Answers for Case Report 1

la. Organophosphate poisoning.

lb. Mechanical ventilation and intravenous atropine.

lc. Exposure to rat poison. On the day of admission the patient had handled rat poison and had presumably ingested the op compound from his unwashed hands while eating.

Organophosphorus poisoning

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Introduction

Organophosphorus poisoning is increasing in incidence and is one of the commonest forms of poisoning responsible for hospital admission in Malawi. It may be fatal. It is therefore extremely important to recognise the characteristic clinical features in order to make the diagnosis and give what may well be life-saving treatment.

Organophosphorus compounds

Organophosphorus compounds are poisons used as insecticides and rodent poisons, are available for agricultural and domestic use and can be bought "over the counter". Control over sales and precautions with use are less stringent, and therefore acute severe poisoning is.more common in developing countries.

Route of poisoning

Poisoning can result from ingestion (accidental or deliberate), inhalation or absorption from skin contact. Ingestion in children usually occurs when younger children pick up and eat poison which is either not properly stored away or is left lying around the house (sometimes disguised with food) for rodents. In adults, poisoning by inhalation or absorption is usually through faulty handling. Deliberate ingestion may be for intentional self-harm; someone may deliberately poison food or drink with criminal intent.

Pharmacoloav

Acetylcholine is the neurotransmitter at neuromuscular junctions, parasympathetic nerve endings, autonomic ganglia and in parts of the central nervous gystem. When nerve endings release acetylcholine at these sites, acetylcholine esterase breaks down acetylcholine and therefore limits the duration of action of acetylcholine at its receptors. Organophosphorus compounds inhibit acetylcholine esterase and therefore prolong the action of acetylcholine at its receptors.

TABLE 1

CLINICAL FEATURES OF ORGANOPHOSPHORUS POISONING ACCORDING TO SITE OF EXCESSIVE ACTION

Site of excessive action	*Clinical features*
neuromuscular junctions	muscle fasciculation smuscular weakness or paralysis (depolarising blockade)
parasympathetic nerve endings and autonomic ganglia	bradycardia, bronchospasm, pulmonary oedema, abdominal colic and diarrhoea, cold sweating, pupil constriction, salivation, lachrymation
central nervous system	confusion, decreased conscious level, convulsions, respiratory depression

Clinical features

The degree of severity of poisoning is variable depending on the amount of organophosphorus absorbed. In mild cases there may only be abdominal diarrhoea and small pupils. In severe cases there is hypersalivation, bradycardia, pinpoint pupils. cold sweating, coma, paralysis and respiratory failure. Death is usually from respiratory failure due to respiratory muscle paralysis and central respiratory depression.

Practice pnint

Hypersalivation is often an important clue to severe organophosphorus poisoning. Drooling of saliva occurs in several conditions e.g. painful swallowing, bulbar and pseudobulbar palsy. However., the continuous production of large quantities of saliva usually occurs as. a result of either the neurotoxic venoms of cobra and mamba bites or organophosphorus poisoning. The sight of a patient continuously drooling and spitting out large quantities of saliva into a bowl or onto the bed should ring a mental alarm bell for organorophosphorus poisoning.

Management

Table 2 shows the aims of management and how to achieve them.

Table 2 Aims of management and how to achieve them

Aim	*Management*
prevent further absorption of poison	gastric lavage (ingestion), washing (skin contact)
reverse the effects of acetylcholine	atropine 600 mcg I.V. every 15 minutes until reversal (for children 20mcg//kg)
reactivate cholinesterase	pralidoxime lg I.V- over 10 minutes
general support	oxygen and I.V. fluids
prevent death from respiratory failure	mechanical ventilation
prevent complications	pressure care, monitor for paralysis signs of pneumonia, catheter

Management depends on the degree of severity of poisoning. In all cases, patients need monitoring of conscious level, respiration, pulse rate and pupil size. Atropine reverses excess parasympathetic activity, and should be given every 15 minutes until the signs of excess parasympathetic activity, hypersalivation, excess lachrymation and bronchial secretions have resolved. In severe cases with coma and respiratory failure, mechanical ventilation is necessary.

