in association with amitriptyline, nortriptyline, desipramine, clomipramine, dothiepin, lofepramine, tranylcypromine, thioridazine, fluphenazine, haloperidol, and carbamazepine.

A number of psychiatric conditions may be complicated by polydipsia, which may in turn lead to hyponatraemia. The literature has been thoroughly reviewed by Illowsky & Kirch (1988). Schizophrenia is the most frequent diagnosis. The prevalence of polydipsia may range from 6% to 17% of chronically ill patients, of whom 25% to 50% may develop symptoms of water intoxication. CPM has been described in a chronic schizophrenic whose hyponatraemia followed self-induced vomiting and subsequent water drinking (Haibach et al, 1986).

Finally, often malnourished and with a documented hyponatraemia, alcoholics are over-represented among cases of CPM (Arieff, 1985). They also constitute a group which tends to gravitate towards the psychiatric services.

We, therefore, recommend that patients with chronic mental illness, and those receiving psychoactive drugs, have periodic review of their electrolyte levels; electrolytes should always be measured on admission to hospital. This is especially important in chronic alcoholics. Where a psychiatric opinion is sought on a patient whose immediate past history includes deranged serum sodium levels, if not alcoholism or malnourishment, a diagnosis of CPM should be entertained.

Acknowledgements

The authors would like to thank those members of the several medical and surgical teams involved in the care of this patient, and also Dr P. R. Beck, Consultant Clinical Biochemist, Dr S. Beck, Consultant Pathologist, and Dr C. M. L. Smith, Consultant Neuropathologist.

References

- ADAMS, R. D., VICTOR, M. & MANCALL, E. L. (1959) Central pontine myelinolysis. Archives of Neurology and Psychiatry, 81, 154-172
- ARIEFF, A. I. (1985) Effects of water, acid-base and electrolyte disorders on the central nervous system. In *Fluid Electrolyte and Acid-Base Disorders* (eds A. I. Arieff & R. A. DeFronzo), pp. 992-1007. New York: Churchill Livingstone.
- ESTOL, C. J., FARIS, A. A., MARTINEZ, A. J., et al (1989) Central pontine myelinolysis after liver transplantation. Neurology, 39, 493-498.
- Gerber, O., Geller, M., Stiller, J., et al (1983) Central pontine myelinolysis: resolution shown by computer tomography. Archives of Neurology, 40, 116-118.
- HAIBACH, H., ANSBACHER, L. E. & DIX, J. D. (1986) Central pontine myelinolysis: a complication of hyponatraemia or of therapeutic intervention? *Journal of Forensic Sciences*, 32, 444-451.
- ILLOWSKY, B. P. & KIRCH, D. G. (1988) Polydipsia and hyponatraemia in psychiatric patients. American Journal of Psychiatry, 145, 675-683.
- LAURENO, R. (1983) Central pontine myelinolysis following rapid correction of hyponatraemia. Annals of Neurology, 13, 232-242.
 MESSERT, B., ORRISON, W. W., HAWKINS, M. J., et al (1979)
 Central pontine myelinolysis: considerations on etiology, diagnosis,
- and treatment. Neurology, 29, 147-160.

 RAGLAND, R. L., DUFFIS, A. W. & GENDLEMAN, S. (1989) Central pontine myelinolysis with clinical recovery: MR documentation. Journal of Computer Assisted Tomography, 13, 316-318.
- WRIGHT, D. G., LAURENO, R. & VICTOR, M. (1979) Pontine and extrapontine myelinolysis. *Brain*, 102, 361-385.

*Christopher L. Kelly, MBChB, MRCPsych, Consultant Psychiatrist, Rotherham District General Hospital, Moorgate Road, Oakwood, Rotherham S60 2UD; Peter McColl, BA, MBChB, Psychiatric Registrar, Rotherham District General Hospital, Moorgate Road, Oakwood, Rotherham S60 2UD

*Correspondence

Chronic Superior Mesenteric Artery Syndrome in Anorexia Nervosa

MOSTAFA H. ELBADAWY

An 18-year-old woman presented with SMAS: an additional diagnosis of anorexia nervosa was later made. Both conditions should be considered when an adolescent presents with weight loss and vomiting. *British Journal of Psychiatry* (1992), 160, 552-554

The superior mesenteric artery syndrome (SMAS), or vascular compression of the duodenum, has been recognised for over 100 years as a cause of

obstruction and gastric dilatation. The transverse portion of the duodenum lies on the vertebral bodies and is crossed anteriorly by the arch of the superior mesenteric artery, which is held away from the aorta by the retroperitoneal pad of fat. Narrowing of the aortomesenteric angle occurs when the retroperitoneal fat is lost in conditions such as burns, prolonged bed rest, rapid growth in children without corresponding weight gain, and anorexia nervosa, or when the body

is held in hyperextension, whether on a frame or in a body cast. However, it is thought that for the syndrome to occur there must be, in addition, predisposing factors such as an abnormally high and fixed position of the duodenum and an abnormal course of the artery (Mansberger, 1986).

