A diabetic haemodialysis patient with dysphagia and weight loss

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Introduction

Tissue diagnosis is essential for differentiating between conditions that present with identical symptoms, e.g. malignancy and chronic infections. Failure to obtain a tissue diagnosis has significant therapeutic implications. We present a case of a patient with chronic renal failure on haemodialysis who presented with dysphagia and weight loss and caused diagnostic difficulties that may have led to inappropriate treatment.

Case

A 61-year-old Asian male with type 2 diabetes mellitus, chronic renal failure, and on haemodialysis presented in July 2001 with a 12-month history of weight loss and a 1-month history of dysphagia, mainly to solids and occasionally to liquids. He had commenced continuous ambulatory peritoneal dialysis in April 1998, but had been transferred to haemodialysis in June 2000 because of ultrafiltration failure. Haemodialysis was performed using an internal jugular Tessio line. Arteriovenous fistula construction failed because of poor vasculature. Blood flow rates on haemodialysis were variable and his average Kt/V was 0.92 over the 12-month period from June 2000 to July 2001 (below the minimum value of 1.2 recommended by the UK Renal Association [1]). In September 2000, the patient was treated with Sertraline 50 mg for depression, associated with which was nausea and anorexia. However, he continued to lose weight and remained anorexic. He first developed symptoms of dysphagia in June 2001, and by July 2001 he had lost 21 kg in weight over the previous 12 months. He was admitted to hospital for further investigations and enteric feeding.

The patient was afebrile with evidence of weight loss but with no palpable lymph nodes. There was clinical evidence of pulmonary congestion and a right pleural effusion. A chest X-ray confirmed the pleural effusion but did not show any other abnormality. Laboratory findings (normal ranges in parentheses) included Hb 11.5 g/dl, white cell count 7.3 × 10³, lymphocyte 0.9 × 10³, platelets 374 × 10³ and ESR 96 mm/h. His C-reactive protein was 183 mg/l (0–6 mg/l) and serum albumin was 28 g/l. Liver function tests were normal. Blood cultures were negative. Diagnostic pleural aspiration and biopsy revealed blood-stained fluid and microscopy revealed macrophages and lymphocytes. There was no bacterial growth and no acid-fast bacilli were identified on ZN staining of pleural fluid or pleura. Biochemical analysis of the pleural fluid showed pH 7.54 (7.35–7.45), protein concentration of 36 g/l, albumin 14 g/l (36–52 g/l), and LDH 375 i.u./l (300–650 i.u./l). A barium swallow examination showed a barium collection outside the mid-esophagus just below the bifurcation of the trachea, indicating esophageal perforation and strongly suggesting the

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Fig. 1. Barium swallow showing barium collection outside the oesophagus, indicating a perforation.
possibility of an oesophageal malignancy (Figure 1). An upper gastrointestinal endoscopy revealed an oesophageal pouch, with an orifice size of ~1 cm, at 27–30 cm. The base of the pouch contained debris, appeared nodular, irregular and macroscopically had the appearance of a carcinoma. Oesophageal brushings were performed and sent for cytology and culture, but no biopsies were taken because of the substantial risk of further perforation.

A computed tomography scan of the patient’s thorax showed a mass lesion associated with the oesophagus below the level of the carina. The oesophageal lumen was distorted by a rounded mass on the left. Para-tracheal lymph nodes were enlarged and the scan confirmed an oesophageal perforation and leak. At this point, the clinical and radiological features were highly suggestive of a primary oesophageal carcinoma with mediastinal lymph node spread (Figure 2).

In order to obtain a tissue diagnosis, a repeat endoscopy was performed, but again tissue biopsy was not performed because of the risk of oesophageal perforation. Nasogastric tube placement was carried out and the patient was started on enteric feeding. Mediastinoscopy and lymph-node biopsy were also considered but were not performed because of the high risks of anaesthesia associated with his poor clinical condition and cardiac dysfunction. An oncology opinion was sought with a view to consideration of chemotherapy and/or radiotherapy for his presumed oesophageal carcinoma.

Four weeks after the patient’s first endoscopy, *Mycobacterium tuberculosis* was cultured from the oesophageal brushings.

**Discussion**

Oesophageal involvement as a presenting feature of tuberculosis is very rare. To our knowledge, this is the first reported case of oesophageal tuberculosis in a patient with chronic renal failure. Oesophageal involvement has been described in the non-nephrological literature, without evidence of pulmonary or overt systemic involvement, as in the case of our patient [2]. The mechanism of oesophageal involvement is felt to be due to retrograde lymphatic spread. The most common presenting symptoms are dysphagia with weight loss, haematemesis and vomiting being less common. The differential diagnosis includes carcinoma of oesophagus, benign stricture, viral and fungal oesophagitis and rarely Crohn’s disease. Endoscopy may show single or multiple superficial ulcers with smooth edges and greyish purulent base, or stricture with fibrotic reaction, and least commonly, multiple small miliary granulomata [3].

Radiological pointers suggestive of tuberculosis include chest X-ray evidence of mediastinal lymph nodes or a mass, or an apical lesion suggestive of past tuberculosis. The changes may be too subtle to appreciate. A computed tomography scan of the thorax will usually identify mediastinal lymph-node enlargements.

Definitive diagnosis is by isolation of tubercle bacilli. Histological evidence of caseating granulomata is unusual. Acid-fast bacilli may be demonstrated on culture of biopsy specimens or from gastric washings [4].

In our patient, oesophageal malignancy was the primary diagnosis, strongly supported by clinical and radiological features until the isolation of acid-fast bacilli in the oesophageal brushings provided the correct diagnosis of tuberculosis oesophagus. Failure to obtain tissue for culture and of histology would have had significant implications. The patient would have been treated for presumed oesophageal carcinoma (inappropriately) with radiotherapy and chemotherapy. Failure of initiation of antituberculous therapy would have led to further deterioration in the patient’s condition and probable demise.

Patients with chronic renal failure are at high risk of tuberculosis because of reduced cellular immunity and other factors including diabetes, malnutrition, ethnicity and dialysis inadequacy. It is likely that a combination of the above factors resulted in the lesion in this patient. Malnutrition is common in patients with end-stage renal failure and contributes to a variety of infections and co-morbidity in dialysis patients. Malnutrition in this patient was probably due to a combination of dysphagia and inadequate dialysis. As in our subject, nasogastric or gastrostomy feeding may be needed in these patients.

Tissue biopsy was not possible because of the risk of oesophageal perforation, and the patient’s poor clinical condition precluded general anaesthesia. However, this case emphasizes the point that tissue biopsy is essential for a diagnosis, whether it is malignancy or tuberculosis, to allow the correct treatment to be given.

**Teaching point**

Oesophageal tuberculosis should be considered in patients with severe and end-stage renal failure with
the above risk factors who present with weight loss, loss of appetite, and dysphagia.

Definitive diagnosis should be based on the isolation of acid-fast bacilli.

Radiological investigations, including computed tomography scans, may produce erroneous results pointing to malignancy, as in this case.

A high index of suspicion is necessary to suggest the diagnosis.

References

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