Letters to the Editor

An oral treatment for lead toxicity

Sir,

We read with interest the paper by Thomas and Ashton\(^1\) which described a patient with lead poisoning in whom comparison was made between the efficacy of intravenous sodium calcium edetate and oral DMSA. The authors claim that an equal amount of lead is excreted by the two drugs. Although both chelating agents produced similar falls in the blood lead concentration, the limited data given in Figure 1 by the authors\(^1\) suggest that the excretion of lead during treatment with sodium calcium edetate was approximately five times greater than with oral DMSA, notwithstanding the fact that both treatments were continued for a similar period.

The efficacy of chelation therapy in lead poisoning cannot be judged by estimating blood lead concentrations alone; determination of urine lead excretion is mandatory. A critical review of the two comparative clinical studies published in the English literature\(^2\) does not support the view that DMSA is superior to sodium calcium edetate in promoting urinary lead excretion. Even though only 12–16 mg/kg/day sodium calcium edetate was given to intoxicated smelter workers,\(^2\) lead excretion was significantly greater than after DMSA administration (18–42 mg/kg/day). In a paediatric study,\(^3\) sodium calcium edetate (29 mg/kg/day) also produced greater urinary lead excretion than DMSA (30 mg/kg/day). Our own data (to be published) demonstrate that intravenous sodium calcium edetate (75 mg/kg/day) is approximately four times more effective in promoting lead excretion than DMSA (30 mg/kg/day). Taking into consideration not only ‘therapeutic’ dose but molar equivalents, we conclude that oral DMSA is less effective than intravenous sodium calcium edetate in promoting urinary lead excretion.

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References


Profound prolonged hypotension following captopril overdose

Sir,

We would like to report a case of captopril overdose complicated by profound hypotension and the failure of naloxone administration as a therapeutic modality. A 22 year old man, with a past history of intravenous drug abuse and several episodes of self-poisoning, presented to our hospital following ingestion of 30, 25 mg tablets captopril and possibly intravenous narcotics. On admission he was unconscious (Glasgow Coma Scale 6), pale and diaphoresing. Systolic blood pressure was 80 mmHg on admission falling to 40 mmHg with a relative bradycardia (60 beats/min). There was no clinical evidence of alcohol ingestion, opiate overdose or recent intravenous injection. Physical examination was otherwise normal. Urea and electrolytes, electrocardiogram and chest radiograph on admission were normal. The patient was given intravenous plasma protein substitute at a rate of 1 litre per hour over 8 hours. A bolus (10 mg) and infusion (0.04 mg/min) of naloxone was given over 1 hour with no effect on conscious level or hypotension. After 8 hours a systolic blood pressure of 90 mmHg was maintained with a concomitant improvement in conscious level.

There are 6 reports of angiotension converting enzyme inhibitor overdose\(^6\)–\(^9\) with variable severity of hypotension and altered conscious level. The duration and severity of the hypotension was unexpected in view of the pharmacokinetics of the drug. Supportive therapy has been advocated with intravenous fluid and, rarely, inotropic support and renal-vasodilator therapy.

It is unlikely that in our patient a concomitant intravenous narcotic overdose may have contributed to the hypotension and the altered conscious level. There was no clinical improvement with intravenous naloxone. Naloxone pre-treatment has been used to block the hypotensive response associated with captopril treatment\(^4\) and we report that, in our patient, naloxone did not reverse captopril-induced hypotension.

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References