

relaxation phases. He was given plentiful fluids and nutritious food and sedated with haloperidol 5 mg eight hourly. Within 48 hours his mental state was much improved and regular sedation was stopped. In view of the hypertensive heart disease thyroxine replacement was started cautiously—25 µg on alternate days, increasing weekly by 25 mg on alternate days to 50 mg daily. Transaminase activities became normal within a week, but renal impairment persisted (see table).

The patient spent one month in hospital, during which his mental state settled gradually and was normal by the time of discharge without psychoactive drugs.

Comment

This case fits the criteria for mania listed by the *Diagnostic and Statistical Manual of Mental Disorders* (3rd edition) and the ninth revision of the *International Classification of Diseases*. The association of mania and myxoedema must be very rare, and we can find no other reported case.^{1,5} The two usual psychiatric syndromes seen in myxoedema are, firstly, confusion and cognitive impairment resembling dementia, sometimes with clouding of consciousness; and, secondly, depressed mood with paranoid delusional ideas and often hallucinations.^{1,2} Irritability and violence are reported only in association with paranoid cases.²

Our patient showed no psychiatric disorder until further metabolic disturbance was superimposed on his thyroid deficiency. After surgery he had a mental disturbance which may have been mild hypomania, which suggests that he may have been particularly susceptible to develop a mental disturbance from metabolic abnormality as an expression of his genetic predisposition indicated by the positive family history.

We thank Professor J T Silverstone for his help and advice in preparing this report.

- 1 Whybrow PC, Prance AJ, Treadaway MD. Mental changes accompanying thyroid gland dysfunction. *Arch Gen Psychiatry* 1969;20:48-63.
- 2 Olivarius BF, Roder E. Reversible psychosis and dementia in myxoedema. *Acta Psychiatr Scand* 1970;46:1-13.
- 3 Silverstone T, Cookson J. The biology of mania. In: Granville Grossman K, ed. *Recent advances in clinical psychiatry* 4. Edinburgh: Churchill Livingstone, 1982:201-41.
- 4 Krauthammer C, Klerman GL. Secondary mania. *Arch Gen Psychiatry* 1978;35:1333-9.
- 5 Stasiek C, Zetin M. Organic manic disorders. *Psychosomatics* 1985;26:394-402.

(Accepted 11 August 1987)

Shenley Hospital, Shenley, Radlett, Hertfordshire WD7 9HB

GABRIELLA ZOLESE, MD, psychiatric registrar

RITA HENRYK-GUTT, MRCP, MRCPsych, consultant psychiatrist

Correspondence to: Dr Zolese.

Faecal peritonitis induced by Picolax

We report a case of faecal peritonitis after the use of Picolax (sodium picosulphate) before barium enema examination. The cause was a perforated diverticulum of the sigmoid colon 10 cm proximal to an obstructive rectosigmoid carcinoma. We believe that this is the first report of faecal peritonitis after the administration of Picolax.

Case report

A 73 year old man was admitted to hospital complaining of abdominal pain of sudden onset that had become worse in the hour before admission. On examination the abdomen was distended, silent, and rigid. Blood pressure was 100/80 mm Hg and the pulse rate 120/min. An erect chest radiograph showed gas under both diaphragms. At laparotomy liquid faeces were found in the peritoneal cavity and the sigmoid colon proximal to an obstructing carcinoma of the rectosigmoid junction was perforated. There was moderate diverticular disease of the descending colon above the perforation. Hartmann's procedure and peritoneal lavage were performed. Histological examination of the resected specimen showed a moderately differentiated adenocarcinoma of the sigmoid colon and a perforated diverticulum 10 cm proximal to the carcinoma; there was spread to the lymph nodes. The intervening colon was reported as being normal. Postoperatively the patient developed septicæmic shock and renal failure, which failed to respond to treatment. He died three weeks later.

