4.0); and IgM 1.58 g/l (normal 0.5-1.9). No monoclonal Ig was detected in serum by immunoelectrophoresis. A chest x-ray film showed a small discrete mass in the left mid-zone, which was reported as almost certainly a benign lesion.

Prednisolone (15 mg thrice daily) was started, and the serum calcium concentration fell but remained above our reference range. After 10 days the prednisolone was gradually stopped but her serum calcium concentration rose further. It was then discovered that she had been taking vitamin D₂ 1.25 mg (50 000 IU) thrice daily for three and a half months before admission. Glutethimide 500 mg at night was started.

Eight days later serum γ -glutamyl transpeptidase activity (a marker of hepatic enzyme induction) rose. The serum calcium concentration fell to within our reference range four days later. Thereafter her confusion and renal function improved considerably. Throughout the period of monitoring there was no biochemical evidence of hepatocellular damage. The figure shows that the raised 25-hydroxy vitamin D concentration fell with enzyme induction but rose again when induction stopped, as indicated by a fall in γ -glutamyl transpeptidase activity, before gradually falling further. There was a significant negative correlation between the corrected serum calcium concentration and γ -glutamyl transferase activity from 10 days before to 10 days after treatment with glutethimide (rank correlation coefficient -0.72 ; $p < 0.001$).

Serum concentrations of corrected total calcium and 25-hydroxy vitamin D and activities of alkaline phosphatase, 5-nucleotidase, and γ -glutamyl transpeptidase before, during, and after 12 days of glutethimide treatment. (Normal ranges: 5-nucleotidase 5-15 U/l; 25-hydroxy vitamin D 7.5-75 nmol/l.)

Conversion: SI to traditional units—Calcium: 1 mmol/l $\approx$ 4 mg/100 ml. 25-Hydroxy vitamin D: 1 nmol/l $\approx$ 0.4 ng/ml.

Comment

Excessive intake of vitamin D₂ may produce an acute illness, while a lower but supplemental intake may be associated with renal calculi.² Hypercalcaemia has been reported to persist up to 14 months after vitamin D₂ has been stopped.³ We suggest that if poisoning has occurred with heavy doses removal of the excess vitamin may be achieved by induction of hepatic microsomal enzymes. The mechanism remains unknown, but rapid metabolism of vitamin D₂ by hydroxylating enzymes has been suggested.¹ Whether overdosage with the more potent metabolites of vitamin D available therapeutically—namely, alfa-calcidol (1 α -hydroxy vitamin D₃) and calcitriol (1 α ,25-dihydroxy cholecalciferol)—could be successfully treated using this method remains uncertain but merits investigation.

As found in a previous study using inducing agents,⁴ the rise in γ -glutamyl transpeptidase activity occurred over about one week. Presumably it takes this time for full alterations in vitamin D metabolism to occur, and therefore enzyme induction would be a useful adjunct to other, more immediate treatment for controlling the acute symptoms of hypercalcaemia from vitamin D poisoning. The rise in plasma alkaline phosphatase and 5-nucleotidase activities is consistent with data suggesting that hepatic membrane-bound enzymes may also be inducible.¹

The principle of using hepatic-enzyme-inducing drugs is well known in the management of disturbances of bilirubin metabolism.¹ Our patient was treated using the same principle, but clearly more

case studies are needed to assess the benefits of glutethimide in vitamin D poisoning. It may be worth considering this form of treatment for intoxication with other exogenous or endogenous fat-soluble compounds that are difficult to excrete, such as vitamin A.

We thank Professor G L Mills, department of geriatric medicine, Royal Liverpool Hospital, for allowing us to report this case, and the SAS Centre Middlesex Hospital for the retrospective estimation of serum 25-hydroxy vitamin D concentrations.

Requests for reprints should be sent to Dr S J Iqbal.

¹ Goldberg DM. The expanding role of microsomal enzyme induction and its implications in clinical chemistry. *Clin Chem* 1980;26:691-9.

² Taylor WH. Renal calculi and self-medication with multivitamin preparations containing vitamin D. *Clin Sci* 1972;42:515-22.

³ Howard JE, Meyer RJ. Intoxication with vitamin D. *J Clin Endocrinol* 1948;8:895-910.

⁴ Whitfield JB, Moss DW, Neale G, Orme M, Breckenridge A. Changes in plasma γ -glutamyl transpeptidase activity associated with alterations in drug metabolism in man. *Br Med J* 1973;i:316-8.

