Phenolphthalein Poisoning

A Case Report

N. BUCHANAN, R. D. CANE, R. GLANTZ, J. A. HUNT

SUMMARY

A case of severe poisoning due to the ingestion of phenolphthalein in the form of a patent medication, is described.

Phenolphthalein is a primary diphenylmethane cathartic, which acts mainly on the colon, about 6 hours after ingestion. Approximately 15% of an oral dose is absorbed, the remainder being excreted in the stools. It is a relatively non-toxic substance, but its administration may lead to derangements of fluid and electrolyte balance and fixed drug eruptions. Two deaths have been reported in association with phenolphthalein ingestion. We wish to report observations made in a 35-year-old Black man who consumed two packets of Brooklax, a chocolate laxative, which was equivalent to 2 g of phenolphthalein.

CASE REPORT

The patient was admitted to a surgical unit on 28 October 1975 with severe abdominal pain, and gave a history of having had diarrhoea and vomiting for a week. He denied enemata, self-medication or treatment by a witchdoctor. Physical examination was unremarkable. An intravenous pyelogram was normal, the serum amylase was 8 Street-Close units, and a peritoneal tap was negative. The next morning the patient's condition had deteriorated, and he was clinically acidotic. Blood gas analysis revealed a pH of 7.1 and a base deficit of 23 mEq/l (Fig. 1). He was given 600 mEq of sodium bicarbonate intravenously, and was transferred to the Intensive Care Unit.

On admission to the Intensive Care Unit the patient was shocked and hypothermic. An isoprenaline drip was started, and a further 100 mEq of sodium bicarbonate was given intravenously. A urinary catheter was passed and 30 ml of blood-stained urine was obtained. Furosemide (40 mg) was given intravenously, with no effect. Investigations at this time showed a serum creatinine concentration of 1.7 mg/100 ml, urea 37 mg/100 ml, sodium 131 mEq/l, potassium 5 mEq/l, calcium 4.3 mEq/l, amylase 6 Street-Close units. A chest radiograph indicated pulmonary oedema and an electrocardiograph showed striking junctional (J) waves (Fig. 2).

Due to the lack of an adequate explanation for the patient's condition, the possibility of a toxin was considered. With this possibility taken into account, as well as the patient's oliguria and severe acidosis, peritoneal dialysis was started. On insertion of the catheter, about 20 ml of blood-stained transudate fluid was obtained (amylase 32 S-C units, protein 1.5 g/100 ml). Dialysis was performed for approximately 24 hours, using 2-litre dialyses, 1 litre being 1.5% Dianeal and 1 litre PlasmaLyte B. Later that day, the patient admitted to taking two slabs of Brooklax, in an attempt to treat his diarrhoea and vomiting.

The patient's biochemical and clinical course is depicted in Fig. 1. Features of interest included hypothermia, hypo-
Discussion

Fig. 2. A sample from the patient's ECG on admission showing a typical J wave.

Dialysis was discontinued after 24 hours, as was the isoprenaline. The patient subsequently made a full recovery, with a return to normal renal function.

REFERENCES


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