One month before admission the patient had attended the outpatient department complaining of diarrhoea and abdominal pain. He did not give a history of passing blood, and a rigid sigmoidoscopy to 15 cm did not show any abnormalities. A barium examination of the colon was planned for the day on which he was admitted. During the evening before admission he had taken two sachets of Picolax in preparation for the barium enema.

Comment

Carcinoma and diverticular disease are common disorders of the colon. Their diagnosis often depends on a barium enema or colonoscopic examination, which in turn depend on good preparation of the bowel. Picolax is a stimulant laxative containing sodium picosulphate and magnesium citrate that has been used for both barium and colonoscopic examinations with good results.^{1,2} The perforation in this case was due to diverticular disease and was precipitated by Picolax and the distal obstructing carcinoma. Rose *et al* stated that Picolax should be used with caution in patients with potentially obstructive lesions or diarrhoea,² and we endorse this view. We would add that any patient suffering from diarrhoea or abdominal pain in whom a carcinoma is suspected should undergo flexible sigmoidoscopy before being given Picolax. If there are clinical signs of obstruction Picolax should not be used. In cases in which the bowel appears normal on flexible sigmoidoscopy we believe that Picolax should be given under medical supervision—that is, while they are in hospital for the ensuing examination.

We emphasise that Picolax is a safe and efficient means of colonic preparation. For its safe record to be maintained patients at risk must be preselected for careful assessment before it is used.

We are grateful to Dr J Burston for the histological examination and to Anne Reavley for secretarial help.

1 De Lacy G, Benson M, Wilkins R. Routine colonic lavage is unnecessary for double contrast barium enema in outpatients. *Br Med J* 1982;284:1021-2.

2 Roe AM, Jamieson MH, MacLennan I. Colonoscopy preparation with Picolax. *J R Coll Surg Edinb* 1984;29:103-4.

(Accepted 26 August 1987)

Department of Surgery, Queen Alexandra Hospital, Portsmouth

R F PHIPPS, FRCS, research fellow

S FRASER, FRCS, surgical registrar

Correspondence to Dr Phipps.

Incidence of the premenstrual syndrome in twins

The premenstrual syndrome is a recognised clinical entity with both psychiatric and physical symptoms,¹ but its cause remains in dispute.² This study was instituted to determine whether a genetic factor is concerned.

Subjects, methods, and results

The premenstrual syndrome is defined as the recurrence of symptoms limited to the premenstruum with complete absence of symptoms for at least seven consecutive days in the postmenstruum.² Subjects were drawn from a premenstrual syndrome clinic run by KD, and all 108 index patients had completed a three month prospective menstrual chart that had resulted in a positive diagnosis of the premenstrual syndrome. There were 15 pairs of monozygous twins and 16 pairs of dizygous female/female twins; their zygosity was determined from their medical records after the diagnosis of the premenstrual syndrome had been confirmed. Controls with confirmed premenstrual syndrome and at least one sister were enlisted between May and October 1986. Siblings were regarded as suffering from the syndrome if they had received treatment for it and it had been confirmed by a menstrual chart. The female siblings of 68 (52%) of the subjects were interviewed, information on the remaining siblings being obtained from the subjects' personal knowledge or from correspondence. No twins or control siblings had been reared separately. Any previous medical and psychiatric illnesses in index subjects and in male and female siblings were also recorded. The data were analysed by the χ^2 test.

Among the 15 sets of monozygous twins both twin siblings suffered from the premenstrual syndrome in every case except one, whereas in the group of dizygous twins only seven of the 16 twin siblings suffered. The 77 controls had 121 female siblings, of whom 38 suffered from the syndrome. This gives a p value of <0.001 for monozygous twins versus controls but no significant difference between dizygous twins and controls. The incidence of previous medical or psychiatric illness among twins, controls, and their siblings was similar.

Comment

Twin studies are a useful method of illustrating "horizontally" the genetic element of disease.³ In the one monozygous twin pair in which only the index patient had the premenstrual syndrome she had started to suffer from it after the birth of the first of her two children, whereas her sibling was nulliparous.