Atropine blocks the action of acetylcholine on muscarinic receptors at parasympathetic nerve endings but not the action of acetylcholine on nicotinic receptors in the central nervous system and at neuromuscular junctions. Pralidoxime is a drug which reactivates acetyl cholinesterase and is useful if given within 24 hours of poisoning, but is not available in Malawi.

Summary

Organophosphorus poisoning is one of the commonest forms of poisoning responsible for hospital admission in Malawi. The clinical features are very distinctive and should prompt the correct diagnosis even when there is no history of organophosphorus ingestion. Atropine is the antidote which reverses some clinical features, but patients with respiratory depression need mechanical ventilation.

Further reading

Watters DAK, Wilson IH, Leaver RJ, Bagshawe A. Care of the critically ill patient in the Tropics and Sub-Tropics. London: Macmillan, 1991:119-120.

Tafuri J and Roberts J. Organophosphate poisoning. Ann Emerg Med 1987; 16: 193-202.

Answers for Case Report 2

2a. Guillain - Barre syndrome

2b. A history of a previous febrile illness may be found. The paralysis is flaccid motor symmetrical and ascending from the lower limbs. There is no bladder involvement. The CSF protein is elevated.

2c. Poliomyelitis - paralysis is flaccid and motor though asymmetrical and painful. Spinal lesion e.g. burkitts lymphoma - bladder control affected and sensory loss. Spinal TB or epidural abscess - painful swelling over spine - paralysis becomes spastic below the level of the lesion which is fixed. Transverse myelitis - lesion is not ascending (fixed), involves bladder and though initially flaccid will become spastic below the level of the lesion.

2d. The prognosis is generally good, with recovery of function in the majority. Mortality is usually due to respiratory paralysis and mechanical ventilation may be life saving if required.

GUILLAIN-BARRE SYNDROME IN THREE MALAWIAN CHILDREN

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GUILLAIN-BARRE SYNDROME (POST INFECTIVE POLYNEURITIS)

The Guillain Barre syndrome (GBS) is characterised by an acute onset of peripheral and cranial nerve dysfunction, with rapidly progressive symmetric weakness and loss of tendon reflexes. Cranial nerve palsy, oropharyngeal and respiratory paresis may occur. Impairment of sensation in the hands and feet, and autonomic dysfunction are uncommon.

The GBS is the most frequently acquired demyelinating neuropathy. The incidence increases with age, but the disease may occur at any age.

From the evidence available, it appears that GBS represents an interaction of an infectious agent and a cell mediated immune response directed against the myelin producing Schwann cells, the Peripheral myelin or one of the myelin components.

In Malawi, there are many causes of paralysis in children. TB (Spine), Epidural Abscesses, Trauma, Burkitts Lymphoma (of spine), Schistosomiasis, HIV, are some of the examples that lead to spastic paralysis. These may cause flaccid weakness and areflexia initially that rapidly give way to spasticity and hyperreflexia. However poliomyelitis, Guillain-Barre syndrome and cauda equina damage all lead to flaccid paralysis with absent reflexes.

A diagnosis of Guillain-Barre syndrome was reached in the three cases presented largely on the clinical presentations and laboratory results of the CSF. A lack of level of sensory loss on the trunk and no impairment of bladder and rectal sphincters function also differentiate spinal cord lesion from GuillainBarre Syndrome.

Poliomyelitis was the most important differential diagnosis. However, in all the three cases vaccinations were up to date and in polio weakness is usually asymmetrical, and the CSF shows an increase white cell count, primarily lymphocytes, usually with a normal protein.

GBS is often preceded by a mild fever associated with viral respiratory or gastrointestinal tract infections. Some workers have found that an influenza-like infection preceded the appearance of the neurologic symptoms in over 80% of their cases. CMV and EBV are some of the more common initiating agents. In addition prior infections with mycoplasma, Hepatitis A and B, Influenza A and B, Echovirus, Coxsackie and Herpes simplex can be linked to the development of GBS.

Two of our cases did give a history of preceding illness. These febrile illnesses subsided without any medication. This raises a possibility that these illnesses could have been viral and that during that period (April to May 1993), there was an epidemic in some parts of Malawi.