Anorexia nervosa and pancreatic ascites

P. Isaacs¹*, A.J. Saunders², B.K. Rosen³ and G.E. Sladen¹*

¹Gastroenterology Unit and Departments of ²Diagnostic Radiology and ³Psychiatry, Guy's Hospital, London SE1 9RT, UK.

Summary: Acute alcoholic pancreatitis was undiagnosed in a patient with anorexia nervosa who subsequently developed pancreatic ascites and oedema, wrongly attributed to protein malnutrition alone. She became hyperphagic in an attempt to reverse the malnutrition and hence the abdominal swelling, indicating that the goal of attaining a thin shape was the major determinant of the eating disorder in this patient.

Measurement of ascitic fluid amylase concentration should be carried out in all patients with unexplained ascites.

Introduction

The disturbance of body image and desire to maintain a thin body shape is characteristic of anorexia nervosa. That the eating pattern is entirely governed by this goal was demonstrated in a patient who began to eat voraciously once she became convinced that this would reverse ascites which was later found to be due to alcoholic pancreatitis with a pseudocyst.

Case report

A 24 year old woman of Latvian parents had, during an unhappy adolescence, developed a habit of frequent fasting. She was obsessed with keeping thin and on leaving school worked as a barmaid. She had not menstruated for 18 months, the menarche having been at age 13. She lived with a close female friend with whom she shared a binge-fast eating pattern. They made excursions together to parties where they would both indulge in casual heterosexual activity and return home together to share a food binge. She felt rejected when the friend developed a permanent heterosexual relationship and she herself went on an alcoholic binge which was followed two days later by moderate abdominal pain for which she did not seek medical advice. Six weeks later she developed swelling of the abdomen and feet. She had a striking appearance being tall (176 cm) having long, thin limbs and a face heavily made-up to further accentuate its thinness in stark contrast with the protruberant abdomen.

There was gross oedema of the legs, vulva and lower

trunk and a tense ascites, body weight 61 kg. Investigations revealed macrocytosis, hyponatraemia and hypoalbuminaemia. Alkaline phosphatase and glutamyl transpeptidase were normal. Ultrasound examination of the abdomen showed only ascites, though the pancreas was not visualized. Clear ascitic fluid was aspirated which was sterile on culture and guinea-pig inoculation and which had a total protein concentration of 20 g/l. Laparoscopy, peritoneal biopsy and liver biopsy showed non-specific inflammation only.

The ascites was attributed to the hypoalbuminaemia secondary to severe malnutrition. The patient was told that reversal of the ascites was therefore dependent upon her eating a large amount of food. She began to eat voraciously and took an average of 3,100 calories per day but the ascites was not decreased by this or by spironolactone (200 mg/day) and a restricted sodium intake (200 mmol/day). Her weight was unchanged by this.

A second abdominal ultrasound examination, 6 months after her initial presentation, revealed the presence of an 11.5 cm diameter cyst in the left hypochondrium; the gallbladder was normal. The ascitic fluid had a high amylase content (1,500 U/l) and plasma amylase was also raised at 980 U/l (normal 330 U/l). As soon as the patient realized that the ascites and the resulting abdominal protruberance were not due to anorexia nervosa she discharged herself from hospital and reverted to her previous eating pattern. The ascites persisted but 3 weeks later she agreed to have the cyst aspirated. The ascites and oedema slowly decreased and she reached an oedema-free weight of 52 kg which she maintained despite continuing in an abnormal eating pattern and retaining her gaunt appearance. The pancreas appeared to

*Correspondence and present address: P. Isaacs M.D., M.R.C.P. Gastroenterology Unit, Victoria Hospital, Blackpool FY3 8NR, UK.

Accepted: 3 July 1986

have recovered in that she had a normal bowel habit with no suggestion of steatorrhoea. Her blood sugar was within the normal range. One year after her initial presentation she presented with epigastric pain. Her weight was 52 kg and scanty menstruation was occurring. On ultrasonography she was found to have a pseudocyst in the tail of the pancreas and three small gallstones in the gallbladder. She refused cholecystectomy and defaulted from follow-up.

Comment

The psychopathology of anorexia nervosa is widely debated (Herzog, 1985) but the characteristic eating behaviour, with fasting and food bingeing, is typically associated with a disturbance of body image and preoccupation with being thin (Garfinkel & Garner, 1983; Herzog, 1985). The importance of the goal of thinness in determining the eating behaviour was highlighted in this patient whose food avoidance was reversed once she became convinced that by eating more she would regain her thin shape. Alternatively, she may have begun eating simply with the intention of recovering her health, although this goal for most anorectics is not usually attractive enough to induce eating normally. She once again reverted to fasting and bingeing when the true aetiology of her ascites became apparent.

References

- BARKIN, J.S., SMITH, F.R., PEREIRAS, R., ISIKOFF, M., LEVI, J., LIVINGSTONE, A., HILL, M. & ROGERS, A.I. (1981). Therapeutic percutaneous aspiration of pancreatic pseudocysts. *Digestive Diseases & Sciences*, **26**, 585.
- COX, K.L., CANNON, R.A., AMENT, M.E., PHILLIPS, H.E. & SCHAFFER, (1983). Biochemical and ultrasonic abnormalities of the pancreas in anorexia nervosa. *Digestive Diseases and Sciences*, **28**, 225.
- EDELL, S.L. (1979). Ultrasonic differentiation of types of ascitic fluid. *American Journal of Radiology*, **133**, 111.
- GARFINKEL, P.E. & GARNER, D.M. (1983). The multideterminate nature of anorexia nervosa. In *Recent Developments in Anorexia Nervosa Research*, Darby, P.L., Garfin-

kel, P.E., Garner, D.M. & Coscina, D.V. (eds). pp. 3–14. Alan R. Liss: New York.

HERZOG, D.B. (1985). Eating disorders. *New England Journal of Medicine*, **313**, 295.

KAPLSON, K.B., MARTIN, E.C. & FANKUCHEN, E.I. (1982). Percutaneous drainage of pancreatic pseudocysts and abscesses. *Radiology*, **142**, 619.

SILVERMAN, J.A. (1983). Medical consequences of starvation; the malnutrition of anorexia nervosa: caveat medicus. In *Recent Developments in Anorexia Nervosa Research*, Darby, P.L., Garfinkel, P.E., Garner, D.M. & Coscina, D.V. (eds). pp. 293–299. Alan R. Liss: New York.

Chronic pancreatic ascites is a rare complication of pancreatitis usually occurring in association with a pseudocyst. The mechanism of fluid accumulation is presumed to be both transudation of pancreatic fluid through the cyst wall and stimulation of peritoneal secretion by the activated enzymes in the peritoneal cavity. This active secretion of fluid presumably accounts for the resistance of a pancreatic ascites to diuretic treatment as seen in this patient. Measurement of peritoneal amylase and an ultrasound examination of the pancreas (Edell, 1979) should be carried out in all cases of ascites of obscure origin to avoid the delay in diagnosis which occurred in this case. Ultrasound-guided needle aspiration (Barkin *et al.*, 1981) may be employed as a safe and effective method of treatment of pancreatic pseudocysts.