Dalton reported that the premenstrual syndrome started after pregnancy in two fifths of 769 women,² so will this monozygous twin sister remain free from it after she has borne children?

Kantero and Widholm found a highly significant correlation for premenstrual symptoms (as opposed to the premenstrual syndrome) in Finnish adolescent daughters and their natural mothers.⁴ This further supports the suggestion that there is a genetic element in the aetiology of the premenstrual syndrome.

Our findings contribute to the theory that the study of nature, not nurture, holds the key to the aetiology of the premenstrual syndrome. Thus probably inquiries into antenatal factors and childhood environment are unfruitful topics for research into its aetiology and resources would be better employed investigating endocrinological factors.

1 Massil H, O'Brien PMS. Premenstrual syndrome. *Br Med J* 1986;293:1289-92.

2 Dalton K. *Premenstrual syndrome and progesterone therapy*. 2nd ed. London: Heinemann Medical, 1984.

3 Erlennmeyer-Kinling L. Genetic approaches to the study of schizophrenia: the genetic evidence as a tool in research. *Acta Genet Med Gemellol (Rome)* 1983;32:53-88.

4 Kantero RL, Widholm O. Correlations of menstrual traits between adolescent girls and their mothers. *Acta Obstet Gynecol Scand* 1971;14 (suppl 14):30-6.

(Accepted 3 September 1987)

London W1

KATHARINA DALTON, MRCS, FRCGP, obstetrician and gynaecologist

St James's University Hospital, Leeds LS9 7TF

MAUREEN E DALTON, MB, MRCOG, lecturer

KATHERINE GUTHRIE, MB, BSC

Correspondence to: Dr Katharina Dalton, 100 Harley Street, London W1.

Continuous arteriovenous haemofiltration in patients with hepatic encephalopathy and renal failure

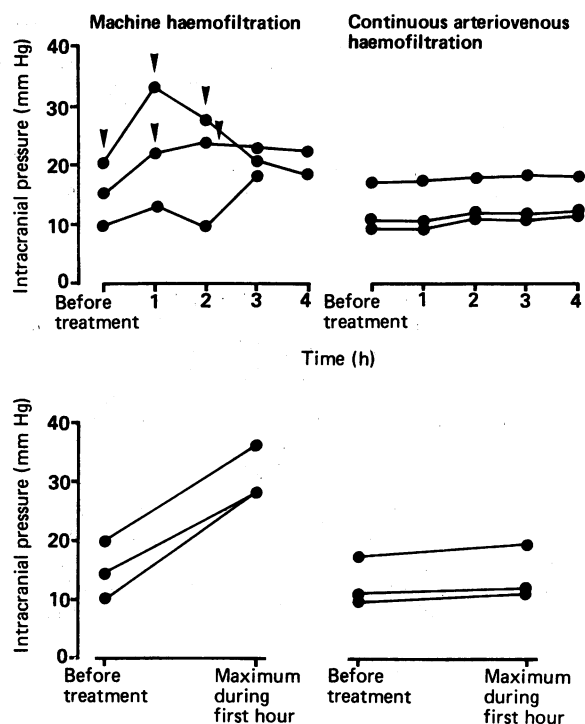
The mortality of patients admitted with fulminant hepatic failure remains high.¹ Charcoal haemoperfusion and dialysis with a polyacrylonitrile membrane have both been reported to increase survival.² Though death is often due to multiple complications, cerebral oedema is a common and serious problem.³ Treatments designed to control raised intracranial pressure are not always successful; hyperventilation does not produce a sustained reduction in pressure, and mannitol, which can control raised intracranial pressure, is not as effective in patients who have renal failure or appreciably increased pressures.⁴

We treated a patient who had hepatorenal failure by two methods of haemofiltration, with different effects on intracranial pressure.

