Medical Memoranda

Fatal Hypernatraemia after a Saline Emetic

Hypertonic saline can be used to provoke vomiting in cases of acute poisoning in attempted suicide.[1] In certain circumstances this may lead to dangerous side-effects as illustrated by the following report.

CASE REPORT

A 74-year-old man was admitted to hospital, having taken 60 mg. of perphenazine ("fentazin") and 750 mg. of imipramine ("tofranil"). This quantity had been intended to represent a ten-day supply. He had been prescribed these tablets by his general practitioner the previous day for what was considered to be mild depression. Apart from having had a transvesical prostatectomy for benign prostatic enlargement in 1957 there was no relevant history of organic disease.

On examination he appeared to be well nourished. He could respond to simple questions and obey simple commands, and could hold a tumbler and drink on request. Agitation and a coarse Parkinsonian tremor of the hands were conspicuous.

The pulse rate was 84/min., regular, and blood-pressure 115/80 mm. Hg (a recording the previous day by his general practitioner gave 170/90 mm. Hg). Crepitations were present at both lung bases. The pupils reacted sluggishly to direct light; there was no neck stiffness, and Kernig's sign was negative. The patient was able to move all limbs equally. Tendon jerks in upper and lower limbs were brisk. Clonus was absent but both plantar responses were extensor.

An attempt to pass a stomach-tube was unsuccessful because of resistance by the patient. To provoke vomiting a tablespoonful of NaCl (30 g.) in 300 ml. of warm water was given and the patient drank this but no vomiting occurred. The dose was repeated, and only when he lay flat did he vomit, approximately 150 ml. being recovered. There was no retching. The vomit was discarded.

An intravenous infusion of NaCl 0.18% in dextrose 4.3% for the purpose of blood lavage,[2] and a course of penicillin, were begun. Urethral catheterization was required 10 hours after admission for acute retention of urine. The urine output for the first 24 hours was 2,100 ml. The patient's condition was unchanged for approximately 20 hours. From this time his condition deteriorated. A tachycardia with frequent ventricular ectopic beats accompanied signs of increasing pulmonary congestion, raised jugulo-venous pressure, and the development of sacral oedema. The systolic blood-pressure remained around 115 mm. Hg until shortly before death. Serial biochemical estimations are shown in the accompanying Table.

	Na (mEq/l.)	Cl (mEq/l.)	K (mEq/l.)	Bic. (mEq/l.)	Urea/mg. 100 ml.
On admission	143	120	4·3	26	37
24 hours after admission ..	174	152	3·7	25	45
27 hours after admission ..	172	152	3·7	25	45

The patient died 32 hours after admission.

The post-mortem findings confirmed the clinical diagnosis of pulmonary oedema. There were lesser degrees of oedema in other organs.

COMMENT

The cause of death in this patient was pulmonary oedema secondary to hypernatraemia, which in turn was caused by the absorption of a large quantity of hypertonic saline used in an attempt to produce vomiting. This attempt failed because the patient had taken an overdose of perphenazine, a chlorpromazine derivative, which is a potent anti-emetic. The Parkinsonian state[3] and hypotension[4] are well-recognized side-effects of phenothiazine drugs.

Imipramine has no anti-emetic effect and may even cause nausea and vomiting as a side-effect.[5] It can cause a Parkinsonian tremor.[6]

The anti-emetic effect of chlorpromazine is well documented[7] and according to Wang[8] perphenazine is 16.6 times more potent as an anti-emetic than the former drug and is more widespread in its anti-emetic action.

The rapid increase in levels of serum sodium and chloride could not be explained by the 10.8 g. of NaCl present in 6 litres of 4.3% dextrose and 0.18% NaCl which the patient received intravenously.

The total intake of NaCl was approximately 70.8 g. (60 g. orally and 10.8 g. intravenously). This is equal to 1,180 mEq Na.

Assuming a total body water of 50 litres, the expected rise in serum sodium would be 23.6 mEq. The observed rise, as the Table shows, was 30 mEq. In view of the possible loss of sodium in the vomit it is suggested that a complex interplay of osmotic forces of sodium in the gut and in the circulation must account for the observed rise in serum sodium.

Hypertonic saline—1 oz. (31 g.) of NaCl to 8 fl. oz. (227 ml.)—is listed in "Drugs of Special Value" section in the H.M.S.O. (1962) publication.[1] That this treatment carries a considerable risk where the new anti-emetic drugs are concerned is illustrated by this case report.

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Foetal Death after Pethidine and Promazine

Encouraged by reports on the value of promazine ("sparine") in obstetrics (Donald, 1959; MacVicar and Murray, 1960; etc.), we have made increasing use of this drug in the maternity department at St. George's Hospital over the past two years. During the five months preceding the admission of the case described below, 160 patients had received one or more injections of promazine 50 mg. with pethidine 150 mg. intramuscularly during the first stage of labour. No ill effects to any mother or child had been observed, and it was our general impression that labour was considerably less of an ordeal for a patient under the influence of this combination of drugs than with pethidine alone. Apart from intramuscular sedation in labour we have also successfully used an intravenous mixture of pethidine, promazine, and levallorphan on many

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