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Childhood drownings in private swimming pools: an avoidable cause of death

Details were obtained on the circumstances in which children were drowned in private swimming pools during 1975-9. An unknown number of children also suffered severe brain damage as a result of near drowning. The main contributory factor in such accidents is the absence of adequate fencing around pools. Legislation requiring such safety measures to be provided exists in many countries and should be introduced here.

Study and results

Since 1975 the Home Office has collected information from police forces on cases of drowning; it kindly supplied us with information on children drowned in private pools during 1975-9. In this period 34 children (21 boys and 13 girls) were recorded as having been drowned in private pools. All were under the age of 7 years with an average of 2.7 years. Twenty-four were drowned at home and 10 elsewhere; of those drowned elsewhere, five, all boys, were trespassing at the time of the accident. Four of the drownings occurred during or shortly after swimming parties at which adults were present. Rainwater accumulating in pools that had been emptied for the winter caused five deaths. In only three cases was there any fencing around the pools; the victims had entered the area through gates that had been left open.

Serious morbidity may be caused by near drowning. We recently saw two boys, both aged 2½ years, who had been rescued from private swimming pools. Despite resuscitation and treatment in intensive care units they had severe brain damage and both required permanent hospital care.

Comment

Drowning in fresh water is rapid. A 50% chance of death has been calculated for unsupervised children who get into difficulties and lose consciousness in fresh water.¹ There is considerable morbidity among survivors of near drowning, and, though the long-term neurological outlook among children who survive may be surprisingly good, brain damage occurred in 20% in one series² of all near drownings in children. Surveys in Australia and Sweden of drownings in private swimming pools showed that the absence of adequate fencing was the main contributory factor, and it has been claimed that 80% of these deaths might be prevented by the provision of such a safety measure.^{3,4} Many countries have legislation that requires owners of domestic pools to ensure that they are adequately fenced. In New South Wales, for example, a 1.5 m fence or wall is required and gates must be self-closing and have a latch that is out of reach of small children. There

are similar regulations in many parts of the USA, Canada, and South Africa.

No such legislation exists in this country; it has been stated that the number of deaths is comparatively small and that it is difficult to justify safety regulations that affect private property. Accidents that leave a child dead or severely handicapped, however, are a tragedy, particularly when they most probably could have been prevented by simple safety measures. Although these deaths are a relatively small proportion of the 50 or so children aged under 5 who are drowned each year,⁵ they are preventable; it is surely not unreasonable, therefore, to require the owners of swimming pools, who are usually relatively wealthy, to ensure that they are safely enclosed.

We thank Mr R Ferguson of the Home Office and Mrs J Burton of the medical statistics unit, Office of Population Censuses and Surveys, for their help. The analysis and conclusions described in this article, however, are ours alone.

¹ Pearn J, Nixon J, Wilkey I. Freshwater drowning and near drowning accidents involving children. A five year total population study. *Med J Aust* 1976;**2**:942-6.

² Peterson B. Morbidity of childhood near drowning. *Pediatrics* 1977;**59**:364-70.

³ Thorson J, Fredin H, Gerdman P, Wiklund R. Accidental drowning among children in Sweden 1958-1967. *Acta Socio-Medica Scandinavica* 1970;**2**:3:149-59.

⁴ Pearn J, Thompson J. Drowning and near drowning in the Australian Capital Territory. A five year total population study of immersion accidents. *Med J Aust* 1977;**1**:130-3.

⁵ Office of Population Censuses and Surveys. *Mortality statistics 1979. Accidents and violence*. London: HMSO, 1981.

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Multiple hepatic abscesses complicating continuous ambulatory peritoneal dialysis

Peritonitis is common during continuous ambulatory peritoneal dialysis^{1,2} and may prove lethal.³ We report a case of relapsing peritonitis complicated by multiple hepatic abscesses.

