Case report

Inflammatory obstruction of oesophageal tubes

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SUMMARY Two patients, intubated for inoperable carcinoma of the oesophagus, recently presented with their tubes blocked by granulation tissue. This complication has not been previously reported. The tissue macroscopically resembled normal mucosa in one and recurrent tumour in the other. The presentation, aetiology, and significance of this granulation tissue is discussed.

Intubation to relieve dysphagia associated with malignant and occasionally benign strictures of the oesophagus is often the only practical option in patients with inoperable carcinomas or severe cardio-respiratory disability. Yet despite its use for upward of a century, complications following intubation often occur, of which obstruction of the tube is one of the most frequently encountered.¹ Food is the usual obstructing object,^{2,3} but other causes have been described – collapse of the tube through structural deterioration of the wall,^{2,4} recurrent tumour,⁵ or prolapse of mucosa into either end of the tube.^{1,6,7}

We recently dealt with two patients presenting with recurrent dysphagia following intubation. At oesophagoscopy the proximal ends of the tubes were blocked by tumour-like tissue which on biopsy proved to consist of granulation tissue alone.

Case reports

CASE 1

A 51 year old man presented with dysphagia for solid food and weight loss over the preceding two months. Barium swallow showed an irregular stricture of the oesophagus and oesophagoscopy showed a bleeding necrotic ulcer at 35 cm. He underwent subtotal oesophagectomy and partial

gastrectomy, an oesophagogastric anastomosis being performed in the neck. At operation enlarged nodes were found around the left gastric artery, and subsequent histology confirmed a mucus secreting adenocarcinoma with nodal metastases. He remained well for three years, but then dysphagia and weight loss recurred and on oesophagoscopy recurrent tumour was found at the anastomosis. The stricture was dilated and an Atkinson prosthetic tube (Key Med, Southend on Sea) inserted perorally. Radiotherapy, from a linear accelerator delivering 8 meV photons, was instituted giving a tumour dose of 5400 rads to the oesophagus in 18 sessions over 37 days.

He remained well for three months, when the onset of retrosternal pain and dysphagia heralded a blocked tube, which was confirmed by a gastrografin swallow. Endoscopy revealed a normal upper oesophagus but there was an operculum of tissue growing over the top of the tube and blocking it. This tissue was removed with biopsy forceps and a loop diathermy. Histology of the tissue showed ulcerated, acanthotic, stratified squamous epithelium without evidence of dysplasia or infiltrating malignancy. This was accompanied by a florid granulation tissue response extending into the submucosa. The areas of ulceration were associated with numerous bacterial colonies and fungal hyphae (Figs 1 and 2).

He made an uneventful recovery, but 10 days after discharge suffered a massive haematemesis at home and died. A postmortem examination was not performed.

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Fig. 1 Acanthotic squamous epithelium adjacent to obstructing lesion. $\times 200$ (original magnification).

CASE 2

A 59 year old woman presented with a three month history of dysphagia. Previous radiotherapy to a laryngeal carcinoma four years earlier made oesophagoscopy difficult due to fibrosis in the upper third of the oesophagus. A carcinoma was seen at 30 cm and this was confirmed by histology. There was associated necrotic tissue which contained fungal hyphae. Because of the radiation damage higher up, radical surgery was felt to be contra-

indicated and as the stricture was short, intubation with a Proctor Livingstone tube (Staniforth, Penarth, S Glam) was performed. Chemotherapy was instituted giving vincristine and adriamycin on the first day, DTIC (Dacarbazine) and 5 FU (Flouro Uracil) on the second day, and 5 FU alone on the third day. Eleven day later she became severely ill with oesophageal candidiasis associated with bone marrow depression and a neutropenia, the white cell count being $0.5 \times 10^9/l$. The infection responded

Fig. 2 Granulation tissue from obstructing lesion. $\times 500$ (original magnification).

slowly to oral nystatin and systemic antibiotics. Two months later she underwent palliative radiotherapy from a linear accelerator delivering 8 meV photons, receiving a tumour dose of 2400 rads to the lower oesophagus in 12 sessions over 33 days.

Two episodes of tube blockage by food debris followed requiring oesophagoscopy to clear the tube. The appearance of the mucosa was suspicious of tumour recurrence, but was not biopsied. Six months after presentation, return of her initial symptoms led to re-endoscopy, where what was thought to be obvious recurrent tumour was seen above the tube but again this was not biopsied. Two weeks later, complete obstruction occurred and oesophagoscopy on this occasion showed a tongue of tumour covering the posterior rim, growing down the tube and blocking it. The tumour mass was snared and removed. Histology showed oedematous squamous epithelium with basal cell hyperplasia and candidal infection. There was no evidence of recurrent malignancy.

Postoperatively she was no better. Repeat endoscopy showed similar tissue growing up into the lower end of the tube. A longer tube was therefore needed to pass beyond the obstruction and consequently an Atkinson prosthesis with a lower flange engaged below the macroscopic limit of the granulation tissue was inserted. She continued to find swallowing difficult, however, and dysphagia to all but liquids necessitated her readmission to hospital one month later. At oesophagoscopy on this occasion the whole oesophagus was seen to contain florid granulation tissue with blockage both above and below the tube. To try and keep a patent passage through to the stomach, the Atkinson tube was replaced by a long Celestin tube (Ambleletin Ltd, Tetbury, Glos). Bony metastases were widespread by this time, but she remained asymptomatic until her death from disseminated carcinoma two months later.

