

Linitis plastica due to gastric diverticulosis

S D Patel MB BS¹ D Semeraro ChB FRCPath²
R I Hall MD FRCS¹

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Linitis plastica is typically due to an infiltrating adenocarcinoma. The term, however, is descriptive rather than diagnostic.

CASE HISTORY

A man of 63 was admitted because of long-term weight loss and worsening symptoms of gastric outlet obstruction. There had been no abdominal pain or change in bowel habit, no haematemesis or melaena. Initial treatment was nasogastric aspiration, intravenous hydration and an intravenous proton pump inhibitor. Endoscopy showed moderate reflux and a poorly distensible stomach with gastric outlet obstruction; the appearances were in keeping with infiltrating adenocarcinoma (linitis plastica) but repeated biopsies showed only inflammatory changes. On CT there was pronounced thickening of the wall of the entire stomach, measuring up to 2 cm, with severe narrowing of the lumen (Figure 1). No gas bubbles were evident in the stomach walls. These findings supported the provisional diagnosis of carcinoma and staging laparoscopy was undertaken. This showed a greatly thickened and rigid stomach with the consistency of rubber. Because there was no evidence of spread beyond the stomach, a total gastrectomy was performed. Postoperative recovery was satisfactory. On histological examination the stomach showed typical changes of diverticular disease, with pouches of mucosa extending through the muscularis propria into adjacent fat (Figure 2). The muscularis propria was hypertrophic and the submucosa showed oedema and fibrosis. In the mucosa there were areas of chronic inflammation with some ulceration; no metaplasia, dysplasia or malignancy was seen.

COMMENT

The typical gastric diverticulum is solitary and does not cause symptoms.^{1,2} Diverticula are found in 0.03–0.3% of necropsies,³ with three-quarters of them high on the posterior wall of the stomach and the remainder in the



Figure 1 CT showing diffusely thickened stomach wall

prepyloric region.^{1,2} Multiple gastric diverticulosis is rarer still and is more likely to cause illness.¹ Of the previously reported cases only two have been associated with gastric outlet obstruction.^{4,5} Our patient represents the third such case and had diverticula throughout the entire stomach with relative sparing of the pylorus—in contrast to the patient of Schweiger *et al.*,⁵ whose diverticula were almost exclusively prepyloric.

With such a rare condition it is difficult to establish an aetiology. Most prepyloric diverticula are associated with peptic ulcer disease, malignant disease or previous surgery² but some may be congenital in origin. In addition, traction diverticula may result from extragastric disease such as pancreatitis or cholecystitis.⁵ With regard to the association with pyloric stenosis, one could hypothesize that the



Figure 2 Section through stomach showing multiple diverticula and diffuse gastric wall thickening

Departments of ¹General Surgery and ²Pathology, Derby City General Hospital, Uttoxeter Road, Derby DE22 3NE, UK

Correspondence to: Mr R I Hall

E-mail: Richard.Hall@derbyhospitals.nhs.uk

diverticula result from high intragastric pressure. Alternatively, prepyloric diverticula might lead to gastric stasis, pyloric ulceration and stenosis. In both instances local muscular weakness must play an important part since even longstanding pyloric stenosis is seldom associated with diverticulosis; gastric diverticula have lately been reported in a patient with Caroli's disease,⁶ a deficiency of the fibromuscular matrix of the biliary tree.

In the present case, the true diagnosis became evident only after gastrectomy. A technique that might possibly have led to earlier diagnosis is endoscopic ultrasound-guided fine-needle aspiration biopsy.⁷

REFERENCES

- Palmer ED. Collective review: gastric diverticula. *Int Abstr Surg* 1951;**92**:417–28
- Eras P, Beranbaum SL. Gastric diverticula: congenital and acquired. *Am J Gastroenterol* 1972;**57**:120–32
- Raffin SB. Diverticula, rupture and volvulus. In: Sleisenger MH, Fordtran JS, eds. *Gastrointestinal Disease*, 4th edn. Philadelphia: W B Saunders, 1989;735–40
- Itai Y, Kogure T, Akiyama H. Multiple gastric diverticulosis: report of a case. *Radiat Med* 1983;**1**:39–41
- Schweiger F, Noonan JS. An unusual case of gastric diverticulosis. *Am J Gastroenterol* 1991;**86**:1817–19
- Naraynsingh V, Maharaj D, Busby GO, Raju GC, Jankey N. Caroli's disease associated with gastric diverticulum. *West Ind Med J* 2000;**49**:175–6
- Vander Noot MR III, Eloubeidi MA, Chen VK, et al. Diagnosis of gastrointestinal tract lesions by endoscopic ultrasound-guided fine-needle aspiration biopsy. *Cancer* 2004;**102**:157–63

