Inflammatory obstruction of oesophageal tubes

T PHILP, A J GUNNING, AND M K BENNETT

From the Department of Thoracic Surgery, Churchill Hospital, Oxford; and Department of Histopathology, John Radcliffe Hospital, Oxford

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. Food is the usual obstructing object, but other causes have been described—collapse of the tube through structural deterioration of the wall, recurrent tumour, or prolapse of mucosa into either end of the tube. 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, anacanthotic, 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.
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 x 10⁹/l. The infection responded...

Fig. 1 Acanthotic squamous epithelium adjacent to obstructing lesion. ×200 (original magnification).

Fig. 2 Granulation tissue from obstructing lesion. ×500 (original magnification).

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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
recurring 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 conse-
quently an Atkinson prosthesis with a lower flap
engaged below the macroscopic limit of the granula-
tion 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 wide-
spread 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 al7 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 pre-
disposing factors – she had oesophageal pathology,
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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, 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 Jenkins 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 Jenkins 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.

References


