Formic acid poisoning

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Summary

Forty-five cases of formic acid poisoning are reviewed. The metabolic and pathological consequences of such overdoses are considered. The consequences are discussed in relation to the amount of the acid ingested. The problems likely to be encountered in the management of such patients are highlighted.

Introduction

Formic acid is a strong acid which is available for domestic use as a constituent of proprietary descaling agents and stain-removing fluids. It is therefore a potential source of both accidental and deliberate poisoning.

The outcome is reported of 45 patients who ingested formic acid.

Patients and results

From 1963 to 78, 45 cases of formic acid ingestion were reported to the National Poisons Information Service (Guy's Hospital). Four proprietary brands of descaling agent containing different formic acid concentrations were involved, Kleenoff (55%), Ataka (44%), Descalite (55%) and Kilrock (60%). Nine children under the age of 12 years accidentally ingested less than 10 g of formic acid. They suffered oropharyngeal burns, which were only superficial, and fully recovered.

Two adults accidentally ingested formic acid, whilst 34 deliberately consumed it. The clinical features were compared with the amount of formic acid ingested. Consumption, by 23 subjects, of between 5 and 30 g of formic acid produced no deaths. The majority (16) developed minor superficial oropharyngeal burns only. Five had more severe symptoms including abdominal pain, vomiting, dyspnoea and dysphagia, whilst 2 sustained haematemeses, pneumonitis and they subsequently developed oesophageal strictures.

Ingestion of 30-45 g of formic acid produced more serious effects. Of the 6 patients recorded, one died, one had reversible disseminated intravascular

coagulation (DIC) and 3 had reversible acute renal failure. All suffered haematemeses and had biochemical evidence of liver impairment. Four needed subsequent treatment for oesophageal strictures.

Ingestion of 45 to 200 g of formic acid was recorded from 16 patients, of whom 14 died. Two recovered after intensive management. Their recoveries were complicated by reversible DIC, pneumonitis and acute renal failure. One has needed frequent re-admissions for oesophageal dilatations. Considering the fatalities, the majority (9) died painfully within the first 36 hr from corrosive perforations of the abdominal viscera and from gastrointestinal haemorrhage. The other 5 developed acute renal failure and this contributed to their death.

Discussion

Despite its ready availability, formic acid, a constituent of proprietary descaling agents and stain-removing fluids, is a relatively rare cause of both accidental and deliberate self-poisoning. Accidental ingestion of formic acid is mainly a problem of childhood and since only small volumes are ingested, there is usually a good prognosis. In this, the experience of the authors accords with 2 previous reported cases (Harvey, 1968; Malizia *et al.*, 1977). Children presumably consume only small quantities of the acid because of its pungency.

The deliberate ingestion of formic acid is poorly documented in the literature. One patient reported from France (Tschantz and Favre, 1975) died after taking formic acid and hydrofluoric acid, whilst a second patient (Wienikowski and Guzik, 1973) recovered after acute renal failure and gastrointestinal haemorrhage. Through the National Poisons Information Service the authors were able to collect 45 instances of formic acid poisoning. In their experience, formic acid poisoning has a high mortality and morbidity. They have assembled a dose-response pattern from the information collected.

The consumption of 60 g or more of formic acid

(approximately 100 ml of the descaling fluid) produced death in all cases.

Formic acid is a strong, corrosive acid and produces extensive tissue burns with a coagulative necrosis. Most patients had corrosive burns to the gastrointestinal epithelium and their extent reflected the quantity of the acid which had been consumed.

Many patients also had corrosive skin burns, some of which were initially overlooked. Five patients who recovered required skin grafting for dermal burns. High doses of formic acid (30 g) caused widespread ulceration, haemorrhage and perforation. As in all cases of corrosive burns, gastric lavage and induced vomiting are contra-indicated. Such procedures carry a very high risk of oesophageal perforation and rupture leading to a serious mediastinitis. It is suggested that milk should be given to buffer the acid rather than titration with an alkali such as lime water.

Respiratory complications were frequently reported in the patients studied. Dyspnoea, low PO_2 and diffuse shadowing on chest X-ray were often reported within 12–24 hr. 'Shock lung' (Stoddart, 1974) or alveolar-capillary leakage was a problem in 5 cases at the 3rd and 4th day. The early lung changes were probably a toxic pneumonitis, resulting from the inhalation of the volatile formic acid. At post-mortem, several had evidence of widespread pneumonitis. Two patients who died had an aspiration pneumonia, gastric lavage having been attempted unsuccessfully.

Metabolic acidosis, haemolysis and DIC occurred in several patients. The haemolysis was probably a direct effect of formic acid on the red cell, whilst the extensive acid necrosis was the likely initiating factor for the DIC.

The high incidence of acute renal failure in the present series (20 patients) reflects both the large fluid losses with resultant hypotension producing tubular necrosis, and the direct nephrotoxic effect of

formic acid. The patients also have some of the features of acute methanol intoxication (Bennet, Cary and Mitchell, 1953) which reflects the fact that methanol is oxidized to formic acid.

The management of formic acid poisoning is supportive with good intensive care. Gastric lavage is contra-indicated, whilst the acid should be buffered with milk. Attention should be paid to skin burns, which are often overlooked. Oesophageal perforation and resulting mediastinitis must be watched for. Maintaining the central venous pressure is important, since gastrointestinal haemorrhage is common. Clotting studies should be checked carefully to detect early DIC. Many of the cases of acute renal failure arose from inadequate fluid replacement rather than the nephrotoxic effect of formic acid. The majority of the patients studied had been given hydrocortisone, although the benefit of steroids in this condition has not been established. Oesophageal stricture was a major long-term problem for those who survived the ingestion of moderate quantities of formic acid.

The ingestion of formic acid, even in moderate quantities, constitutes a serious tissue and metabolic insult. The mortality and morbidity will remain high whilst such a strong acid is widely available in the home.

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