Module 5.3
Clonidine

Introduction

Clonidine is a centrally acting alpha receptor agonist with a wide range of clinical uses including treatment for hypertension ADHD, conduct disorder, narcotic withdrawal and Tourette's syndrome.

Toxicokinetics

Clonidine is rapidly and well absorbed, with peak concentrations at 90 minutes. About 20-40% is protein bound and it has a volume of distribution of 3.2-5.6L/kg. It is largely eliminated unchanged by the kidneys[1].

Risk Assessment

Clonidine toxicity is manifest by CNS depression, miosis, bradycardia, hypotension and hypothermia[1]. Onset is rapid, normally within 30-60 minutes.

A recent Australian series of clonidine overdose showed that clonidine intoxication does not cause severe toxicity but does cause prolonged bradycardia (median duration 20h) and sedation[2].

While there is no clear correlation between dose ingested and toxicity, one small case series of clonidine poisoning in paediatric patients found ingestions of <10mcg/kg caused minimal toxicity[3].

Management

Resuscitation

Hypotension should respond to fluid resuscitation. Bradycardia is typically mild and rarely requires therapy, however if there is haemodynamic compromise atropine can be given.
Airway protection is necessary for coma, however this unlikely to occur unless there are sedating co-ingestants.

Decontamination

Rapid onset of toxicity typically precludes administration of activated charcoal.

Supportive Care

Standard supportive measures should be instituted including bladder cares, thromboprophylaxis if required and maintenance fluids.

Disposition

Most patients can be managed safely in the Short Stay Unit. Toxicity typically resolves over 24 to 48 hours. Care needs to be taken to manage clonidine withdrawal after this time.

Additional Information

- Clonidine withdrawal can occur. It is characterised by anxiety, headache, sweating, tachycardia, hypertension and nausea. Restarting clonidine is the most effective treatment[2].

- Naloxone has been used in clonidine overdose to reverse toxicity, particularly sedation[1], however recent evidence suggests it is ineffective and is not recommended[4].

Further reading

- Isbister GK, Heppell SP, Page CB & Ryan NM "Adult clonidine overdose: prolonged bradycardia and central nervous system depression, but not severe toxicity" *Clinical Toxicology*, 2017; DOI: 10.1080/15563650.2016.1277234

References

1. Nelson L et al. Goldfrank's Toxicologic Emergencies. 9th Ed. 2010. McGrawHill Medical: Sydney.

2. Isbister GK, Heppell SP, Page CB & Ryan NM "Adult clonidine overdose: prolonged bradycardia and central nervous system depression, but not severe toxicity" *Clinical Toxicology*, 2017; DOI: 10.1080/15563650.2016.1277234

3. Fiser DH, Moss MM, Walker W. "Critical care for clonidine poisoning in toddlers." *Crit Care Med* 1990; Oct;18(10):1124-1128

4. Kristin M. Engebretsen, Kathleen M. Kaczmarek, Jenifer Morgan & Joel S. Holger (2011) High-dose insulin therapy in beta-blocker and calcium channel-blocker poisoning, Clinical Toxicology, 49:4, 277-283, DOI: 10.3109/15563650.2011.582471