**DECONGESTANTS**

**1- IMIDAZOLINE DECONGESTANTS**

The imidazoline derivatives, **oxymetazoline, xylometazoline, tetrahydrozoline and naphazoline,** are found in topical ophthalmic and nasal decongestants available OTC. They are generally used as topical vasoconstrictors in the nose and eyes for temporary relief of nasal congestion due to colds, hay fever or other upper respiratory allergies, or sinusitis.

Imidazolines are sympathomimetic agents, with primary effects on α-adrenergic receptors and little if any effect on β-adrenergic receptors. Oxymetazoline is readily absorbed orally. Effects on α-receptors from systemically absorbed oxymetazoline hydrochloride may persist for up to 7 hr after a single dose. The elimination half-life in people is 5–8 hr. It is excreted unchanged both by the kidneys (30%) and in feces (10%).

**Clinical Findings**

signs of intoxication may include vomiting, bradycardia, cardiac arrhythmias, hypotension or hypertension, panting, increased upper respiratory sounds, depression, nervousness and weakness. These signs usually appear within 30 min to 4 hr after exposure.

**Treatment**

* Decontamination (induction of emesis and administration of activated charcoal) may not be practical because of the rapid absorption and onset of clinical signs.
* Heart rate and rhythm and blood pressure should be assessed, and an ECG obtained if needed. IV fluids should be given, along with atropine at 0.02 mg/kg, IV, if bradycardia is present.
* Diazepam (0.25–0.5 mg/kg, IV) can be given if CNS signs are present.
* Serum electrolytes (ie, potassium, sodium, chloride) should be assessed and corrected as needed.
* Yohimbine, a specific α2-adrenergic antagonist, can also be used at 0.1 mg/kg, IV, and repeated in 2–3 hr if needed.

**2- Phenylephrine**

Phenylephrine is a sympathomimetic amine with mainly an α1-adrenergic receptor agonist effect, available OTC as a decongestant in oral tablets, nasal sprays, or eye drops. It has poor oral bioavailability (38%) in people because of a significant first-pass effect and extensive metabolism by monoamine oxidases in the GI tract and liver. The half-life is 2–3 hr. CNS stimulation, agitation, nervousness, and hypertension are possible but less frequent with phenylephrine than with pseudoephedrine. Treatment is mainly symptomatic care and is similar to that for pseudoephedrine toxicosis (see below).

**PSEUDOEPHEDRINE AND EPHEDRINE**

Pseudoephedrine is a sympathomimetic drug found naturally in plants of the genus *Ephedra*. Several states in the USA have limited the availability and use of pseudoephedrine as an OTC decongestant because of its use as a precursor in illegal amphetamine synthesis. It is being replaced with other decongestants such as phenylephrine.

Pseudoephedrine is a stereoisomer of ephedrine and is available as the hydrochloride or sulfate salt. Both ephedrine and pseudoephedrine have α- and β-adrenergic agonist effects. The pharmacologic effects of the drugs are due to direct stimulation of adrenergic receptors and the release of norepinephrine.

Pseudoephedrine is rapidly absorbed orally. The onset of action is 15–30 min, with peak effects within 30–60 min. It is incompletely metabolized in the liver. Approximately 90% of the drug is eliminated through the kidneys. Renal excretion is accelerated in acidic urine.

**Clinical Findings**

Pseudoephedrine and ephedrine overdose can result in mainly sympathomimetic effects, including agitation, hyperactivity, mydriasis, tachycardia, hypertension, sinus arrhythmias, anxiety, tremors, hyperthermia. Clinical signs can be seen at dosages of 5–6 mg/kg, and death may occur at 10–12 mg/kg.

**Treatment**

Treatment of pseudoephedrine poisoning consists of **decontamination**, controlling the **CNS** and **cardiovascular effects**, and **supportive care**. Vomiting should be induced only in asymptomatic patients, followed by administration of activated charcoal with a cathartic. If emesis is contraindicated, a gastric lavage should be performed.

Hyperactivity, nervousness, or seizures can be controlled with chlorpromazine, phenobarbital, or pentobarbital. Diazepam should be avoided, because it can exaggerate hyperactivity. Phenothiazines should be used with caution because they can lower the seizure threshold. Tachycardia can be controlled with propranolol, repeated if needed, or with esmolol. Acidifying the urine with ammonium chloride or ascorbic acid may enhance urinary excretion of pseudoephedrine.

Electrolytes, heart rate and rhythm, and blood pressure should be monitored. The presence of pseudoephedrine in urine can support the diagnosis.