**PHARM PROFILE**

**DIMERCAPROL**

- Used as the specific antidote for treating acute arsenic poisoning
- Used for treating poisoning caused by mercury, lead, copper, antimony, chromium, and zinc

Dimercaprol (2,3-dimercaptopropanol), also known as **BAL** (British antilewisite), is the specific antidote for treating arsenic poisoning. In small animals, the common sources of exposure to arsenic are home arsenical herbicidal and pesticidal preparations, such as roach and ant baits. The route of exposure is usually oral, but systemic toxicosis can result from percutaneous exposure. Arsenic poisoning is seen more frequently in cats than in dogs. Cats are susceptible because their lethal dose is less than 5 mg/kg of sodium arsenite. Dimercaprol is also used as an antidote in treating mercury and lead intoxication.

**PHARMACOKINETICS AND PHARMACOLOGY**

After IM administration of dimercaprol, peak concentrations in blood occur in 30 to 60 minutes. Dimercaprol is distributed to intracellular fluid. Highest tissue levels of the drug are found in the kidneys and liver. Dimercaprol diffuses into the brain. The drug is metabolized by glucuronidation to inactive compounds and is excreted in urine and feces. Dimercaprol is completely excreted within 4 hours. The plasma half-life is short, and repeated daily doses may be needed.

Dimercaprol is a heavy metal chelating agent. The pharmacologic actions are the result of formation of chelate between its sulfhydryl groups and heavy metals (i.e., arsenic, lead, mercury). Dimercaprol has a high affinity for arsenic. The two sulfhydryl groups of dimercaprol bind arsenic, forming a stable heterocyclic ring complex. One molecule of dimercaprol chelates with one of arsenic. Arsenic is removed from binding sites on sulfhydryl enzyme systems essential to cellular metabolism, such as pyruvate dehydrogenase. The dimercaprol–arsenic complex is nontoxic and is excreted in the urine and feces.

Chelation to dimercaprol is reversible. An insufficient concentration of dimercaprol may allow dissociation of the chelate. Adequate doses must be administered to ensure an excess of free dimercaprol. Dissociation can also occur in acidic urine and may cause renal damage. Alkalization of the urine stabilizes this chelate and promotes its excretion.

In mercury poisoning, two molecules of dimercaprol chelate with one of mercury. This chelate is more water-soluble and is excreted by the kidneys.

**INDICATIONS**

The principal indication for dimercaprol in veterinary medicine is the treatment of acute inorganic arsenic poisoning. However, dimercaprol therapy has not been proven effective for chronic arsenic poisoning.

Chelation therapy with dimercaprol is also used to treat poisoning with either inorganic or elemental mercury. However, dimercaprol is not recommended for chelation of methylmercury. Chelation may facilitate the redistribution of mercury into the central nervous system.

Dimercaprol is also recommended for treating acute lead poisoning associated with encephalopathy.

**CAUTIONS**

IM injections are very painful, particularly if the drug is not administered deeply. Pain may be reduced by using a local anesthetic (e.g., procaine) before the injection.

Sterile or pyogenic abscess formation at the injection site is possible. Other adverse effects (e.g., vomiting, lacrimation, salivation, blepharospasm, tachycardia, elevation of blood pressure) are transient and successfully treated with injectable epinephrine.
In addition, dimercaprol can cause metabolic disturbances (i.e., lactic acidemia, acidosis, hypoglycemia). Long-term use is associated with thrombocytopenia and increased prothrombin time. Dimercaprol interferes with iodine accumulation by the thyroid.

Dimercaprol is contraindicated in iron, cadmium, and selenium poisoning. In these poisonings, the dimercaprol–metal complex is more toxic than the metal alone. Dimercaprol is also contraindicated in animals with hepatic insufficiency unless it is due to arsenic poisoning. Dimercaprol is embryotoxic in mice.

**ACUTE TOXICITY**

The LD₅₀ of dimercaprol is 86.7 mg/kg in rats following IM administration. In animals, signs of overdosage include ataxia, nystagmus, tremors, tetanic seizures, coma, and death. Dimercaprol is also potentially nephrotoxic.

**DOSEAGE AND ADMINISTRATION**

Dimercaprol is much more effective when given as soon as possible after exposure to arsenic because it is more effective in preventing inhibition of sulphydryl enzymes than in reactivating them. Dimercaprol should be administered by deep IM injection. The loading dose is 5 mg/kg IM followed by 2.5 mg/kg IM at 3- to 4-hour intervals for 2 days; then the dosing interval is progressively lengthened to 12 hours until recovery. Dimercaprol dosing may be discontinued when signs of arsenic toxicity resolve. Disappointing therapeutic results may occur because veterinarians have not repeated the treatment.

In addition to dimercaprol therapy, sodium thiosulfate should be given. The sulfur of thiosulfate might react with arsenic and immobilize the metal. The initial dose is 40 to 50 mg/kg IV as a 20% solution. It should be repeated two or three times daily until recovery (i.e., 3 to 4 days).

**STORAGE AND HANDLING**

Dimercaprol should be stored at room temperature (59°F to 86°F; 15°C to 30°C) and protected from light. Sediment may develop during sterilization, but it does not indicate deterioration of the product.

**REFERENCES**

10. El Bahri L, Ben Romdane S: Arsenic


