Poisons

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Epidemiology and clinical presentation

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Epidemiology

In the UK, poisoning causes about 10% of acute hospital medical admissions. This usually involves self-administration of a prescribed or over-the-counter medicine or an illicit drug. Most patients have taken (usually ingested) more than one drug, and alcohol is the most commonly implicated second agent. As deliberate self-harm is a risk factor for further episodes of self-harm, about 20% of these episodes occur in the same patient group.

Substances may also be administered deliberately to cause harm or for financial or sexual gain. Poisoning is sometimes iatrogenic – for example, digoxin toxicity. Occupational poisoning is common in developing countries and continues to occur in the developed world. Poisoning in young children is usually accidental, but it may be iatrogenic in those younger than six months, involving, for example, overtreatment with paracetamol.

The type of agent taken is influenced predominantly by availability. For example, in the UK, paracetamol poisoning is responsible for about one third of all admissions for poisoning, whereas the agents ingested in Sri Lanka are more often pesticides or plants, such as oleander. Pesticides cause less than 0.05% of hospital admissions for poisoning in England and Wales, whereas pesticides are associated with around 50% of all cases of poisoning and substantial mortality in Sri Lanka.[1]

Key Points

- **Acute poisoning is responsible for about 10% of acute hospital medical admissions in the UK**
- **The type of agent taken is influenced predominantly by availability. For example, in the UK, paracetamol poisoning is responsible for about one third of all admissions, whereas the agents ingested in Sri Lanka are more often pesticides or plants, such as oleander**
- **In the UK, deaths are due predominantly to carbon monoxide, antidepressants, paracetamol, analgesic combinations that contain paracetamol and an opioid, heroin, methadone or cocaine**
- **Most deaths occur before admission to hospital; less than 1% of poisoned patients admitted to hospital die**
- **Neurological signs, particularly those normally indicative of brain stem damage, may be observed but do not necessarily imply a poor prognosis**
- **Acid–base and electrolyte abnormalities may be diagnostically important**

KEY WORDS: acid–base and electrolyte abnormalities, blisters, deliberate self-harm, epidemiology, poisoning, rhabdomyolysis

Deaths from poisoning

In the EU as a whole, poisoning is a major cause of death among men and women aged 20–44 years.[2] Fatalities in the UK are due predominantly to carbon monoxide, antidepressants, paracetamol, analgesic combinations that contain paracetamol and an opioid, heroin, methadone or cocaine.[3] Most poison-related deaths occur before admission to hospital, and less than 1% of poisoned patients admitted to hospital die.

Deaths from poisoning in children are usually due to inappropriate storage of drugs such as digoxin and quinine and drugs of abuse prescribed or purchased for a parent or carer.

Clinical presentation

General observation can provide vital information – for example, alcohol or solvents may be smelled on the breath, needle track marks may reveal undisclosed substance abuse, atypical bruising may warn of domestic or other violence and the stigmata of alcoholic liver disease may be revealed. Acutely poisoned patients may also be emotionally and psychiatrically distressed.

With many poisons, only a single feature supports the diagnosis (Table 1). In other cases, the cluster of features on presentation is distinctive and diagnostic. For example, fixed dilated pupils, exaggerated tendon reflexes, extensor plantar responses, depressed respiration and cardiac arrhythmias suggest poisoning with a tricyclic antidepressant (Table 2).

Hypotension and hypothermia

Although hypotension is a recognised feature of acute poisoning, the classic features

Table 1. Common features in acute poisoning.

Features	Poisons
Miosis	Opioids, organophosphorus insecticides, nerve agents
Mydriasis	Tricyclic antidepressants, amfetamines, cocaine, anticholinergic drugs
Blindness	Methanol, quinine
Papilloedema	Carbon monoxide, methanol
Convulsions	Tricyclic antidepressants, theophylline, opioids, mefenamic acid, isoniazid, amfetamines
Dystonic reactions	Metoclopramide, phenothiazines
Divergent strabismus	Tricyclic antidepressants
Nystagmus	Phenytoin, carbamazepine
Hypertonia and hyperreflexia	Tricyclic antidepressants and other anticholinergic drugs
Hyperventilation	Salicylates, phenoxyacetate herbicides, theophylline
Hyperthermia	Ecstasy (MDMA)
Blisters	Usually occur in comatose patients

MDMA = 3,4-methylenedioxymethamphetamine.

of shock are seen rarely, because only a minority of patients are severely poisoned. A core temperature <35°C may be recorded in deeply unconscious patients who have been exposed, particularly in cold weather, for several hours.

Blisters

Skin blisters may be found in poisoned patients who are, or have been, unconscious.[4,5] Such lesions are not diagnostic of specific poisons but are sufficiently common in poisoned patients (and sufficiently uncommon in patients unconscious from other causes) to be of diagnostic value.

