A case of self-poisoning with Carbrital

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Case Report
A 67 year-old woman was admitted one-and-a-half hours after taking 50 capsules of Carbrital, each containing pentobarbitone sodium 100 mg and carbromal 250 mg.

On admission she was comatose and areflexic except for the preservation of the cough reflex. The pulse was 72/minute, sinus rhythm and blood pressure 90/50 mmHg. There were no signs of cardiac or peripheral circulatory failure at this time.

In the accident and emergency department gastric lavage was performed with a wide-bore tube. An intravenous infusion was set up with 1/6 molar sodium lactate solution and frusemide 20 mg was given intravenously. The patient was then transferred to the Medical Intensive Care Unit for further treatment.

First 24 hr
For the first 24 hr after admission an alkaline diuresis was produced using alternate 540 ml bottles of 1/6 molar sodium lactate solution and 5% dextrose solution. Potassium chloride 0-75 g. (10 mEq.) was added to alternate bottles and the diuresis was maintained by intermittent intravenous frusemide, 380 mg being administered in 24 hr. In addition the patient was given hydrocortisone 100 mg intramuscularly 4-hourly and 35% oxygen by Ventimask.

A chest X-ray taken during this period showed a vaguely outlined radio-opaque mass in the left hypochondrium. It was suggested that this could possibly be the ingested capsules; subsequent events lent support to this suggestion.

Throughout this period the blood pressure never rose above 95/60 mmHg and the rectal temperature was consistently below 94°F at one point. The patient remained deeply comatose but the cough reflex was preserved.

Second 24 hr
It was decided to abandon the alkaline diuresis and the intravenous infusion was continued at a slower rate with alternating bottles of normal saline and 5% dextrose with similar quantities of potassium chloride in alternate bottles.

After 40 hr spontaneous respiration ceased and the patient developed atrial fibrillation. An endotracheal tube was passed and respirations were maintained using an East Radcliffe Positive Pressure Ventilator. Following this the patient reverted to sinus rhythm spontaneously.

46 hr after admission the patient suffered a cardiac arrest but was restored to sinus rhythm after a brief period of external cardiac massage and one DC countershock of 100 Joules.

After 48 hr
Following this episode of cardiac arrest no further cardiac dysrhythmias were noted and the level of consciousness slowly rose. At 74 hr the endotracheal tube was removed and spontaneous respiration was restored. 90 hr after admission the patient was sufficiently conscious to answer simple questions coherently and was then transferred to a general medical ward. The patient was referred to a Consultant Psychiatrist and was discharged 24 days after admission to receive outpatient treatment for depression.

Findings
In the first 24-hr 11,200 ml of fluid were administered by intravenous infusion and 11 litres of urine were produced. The pH of each 500 ml collected was shown to be greater than 8.

During this period the plasma potassium was consistently low reaching 2.3 mEq/l at one point. The plasma bicarbonate (auto-analyser) was raised...
to 58 mEq/l and remained elevated for the first 48 hr. Throughout this period the range of the plasma pH was 7-41–7-51 and for the Paco₂, 77–93 mmHg.

At the time of the cardiac arrest (46 hr) the plasma potassium was 2·6 mEq/l and the plasma levels of pentobarbitone and bromide were 2·75 mg/100 ml and 6·7 mg/100 ml respectively.

In the second 24-hr 4070 ml of fluid were administered by intravenous infusion and 2475 ml of urine were collected.

At the time the patient regained consciousness the plasma pentobarbitone level was 0·8 mg/100 ml.

A total of 17 litres of fluid was administered by intravenous infusion and 16 litres of urine were collected. In 11 litres of the urine 104·4 mg of pentobarbitone were recovered and the total in 17 litres of urine was unlikely to be in excess of 153 mg.

Fig. 1 shows the chest X-ray taken on admission and the radio-opaque mass can be seen in the left hypochondrium. This mass was visible on serial X-rays during the first 72-hr after admission but 90 hr after admission it was no longer visible on X-ray.

To test the suggestion that this mass consisted of partially digested Carbrital capsules, a mass of these were incubated with dilute hydrochloric acid at 38°C and this mass was then X-rayed. This X-ray is shown in Fig. 2. This supports the suggested explanation of the opacity on the abdominal X-rays.

**FIG. 1.** Chest X-ray on admission showing an opacity in the left hypochondrium.

**FIG. 2.** X-ray of a mass of Carbrital capsules digested with N HCl for 2 hr.

**Discussion**

There are several interesting features about this case. Although gastric lavage using a wide-bore tube was carried out within 2 hr of ingestion of the capsules, it failed to remove the large bulk of them. As a result the patient continued to absorb the drugs over a prolonged period.

With the collection of 11 litres of urine in the first 24-hr there was no doubt that the diuresis was massive and with a urine pH of 8 it was certainly alkaline, but the quantity of pentobarbitone eliminated by this technique was only 104·4 mg, which is equivalent to the content of one Carbrital capsule. In spite of intravenous potassium supplements the technique produced a severe hypokalaemic alkalosis in the presence of respiratory carbon dioxide retention.

The respiratory arrest occurred when the plasma content of each drug, if present alone, was sufficient to produce coma.
Case reports

**Clostridium septicum** infection of the thyroid gland

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A case of infection of the thyroid gland by *Clostridium septicum* is described, which is only the fourth case of gas-forming suppurative thyroiditis to be reported and the first in which the causative organism has been identified.

The case also illustrates a most unusual presentation of carcinoma of the colon.

Case report

A 58-year-old man who worked as a cold store attendant was admitted to hospital with a painful inflamed swelling of his neck. The swelling was in the region of the thyroid gland and had developed in 12 hr with dysphagia and positional stridor. Rigors had occurred at the onset of the illness. His thyroid gland had been enlarged for 20 years and a firm enlargement of the right lobe had been noted on admission to hospital 2 years earlier. The patient looked ill and pale, and had a temperature of 103°F. He was clinically euthyroid. His neck was swollen, inflamed and tender due to enlargement of the thyroid gland, particularly on the right side. There

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References


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