Discussion

To our knowledge, these are the first reported cases of oesophageal tube blockage caused by granulation tissue alone. Previous reports (*vide supra*) have all stressed tube blockage as a major complication of intubation, with food, recurrent tumour and mucosa being the agents most commonly cited. It is impossible, however, to imagine that granulation tissue formation has only been confined to these two cases.

In both patients, the appearances of the obstructing masses at oesophagoscopy have been unlike granulation tissue. In both patients the oesophageal mucous membrane proximal to the

obstruction was apparently quite normal.

In the first patient, the tissue macroscopically resembled normal mucosa. In their original paper, Adams and Enerson⁶ describing one case of mucosal prolapse into the funnel of a Mousseau-Barbin tube, noted that the obstruction was a 'polypoid, soft reddish mass' which microscopically showed 'diffuse oedema, hyperaemia, a low grade infiltrate of round cells and polymorphonuclear leucocytes . . . (and) . . . generally thinned but intact epithelium with numerous villiform projections.' The authors thought this represented normal mucosa but we feel this description could be reasonably interpreted as representative of organising granulation tissue. Weisel *et al*⁷ quoting two cases of mucosal prolapse into the funnel of the tubes discovered at necropsy, did not examine this tissue histologically.

In our second patient a confident diagnosis of recurrent tumour was made at oesophagoscopy. Intubation is performed when palliation is felt to be the only practical treatment. Oesophageal carcinoma is known to spread longitudinally, both by direct infiltration and lymphatic permeation. Should tube blockage subsequently occur, the finding of tissue resembling recurrent tumour above or below the prosthesis may well excite no comment and consequently not be biopsied, as in our patient. Our experience would suggest that in the intubated patient, the assumption that growth of tissue at either end of the tube represents tumour may be fallacious; the true incidence of recurrent carcinoma in these patients may, therefore, be much lower.

The aetiology of the granulation tissue is unknown. It seems reasonable to assume that prolonged trauma by the neck of the funnel against the mucosa was the main factor. The Proctor Livingstone tube has a 'fish mouth' at the proximal and distal ends, in an attempt to prevent pressure being exerted on the oesophageal wall by the rim of the prosthesis. In spite of this, granulation tissue developed in one patient with this tube in place. Both our patients underwent radiotherapy after intubation and this may also have been contributory.

The Atkinson prosthesis is manufactured from silastic rubber. The Proctor Livingstone and Celestin tubes are manufactured from latex rubber. Whether these materials are significant in the development of granulation tissue is unknown.

The presence of fungal hyphae in the granulations in both patients was an interesting finding. Acute oesophagitis due to candida is well described.⁸ Our second patient, who suffered an attack of acute oesophageal candidiasis during which she became extremely ill, showed most of the recognised predisposing factors – she had oesophageal pathology,

with candida associated with the carcinoma at the time of diagnosis, and she had recently undergone three days of chemotherapy which had caused a dramatic fall in the white count.

Although she responded to oral nystatin, it would seem likely in the light of subsequent events that her oesophagus was never fully cleared of fungus and the presence of the oesophageal tube undoubtedly aided the continuation of chronic infection and ultimately the formation of granulation tissue.

In the first patient, the role of candida in the aetiology of the granulation tissue was less certain. Symptomless forms of oesophageal candidiasis have been described,⁹ but these usually present with upper oesophageal benign strictures. In a recent prospective review of all biopsies in 465 consecutive upper gastrointestinal endoscopies however, all investigated for the presence of candida, Scott and Jenkins¹⁰ found fungal hyphae invading tissue or ulcer slough in 4%. In none was oesophageal candidiasis suspected clinically and in all but two patients there was associated local pathology. Of 11 patients with carcinoma of the oesophagus, three showed candida microscopically. Our experience would support their contention that the presence of intramural candida is secondary to local mucosal injury, but that it may itself aggravate and perpetuate the underlying mucosal damage.

From two patients it is difficult to make any firm recommendations regarding the management of this complication of intubation, particularly as the prognosis in this group of patients is poor.

It is alarming, however, that formation of a large inflammatory mass in our first patient shortly preceded probable erosion of the tube into a major artery. Florid granulation tissue formation may therefore indicate extensive local damage to the oesophageal wall and presage perforation or erosion into an adjacent viscus.

In our second patient, dysphagia continued largely unaffected by local measures, and it was only when a short tube was replaced by a long Celestin tube, with the distal end sited in the stomach, that she experienced any benefit from intubation. In retrospect, effective control of the granulation tissue and eradication of the infection would seem to have required at least temporary removal of the

prosthetic tube, dilatation of the oesophagus and intensive antifungal treatment.

Scott and Jenkins¹⁰ concluded that routine antifungal therapy might be justified in patients intubated for carcinoma of the oesophagus. Although from our overall clinical experience of palliative oesophageal intubation we would not necessarily support this view, we would certainly recommend routine oesophageal biopsy should these patients require endoscopy, and appropriate treatment instituted if candida is found, in an attempt to prevent granulation tissue formation. In the presence of significant amounts of granulation tissue we would suggest removal of the prosthesis, intensive antifungal therapy if candida is present and replacement with a longer tube only if recurrent tumour growth makes it necessary.

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