Case report

A 41-year-old man had been receiving haemodialysis from 1970 to 1977. Continuous ambulatory peritoneal dialysis was started in March 1980 for terminal renal failure due to chronic rejection causing nephrotic syndrome. During the first nine months of continuous ambulatory peritoneal dialysis three episodes of peritonitis were recorded (overall incidence at this unit—one episode every 16 patient-months). After 11 months the patient was admitted to hospital for peritonitis; culture grew no pathogens. Continuous peritoneal lavage with cefalexin, in association with parenteral cefalexin and tobramycin, resulted in clinical improvement and after three days continuous ambulatory peritoneal dialysis was started again with the same antibiotic treatment. Despite the persistence of slight fever, antibiotics were stopped on the 14th day. Three days later, epigastric pain returned, peritoneal effluent became cloudy, and culture was positive for *Staphylococcus albus*, sensitive to cephalosporins. Intraperitoneal and parenteral treatment were given in accordance with "antibiogram," but despite initial relief abdominal pain reappeared after 11 days, localised in the right upper quadrant, radiating to the shoulder; palpation elicited extreme pain at the hepatic border, localised at the intersection of the outer edge of the rectus abdominis. Peritoneal fluid was purulent with a high leucocyte count; intramuscular tobramycin was started. Abdominal echotomography gave negative results.

Nineteen days after operation the patient underwent exploratory laparotomy. A corpuscolate fluid was found in the peritoneal cavity and a small area (8 × 3 cm) of swelling and reddening with microabscesses was found at the lower side of the right hepatic lobe, along the posterior edge (segments V, VII, VIII). Exploration puncture gave scant bloody fluid, but culture was positive for *Staph albus*. The abscesses were too small (1-4 mm in

diameter) to warrant surgical drainage, so the Tenckhoff catheter was removed and replaced by a stylet catheter. Antibiotic treatment included intraperitoneal and intravenous vancomycin and intravenous amikacin for three weeks, and erythromycin orally for four weeks. From the second postoperative day the patient underwent haemodialysis through an external femoral bypass. Thirteen days after operation hepatic scintigraphy showed no alteration attributable to abscess and the patient was discharged without antibiotic treatment. Twelve months after discharge the patient is undergoing haemodialysis and is rehabilitated to grade A 1 (code of the European Dialysis and Transplant Association).

Comment

Gram-positive micro-organisms, particularly *Staph albus*, are the most frequent causal agent of peritonitis during continuous ambulatory peritoneal dialysis.^{1,2} Generally, these agents are responsive to cephalosporins, which are used as initial treatment until the results of bacteriological culture are available.² The case described here illustrates that peritonitis due to *Staph albus* may lead to severe infectious complications, such as hepatic abscess. This patient presented predisposing factors such as nephrotic syndrome and a long period of immunosuppressive treatment before starting continuous ambulatory peritoneal dialysis. We believe that antibiotics were discontinued too soon, before the septic focus in the liver had been eradicated. Staphylococci are not usually the cause of hepatic abscesses.⁴ In the case described micro-organisms presumably reached the liver through the portal vein, though no such occurrence has been reported in patients undergoing continuous ambulatory peritoneal dialysis.

We recommend exploratory laparotomy for making a prompt diagnosis when other methods fail to show the cause of a persisting peritonitis.⁵ This may be of considerable help in establishing appropriate treatment for diseases such as hepatic abscesses which have a poor prognosis. Multiple hepatic abscesses do not lend themselves to adequate surgical drainage, and, though they can sometimes be evacuated by syringe, an aggressive schedule of specific antibiotic treatment maintained for a sufficiently long period remains the best possible way of achieving a cure.

¹ Rubin J, Rogers WA, Taylor HM, et al. Peritonitis during continuous ambulatory dialysis. *Ann Intern Med* 1980;**92**:7-13.

² Vas SI, Low DE, Oreopulos DG. Peritonitis. In: Nolph KD, ed. *Peritoneal dialysis*. The Hague: Martinus Nijhoff, 1981:344-65.

³ Oreopulos DG, Vas S, Khanna R. Treatment of peritonitis. In: Moncrief JW, Popowich RP, eds. *CAPD update*. New York: Masson Publishing, 1981:259-64.

⁴ Palmer ED. The changing manifestations of pyogenic liver abscess. *JAMA* 1975;**231**:192-4.

⁵ Fridday RO, Barriga P, Crummy AB. Detection and localization of intra-abdominal abscesses by diagnostic ultrasound. *Arch Surg* 1975;**110**:335-8.

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Myxoedema coma induced by beta-adrenoreceptor-blocking agent

Although the pathogenesis of myxoedema coma is uncertain, precipitating factors such as exposure to cold, infection, administration of central nervous system depressants, alveolar hypoventilation, and hyponatraemia are reported to predispose to its development.¹ Myxoedema coma induced by administration of beta-adrenoreceptor-blocking agents has not, however, been reported. We describe a patient with myxoedema coma induced by a beta-blocker who recovered consciousness after withdrawal of the drug.