Low TSH in a patient with primary hypothyroidism

D Morganstein MA MRCP¹ N Mendoza MBBS FRCS²
N Strickland FRCP FRCS³ K Meeran MD FRCP¹

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For routine thyroid function testing many laboratories now confine themselves to assay of thyroid stimulating hormone (TSH). This will identify the great majority of affected patients¹ though it can miss secondary hypothyroidism.^{2,3} TSH measurement is also used to monitor thyroxine replacement, and on rare occasions this too can be misleading.

¹Endocrine Unit, Imperial College Faculty of Medicine, Charing Cross Hospital, London, ²Department of Neurosurgery, Charing Cross Hospital, London, ³Department of Imaging, Imperial College Faculty of Medicine, Hammersmith Hospital, London, UK

Correspondence to: Dr D Morganstein, Molecular Endocrinology, IRDB, Imperial College, Hammersmith Campus, Du Cane Road, London W12 0NN, UK

E-mail: d.morganstein@ic.ac.uk

CASE HISTORY

A woman of 66 was seen in the emergency department with a history of recurrent falls, urinary incontinence and new-onset confusion. She walked with a shuffling small-stepped gait and her abbreviated mental test score was 1 out of 10. 10 years earlier she had been found to have primary hypothyroidism, TSH 12 mU/L (reference range 0.3–4.2), and on treatment with thyroxine 100 µg daily the TSH had become normal. A year before the present episode her TSH was noted to have fallen to 0.49 mU/L, which in the absence of a free T4 was interpreted as suggesting that she was taking too high a dose of thyroxine. The dose was reduced to 75 µg daily and three months later the TSH was 1.26 mU/L. However, a further six months later the TSH had fallen to 0.2 mU/L and the thyroxine dose was once again reduced, to 50 µg daily.

CT of her head showed hydrocephalus and a large cystic lesion in the region of the pituitary. MRI confirmed these findings (Figure 1a,b). Her baseline pituitary hormonal profile showed a very high prolactin of 398 500 mU/L (<650). The TSH was now raised at 22.0 mU/L, with a free thyroxine of 9.6 pmol/L (9.0–26.0); she had not taken thyroxine for about two weeks because of her confusion. Further assessment revealed a follicle stimulating hormone of 0.4 IU/mL, undetectable luteinizing hormone, growth hormone 0.3 IU/L with an IGF-1 4.0 nmol/L (6.0–30.0), and cortisol 765 nmol/L. These results indicated partial hypopituitarism. Thyroid peroxidase antibodies were strongly positive. She had a bitemporal hemianopia on perimetry. She was restarted on thyroxine replacement.

Transcranial drainage of the intraventricular cystic lesion did not improve her clinical state or lessen the hydrocephalus (Figure 1c). She was therefore started on cabergoline 500 µg twice weekly, and over the subsequent nine months her tumour shrank (Figure 1d–f), her visual fields improved, and her confusion resolved completely, to the extent that she was able to proof-read this paper.

COMMENT

This patient with primary autoimmune hypothyroidism went on to develop a giant macroprolactinoma. We propose that the continuous decline in her TSH despite reduction of the thyroxine dose was due to partial pituitary failure. Such a phenomenon has been observed by others;⁴ the low TSH in this patient reflected the development of hypopituitarism, not over-replacement leading to thyrotoxicosis.

The routine measurement of TSH to monitor the adequacy of thyroxine replacement has in this case given a fascinating insight into the changes occurring in the thyroid axis as hypopituitarism developed. The use of TSH as the sole index of function led to erroneous interpretation and inappropriate reductions of thyroxine dosage. Only when