Rhabdomyolysis

In patients who are poisoned, non-traumatic rhabdomyolysis may result. This may be accompanied by acute renal failure (which may be non-oliguric) and peripheral nerve damage (secondary to compartment syndrome), which predominantly results in wrist or foot drop.

Neurological features

Pyramidal tract signs. The usual features of pyramidal tract involvement (hypertonia, hyperreflexia and extensor plantar responses) are commonly found in poisoning with tricyclic antidepressants and other drugs with marked anticholinergic actions (for example, the older antihistamines). However, all of these signs may be abolished in deep coma.

Abnormal movements. Decerebrate and decorticate movements of the limbs often occur in unconscious poisoned patients, but in most cases there is no irreversible brain damage and the patient recovers fully. Acute dystonic movements (including acute torticollis, orolingual dyskinesias and oculogyric crises) are also produced; these are usually caused by metoclopramide[6] or, less commonly, haloperidol, droperidol, prochlorperazine or trifluoperazine. Choreoathetosis has been reported as a rare presenting feature of poisoning with organophosphorus insecticides.[7]

Pupillary changes. Inequality of the pupils is not uncommon in poisoned patients. Widely dilated pupils that react poorly to light may be caused by poisons with anticholinergic actions (for example, tricyclic antidepressants), poisons with sympathomimetic effects (for example, amfetamines) and poisons that cause blindness due to retinal toxicity (for example, quinine and ethanol). Miosis is usually caused by opioid analgesics or poisons with cholinergic or anticholinesterase actions (for example, organophosphorus insecticides and nerve agents). The degree and speed of reaction of the pupils to light is of no clinical value.

Ocular movements. Strabismus, internuclear ophthalmoplegia and total external ophthalmoplegia have been described in patients with impairment of consciousness due to acute poisoning.[8] Transient and variable strabismus (usually with the optic axes divergent in the horizontal plane) has followed poisoning with

Table 2. Common feature clusters in acute poisoning.

Feature clusters	Poisons
Coma, hypertonia, hyperreflexia, extensor plantar responses, myoclonus, strabismus, mydriasis, sinus tachycardia	Tricyclic antidepressants; less commonly antihistamines, orphenadrine, thioridazine
Coma, hypotonia, hyporeflexia, plantar responses, flexor or non-elicitable hypotension	Barbiturates, benzodiazepine and alcohol combinations, tricyclic antidepressants
Coma, miosis, reduced respiratory rate	Opioid analgesics
Nausea, vomiting, tinnitus, deafness, sweating, hyperventilation, vasodilation, tachycardia	Salicylates
Hyperthermia, tachycardia, delirium, agitation, mydriasis	Ecstasy (MDMA) or other amfetamine
Miosis, hypersalivation, bronchorrhoea	Organophosphorus and carbamate insecticides, nerve agents
Delirium and hallucinations	Anticholinergic drugs, amfetamines, cannabis, recovery from tricyclic antidepressant poisoning

MDMA = 3,4-methylenedioxymethamphetamine.

phenytoin, carbamazepine and tricyclic antidepressants. Occasionally there may be total external ophthalmoplegia, even in patients in whom consciousness is no more than minimally impaired.

Dysconjugate eye movements have been reported in poisoning with tricyclic antidepressants, phenothiazines, benzodiazepines, barbiturates and ethanol. It may become apparent, however, only when the oculovestibular reflexes are examined with caloric stimuli.

Loss of oculocephalic and oculovestibular reflexes. Loss of these reflexes is usually regarded as evidence of severe brain stem damage and brain stem death. In acute poisoning, however, such a conclusion is not justified. Poisoning with carbamazepine, phenytoin and tricyclic antidepressants can be associated with loss of these reflexes, but patients recover completely.

Visual impairment. Visual impairment is associated most commonly with poisoning with quinine or methanol.[9,10]

Acid–base and biochemical abnormalities

Acid–base abnormalities, particularly respiratory and metabolic acidoses, are common presentations in acute poisoning. Respiratory acidosis due to depression of the central nervous system or pulmonary toxicity and metabolic acidosis due to lactic acidaemia or derangements of intermediary metabolism are particular features of poisoning. Respiratory alkalosis is a feature of salicylate poisoning. Hypokalaemia and hyperkalaemia are most often due to redistribution of potassium across cell membranes, such as may occur in poisoning with β_2-agonists (hypokalaemia) and digoxin (hyperkalaemia). Hyponatraemia is a recognised complication of the use of ecstasy (3,4-methylenedioxymethamphetamine (MDMA)) and is caused by inappropriate secretion of antidiuretic hormone. Hypoglycaemia may follow an overdose of insulin, sulphonylurea or ethanol and may occur in paracetamol-induced liver failure.

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