Myxoedema ascites

J. A. McM. TURNER
M.B., B.S., M.R.C.P.

J. RAPOPORT*
M.B., B.S., M.R.C.P.

Cardiothoracic Department, Central Middlesex Hospital, London NW10

Summary

A case of myxoedema ascites complicating ischaemic heart disease is reported. Partial thyroid hormone replacement therapy, given slowly, eventually produced a diuresis and complete resolution of the ascites after a delay of 4 weeks.

Introduction

Although effusions into body cavities are common in patients with myxoedema (Sachdev and Hall, 1975), ascites is decidedly less common. Kochen and Atkinson in 1963 in a review of the literature found only sixteen previously reported cases, and since then one case report has been published (Waskilewska and Komainicka, 1974). The diagnosis of myxoedema ascites is difficult since it often occurs in the elderly where other diseases are usually thought to be responsible. The cause of ascites in myxoedema remains unknown and the ascites may be massive (Paddock, 1950). Response to thyroid replacement therapy is characteristic and diagnostic, and occurs despite considerable delay in diagnosis (Pathy, 1955; Marzullo and Franco, 1939).

Case history

A 62-year-old male was admitted to the Central Middlesex Hospital with a 3-week history of abdominal swelling and increasing angina of effort. There was a past history of ischaemic heart disease, gout and ureteric colic. He had physical signs of gross ascites, but his jugular venous pressure was normal and he was not in congestive cardiac failure. Apart from his massive ascites no other abnormal physical signs were noted. Investigations revealed a normochromic normocytic anaemia (Hb, 11 g/dl) and a raised blood urea 13.2 mmol/l. His chest radiograph revealed cardiomegaly (cardiothoracic ratio 16.5 : 28 cm), but clear lung fields. His serum albumin was 36 g/l, liver function tests were normal and there was no proteinuria. Repeated bacteriological and cytological examination of the ascitic fluid showed no evidence of infection or malignancy. The protein content of the ascitic fluid was 46 g/l.

Correspondence: Dr J. A. McM. Turner, Department of Medicine, Middlesex Hospital, London W1N 8AA.

* Present address: Department of Nephrology, Rambam Government Hospital, Haifa, Israel.

Laparoscopy revealed no evidence of portal hypertension or cirrhosis and a liver biopsy was normal. A barium enema and a barium meal were both normal. An electrocardiogram showed low voltages and evidence of old inferior and anterior myocardial infarctions.

No clear diagnosis was made and initially he was treated with diuretics and trinitrin, but failed to show any improvement over a 2-week period and his weight and abdominal girth increased. Further questioning at this time revealed that his mental function had deteriorated over the past year, his voice had become low pitched, his speech slow and he had noted his skin had become dry. Investigations at this stage revealed an effective thyroid ratio 0.75 (normal range 0.86-1.13), protein-bound iodine 100 mmol/l, an exaggerated thyroid stimulating hormone response to thyroid releasing hormone. Thyroid antibodies were positive.

A diagnosis of ascites secondary to hypothyroidism was then considered. Because of his ischaemic heart disease replacement therapy was introduced cautiously. Initially triiodothyronine 10 µg/day increasing by 5 µg every 3 days for 2 weeks and then L-thyroxine starting at 50 µg/day and increasing to 75 µg after 2 weeks. There was no response for the first 26 days, but then his weight began to fall dramatically and fell from 86 kg to 68 kg over the next 6 weeks and the ascites disappeared.

Three months after discharge the patient was well, clinically euthyroid and had no evidence of ascites. At this time his therapy was L-thyroxine 150 µg/day, allopurinol 100 mg b.d. and practolol 100 mg b.d. However, 7 months after discharge he died suddenly at home and no post-mortem was held.

