Management of the poisoned patient

Andrew J Goddard

James Dear

Abstract

Poisoning is a common hospital presentation that is increasing in incidence. The clinical effects depend on a number of variables including the nature of the poison, total dose and timing, and the age and underlying health of the patient. These factors contribute to the toxicities encountered, the supportive measures required, and the requirement for admission to a critical care area. Patients presenting early after poisoning can be considered for decontamination. Not all poisons are amenable to such measures, but when they are, the toxic effects may be reduced or even prevented. When the agent causing poisoning is known, specific antidotes may be available and these offer the opportunity to reduce morbidity and mortality. When the agent is unknown the clinical syndrome of symptoms and signs may suggest one of the recognizable toxidromes that can guide treatment. Patients can be rendered incapacitated as a result of the toxic effects of poisons and this may impact upon informed consent. Once medically able, all deliberate self-poisoned patients should be referred for psychiatric assessment.

Keywords Antidote; decontamination; OD; overdose; poison; toxbase; toxicity; toxicology; toxidrome

Learning objectives

After reading this article, you should be able to:

- describe in a system-based manner the potential toxic effects of poisons along with common therapies for their management
- list the common toxidromes, their receptor system, clinical features and examples of causative poisons
- outline the indications for decontamination and gastric lavage and be aware of the available antidotes to some common poisons.

The poisoning problem

Poisoning is one of the commonest medical presentations to hospital in the UK accounting for over 500,000 NHS hospital bed-days in 2007/08.[1] Most patients can be managed in emergency department observation units, acute medical admission units or dedicated toxicology units. However, certain patients may require admission to a critical care area. It is important to note that most poisoned patients have acutely reversible conditions.

Considerations when assessing poisoned patients

Several factors must be considered: the poisons ingested, their quantity and timing, the presence or potential for poison induced end-organ toxicity, as well as certain poison and patient characteristics. However, the initial assessment of any known or suspected poisoned critically ill patient should, as with all such patients, begin with a rapid evaluation of airway patency, breathing and circulation adequacy, fluid balance and neurological status as well as an objective measurement of blood glucose. Consideration of all of these will dictate the most appropriate place for the patient's ongoing management.

System-based potential poison-induced toxicities and example therapies

System	Potential toxic effects	Example therapies
Respiratory	Hypoxia	Oxygen, CPAP
	Hypoventilation	Non-invasive ventilation,
	Acute lung injury/ARDS	endotracheal intubation and ventilation
Cardiovascular	Hypertension	Vasodilators, Ca channel/β/α-blockers
	Hypotension	Circulating volume resuscitation, vasopressors, inotropes
	Bradyarrhythmia	Chronotropes, external cardiac pacing, temporary transvenous cardiac pacing
Neurological	Depressed consciousness	ETT (if airway compromised)
	Delirium	Neuroleptics, short-acting sedatives
	Status epilepticus	Benzodiazepines, hydantoins, barbituric coma, cooling, paralysis
Gastrointestinal	Acute hepatic failure	Support bridge to transplantation
	Vomiting	NG tube, ETT (if aspiration risk)
Genitourinary	Acute kidney injury	Renal replacement therapy
Metabolic	Metabolic acidosis	Sodium bicarbonate,
	Electrolyte disturbance	renal replacement therapy, CVC (for rapid correction)

ARDS, acute respiratory distress syndrome; CPAP, continuous positive airway pressure; CVC, central venous catheter; ETI, endotracheal intubation; NG, nasogastric tube

Table 1

Andrew J Goddard *MB ChB MRCP(UK) is a Specialty Registrar in Acute Medicine and General (Internal) Medicine at Queen Margaret Hospital, Dunfermline, UK. Conflicts of interest: none declared.*

James Dear *BM BCh PhD MRCP(UK) is a Consultant in Clinical Pharmacology and General (Internal) Medicine at the Royal Infirmary of Edinburgh, UK. Conflicts of interest: none declared.*

The main clinical toxidromes, their receptor system, the key clinical features and example agents

Toxidrome	Receptors	Effect	Clinical features	Example agent(s)
Anticholinergic	Nicotinic Muscarinic	Antagonist	Dry skin Hyperthermia Thirst Dry mouth Dilated pupils Tachycardia Urinary retention Decreased bowel sounds	Tricyclics Antipsychotics Antihistamines
Cholinergic	Nicotinic Muscarinic	Agonist	Defaecation Urination Miosis Bradycardia[a] Tachycardia[a] Emesis Lacrimation Hyperhidrosis	Organophosphates
Opioid	Opioid	Agonist	Hypoventilation Pulmonary oedema[b] Hypotension Depressed consciousness Pinpoint pupils Naloxone response	Opiates
Sympathomimetic	Multiple molecular effects	Agonist	Hyper/hypotension Tachycardia Neurological excitation Tremor Hyperreflexia Seizures	Cocaine Amphetamines
Serotonin[c]	$5HT_2$	Agonist	Restlessness Agitation Confusion Hyperreflexia Clonus Tremor Shivering Hypertonia Fever Flushing	SSRI MAOI Tricyclics Venlafaxine MDMA Amphetamines Cocaine Tramadol Triptans Linezolid St John's wort

MAOI, monoamine oxidase inhibitors; MDMA, 3,4-methylenedioxymethamphetamine (ecstasy); SSRI, selective serotonin reuptake inhibitors
[a] The clinical picture depends on whether nicotinic (tachycardia) or muscarinic (bradycardia) receptor stimulation predominates.
[b] Rare, mechanism unclear but appears not to be cardiac in origin.
[c] Typically occurs when two or more drugs that affect the serotonin system are ingested.

Table 2

Poisons ingested; quantity and timings

These details may not always be immediately, or indeed ever, known. All efforts must be made to elucidate as much information as possible and an accurate collateral history is vital. Whilst this is being carried out, simultaneous initial management should focus on supporting organ function and correction of physiological derangement rather than treating the poison(s) themselves. This is especially true in the case of poly-agent poisoning where physiological effects can be unpredictable.

Presence or potential for end-organ toxicity

Table 1 provides a system-based list of possible poison-induced end-organ toxicities and examples of therapies for each. When presented with a patient displaying any of these where no other