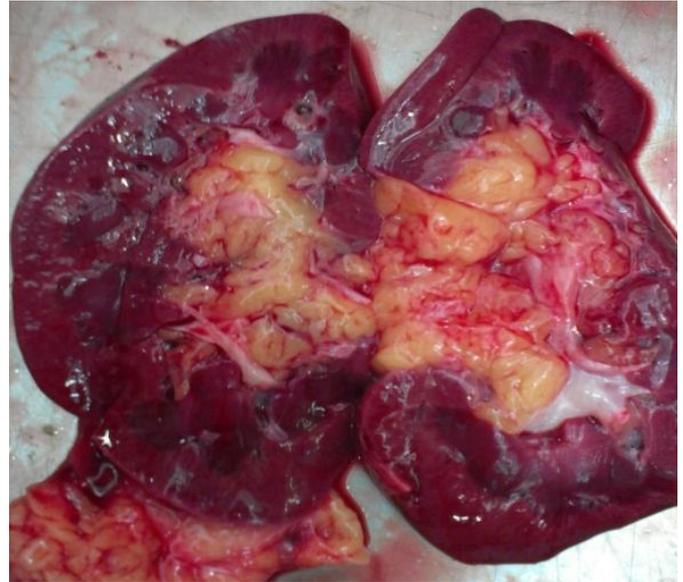
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**Figure 1.** Haemorrhagic gastritis due to boric acid toxicity on autopsy examination



**Figure 2.** Congestion of kidneys due to boric acid toxicity on autopsy examination

BA-containing roach pesticide product (2). Wong et al. reported death of five infants who accidentally ingested 4.5-14 g of BA-containing products; and exhibited serum levels of 200-1600 mg/L. The patients manifested with diarrhea, vomiting, erythema, exfoliation, desquamation of the skin, and marked central nervous system irritation (9).

Nevertheless, massive acute BA poisoning mostly occurs in adults. Corradi et al. reported an 82 year old man who was brought to emergency department after unintentional ingestion of a large amount of BA (10). The patient was treated with hemodialysis and forced diuresis. The patient's condition steadily improved over 84 hours post-admission. They concluded that early treatment with forced diuresis and hemodialysis, even if signs of renal dysfunction are not apparent, can prevent severe renal damage and subsequent complications (10). Naderi and Palmer also reported usefulness of hemodialysis in the treatment of BA poisoning (11). Restuccio et al. reported a case of suicidal attempt in which a 45 year old man ingested approximately two cups of BA crystals dissolved in water (12). The patient developed nausea, vomiting and greenish diarrhea; and dehydration occurred shortly thereafter. The patient was admitted to hospital with hypotension, metabolic acidosis, oliguric renal failure, a generalized erythematous rash, and several superficial skin abrasions. He subsequently developed dysrhythmia and he failed to treatments administered and thus he died after 17 hours of admission. His postmortem urine boron concentration was 29.4 mg/dL (12).

A teaspoonful of 99% BA is equivalent to 3 to 4 g BA. The single oral toxic dose of BA for acute toxicity is highly variable but serious poisoning is reported to occur with 1-3g in newborns, 5g in infants and 20g in adults (13). According to the United States Environmental Protection

Agency and the Agency for Toxic Substances and Disease Registry (ATSDR) minimal fatal dose of ingested boron as BA is 2-3 g in infants, 5-6 g in children and 15-20 g in adults (1,14).

BA toxicity causes gastrointestinal irritation with vomiting and diarrhea that is bluish green in color (5). Death in acute toxicity may be caused by shock resulting from dehydration and renal failure (1), though renal toxicity may not occur following ingestion of minimal amounts of BA (1). Neurologic complications including agitation, seizure and encephalopathy may also occur. Dermal complications of BA toxicity includes erythrodermic rash similar to boiled lobster which could be generalized or localized to the palms of the hands, soles of the feet, buttock, scrotum or face followed by exfoliation after 2-5 days resembling toxic epidermal necrolysis or scalded skin syndrome of newborns (15,16). Alopecia totalis has also been reported in some of the cases (15).

Diagnosis of BA toxicity is based on history of occupational, accidental or suicidal exposure to the compound, the presence of gastroenteritis (with bluish green vomitus) bluish green discoloration of the fecal matter and vomitus, erythrodermic exfoliative rash (boiled lobster appearance), and elevated serum borate levels more than 5 mg/L (1,13). On autopsy examination, there are external findings of erythematous rash and redness of skin and bluish green color of vomitus and fecal matter (12). On internal examination, there are findings of cherry red discoloration of blood, bright red gastric mucosal edema of lungs and visceral congestion. In addition, degenerative changes in the liver, kidney, and brain and also intracytoplasmic inclusion bodies in the pancreatic acinar cells might be observed (9,17).

## LIMITATIONS

We could not determine the exact amount of ingested BA as the body was found by the police force and the details were not reported by them.

## CONCLUSION

Ingestion of BA-based products can be fatal. A patient with severe BA poisoning can be treated with early aggressive hydration and hemodialysis. A dead body with erythematous rash, redness of skin, bluish green color of vomitus and fecal matter, corrosive changes in oral and nasal cavity and multiple organ damages can be highly suggestive of BA poisoning.

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