Decongestants

A decongestant (or nasal decongestant) is used to relieve nasal congestion in the upper respiratory tract.

These are alpha-agonists which on topical application as dilute solution (0.05-0.1%) produce local vasoconstriction.
Regular use of these agents for long periods should be avoided .

Decongestants can be absorbed from the nose via an inhaler and produce systemic effects,

mainly CNS stimulation and rise in blood pressure.

These drugs should be used cautiously in hypertensives and in those receiving MAO inhibitors.

The vast majority of decongestants act via enhancing norepinephrine (noradrenaline) and epinephrine (adrenaline) or adrenergic activity by stimulating the α-adrenergic receptors.

This induces vasoconstriction of the blood vessels in the nose, throat, and paranasal sinuses, which results in reduced inflammation (swelling) and mucus formation in these areas.

Topical nasal or ophthalmic decongestants quickly develop tachyphylaxis
(a rapid decrease in the response to a drug after repeated doses over a short period of time).

Long-term use is not recommended, since these agents lose effectiveness after a few days.

Common decongestants:

- Ephedrine
- Levomethamphetamine
- Naphazoline
- Oxymetazoline
- Phenylephrine
- Phenylpropanolamine
- Propylhexedrine
- Synephrine
- Tetrahydrozoline
- Xylometazoline
- Pseudoephedrine—controlled in some jurisdictions for over the counter use
- Tramazoline

Pseudoephedrine is the d-isomer of ephedrine and has only 25% of the adrenergic receptor activity of ephedrine.

While both ephedrine and pseudoephedrine are stimulants of alpha as well as beta adrenergic receptors.

Phenylpropanolamine is a sympathomimetic agent with primarily direct alpha-adrenergic agonist effects, but also indirect stimulation of noradrenaline release. It also has weak beta-1 agonist effects but lacks beta-2 agonist properties.
It is used as an oral and topical decongestant and an anorexiant.

Locally instilled nasal decongestants comprise imidazoline compounds such as naphazoline, oxymetazoline, tetrahydrozoline, and xylometazoline, which are powerful alpha2- adrenergic receptor stimulants.

Common signs of poisoning:

Vomiting

Dilated pupils

Severe blood pressure changes

Elevated or really slow heart rate

Tremors

Seizures

Acute death in dogs and cats

Clinical (Toxic) Features

1. Toxicity usually manifests as CNS stimulation, hypertension, and tachycardia (bradycardia with phenylpropanolamine). Headache is common. Serious manifestations include seizures, dysrhythmias, cerebral haemorrhage, and psychosis.

2. Imidazoline decongestants cause CNS depression, hypotension, bradycardia, and respiratory depression. Imidazolines may also be used in combination with other sympathomimetics (e.g. phenylephrine or ephedrine), in which case, hypertension may be seen. Miosis may be present. Mydriasis has also occurred.

3. Imidazolines have presynaptic alpha-2 stimulant effects, like clonidine.

Overdose or intoxication from oral ingestion or excessive topical administration can result in severe drowsiness with diaphoresis, hypotension or shock, bradycardia, respiratory depression, and coma. These manifestations may occur in both adults and children; however, young children (especially infants) are more susceptible to toxicity.

Recovery may be expected in 12 to 36 hours.

CNS depression ranging from sleepiness, hypotonia, and hyporeflexia to coma is common in children.

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Headache, nervousness, tremors, and insomnia are frequently reported.

4. Chronic over use may result in reactive vasodilation of the nasal mucosa. Acute psychosis and hypertension have been reported with chronic abuse.

5. Signs and symptoms of phenylpropanolamine overdose comprise hypertension, mydriasis, arrhythmias, anxiety, chest pain, auditory and visual hallucinations, paranoid ideation, occasionally delirium and psychosis, seizures, haemorrhagic and non-haemorrhagic cerebral infarctions, rhabdomyolysis, urinary retention, and renal failure.

In fact, phenylpropanolamine has a propensity to cause significant hypertension, and may result in reflex bradycardia, extensive myocardial ischaemia, cerebral haemorrhage, or renal toxicity.

Tachycardia can also occur. Peak blood pressure effects of phenylpropanolamine occur at about 2.5 hours after PPA ingestion with individual times ranging from 0.5 to 4.5 hours.

6. Psychiatric disturbances, particularly in children, have been reported after ingestion of phenylpropanolamine including restlessness, irritability, aggressiveness, sleep disturbances, psychotic episodes, confusion, acute mania and hallucinations.

Treatment

Because drowsiness and coma may occur rapidly, emesis is not indicated even when nasal decongestants have been ingested.

Emesis is contraindicated in patients with hypertension, since protracted vomiting may increase intracranial pressure.

Activated charcoal and gastric lavage are also not routinely recommended, though they may be of value in phenylpropanolamine ingestions.

Monitor serum CPK and renal function in severely symptomatic patients, and those with prolonged seizures or coma.

A CT scan is indicated in patients with severe headache, neurologic deficits, or abnormal mental status (especially in the case of phenylpropanolamine).

1. Seizures, agitation, and psychosis should be treated with IV diazepam..

2. Severe symptomatic palpitations, tremor, and associated anxiety may respond to propranolol, particularly in patients with combination overdose of phenylpropanolamine and other sympathomimetic agents.

3. Persistent or highly elevated hypertension should be treated with nitroprusside or nifedipine. Nitroglycerin and phentolamine are possible alternatives.

4. Hypotension can be managed with isotonic fluids, Trendelenberg position, and dopamine infusions.

5. Dysrhythmias usually respond to standard doses of lignocaine or bretylium. Propranolol can also be used.

Lignocaine and amiodarone are generally first line agents for stable monomorphic ventricular tachycardia, particularly in patients with underlying impaired cardiac function.

Sotalol is an alternative for stable monomorphic ventricular tachycardia.

Sinus tachycardia does not generally require treatment unless haemodynamic compromise develops. If therapy is required, a short acting, cardioselective agent such as esmolol is generally preferred.

Bradycardia generally does not require treatment. Since the bradycardia is a reflex response, atropine should theoretically be avoided as it may worsen hypertension.

6. Because the imidazoline decongestants produce sedation, hypotension, and bradycardia via acentral alpha-adrenoreceptor stimulation, similar to clonidine, the administration of naloxone may theoretically be beneficial.

7. Dialysis may be beneficial in phenylpropanolamine overdose.