

CASE REPORTS

Fatal outcome from administration of a salt emetic

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Summary

A case is described of the death of a young female patient following the administration of a salt emetic after a relatively minor overdose of a proprietary analgesic containing aspirin. It is suggested that death occurred as a direct consequence of the salt ingestion and that the dangers of this method of inducing emesis should be more widely appreciated.

Introduction

The first recorded case of death from salt poisoning was in the London Courier October 1 1828. 'A man proposed to some of his comrades to sup a pound of common salt in a pint of ale'. Soon after accomplishing his feat 'he was seized with all the symptoms of irritant poisoning and died within twenty-four hours'.

Accidental poisoning with sodium chloride in infants and young children is a well known entity. Two disasters have been reported in which salt was used instead of sugar in preparing infant feeds. In these two disasters twelve children died (Elton, Elton and Nazareno, 1963; Finberg, Kiley and Luttrell, 1963). There have been several reports of death in infants due to the administration of saline emetics and in particular DeGenaro and Nyhan (1971) emphasized the danger of this form of emesis. A further warning was given by Robertson (1971).

There have been relatively few reports concerning the dangers of salt emetics when administered to adults. The case described by Ward (1963) was of a 74-year-old man who died from pulmonary oedema as a result of administration of a saline emetic after an overdose of perphenazine and imipramine.

Laurence and Hopkins (1969) described a patient in whom the mode of death had been similar to that in the present instance. Their patient was a 35-year-old woman who had taken an overdose of thiori-

dazine. An emetic had been given containing 3000 mEq of sodium chloride and death occurred in coma 8 days after admission. Robertson (1971) mentioned the death of a 23-year-old woman consequent to the administration of a salt emetic after taking an overdose of chlordiazepoxide. Three patients dying after the intra-uterine injection of hypertonic saline as an abortifacient have been described (Cameron and Dayan, 1966; Dayan, Cameron and Phillipp, 1967).

Despite these reports, household salt continues to be used as an emetic in the first aid treatment of overdose.

Case report

A 26-year-old housewife who had, for the past 4 months, been receiving voluntary inpatient treatment for depression at a local psychiatric unit took an overdose of approximately 40 Anadin tablets. Each tablet contains 65 mg salicylamide, 325 mg acetylsalicylic acid, 14.8 mg caffeine and 1 mg quinine sulphate. Her depressive illness had begun 2 years previously following the birth of her second child. Her current drug therapy consisted of trimipramine, clomipramine and nitrazepam. There was no past history of significant organic illness.

On discovery of her overdose a saline emetic was administered using six tumblerfuls of water each containing a dessertspoonful of salt. This is equivalent to 150 g sodium chloride or 2600 mEq sodium. The patient began vomiting on the third glass but did not bring up any tablets. Further glasses of saline were ordered and gastric lavage was performed. She was then transferred to an acute medical unit.

On arrival an estimated 3 hr after the overdose and 2 hr after administration of the saline, the patient was drowsy but able to give a history. She complained of tinnitus and parasthesiae. Myoclonic jerks of the arms and twitching of the eyelids were noted. Muscle tone was generally increased and there was hyperreflexia. She was hyperventilating at 48 breaths/min, had a tachycardia of 120 beats/min and a blood

pressure of 140/100 mmHg. Her abdomen was distended and bowel sounds were absent.

Investigations at this time were as follows:

| | |
|-------------------------|----------------|
| Plasma sodium | 172 mEq/l |
| potassium | 4.6 mEq/l |
| chloride | 155 mEq/l |
| bicarbonate | 17 mEq/l |
| urea | 24 mg/100 ml |
| Plasma salicylate level | 17.1 mg/100 ml |
| Arterial pH | 7.26 |
| Standard bicarbonate | 16.5 mEq/l |
| PaO ₂ | 75 mmHg |
| Paco ₂ | 35 mmHg. |

Plain X-ray of the abdomen showed distended loops of small bowel with multiple fluid levels. Chest X-ray was normal.

