Comment

Phaeochromocytoma presenting as a cardiac or abdominal emergency has been described in adults but is less common in children. All 85 children reviewed by Hume had symptoms for at least two weeks, although in several of the children the condition ran a rapid course and was not diagnosed until sudden death occurred.[4] Minor trauma and surgery are common precipitants of symptoms in phaeochromocytoma, and deliberate massage of the tumour by a physician may provoke an attack.[5]

Our patient was examined by several different doctors, which may have caused sustained release of catecholamines. More probably, however, an appreciable discharge at the time of the initial trauma produced a catecholamine crisis. A phaeochromocytoma might have been suspected if more weight had been given to the initial hypertension and persistent dilated pupils. Although treatment with α and β adrenergic blockade might have reversed the outcome, the myocardial injury caused by the initial release of the catecholamine may have been too advanced at presentation for this to have been of any great benefit.

We thank Mr J A S Dickson and Professor R D G Milner for permission to publish the clinical details of this case. We also thank Miss J Massey for carrying out the catecholamine assays.

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Opiate toxicity after self poisoning with aspirin and codeine

Compound analgesic preparations containing aspirin and codeine have been widely available for many years, but to our knowledge there is no record of serious toxicity in adults from any component other than salicylate. We report on two patients who developed severe opiate toxicity after overdoses of aspirin and codeine tablets *BP* (aspirin 300 mg, codeine 8 mg).

Case reports

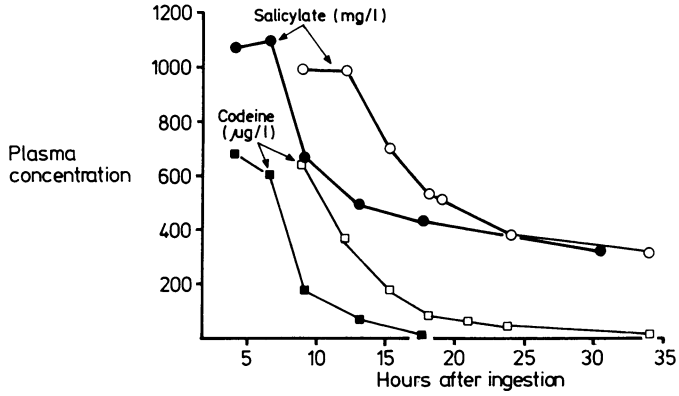
CASE 1

A 34 year old woman was admitted four hours after having ingested 80 tablets (24 g aspirin, 640 mg codeine) with 100 mg metoclopramide and alcohol. On examination she was unresponsive to painful stimuli, had pinpoint pupils, and was sweating. Her pulse rate was 130/min and blood pressure 115/80 mm Hg, and respiration was shallow at a rate of 38/min. Arterial blood gas analysis showed a mixed metabolic and respiratory acidosis (pH 7·24, carbon dioxide tension 5·9 kPa (44 mm Hg), oxygen tension 10·2 kPa (77 mm Hg), and bicarbonate 18·9 mmol (mEq)/l). After administration of naloxone (0·8 mg intravenously) her conscious level improved such that she responded to verbal commands, the pupils dilated, and the respiratory component of the acidosis was reversed within 30 minutes (pH 7·4, carbon dioxide tension 2·8 kPa (21 mm Hg), oxygen tension 14·1 kPa (106 mm Hg), and bicarbonate 14 mmol/l. A further 1·2 mg naloxone was given to prevent deterioration of conscious level.

Plasma salicylate and codeine concentrations on admission were 1083 mg/l and 590 μg/l respectively. Plasma urea, sodium, potassium, and glucose concentrations were normal, and the blood alcohol concentration was 47 mmol/l (2·15 g/l). Forced alkaline diuresis with 8 litres of a solution of isotonic saline and dextrose containing 37·5 mmol (3·2 g) sodium bicarbonate followed by 0·5 litre 1·26% sodium bicarbonate caused the plasma salicylate concentration to fall to 508 mg/l (figure). She thereafter made an uneventful recovery and was discharged home after psychiatric review.

CASE 2

A 30 year old woman was admitted after having ingested 100 tablets (30 g aspirin, 800 mg codeine) and an unknown amount of diazepam. She was drowsy but responded to verbal commands, had pinpoint pupils, and was sweating and hyperventilating with a pulse rate of 115/min and blood pressure of 120/80 mm Hg. Arterial blood gas analysis showed a mixed respiratory alkalosis and metabolic acidosis pH 7·4, carbon dioxide tension 3·1 kPa (23 mm Hg), oxygen tension 13·6 kPa (102 mm Hg), and bicarbonate 15 mmol/l. Immediately after administration of naloxone (0·8 mg intravenously) the pupils dilated and she became fully conscious. Plasma salicylate and codeine concentrations were 1006 mg/l and 650 μg/l respectively. Plasma urea, sodium, and potassium concentrations were normal. Diazepam and nordiazepam concentrations were 0·38 mg/l and 0·84 mg/l respectively. Forced alkaline diuresis with 6 litres of a solution similar to that used in case 1 with 1·5 litres 1·26% sodium bicarbonate was carried out; 21 hours after her admission the plasma salicylate concentration measured 462 mg/l (figure).



Salicylate and codeine concentrations in cases 1 (black circles and squares) and 2 (open circles and squares).

On three occasions during forced alkaline diuresis her conscious level deteriorated such that she responded only to painful stimuli and had pinpoint pupils. On each occasion these signs were reversed by naloxone, a further 4·0 mg in total being given intravenously.

Comment

The amount of codeine in combination analgesics available without prescription is generally considered to be toxicologically unimportant in the context of overdosage.[1] In both our cases, however, severe salicylate poisoning was associated with appreciable depression of consciousness and miosis, which were rapidly reversed by naloxone. Furthermore, if the central depression had been attributed solely to intoxication with salicylate haemodialysis might have been inappropriately begun because of the poor prognosis of coma induced by salicylate.[2]

The initial plasma codeine concentrations were some five times the peak concentrations reached one hour after a 60 mg oral dose in volunteers[3] but considerably less than those reported after fatal overdose complicating abuse of narcotics (1400-4800 μg/l).[4,5]

Appreciable opiate toxicity may therefore complicate large overdoses of proprietary preparations containing salicylate and codeine that are widely available over the counter. Depression of consciousness after ingestion of these combination analgesics should not be attributed to salicylate acidaemia without a trial of naloxone in adequate dosage.

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