Case report

A previously healthy 25 year old woman took 100 paracetamol tablets (0.5 g each) pending a divorce settlement. Three days later she started to feel unwell, became confused, and was admitted to the regional liver unit with grade IV hepatic encephalopathy. On arrival she responded only to deep pain and showed icterus with anuria. Blood biochemical measurements showed: paracetamol 36 mg/l, urea 18.4 mmol/l, creatinine 664 µmol/l, maximum prothrombin ratio 4.9, maximum alanine aminotransferase >3000 IU/l, and glucose 5.1 mmol/l. She was ventilated, and an intradural pressure transducer was inserted to monitor intracranial pressure. She was initially treated by machine driven haemofiltration with a Gambro FH77 haemofilter and a 17 litre exchange of fluid. Her intracranial pressure increased from 20 mmHg to 36 mmHg during the first hour of treatment despite bolus mannitol and then decreased to 19 mmHg after further mannitol.

The next day she was treated by continuous arteriovenous haemofiltration; there was no change in intracranial pressure during the procedure. On each of the following two days she received machine driven haemofiltration, and on both occasions her intracranial pressure increased during the first hour. Despite mannitol the intracranial pressure increased during treatment. She was then treated by continuous arteriovenous haemofiltration, and once again no change was noted in intracranial pressure (figure). After five days her urine output improved, and after a further week continuous arteriovenous haemofiltration was stopped. She made a rapid recovery and was discharged home with no residual clinical effects.



Changes in mean intracranial pressure during machine haemofiltration and continuous arteriovenous haemofiltration and maximum sustained intracranial pressure during first hour of treatment. ▼ = Points at which mannitol was given.

Comment

Evidence from the use of extracorporeal membranes in other studies is relevant to these findings. Patients who have chronic renal failure and are undergoing regular haemodialysis have been shown to develop cerebral oedema, and patients who have acute renal failure complicated by an increased intracranial pressure may deteriorate rapidly during haemodialysis.⁵ An increase in intracranial pressure has been reported in patients who have grade IV hepatic coma treated by polyacrylonitrile membrane haemodialysis.

It has been suggested that haemofiltration, by minimising systemic osmotic gradients, would be a better approach than haemodialysis and would diminish both the incidence and severity of cerebral oedema. In this case continuous arteriovenous haemofiltration, with an ultrafiltration rate of 1000 ml/hour, caused less change in intracranial pressure than machine driven haemofiltration, with a 17 litre exchange of fluid in three to four hours. Continuous arteriovenous haemofiltration may therefore have an advantage in the treatment of patients who have hepatic encephalopathy complicated by acute renal failure.

1 Silk DBA, Williams R. Experiences in the treatment of fulminant hepatic failure by conservative therapy, charcoal haemoperfusion and polyacrylonitrile haemodialysis. *Int J Artif Organs* 1978;1:29-33.

2 Silk DBA, Trewby PN, Chase RA, et al. Treatment of fulminant hepatic failure by polyacrylonitrile membrane haemodialysis. *Lancet* 1977;ii:1-3.

3 Ware AJ, D'Agostino A, Combes B. Cerebral oedema: a major complication of massive hepatic necrosis. *Gastroenterology* 1971;61:877-84.

4 Canalese J, Gimson AES, Davis C, et al. Controlled trial of dexamethasone and mannitol for the cerebral oedema of fulminant hepatic failure. *Gut* 1982;23:625-9.

5 Davenport A, Goldsmith HJ. Haemofiltration in the management of patients with acute renal failure complicated by raised intracranial pressure. *Lancet* 1987;ii:216.

(Accepted 11 August 1987)

Departments of Renal Medicine, Medicine, and Anaesthetics, St James's University Hospital, Leeds LS9 7TF

A DAVENPORT, MA, MRCP, research registrar in renal medicine

E J WILL, BM, MRCP, consultant nephrologist

M S LOSOWSKY, MD, FRCP, professor of medicine

S SWINDELLS, BSC, FFARCS, consultant anaesthetist

Correspondence to: Dr Davenport.