Antihistamines

Overview

Sedation, anticholinergic and cardiotoxity are possible depending on dose and whether older (sedating) or newer (non-sedating) generation of antihistmines.

Toxic mechanism

Competitive inhibition at H1 receptors. Sedating AH are lipophilic and cross BBB, they also block M1, α & 5HT receptors. Non-sedating AH are less lipophilic and H1 selective, although OD may ↓selectivity and still cause CNS sedation. Cardiac Na & K channel blockade occurs in large/massive OD.

Toxicokinetics

Well abs. Lipophilic with large Vd (Sedating AH), or small Vd (Non-sedating). Variable liver met. Variable $T\frac{1}{2}$.

Clinical features

CNS depression/sedation: minor with non-sedating AH
Anticholinergic toxidrome: minor with non-sedating AH
Seizures: rare – only really a risk with sedating AH
CVS:

- ↑HR
- Hypotension 2° to NaBlockade rare after massive OD of sedating AH.
- ↑QTc 2° to KBlockade rare after large OD of non-sedating AH.

Investigations

Screening: serial ECG, paracetamol, BSL

Risk assessment

Large OD may → CVS toxicity otherwise sedation/anticholinergic effects more likely with sedating AH. Children may gate anticholinergic delirium.

Management

Resus: Rarely required but should be cardiac monitored for 6h (12h non-sedating).
Supportive Care:

- Treat seizures with BDZs,
- For ↑QRS/NaBlockade: **Sodium bicarbonate** 1-2mmol/kg boluses over 1-2min.
- For ↑QTc: **$MgSO_4$**, **isoprenaline** or overdrive pacing.

Antidote: **Physostigmine** if severe anticholinergic delirium unresponsive to BDZ.

Disposition

If asymptomatic and normal ECG at 6hr → d/c else admit. If sig. delirium/sedation or dysrhythmia → HDU/ICU.