serum lithium concentrations in patients taking the drug and indicates that, although often reversible, metabolic disturbances may persist for more than two years after treatment is stopped.

We thank Dr D Pariente for referring the patient and dealing with the psychiatric problems, and Professor J L H O'Riordan, Middlesex Hospital, for advice on the endocrinological aspects of the case.

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Severe leucopenia in fatal lithium poisoning

Lithium carbonate is commonly used to treat manic depressive psychosis.[1] Numerous adverse effects (some related to dose) have been described, and it is important that plasma lithium concentrations are monitored regularly so that they may be maintained within the therapeutic range of 0·6-1·2 mmol/l (0·42-0·83 mg/100 ml).[1] Gastrointestinal, endocrine, neurological, and renal side effects are well recognised,[1] and haematological abnormalities have been less commonly described.[2-5] Severe toxicity may occur with plasma lithium concentrations over 2·0-2·5 mmol/l (1·4-1·7 mg/100 ml) and is potentially lethal.[1] Therapeutic efforts to reduce plasma lithium concentrations may be hampered by continued absorption from sustained release preparations.[1]

We describe a fatal case of self poisoning with lithium carbonate that resulted in severe leucopenia before death.

Case report

A 49 year old man with a long history of depression was admitted as an emergency in a stuporous condition (day 1). His general health had been good, and his only regular medication was lithium carbonate (sustained release) 800 mg a day, although on occasions he had received chlorpromazine, thioridazine, flupenthixol, and benzodiazepines without ill effect. He had been found unconscious and was estimated to have ingested more than 30 lithium carbonate 400 mg tablets. He had also taken an undetermined number of tablets of chlorpromazine 25 mg, flurazepam 1 mg, and temazepam 10 mg, but there had been no more than three tablets in each container before overdosage.

On examination he was polyuric, haemodynamically stable, and breathing spontaneously. Serum lithium concentration was 3·32 mmol/l (2·3 mg/100 ml) (no other drugs detectable), haemoglobin concentration 14·1 g/dl, red cell count $4{\cdot}99\times10^{12}$/l, white cell count $10{\cdot}0\times10^{9}$/l, and platelets 283×10^{9}/l. Gastric lavage was performed, intravenous fluids started, and 50 ml lactulose given orally. No other drugs were administered. Five hours later the serum lithium concentration reached 5·54 mmol/l (3·8 mg/100 ml) (haemoglobin concentration 17·5 g/dl, red cell count $5{\cdot}58\times10^{12}$/l, white cells $11{\cdot}2\times10^{9}$/l, platelets 111×10^{9}/l) and peritoneal dialysis was started. On the morning of day 2 the serum lithium concentration reached 5·8 mmol/l (4·0 mg/100 ml) while the white cell count had fallen to $1{\cdot}4\times10^{9}$/l with an unremarkable differential count (74% neutrophils, 22% lymphocytes, 2% eosinophils, 2% monocytes, no basophils). The red cell count was $4{\cdot}33\times10^{12}$/l, haemoglobin concentration 12·6 g/dl, and platelets 242×10^{9}/l.

He subsequently became hypotensive and developed type 1 respiratory failure, although there was no suggestion of any infective process at any site and the temperature remained between 35°C and 37°C. Positive end expiratory pressure ventilation and intravenous dopamine were started. Peritoneal dialysis was continued. By day 3 serum lithium concentration had fallen to 3·9 mmol/l (2·7 mg/100 ml) but the white cell count was $0{\cdot}2\times10^{9}$/l (haemoglobin concentration 11·5 g/dl, red cells $3{\cdot}97\times10^{12}$/l, platelets 111×10^{9}/l). Shortly after, he had an asystolic cardiac arrest and attempts at resuscitation were unsuccessful.

Comment

The major point of interest was the development of pronounced leucopenia (without appreciable changes in circulating red cell and platelet counts) associated with an acute rise in plasma lithium concentrations, which had previously been within the therapeutic range. This does not appear to have been described previously. Conversely, circulatory failure is well recognised.[1]

Haematological abnormalities have been described in association with long term lithium treatment. A reversible increase in circulating leucocytes may occur, and lithium might be of value in treating neutropenia induced by chemotherapy.[2] Associations with leukaemic conditions and megaloblastic anaemia have also been noted.[3,4] Hussain *et al* reported on a patient maintained on imipramine and thioridazine who developed fatal aplastic anaemia 12 weeks after starting lithium 300 mg daily; the serum lithium concentration did not exceed 0·76 mmol/l (0·53 mg/100 ml).[5] The exact mechanism of the rapid fall in the white cell count with a normal differential described here is unclear, but it may represent a direct toxic effect of lithium at high plasma concentrations on the circulating leucocytes. Whatever the mechanism, we would recommend close monitoring of the white cell count in all cases of lithium overdosage.

We thank Dr A I Macdougall, consultant renal physician at this hospital, for his helpful advice in the preparation of this case report.

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Inadvertent duplicate publication

Treatment of severe poisoning with slow release theophylline

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The *BMJ* regrets that the case report in the above article (19 May 1984, p 1497) was substantially the same as that published in *Krankenhausarzt* (1983;**56**:413-23). The authors inform us that it was also published in *Attemwegs und Lungenkrankenheiten* (1983;**9**:93-8) and reported at a symposium in Vienna (Wiener Intensive-medizinische Tage 1983). They did not, however, tell us this when the article was submitted for publication, their article did not contain any reference to the earlier papers, and all authors signed our copyright form, which states, among other things, that "papers are accepted on condition that they have not been published by any other journal."

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