

Overview

Opioids cause CNS & respiratory depression which can lead to respiratory arrest & death. Some opioids have unexpected toxic effects (see below). May be recreational OD.

Toxic mechanism

Mu receptor activity is responsible for sedation and respiratory depression. Other opioid actions can cause N & V (dopamine receptors), constipation (peripheral mu receptors), pruritus (histamine release) & seizures.

Toxicokinetics

Variable oral abs. Large VDs (codeine 2.4L/kg, morphine 3.4L/kg). Hepatic met. often to active metabolites excreted in urine. Variable duration of effect (heroin <6hrs, methadone ~24hrs)

Clinical features

Classically: CNS depression, shallow & slow respirations, miosis.

Others: Hypoxia, N & V (\pm aspiration), hypothermia & pressure injury (skin necrosis, compartment syndrome, rhabdomyolysis) from prolonged prostration. APO.

Other specific toxic effects:

- Dextropropoxyphene (seizures, hypoBP, sodium-block cardiac arrhythmias)
- Tramadol (esp dose >10mg/kg: seizures, serotonin syndrome)
- Pethidine (rpt doses \rightarrow accumulation of norpethidine: seizures. Also serotonin syndrome)

Investigations

Deliberate OD screening tests: ECG, BSL, paracetamol level.

Specific: Only for complications.

Risk assessment

CNS/respiratory depression can occur just above analgesic dose.

Opioid naïve patients or if other depressants co-ingested increases risk of depression.

In children a single tablet can cause respiratory arrest or, in the case of tramadol, seizures.

Management

Resuscitation: Particular attention to A & B. Dextropropoxyphene ventricular arrhythmias may need IV **sodium bicarbonate**.

Supportive: Fluids, seizure control (BDZ), close monitoring.

Decontamination: Activated charcoal is not routinely indicated.

Elimination: Not clinically useful.

Antidotes: **Naloxone** (see Antidotes).

Disposition

Children, and any morphine, tramadol, or controlled release opioid OD need 12-24hrs observation.