Discussion

Ascites occurring as a result of hypothyroidism is well documented, but its cause remains obscure. The actual mechanisms of production of fluid are unknown, but increased capillary permeability has been shown (McGavack and Schwimmer, 1944) and inappropriate anti-diuretic hormone secretion has been suggested (Leichty, Miller and Cohen, 1970). Nevertheless it appears that the extravasation of hygroscopic mucopolysaccharides into body cavities is largely responsible for the effusions of myxoedema

(Byrom, 1933). The reasons for abdominal localization of the fluid are not clear. The fluid of myxoedema ascites is characteristically straw-coloured and has the properties of an exudate with a protein concentration of 40–60 g/l and an electrophoretic pattern similar to that of serum (Taipole and Hokkanen, 1956). This may be helpful in the differential diagnosis in patients with cirrhosis and congestive cardiac failure where the protein content of the ascitic fluid is low and the fluid has the characteristic of a transudate.

Other workers (Paddock, 1950; Pathy, 1955) have shown a diuresis beginning at about 2 weeks after the initiation of L-thyroxine therapy. The initial response to L-thyroxine was disappointingly slow in the present case. Full thyroid replacement therapy had, however, not been attained in the patient at the stage when complete resolution of the ascites occurred.

Myxoedema ascites is rare, and its onset may be insidious, but when it occurs it is usually massive and the diagnosis is often delayed. Myxoedema should always be considered in the differential diagnosis of ascites since it is one of the few diseases for which treatment is simple and curative and the need for repeated paracentesis is thereby avoided.

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References

- BYROM, F.B. (1933) The nature of myxoedema. *Clinical Science*, **1**, 273.
- KOCHEN, R.S. & ATKINSON, M. (1963) Ascites in hypothyroidism. *Lancet*, **i**, 527.
- LEICHTY, R.D., MILLER, R.F. & COHEN, W.N. (1970) Myxoedema causing adynamic ileus, serous effusions and inappropriate secretion of anti-diuretic hormone. *Surgical Clinics of North America*, **50** (5), 1087.
- MCGAVACK, T.H. & SCHWIMMER, D. (1944) Problems in the treatment of cardiac failure in myxoedema. *Journal of Clinical Endocrinology*, **4**, 427.
- MARZULLO, E.R. & FRANCO, S. (1939) Myxoedema with multiple serous effusions and cardiac involvement (myxoedema heart). *American Heart Journal*, **17**, 369.
- PADDOCK, F.K. (1950) Massive ascites due to myxoedema. *New England Journal of Medicine*, **242**, 822.
- PATHY, M.S. (1955) A case of myxoedema with serous effusions. *British Medical Journal*, **2**, 772.
- SACHDEV, Y. & HALL, R. (1975) Effusions into body cavities in hypothyroidism. *Lancet*, **i**, 564.
- TAIPOLE, E. & HOKKANEN, E. (1956) The muco-protein levels of the ascitic and pleural fluids and their clinical significance. *Acta medica Scandinavica*, **55**, 113.
- WASKILEWSKA, J. & KOMAINICKA, R. (1974) A case of hypothyroidism with concomitant ascites. *Przegląd Lekarski (Krakow)*, **38** (8), 766.

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Free perforation of the small bowel in Crohn's disease

RODNEY J. CROFT
M.A., M.B., F.R.C.S.

Central Middlesex Hospital

Summary

A rare case of free double ileal perforation in previously asymptomatic and undiagnosed Crohn's disease of the terminal ileum is described. At operation, a primary resection and anastomosis of the diseased bowel was performed. The management of free perforation in Crohn's disease is discussed, together with a review of the literature. It is suggested that the absence of steroids in a previously undiagnosed case may favour primary resection and anastomosis.

Introduction

The first case of free perforation in ileitis was

Correspondence: Mr Rodney J. Croft, Senior Surgical Registrar, The Middlesex Hospital, Mortimer Street, London W1.

recorded by Arnheim (1935), and since then, more than 100 cases of this complication have been recorded (Menguy, 1972), and the incidence is estimated to be 1–2% (Steinberg, Trevor-Cooke and Alexander-Williams, 1973).

Most authors agree that primary resection of the diseased segment is the surgical treatment of choice, but in view of the peritoneal contamination, controversy exists regarding the timing of the anastomosis. Primary anastomosis is favoured by Waye and Lithgow (1967), and Graham and Baugh (1968), but Menguy (1972) favours an initial double-barrelled ileocolostomy with subsequent elective closure. The author's experience with a recent case has raised further points regarding this controversy.