patients who had estimated their bowel frequency at eight or more a week there was no systematic difference between recalled and recorded frequencies ($\chi^2 = 0{\cdot}471$). When the imprecisely documented group with 15 or more stools a week was excluded, however, a different picture emerged. Of those who predicted 8, 9 or 10, and 11 to 14 bowel actions a week, half were wrong by two bowel actions a week or more and most of the errors were overestimates.

Two consecutive days with no bowel action—Fourteen patients predicted that at some stage in the next two weeks they would experience two consecutive days with no bowel action. Only nine, however, recorded themselves as doing this. Of the 67 patients who had predicted they would not miss two consecutive days, two in fact did so.

Two or more bowel actions between meals—This was predicted by 16 patients but experienced by only seven of them. It was also experienced by five of the 65 patients who had predicted it would not happen in a two-week period. Of these five, three had denied that it ever happened.

Bowel actions after rising but before breakfast—Since 29 of the 81 patients usually had something to eat or drink in bed before rising and six more had no breakfast, only 46 patients were eligible for analysis. Only 18 of these predicted that they would open their bowels before breakfast during a two-week period. In fact 38 patients were recorded as doing so. This included all 18 who had expected it, but also 20 of the 28 who had not expected it. These 20 patients had denied that they ever opened their bowels before breakfast. Despite this, one of them did so every day for two weeks.

Discussion

The accuracy of diary recordings is obviously crucial to the evaluation of our data. We made a special effort to detect and exclude any errors and omissions and included only subjects who, when given the opportunity to confess without risk of embarrassment, denied any peccadillo, however slight. More rigorous surveillance would have been impracticable without changing the patients' life-style and therefore, probably, their bowel habits, which would have negated the object of this study.

Overall, there was reasonably close agreement between the number of bowel actions patients stated they had each week and the number they recorded. Nevertheless, one in six patients were wrong in their predictions by as much as three motions a week. This degree of error is probably unimportant clinically, but implies that patients' estimates should not be regarded as precise in, for example, epidemiological surveys. Patients whose usual bowel frequency deviated from the norm of seven bowel actions a week tended at interview to exaggerate the extent of this deviation.

The discrepancy between recalled and recorded information was particularly pronounced in relation to the passage of stools after rising from sleep but before breakfast. Many people seem to be remarkably unaware of the times at which they defecate. This suggests that patients' statements about the timing of their bowel actions should be accepted with caution.

The passage of multiple stools between one meal and the next would be regarded as abnormal by most patients as well as most doctors. Nevertheless, patients were notably bad at predicting this. Half the recordings of it had not been expected, and half of those who expected it failed to record it. Similarly, the occurrence of two consecutive days with no bowel action was considerably overpredicted. Since episodes of infrequent and frequent bowel action are central to the diagnosis of the irritable bowel syndrome, these findings suggest that this disorder is apt to be misdiagnosed unless patients are asked to record their bowel actions in a diary.

Investigations of bowel habit in the general population[3–6] have all assumed that the questionnaire technique is satisfactory. Our results suggest that it is not satisfactory, particularly if reliable information is required beyond the mere number of bowel actions a week. Perhaps future epidemiological studies should include actual recordings of bowel motions.

We gratefully acknowledge help in planning this study and in analysing the data from Miss E H L Duncan and Mrs A Morris of the department of medical statistics, University of Bristol. The investigation was supported by a grant from Kellogg of Great Britain Limited. We thank the doctors who helped with the questionnaire.

Copies of the questionnaire, diary, and honesty letter are available from the authors on request.

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SHORT REPORTS

Amodiaquine-induced involuntary movements

Amodiaquine hydrochloride is similar to chloroquine and is an effective, relatively non-toxic antimalarial drug. Side effects from therapeutic doses are nausea, vomiting, diarrhoea, and lethargy.[1] Prolonged use may cause corneal deposits and bluish grey pigmentation of the fingernails, skin, and hard palate.[2] Absolute neutrophenia has been reported in a girl aged 2 given 200 mg amodiaquine base daily for seven days.[3] We report other side effects seen in four patients given this drug.

Case reports

Case 1—A young woman presented with a protruding tongue, difficulty in speaking, and excessive salivation. Three days previously she had been given three tablets of amodiaquine initially and thereafter three tablets daily for malaria. She had had similar symptoms once before after taking this drug. There was fasciculation of the tongue and facial muscles and saliva dripped from her mouth. The involuntary movements were thought to be drug-induced. She was given benztropine mesylate 2 mg intravenously. Within 30 minutes the fasciculation ceased, salivation had greatly decreased, and the tongue began to recede. The dose was repeated six hours later with further benefit. Because the patient had taken aspirin with the amodiaquine she agreed to take amodiaquine again if she had another malaria attack. Six months later one occurred and the side effects were exactly repeated.

Case 2—A young woman presented with a three-day history of fever, diagnosed as malaria, for which she had had chloroquine followed by three tablets of amodiaquine. Within 24 hours she was nauseated and dizzy, her gait was wobbly, she had intention tremor, she stammered, and her tongue felt heavy and later protruded. She was thought to have hysteria, but a psychiatrist diagnosed drug-induced pseudo-Parkinsonism. After benztropine mesylate 2 mg intravenously she rapidly improved.

Case 3—A young woman was given three tablets of amodiaquine for suspected malaria. Within three hours she developed tremor, difficulty with speech, and excessive salivation. She was prescribed oral benzhexol 10 mg and the symptoms disappeared within two hours. She recalled having had similar symptoms about three hours after taking amodiaquine previously. They had decreased gradually and disappeared over 24 hours.