There are acute, subacute, and chronic forms of the disorder, in all of which the patient suffers from post-prandial bloating and pain, followed by nausea and vomiting of bile-stained material. Chronic patients gradually learn to limit their food intake to reduce the discomfort and the vomiting.

The diagnosis is usually confirmed radiologically, the classic signs to be seen on barium examination being dilatation of the stomach and the duodenum down to the level of the superior mesenteric artery, delay in barium passage, a sharp oblique extrinsic defect corresponding to the course of the artery, and relief of the obstruction by turning the patient to the left lateral or prone positions. Simultaneous aortography gives more definitive information but is rarely required in clinical practice.

There are reports of the association of anorexia nervosa with the acute (Froese et al, 1978; Pentlow & Dent, 1981) and subacute forms (Sours & Vorhaus, 1981), but none where it is associated with the chronic form. The following report is of an adolescent girl in whom the syndrome developed insidiously and took a chronic course.

Case report

An 18-year-old single woman presented to the general surgeon, giving a four-year history of heartburn, epigastric pain, and vomiting following the ingestion of any solid food. She avoided eating so as not to exacerbate her symptoms. She had been treated in various clinics and hospitals, but was unable to furnish any details. Her blood count, electrolytes, and liver function were all within normal range. A barium meal revealed a "stomach dilated down to the symphysis pubis, gross dilatation of the proximal two parts of the duodenum, delay in gastric emptying, and a straight extrinsic defect across the third part of the duodenum". Fluoroscopy revealed that "turning the patient to her left side allowed the barium to flow into the distal duodenum". The surgeon made a diagnosis of SMAS and began to treat her conservatively. However, he felt she needed psychiatric help and referred her for psychiatric assessment.

The patient complained to the psychiatrist that she suffered from palpitations, tremors, tightness in the chest, disturbed nights, and frequent nightmares about fires and snakes which woke her, screaming. She denied she had ever tried to lose weight, and said she often felt distressed for no obvious reason.

Both herself and her parents are uneducated, and the exact duration and chronological order of appearance of the symptoms were difficult to elucidate. She said she had been menstruating for a number of years and denied that anything was abnormal with her periods. Her weight was 35.8 kg and she was 168 cm tall. At that stage her symptoms were felt to be attributable to a prolonged physical illness and she was put on trifluoperazine (1 mg t.d.s.).

Three months later it was decided that conservative treatment had failed and a gastrojejunostomy was performed, following which her physical symptoms were greatly improved and her weight began to increase. She failed to attend a psychiatric follow-up appointment. A few months later another psychiatric consultation was requested since her weight had not increased as expected. She was still living on fluids, was felt to be neglecting herself, and her periods were said to be becoming irregular. When seen again by the psychiatrist she declared that, contrary to her previous statements, it had not been because of the pain that she did not eat before the operation - she never felt hungry and felt her weight to be quite adequate. She later said that long before surgery her periods had not been regular and that now they recurred every 15-18 days, with little bleeding. She still suffered from insomnia and dyspnoea, and in addition complained of lower abdominal pain with each period. Both the patient and her parents became extremely defensive whenever an attempt was made to explore the family relationships. Her weight was then 36.7 kg.

She was admitted for two days, during which she refused to eat, despite reassurance that with the medication and special regime she was prescribed there would be no pain. She would not be coaxed by the nursing staff into tasting anything, and became extremely hostile when the attempts were repeated at short intervals. She evaded them by remaining in the bathroom for long periods, promising to eat the next meal but not this one, demanding that her parents bring her home-made food, which she then refused.

She then convinced her parents to discharge her against medical advice. Six months later she presented to the general physician with the same abdominal complaints. An endoscopy revealed that her stomach had returned to a normal size, but there was evidence of mild duodenitis. He felt that her complaints were disproportionate and should have responded more easily to symptomatic treatment; he referred her back to the psychiatrist. She remained silent for most of the interview, letting her parents reiterate their doubts that there was anything psychiatric in her complaints. She could not consider any kind of nutritional programme, maintaining that her weight was normal. She continued to appear in the casualty department every few months, was seen by the orthopaedic surgeon on many occasions for complaints of joint pains, and refused his suggestion that she might need psychiatric help.