Initial intravenous therapy consisted of 500 ml normal saline given over 2 hr and then infusion of one litre 5% dextrose over approximately 45 min. Soon afterwards 2% glycosuria was noted and the blood glucose was found to be 420 mg/100 ml. She was given 50 units of soluble insulin intramuscularly and 500 ml of 4.2% sodium bicarbonate solution. Approximately 330 mEq sodium had therefore been given intravenously. When the diagnosis of salt poisoning became clear, intravenous therapy was continued with 5% dextrose solution at a rate to maintain the central venous pressure within normal limits.

Four hours after admission she was barely responding to painful stimuli and then developed a series of grand mal convulsions and her temperature began to rise. Intravenous diazepam failed to control her convulsions and it was necessary to paralyse her and establish artificial ventilation.

Peritoneal dialysis was commenced using 5% dextrose solution with 4 mEq potassium chloride per litre. Twenty per cent mannitol solution, frusemide and dexamethasone were also administered intravenously in an attempt to reduce cerebral oedema.

Fourteen hours after admission circulatory collapse occurred associated with the onset of anuria. Her previous urine output had ranged from 120 to 400 ml/hr totalling 5 l. Her temperature had risen to 41.5°C in spite of measures to cool her, including application of damp sheets. At this time her plasma electrolytes were:

| | |
|-------------|--------------|
| Sodium | 162 mEq/l |
| Potassium | 3.6 mEq/l |
| Chloride | 140 mEq/l |
| Bicarbonate | 19 mEq/l |
| Urea | 46 mg/100 ml |

and with a further 6 hr dialysis were:

| | |
|-------------|---------------|
| Sodium | 152 mEq/l |
| Potassium | 4.0 mEq/l |
| Chloride | 119 mEq/l |
| Bicarbonate | 21 mEq/l |
| Urea | 63 mg/100 ml. |

An electro-encephalogram 24 hr after the initial event recorded no cerebral activity at all. The patient died 37 hr after receiving the emetic.

Biochemical monitoring during her admission showed a falling plasma salicylate level, correction of the metabolic acidosis, satisfactory arterial blood gas tensions and blood glucose levels.

At necropsy the brain was swollen and congested, weighing 1450 g and there was patchy haemorrhage under the pia mater. There was no visible cerebral necrosis but in the right caudate nucleus a recent globular haemorrhage 1.2 cm in diameter was present.

Histological examination of the brain failed to show the frank neuronal necrosis reported by Cameron and Dayan (1966) and sections of Ammon's horn were normal. The only abnormalities apart from the recent haemorrhage were signs of early reactive changes in the astrocytes of the white matter.

The thoracic organs were normal and the lungs were not unduly oedematous. The whole of the gastric mucosa was thickened and haemorrhagic and there were a few small areas of sub-mucosal haemorrhage in the jejunum and ileum. The large intestine was dilated and contained a large amount of very watery faecal fluid. The cortices of both kidneys were slightly swollen but the kidneys were normal histologically.

Discussion

The diagnosis of salt poisoning was suspected because of the high plasma sodium and a plasma salicylate level inconsistent with severe salicylism. The diagnosis was confirmed when the history of salt administration was ascertained.

The symptoms of salt poisoning have been well documented. The main clinical manifestations are due to a cerebral disorder with convulsions and hyperpyrexia, thought to be caused by osmolar effects on the central nervous system. Metabolic acidosis is another feature and the clinical presentation of this case illustrates the similarity between salicylism and salt poisoning. It is possible in view of the common use of salt emetics that occasional deaths attributed to aspirin poisoning have in fact been examples of salt poisoning.

Treatment of salt poisoning is difficult. Peritoneal dialysis using 7% or 8% dextrose solution is recommended as the most effective measure for removing the salt (Finberg *et al.*, 1963), but correction of the metabolic abnormality by this means is slow. The deterioration in the patient's condition, however, is rapid; our patient, for example, appeared to suffer severe brain damage some 6 hr after the ingestion of the salt. The use of haemodialysis has not been described in this condition, but it would seem on theoretical grounds that it might offer no advantage

over peritoneal dialysis. Correction of the metabolic state, control of convulsions and maintenance of oxygenation and hydration appear to be the most important aspects of therapy.