Case 4—A 7-year-old boy presented with a protruding tongue. He had been given two tablets of amodiaquine on each of the two preceding days. He was given benztropine 0·5 mg intravenously and rapidly improved.

Comment

Involuntary movements are common in extrapyramidal syndromes such as Parkinsonism, hepatolenticular degeneration, chorea, and

athetosis[4] and may occur in psychological illnesses, especially hysteria, and with drugs such as phenothiazines and levodopa.[5] This report adds amodiaquine to the list. Involuntary movements after taking phenothiazines have been attributed to toxic overdose or to individual idiosyncrasy. The doses of amodiaquine in our cases were large but not large enough to be toxic. The patients' reactions were therefore probably idiosyncratic. The rarity of such cases supports this view.

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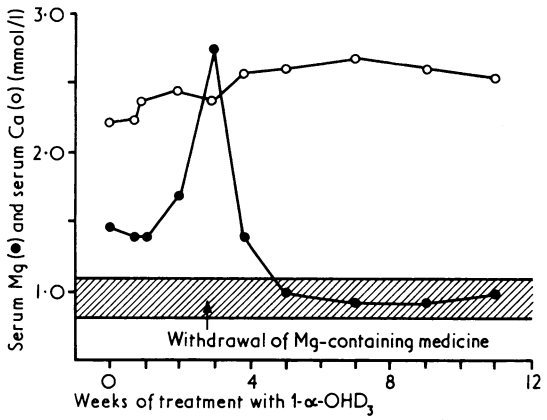
Iatrogenic magnesium intoxication during 1-α- hydroxycholecalciferol treatment

During a therapeutic trial with 1-α-hydroxycholecaliferol (1-α-OHD_3) in chronic renal failure one of our patients became seriously ill because of magnesium intoxication. We therefore want to draw attention to the special risk of magnesium intoxication in patients with chronic renal failure treated with 1-α-OHD_3.

Case report

The patient was a 43-year-old woman with long-standing severe chronic pyelonephritis. The glomerular filtration rate (GFR) was 5 ml/min during the period of treatment with 1-α-OHD_3. Serum magnesium and calcium values are shown in the figure. During treatment with 1-α-OHD_3 (1 μg/day by mouth) the serum calcium concentration began to rise slowly until a maximum was reached after eight weeks' treatment. Before the start of treatment the serum magnesium concentration was moderately increased.



Serum calcium and magnesium values during treatment with 1-α-OHD_3. Shaded area represents normal range of serum magnesium values.

Conversion: SI to traditional units—Magnesium: 1 mmol/l ≈ 2·4 mg/100 ml. Calcium: 1 mmol/l ≈ 4 mg/100 ml.

About two weeks later the patient became exhausted and confused and developed muscle weakness and tremor, and there was an abrupt increase in the serum magnesium concentration. The patient was thoroughly questioned about her medicine intake, and she revealed that for many years she had regularly taken a magnesium-containing antacid powder recommended by a healer. All magnesium-containing medicines were withheld, and the serum magnesium levels returned to normal during the following week, when the symptoms of intoxication disappeared. The 1-α-OHD_3 treatment was continued unchanged.

Discussion

Patients with chronic renal failure tend to retain magnesium.[1] This patient who had regularly taken magnesium-containing medicine had never shown any signs of intoxication, although she had had the same low GFR for a long time. The treatment with 1-α-OHD_3 no doubt contributed to the severe hypermagnesaemia with magnesium intoxication. The increase in serum magnesium concentrations was probably due to an increased intestinal absorption of magnesium similar to the effect of 1-α-OHD_3 on the intestinal absorption of calcium.[2] Magnesium intoxication may likewise be provoked by other Vitamin-D metabolites—for example, 1, 25-dihydroxycholecalciferol and 25-hydroxycholecalciferol—both of which, together with 1-α-OHD_3, are gaining popularity as drugs for treatment and prophylaxis of renal bone disease.

We find it advisable to control serum magnesium values before the start of treatment with 1-α-OHD_3. High values may indicate an intake of magnesium-containing medicine. If general symptoms of magnesium intoxication occur during treatment with 1-α-OHD_3 serum magnesium as well as serum calcium should be controlled.

We thank Leo Pharmaceutical Products, Copenhagen, Denmark, for providing 1-α-OHD_3, and the department of clinical chemistry, Aalborg Hospital, Aalborg, for blood analyses.

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Subclinical coeliac disease and infertility

Three women under the care of one of us (RW), who had been investigated for infertility, were found to be suffering from coeliac disease. One patient had a healthy baby after treatment with a gluten-free diet. We were impressed by the lack of symptoms in another patient and the mild gastrointestinal symptoms in the other two when they presented with infertility, and we therefore decided to screen a group of women attending an infertility clinic for coeliac disease.

Patients, methods, and results

Seventy-seven women with primary or secondary infertility without overt disease were screened for coeliac disease, using a questionnaire, measurement of the haemoglobin concentration, and examination of a blood film for Howell-Jolly bodies; tests were also done for red cell folate and reticulin antibody.[1] Jejunal biopsy was performed if a screening test proved positive.

Details of the nine patients selected for jejunal biopsy are given in the table. The patient with subtotal villous atrophy had had anaemia and spells of diarrhoea as a child, but had been completely asymptomatic since she was 12. She became pregnant with twins after clomiphene treatment at the time