for serum ferritin estimation was taken either before dialysis or on a non-dialysis day into iron-free containers, and serum was stored at -20°C until assayed. Haemoglobin concentration was measured simultaneously. Serum aspartate transaminase concentration measured by autoanalyser in all patients ranged from four to 57 IU/l; in only three patients did the concentration exceed 50 IU/l. Serum ferritin concentration was measured by radioimmunoassay on all samples as a single batch.³ The normal range using this method is 15-250 $\mu\text{g/l}$. Serum ferritin concentration is normally distributed on a logarithmic scale. Statistical analysis by Student's *t* test was therefore performed on logarithms, and geometric means are quoted.

Mean serum ferritin concentrations after low, intermediate, and high intakes of elemental iron. Shaded areas indicate 95% confidence limits.

Mean serum ferritin concentration was 493 $\mu\text{g/l}$ (range 10-8600 $\mu\text{g/l}$). Serum ferritin was raised in 44 patients and normal in 16 patients; only in the patient using an X23 coil dialyser was the value subnormal. There was no significant correlation between haemoglobin and serum ferritin concentrations. There was no significant difference in serum ferritin concentration between patients haemodialysed for less than 12 months and the remainder, between transfused and non-transfused patients, between men and women, between menstruating and non-menstruating women, or between the nine patients with polycystic kidneys and the remainder. There was no significant correlation between total amount of oral iron received since starting haemodialysis and serum ferritin concentration. Patients were divided into three groups according to average daily intake of elemental iron since starting haemodialysis or in the 12 months preceding the study in the case of patients dialysed for more than a year. No patient admitted to taking less than 60 mg elemental iron daily. A low-intake group (15 patients) ingested 60-120 mg daily, an intermediate group (28) ingested 121-240 mg daily, and a high intake group (18) ingested 241-360 mg daily. Significant differences in mean serum ferritin concentration were found between the low-intake and the other two groups ($P < 0.001$ in each case; see figure).

Comment

We measured serum ferritin to assess iron state since it correlates well with the state of marrow iron stores in patients treated with haemodialysis.⁴ Our results indicate that most patients were receiving unnecessarily large amounts of oral iron. While there is no proof that any harm has resulted from excessive iron ingestion, it would seem wise to avoid overtreatment on grounds of possible risk, patient convenience, and economy. It is unnecessary to exceed 120 mg elemental iron daily as maintenance treatment in patients treated as described by flat-board haemodialysis. This dose is too large in some patients. An adequate maintenance dose for most patients is probably 60 mg elemental iron (one ferrous sulphate tablet) daily.

Dialysis carried out as described is associated with the loss of about

20 ml blood in dialysis membranes and lines per dialysis and with a mean percentage daily loss of iron ranging from 0.02 to 1.3%.⁵ Our conclusions are therefore applicable only to patients undergoing haemodialysis whose iron losses are similar to this. The single patient with a low serum ferritin concentration had both used a coil dialyser and taken only 60 mg of elemental iron daily for three years before the study and also menstruated regularly. Iron requirements in patients using coil dialysers need further investigation.

These observations relate to maintenance treatment. Many patients starting haemodialysis who have not had iron supplementation are iron depleted.¹ In such patients higher doses of oral iron may be required initially.

We thank the patients and staff of the regional renal unit, St Bartholomew's/St Leonard's Hospital, for their co-operation and Dr J Allawi, Miss R Minchinton, Professor D L Mollin, Mrs J Jessop, and Mrs J Wadsworth for their help.

Requests for reprints should be addressed to Dr L R I Baker.

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A case of Dettol addiction

Joubert *et al*¹ described a case of severe poisoning from ingestion of Dettol. We report a case of Dettol addiction, which we believe to be the first such case to be recorded.

Case report

A 56-year-old cachectic woman was admitted to hospital with anorexia, weight loss, and abdominal distension for three months. There were no other gastrointestinal symptoms. Her medical history contained nothing of note; she worked as a cook and had been divorced for 10 years. She was emaciated with gross ascites and oedema of the legs. A large mass was present in the right iliac fossa and the liver was slightly enlarged. There was no abnormality in the cardiovascular or central nervous system but she had pneumonic changes in both lungs. Alkaline phosphatase activity was 180 IU/l and γ -glutamyltransferase activity 84 IU/l, and isoenzymes were of liver origin; other biochemical values were within normal limits, as was a full blood count. There was no abnormality of renal function and no evidence of sexually transmitted disease.

While an inpatient she was observed drinking undiluted Dettol (4.8% chloroxylenol in isopropyl alcohol) and also to use Dettol-soaked pessaries. Three bottles of Dettol (500 ml) were found in her locker. She admitted to drinking Dettol for at least 10 years to "purify" herself but denied ever drinking more than half a bottle a day. Her 25-year-old son stated that she had been well until seven or eight years before, when she developed an overwhelming fear of acquiring syphilis; this occurred after her marital disharmony and coincided with the onset of Dettol abuse, which was limited initially to small amounts only and increased gradually. Over the year before admission she had become confused and disorientated. While in hospital she underwent psychiatric assessment and a schizophrenic illness was diagnosed.

Comment

We can find no other case of long-term Dettol ingestion leading to probable addiction. Dettol contains some isopropyl alcohol in addition to chloroxylenol, and this is the next most common alcohol after ethyl and methyl to which addiction occurs. Ingestion of isopropyl alcohol occurred in two-thirds of the non-ethyl-alcohol addicts described by Mendelson *et al*.² The motivation in our case was

cleansing and may have stemmed from a schizophrenic illness characterised by an overwhelming fear of developing syphilis; addiction subsequently occurred. The pattern of addiction was similar to that with other addictive substances, including a reluctance to admit the quantity consumed, drinking secretly alone, and consuming gradually increasing amounts. There was no information on attempts to abstain, nor were there obvious withdrawal symptoms while in hospital.

