deficit; surprisingly, this was only clinically apparent in the skin overlying the angioliomata, presumably secondary to extravasation from infarcted capillaries.

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Fatal self-poisoning with Dettol

DAVID MEEK
M.B., Ch.B.

ROGER GABRIEL
M.Sc., M.B. M.R.C.P.

D. M. PIERCY
M.B., M.R.C.Path

Departments of Medicine and Pathology, Royal Infirmary, Hull

DETTOL is a common household disinfectant. A fatal case of self-poisoning with this agent is reported.

Case history

A 66-year-old housewife was admitted 90 min after ingesting, it was said, about 300 ml of Dettol. She was unconscious with flaccid areflexic limbs but responded to deep and painful stimuli. The pupils were equal and constricted, responding sluggishly to light. The blood pressure was 90/0 mmHg, pulse 64 beats/min and respirations 18/min. The cardiovascular and respiratory systems were otherwise normal.

The initial plasma biochemistry showed a urea of 9.5 mmol/l, sodium 136 mmol/l, bicarbonate 9 mmol/l, chloride 100 mmol/l, SGOT 58 u./l (normal range 8–22) and the SHBD 620 u./l (normal range 90–190). A urine sample obtained by catheterization was of beetroot colour and contained blood, sugar and ketone bodies. Red blood cells were seen on microscopy. The urine sodium concentration was 70 mmol/l.

Six and a half hours after taking the poison the conscious level lightened and the patient became cerebrally irritable with hypertonic limbs. The pupils were normal, equal in size and responded to

light. The blood pressure had risen to 130/60 mmHg. Two hours later she was fully conscious although drowsy. An irregular bradycardia of 50 beats/min developed and the blood pressure fell to 100/0 mmHg.

The following morning (21 hr after ingestion of Dettol) the patient was confused but there was no evidence of any neurological deficit. Signs of pulmonary oedema were present. An ECG showed a sinus bradycardia. The blood urea was 13 mmol/l and the plasma electrolytes normal. In order to exclude the possibility of poisoning from other sources, plasma and urine samples were examined for a wide variety of drugs which are commonly encountered in overdose (Berry and Grove, 1973; Flanagan and Withers, 1972), but none was detected.

Since catheterization, the patient had passed only 40 ml of urine: haematuria, glycosuria and ketonuria were still present and the oliguria was unresponsive to intravenous frusemide. In view of the oliguria, chest signs and rapidly rising blood urea, peritoneal dialysis was commenced. Despite a technically successful dialysis 37 hr after taking the Dettol the patient deteriorated, with increasing acidosis, tachypnoea, tachycardia and hypotension and died within 1 hr.

Post-mortem and histology findings

Isolated areas of corrosive staining were present on the lips and chin, mouth, oesophagus, trachea

Correspondence: Dr Roger Gabriel, Renal Unit, St Mary's Hospital, London W2.

and main bronchi. Patchy hyperaemia and erosions were present in the stomach which contained 'coffee-ground' material. The lungs showed evidence of pulmonary oedema and bronchopneumonia.

Histology of the stomach showed autolysis of mucosal epithelium. The liver showed no necrosis, macroscopically or microscopically. There was some renal cortical thinning and the glomeruli were normal apart from a few which were hyalinized. Many of the tubular epithelial cells contained granular pigments. Brown-tinted hyaline casts were present in the tubular lumina mainly in the loops of Henle. Attempts were made to demonstrate the presence of phenolic metabolites in the tubular casts. The results were equivocal.

Discussion

Dettol contains 4.8% parachlorometaxylene (PCMX), isopropyl alcohol, essential pine oils, castor oil, soap, and burnt sugar. There is an emetic action and perhaps this explains the absence of reported cases of poisoning with Dettol in adults.

PCMX has a formula similar to that of the other phenolic disinfectants—cresols, xylenols and carbolic acid. The clinical course of this patient—unconsciousness followed by a spontaneous recovery of consciousness with later coma and death—is similar to that seen in poisoning with cresols (Cooper, 1958), xylenols (Graham, 1962) and carbolic acid (Turtle and Dolan, 1922). Presumably unconsciousness associated with phenolic poisoning results from the solubility of phenols in brain lipids.