Poisoning by salt emetics is particularly regrettable as other methods of inducing vomiting exist. Syrup of ipecacuanha has been used extensively in paediatric practice with no reported adverse effect and many patients can be made to vomit by pharyngeal stimulation. Salt usually induces vomiting even in small quantities but the amounts and dilutions recommended in first aid books vary widely and the recipes are often vague. In our view, either the recommendation of the use of salt emetics should be abandoned, or very strict warning given of the danger of overdose.

Acknowledgment

We are grateful to Dr A. C. Hunt for supplying details of the necropsy and for his helpful advice, and to Dr G. R. Steed for allowing us to report a patient under his care.

References

- CAMERON, J.M. & DAYAN, A.D. (1966) Association of brain damage with therapeutic abortion induced by amniotic-fluid-replacement. Report of two cases. *British Medical Journal*, **1**, 1010.
- DAYAN, A.D., CAMERON, M.M. & PHILLIPP, E. (1967) Fatal brain damage associated with therapeutic abortion induced by amniocentesis. Report of one case. *Medicine, Science and Law*, **7**, 70.
- DEGENARO, F. & NYHAN, W.L. (1971) Salt—a dangerous 'antidote'. *Journal of Pediatrics*, **78**, 1048.
- ELTON, N.W., ELTON, W.J. & NAZARENO, J.P. (1963) Pathology of acute salt poisoning in infants. *American Journal of Clinical Pathology*, **39**, 252.
- FINBERG, L., KILEY, J. & LUTTRELL, C.N. (1963) Mass accidental salt poisoning in infancy. *Journal of the American Medical Association*, **184**, 187.
- LAURENCE, B.H. & HOPKINS, B.E. (1969) Hypernatraemia following a saline emetic. *Medical Journal of Australia*, **1**, 1301.
- ROBERTSON, W.O. (1971) A further warning on the use of salt as an emetic agent. *Journal of Pediatrics*, **79**, 877.
- WARD, D.J. (1963) Fatal hypernatraemia after a saline emetic. *British Medical Journal*, **2**, 432.

Postgraduate Medical Journal (August 1974) **50**, 515–517.

Acute ethanol poisoning treated by haemodialysis

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Summary

A non-alcoholic patient with severe ethanol poisoning who was deteriorating clinically despite adequate supportive therapy was successfully treated by haemodialysis.

Case history

A 23-year-old man was admitted at 7 p.m. Since the morning he had drunk 10 pints of beer. He was then 'dared' to drink half a bottle of gin, which he did quickly. At 5 p.m. he suddenly fell unconscious to the floor.

On examination. There were superficial abrasions over the anterior chest wall and right temple sustained when he fell. Respiration rate was 20/min with adequate ventilation of both lungs, pulse 132, B.P. 140/80 mmHg, heart sounds normal. He was unrousable and unresponsive to painful stimuli. Limb reflexes were brisk and equal. Pupils were equal

and reacted to light. He was intubated with a cuffed endotracheal tube and his stomach washed out.

Biochemical tests on admission showed blood ethanol 465 mg%, blood sugar 93 mg%, plasma urea and electrolytes normal. Arterial pH 7.27, actual PCO₂ 35 mmHg, base excess 10 mEq/l, buffer base 38 mEq/l, standard bicarbonate 17 mEq/l, actual bicarbonate 15 mEq/l, indicating a moderate metabolic acidosis.

Over the next 2 hr his condition deteriorated. The pupils became widely dilated and unresponsive to light. The limb reflexes became unobtainable. Respiration slowed and became more shallow. Blood pressure fell to 90/60 mmHg, but peripheral perfusion remained satisfactory. This depression of the central nervous system could not be attributed to hypoventilation as at this time an arterial Po₂ was 91 mmHg, and the PCO₂ was 25 mmHg.

Because of this clinical deterioration and his