In the case reported by Joubert *et al* acute ingestion of 350 ml household Dettol produced deep coma for six to eight hours, then diarrhoea for 48 hours; ultimate recovery, however, was uneventful. We could not ascertain the exact quantity that our patient was consuming, but apparently she had been regularly intoxicated for at least a year. We do not know the long-term effects of this substance, but at necropsy the liver and kidneys were normal, though the γ -glutamyl-transferase activity—a sensitive index of alcoholic liver drainage—was increased.

Patients in the series of Mendelson *et al* were all addicted to ethyl alcohol primarily and subsequently used isopropyl alcohol because of its greater effects. The present case appears to be unique, as the addiction supervened on a morbid cleansing obsession.

We are grateful for the help of Mrs A Irwin, staff pharmacist, Royal Infirmary, Edinburgh.

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Treatment of ulcerative colitis with thalidomide

The only generally accepted ethical use of thalidomide is in treating erythema nodosum leprosum (ENL),¹ the immune-complex complication of lepromatous leprosy. Its value has been proved by double-blind, internally controlled trials^{2,3} and it is the preferred treatment of severe chronic ENL in light-skinned male patients and postmenopausal women, being less toxic than corticosteroids. Its mode of action, however, remains uncertain.

Having observed over many years the excellent results obtained with thalidomide in ENL, a leprosarium staff member with chronic ulcerative colitis asked that she might undergo clinical trial of the drug. Since immune complexes may play a part in the pathogenesis of ulcerative colitis⁴ her request was accepted; we report here the results after 18 months' continuous treatment.

Case report

In October 1972 a 28-year-old married woman with three children and a history of asthma developed abdominal pain and diarrhoea with blood and mucus in her stools. No parasites were detected, metronidazole was unhelpful, and sigmoidoscopy and barium-enema examinations confirmed the diagnosis of ulcerative colitis. She was given oral steroids, hydrocortisone-retention enemas, and sulphasalazine. Over the next four years, however, she felt continuously unwell with lassitude, abdominal pain, and chronic variable diarrhoea. She also began to have pain in the sacroiliac joints whenever oral steroids were stopped.

Before beginning thalidomide in April 1977 she felt tired and was passing three or four bloody motions a day despite treatment with prednisolone 5 mg daily and sulphasalazine 1 g thrice daily. Her haemoglobin concentration was 8.9 g/dl and white cell count $13.4 \times 10^9/l$ ($13\,400/mm^3$). Barium-enema examination disclosed absence of haustrations from the rectum to the hepatic flexure, with extensive mucosal ulceration. Limited colonoscopy showed moderate to severe inflammation of rectal, sigmoid, and descending colonic mucosa, which exuded pus and bled easily. Representative biopsy samples were taken at 10 cm intervals.

Thalidomide 300 mg daily was begun under contraceptive cover, but since there was no immediate response the dosage was increased to 400 mg daily after five weeks. The patient then steadily improved. By eight weeks she was having only one bowel motion daily, by two and a half months macroscopic blood had disappeared, and for the last year she considered her motions to be normal, save for occasional constipation. Her general condition was very satisfactory, although she continued to have stiff fingers and back on first waking and occasional episodes of asthma. Full independent neurological and electromyographic examinations failed to detect any sign of thalidomide neuropathy. Her haemoglobin concentration was 13.2 g/dl and white cell count $4.75 \times 10^9/l$ with 8% eosinophils, probably due to the thalidomide.³ On repeat colonoscopy after six months the mucosa of the rectum, sigmoid, and descending colon looked normal, with no pus and no bleeding on instrumentation. Mucosal biopsy at comparable sites showed substantial improvement. Although severe mucosal atrophy persisted, with epithelial hyperplasia, there was only very little inflammation, which was chronic in character, the findings being graded as "chronic ulcerative colitis in remission." Prednisolone and sulphasalazine were stopped at 5 and 15 months respectively and the dosage of thalidomide reduced gradually to 200 mg daily without clinical relapse.

Comment

Ulcerative colitis is noted for its episodes of remission and exacerbation. Nevertheless, our patient's apparent response to thalidomide was impressive, since after four and a half years of ill health she became fit apart from morning joint pain and stiffness and had stopped taking steroids and sulphasalazine without any sign of relapse. A well-planned clinical trial in carefully selected patients with moderate to severe disease not warranting surgery appears to be indicated.

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ONE HUNDRED YEARS AGO We are informed that the Commissioners appointed by the Home Secretary to inquire into the circumstances of the death of John Nolan in the Clerkenwell Prison have completed their investigations and prepared their report. It will be remembered that a coroner's jury recorded a verdict that this lad, who was convicted of a trivial offence and sentenced to four months' hard labour, "died from inflammation and congestion of the lungs, accelerated by repeated punishment on dry bread and water, ordered by the governor and sanctioned by the surgeon of the prison." Considerable feeling was excited; and at the inquest the Chairman of the Visiting Justices stated that their control over such punishment was now nullified. Mr Cross directed a special investigation by Dr Risdon Bennett (the President of the Royal College of Physicians), Dr Guy, FRS, and Dr Bristowe, senior physician of St Thomas's Hospital. The conclusions of their report are entirely adverse to the verdict; they consider that there is no evidence whatever to justify the allegation that death was accelerated in the manner stated; they acquit the governor and the medical officer of blame; and they comment severely upon the fact that the coroner did not order a *post mortem* examination in the case. (*British Medical Journal*, 1879.)