No explanation could be found for renal failure following phenolic poisoning but it is highly probable that these compounds are nephrotoxic and in the case reported here periods of hypotension occurred which may have contributed to the renal functional impairment. The oliguria and haematuria of this patient are similar to those following carbolic acid poisoning as described by Turtle and Dolan (1922). The renal morphology of Turtle and Dolan's patient was interpreted as 'acute haemorrhagic nephritis' while the histology in the present patient was normal. This difference may represent increased understanding of renal morphology. Lack of abnormality to light microscopy of the glomeruli from a patient who has developed acute renal failure is not unusual (Dunhill, 1974), although abnormalities of the tubules are more frequently present (Olsen, 1967).

The isopropyl alcohol content of the Dettol probably has an additive effect to the toxicity of PCMX. Isopropyl alcohol is twice as toxic as ethanol because it has a slow rate of metabolism (Williams, 1959). This alcohol is largely oxidized to acetone and excreted in the urine. Ketonuria was

present in the patient. The pre-dialysis isopropyl alcohol level was 232 mg/100 ml, and it has previously been found in fatal cases in concentrations up to 440 mg/100 ml (Adelson, 1962; King, Bradley and Shires, 1970). There is, however, no correlation between plasma levels and mortality (King *et al.*, 1970). It has been shown (Freireich *et al.*, 1967; King *et al.*, 1970) that isopropyl alcohol is removed by haemodialysis and presumably also by peritoneal dialysis. The patient in this case received about 9 hr of peritoneal dialysis before she lost consciousness for the second time and it therefore seems unlikely that isopropyl alcohol contributed greatly to her death. Juncos and Taguchi (1968) reported elevated SHBD and SGOT following a non-fatal poisoning with isopropyl alcohol; their patient subsequently developed a myopathy. The elevated enzymes in that patient parallel the observations in the present case.

The third toxic component of Dettol is the essential pine oil. These oils are comprised of tertiary and secondary terpene alcohols. Poisoning with essential pine oils may be fatal owing to respiratory failure secondary to depression of the central nervous system (Gornel and Goldman, 1968). These authors reported a non-fatal case of renal failure following poisoning with terpene alcohol which was used as an abortifacient. This patient also had an elevated SGOT in the early phase of her illness.

Dettol thus contains three poisonous constituents, PCMX, isopropyl alcohol and essential pine oils. Much of the isopropyl alcohol was probably removed so it appears that the death of the patient was more likely due to PCMX and essential pine oil, although the amount of terpene alcohol in Dettol is undisclosed. Serious self-poisoning with household disinfectants is uncommon, as in Great Britain, perhaps surprisingly, Martindale (1972) lists twenty-eight similar preparations which are freely available.

From the experience with this patient it is suggested that the following steps should be taken in any further cases:

- (1) The patient should be given oral liquid paraffin because PCMX is soluble in this oil (Graham, 1962) and gut absorption should be diminished thereby.
- (2) A forced alkaline diuresis. PCMX is insoluble in acid urine and moderately soluble in alkaline urine (Zondek, 1942a).
- (3) Exchange transfusion. Zondek (1942b) has shown that PCMX is concentrated in erythrocytes. In the present patient the plasma PCMX was 11 mg/100 ml but after hydrolysis of the red cells the concentration was 490 mg/100 ml. Exchange transfusion, therefore, should remove a large quantity of the ingested PCMX.

(4) Early dialysis to remove isopropyl alcohol (King *et al.*, 1970).

(5) Intermittent positive pressure respiration should be considered to support the patient while other measures are being undertaken.

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Spontaneous rupture of the abdominal aorta

T. G. WILLIAMS*
M.B., B.Chir., F.R.C.S.

The Ipswich Group of Hospitals

Summary

Fatal spontaneous rupture of the lower abdominal aorta in a previously healthy 61-year-old woman is reported; the possibility that she had the Ehlers-Danlos syndrome is discussed.

Introduction

Rupture of the abdominal aorta has not hitherto been reported in the absence of trauma, hypertension, overt aortic disease or prior clinical evidence of a connective tissue disorder. None of these features was present in the following case.

* Present address: 15 Thorley Gardens, Pyrford Road, Pyrford, Surrey GU22 8UL.

Case report

A 61-year-old woman, previously normotensive and in excellent physical health although treated with amitriptyline for a year because of depression, was admitted urgently with a 12-hr history of epigastric pain radiating to the back. She had never previously been in hospital and there was no relevant family history. She had always bruised easily, as when her dog jumped at her or someone held her arm. On two occasions her gums had to be stitched because of persistent bleeding after tooth extraction. She had a normal 30-year-old son and two subsequent miscarriages, all delivered uneventfully at home.