

# Survival after severe self poisoning with sodium valproate

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**Summary:** A 48 year old patient deliberately poisoned herself with 25 g of sodium valproate and survived with supportive measures only. This case contradicts the experience of those who advocate aggressive management of such severe overdoses.

## Introduction

Although sodium valproate is now widely used as an anticonvulsant, experience of self poisoning with it is limited and there is a lack of reliable data on its effects in overdose (Volans *et al.*, 1983). We describe a patient who survived ingestion of 25 g of sodium valproate with supportive therapy alone.

## Case history

A 48 year old woman presented 4 hours after deliberate ingestion of a mixed overdose of 25 g sodium valproate (enteric coated), 40 mg diazepam and 50 mg temazepam. Idiopathic temporal lobe epilepsy had been diagnosed 15 years previously for which she was treated with sodium valproate 500 mg t.i.d. On admission she was drowsy but rouseable, with generalized hypotonicity but no other abnormal physical findings. A 20 litre gastric lavage was performed but no tablets were recovered. Her conscious level gradually deteriorated and 3 hours after admission she was deeply unconscious, and remained so for 30 hours.

Serum concentration of sodium valproate was 1040  $\mu\text{mol/l}$  at 4.5 hours and 5100  $\mu\text{mol/l}$  18 hours after ingestion. Electrocardiogram, minute ventilation, arterial blood gases, urea and electrolytes and haematological investigations including clotting studies and platelet counts remained normal throughout.

Liver function tests, with the exception of aspartate transaminase (AST) were normal. The AST level was elevated from day three to day nine after ingestion with a peak value of 69 U/l on day three. Despite a normal serum calcium a reduction in serum phosphate was observed from day four to day nine, with a

minimum value of 0.50 mmol/l on day five (reference range 0.80–1.55 mmol/l). The patient made a complete recovery and subsequent to psychiatric assessment was discharged from hospital after 11 days.

## Discussion

Although self poisoning among the epileptic population is relatively common (Mackay, 1979), experience with non-barbiturate anticonvulsants is limited. To our knowledge only three fatalities have been described with sodium valproate (Gourru, 1980; Boillot *et al.*, 1981; Schnabel *et al.*, 1984). Coma and respiratory depression, convulsions, transitory disturbance of liver function tests, hypocalcaemia and thrombocytopenia have been described. In the patient we describe, coma did occur but presumably this was due in part to concurrent ingestion of benzodiazepines.

A variety of 'active treatments' for valproate poisoning have been advocated including forced diuresis (ABPI, 1983), naloxone (Steinman *et al.*, 1979), and haemodialysis/haemoperfusion (Mortensen *et al.*, 1983). However, the experience of Volans *et al.* (1983), Karlsen *et al.* (1983) and Eeg-Olofsson & Lindskog (1982) indicate that supportive therapy alone is adequate, even in massive overdose. Volans *et al.* (1983) question the need for and the evidence of efficacy for more active measures. The survival of our patient lends support to this contention.

This highlights the need for continued reporting and monitoring of outcome in self poisoning with sodium valproate to enable the management of choice to be properly defined.

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