DIMERCAPROL - (BAL)

Introduction

This agent was developed in Great Britain during World War II as a therapeutic antidote against poisoning by the arsenic-containing warfare agent lewisite. It thus became known as British anti-Lewisite, or BAL.

It is a rarely used intramuscular chelator of heavy metals.

It is the most toxic of all the chelating agents.

It is reserved for the treatment of severe poisoning from **lead, inorganic arsenic and mercury.**

Dimercaprol is also known by the terms:

- 2, 3 dimercaptopropanol.
- British anti-lewisite (BAL)

Preparation

- Dimercaprol 300 mg in peanut oil/ 3 ml ampoules.

Action

- Dimercaprol binds metal ions to form stable dimercaptides, which can then be excreted in the urine.
- Sodium Calcium Edetate predominantly chelates *extracellular* lead and so there is a risk of mobilization of lead from soft tissues into the blood stream which may then exacerbate CNS symptoms (lead encephalopathy). BAL on the other hand mobilizes lead from *intracellular as well as extracellular* sites and it is for this reason that BAL is administered before Sodium Calcium Edetate in order to address this concern. [2]

Indications

It is reserved for the treatment of severe poisoning from

- **Lead**

- **Arsenic**
- **Inorganic mercury**

In cases of **lead encephalopathy** it is used in *combination* with Sodium Calcium Edetate It has also been used in a wide range of other heavy metal poisonings, however clinical experience with these is limited.

They include:

- Gold, bismuth, antimony, chromium, nickel, tungsten and zinc.

Contra-indications / Precautions

- Peanut allergy
- G6PD deficiency.

Adverse Reactions

Dimercaprol has a high incidence (up to 50%) of adverse effects, including:

- Local pain and sterile abscess
- Non-specific constitutional symptoms, fever and myalgias, headache, nausea and vomiting.
- Hypertension
- Excessive lacrimation, rhinorrhea and salivation
- Renal impairment.
- Peripheral paraesthesias
- All chelation therapies carry the risk of micronutrient deficiency, including iron, zinc and copper, so these blood concentrations should be checked before and during chelation therapy, and any deficiencies corrected.

Dosing

Dimercaprol can only be administered IM.

A clinical toxicologist should be consulted on precise dosing regimes.

Alkalinise the urine prior to commencing therapy to reduce the risk of nephrotoxicity, (prevents the dissociation of dimercaprol - metal complexes in the urine).

In *general* terms for lead encephalopathy:

- Commence BAL 4 mg/kg IM every 4 hours, for 5 days.
- Follow with EDTA therapy.

References

1. Lead poisoning in: Murray L et al. Toxicology Handbook 2nd ed 2011.
2. Lead poisoning in: Emergency Medicine 4th ed Tintinalli et al, 1996, p. 835.
3. Dimercaprol in: Murray L et al. Toxicology Handbook 2nd ed 2011.
4. Sodium Calcium Edetate in: Murray L et al. Toxicology Handbook 2nd ed 2011.
5. Succimer in: Murray L et al. Toxicology Handbook 2nd ed 2011.
6. eTG - Toxicology and Wilderness, 2nd ed, 2012

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