Discussion

Superior mesenteric artery syndrome may be associated with anorexia nervosa in four possible ways. Acute or subacute SMAS may complicate a diagnosed case of anorexia, the danger here lying in regarding the vomiting as psychogenic and part of the anorexia syndrome (Froese *et al*, 1978; Pentlow & Dent, 1981; Sours & Vorhaus, 1981). Secondly, chronic SMAS

554 ELBADAWY

may simulate anorexia (Kornmehl et al, 1988). In both these instances, there is a danger of missing the obstruction, with resultant delays in treatment. Thirdly, chronic SMAS may complicate anorexia, in which case either syndrome may be diagnosed but probably not both. The fourth possibility is for chronic SMAS to precipitate anorexia.

The delay in diagnosing anorexia nervosa in this case was partly due to the attitude of the patient, who was reluctant to provide the information required, and that of the parents, who thought it to be irrelevant, and partly because of the severity of the organic condition. Although some residual symptoms of the same nature as the original ones are expected to persist postoperatively, there are two main differences between the pre- and post-operative states: the first is that all patients are expected to regain normal weight (Ylinen et al, 1989), and the second that the vomiting mostly stops altogether and the other symptoms are milder and respond to symptomatic treatment. In this patient the vomiting did stop, and in the immediate post-operative period she complained of very little pain or distension. However, she failed to gain any weight, and her gastric complaints increased and failed to respond to symptomatic treatment, which induced the treating physician to seek further psychiatric help. The resistance then exhibited by the patient to such help, her refusal, against all evidence, to admit that she was underweight, and the fact that her menstrual periods had been irregular for much longer than she at first admitted and, at the time of her presentation, were not far from amenorrhoea, all indicate a diagnosis of anorexia nervosa.

There remains the question of which of the two conditions, SMAS or anorexia nervosa, preceded and helped to precipitate the other. This patient's

symptoms made their first appearance when she was about 14 years of age, which is indeed a period of rapid growth. She was presumably anatomically predisposed to develop SMAS should any factor intervene to prevent her weight from increasing to match the increase in height, with a consequent failure to build up an adequate retroperitoneal pad of fat. As there seems to be no other apparent reason for it, it may be surmised that it was the onset of anorexia at that stage that provided that factor.

As the number of reports associating SMAS with anorexia increase, it becomes essential to consider both conditions whenever faced with weight loss and vomiting in an adolescent.

References

FROESE, A. P., SZMUILOWICZ, J. & BAILEY, J. D. (1978) The superior mesenteric artery syndrome: cause or complication of anorexia nervosa. Canadian Psychiatric Association Journal, 23, 325-327.

KORNMEHL, P., WEIZMAN, Z., LISS, Z., et al (1988) Superior mesenteric artery syndrome presenting as an anorexia nervosa illness. Journal of Adolescent Health Care, 9, 340-343.

Mansberger, Jr, A. R. (1986) Vascular compression of the duodenum. In *Textbook of Surgery* (13th edn) (ed. D. C. Sabiston, Jr). Tokyo: Igaku-Shoin/Saunders.

PENTLOW, B. D. & DENT, R. G. (1981) Acute vascular compression of the duodenum in anorexia nervosa. *British Journal of Surgery*, 68, 665-666.

SOURS, J. A. & VORHAUS, L. J. (1981) Superior mesenteric artery syndrome in anorexia nervosa: a case report. *American Journal* of Psychiatry, 138, 519-520.

YLINEN, P., KINNUNEN, J. & HOCKERSTEDT, K. (1989) Superior mesenteric artery syndrome: a follow up study of 16 operated patients. *Journal of Clinical Gastroenterology*, 11, 386-391.

M. H. Elbadawy, MB, BCh, MRCPsych, Consultant Psychiatrist, Madina National Hospital, PO Box 1969, Madina, Saudi Arabia

The Association Between Triple X and Psychosis

WENDY J. WOODHOUSE, ANTHONY J. HOLLAND, GREG McLEAN and ADRIANNE M. REVELEY

Two cases of psychotic illness in association with the karyotype triple X showed specific diagnostic and management problems as well as obstetric complications, EEG abnormalities, and lack of a family history of psychiatric disorder. Routine karyotyping during the investigation of psychosis is becoming relevant to psychiatric practice as research reports increasingly feature genetic and chromosome anomalies in association with schizophrenic psychoses.

British Journal of Psychiatry (1992), 160, 554-557

The search for congenital and acquired organic abnormalities in the functional psychoses has a long

history. Pharmacological agents and cerebral pathology have regularly been reported in association with schizophrenia-like psychoses. Initially, these were seen as rare syndromes, but this did not decrease their interest to those involved in the search for an aetiopathology of schizophrenia. It has now become clear that cerebral abnormality is quite common in schizophrenia, with up to one-third of cases showing enlarged lateral ventricles (Johnstone et al, 1976), and minor physical anomalies are also thought to occur more often among schizophrenics (Gualtieri et al, 1982).