Dysphagia and regurgitation in a 10-year-old child

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A 10-year-old girl presented with a four-month history of progressive difficulties with swallowing, regurgitation and weight loss. Medical history did not reveal any episode of choking, dysphagia or respiratory symptoms. Dysphagia had worsened during the last two weeks. On admission physical examination was unremarkable. There was no swelling, tenderness or crepitus in her neck. Routine blood chemistry, tumour markers and urinary catecholamine metabolites were considered within normal limits. A barium swallow (figure 1) and thoracic computed tomography (CT) were performed (figure 2).

Questions

1 List the oesophageal disorders associated with dysphagia and regurgitation in infants and children.
2 What further investigations would be helpful?
3 Surgical exploration revealed an inflammatory para-oesophageal tumour in the mid-oesophagus region. What is the most likely diagnosis?
Answers

QUESTION 1
A wide variety of disease processes have to be considered in diagnostic work-up of a child presenting with dysphagia and regurgitation (box 1). Many of these possibilities can be ruled out by a precise medical history, physical examination, and basic investigations.

QUESTION 2
Oesophagoscopy gives very valuable information and sometimes offers, in addition, a therapeutic approach. In our case a small amount of granulation tissue and rigidity of the wall were found at the site of the stenosis. We were unable to explain a clashing sound produced when the wall of the oesophagus was touched at the region of the stricture.

QUESTION 3
The most likely diagnosis to be considered would be an inflamed foregut duplication or a congenital diverticulum. A right exploratory thoracotomy revealed a para-oesophageal inflammatory tumour in the region of the mid-oesophagus. We were unable to separate the mass from the oesophagus, so it was resected, together with the adherent part of the oesophagus. End-to-end anastomosis could be performed without tension. Analysis of the resected specimen surprisingly revealed a green, sharp-edged plastic lid within an inflammatory diverticulum. The entrance to this diverticulum was completely covered by hypertrophic mucosa. Only a narrow fistula track could be demonstrated. This diverticulum appeared to be the result of impaction and penetration of the radiolucent foreign body and subsequent mediastinitis.

Discussion
Ingestion of a foreign body into the oesophagus is a very common event in children. Generally, ingestion is associated with a witnessed choking episode and the foreign body is removed soon after. The common presenting symptom in the ‘acute’ phase are well known (excess drooling, poor feeding, dysphagia, inability to swallow, regurgitation, and oesophageal vomiting). Some patients may present with predominant respiratory symptoms such as stridor, wheezing or chronic pneumonia. In about 5% there is only a brief period of coughing and choking at the moment of ingestion which may not be witnessed or can be misinterpreted. Therefore diagnosis and adequate treatment can be delayed in these cases.

Some foreign bodies can penetrate the wall of the oesophagus and migrate extraluminally. Time, pressure and foreign tissue reaction allow a nearly symptomless penetration without a clinically obvious mediastinitis. The presenting features of such an ‘occult’ foreign body may be paradoxical and a wide spectrum of complications in chronic oesophageal foreign body have been reported (including stridor, perforation, mediastinitis, stenosis, tracheo-oesophageal fistula, damage of great vessels, aorto-oesophageal fistula). Progressive oesophageal obstruction and diverticulum in the mid-oesophagus as a result of penetration is uncommon.

Diagnostic work-up in patients with a suspected ‘occult’ foreign body remains difficult, especially in the case of a radiolucent object. Displacement and narrowing of the trachea by a soft tissue mass can be demonstrated in a lateral view X-ray. A barium oesophagram may show deviation, dilatation, and stenosis as well as irregularity of the wall of the oesophagus. The radiolucent foreign body itself can only be demonstrated if it produces a mass effect, filling defect or extravasation of contrast. Having completely eroded the oesophageal wall, however, it may be unrecognisable.

As stated earlier, oesophagoscopy gives very valuable information and sometimes offers a therapeutic approach. However, in most reported instances the original endoscopist could not find the impacted foreign body. Granulation tissue mostly covers the superior edge of the foreign body leading to poor visualisation in antegrade oesophagoscopy. CT examination reveals a soft tissue mass displacing the oesophagus and/or trachea. Identifying the foreign body itself can be difficult, especially with plastic, as it presents a similar picture to air in an abscess.

Differential diagnosis of a para-oesophageal mass must include neuroblastoma or ganglioneuroma, foregut duplication of the oesophagus and congenital diverticulum. A secondary inflammatory diverticulum as a result of penetration of a foreign body and consequent mediastinitis represents a rather uncommon condition. Nevertheless, when confronted by

Oesophageal disorders associated with dysphagia and regurgitation in infants and children

• congenital oesophageal stenosis: oesophageal webs, fibromuscular hyperplasia, strictures by tracheo-bronchial remnants
• achalasia of the cardia
• motility disorders: primary or secondary, after oesophageal surgery
• oesophageal emesis in neurologically impaired children
• postoperative strictures: oesophageal atresia, oesophageal replacement
• peptic strictures due to gastro-oesophageal reflux (GER)
• corrosive strictures
• strictures after long-time nasogastric intubation (without GER)
• obstruction caused by foreign body impaction
• tumours: leiomyoma, rhabdomyoma, neuroblastoma, ganglioneuroma, foregut duplication, discitis, mediastinitis
• vascular compression (e.g. right-side aorta, double aortic arch)
• Crohn’s disease
• aggressive fibromatosis
• immunologically comprised patient
• epidermolysis bullosa
• dermatomyositis
• scleroderma
• Stevens-Johnson syndrome

Box 1
Self-assessment questions

children with acquired oesophageal strictures, the possibility of a chronic impacted foreign body should be kept in mind.

**Final diagnosis**

Stricture of the oesophagus by a mediastinal mass caused by a chronically impacted radiolucent foreign body.

**Keywords:** oesophageal strictures; foreign body ingestion

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**Learning points**

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<thead>
<tr>
<th>In children with acquired oesophageal strictures</th>
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<tr>
<td>• even without a definite history of ingestion, there could be a foreign body in the oesophagus</td>
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<tr>
<td>• diagnostic work-up in cases of suspected ‘occult foreign body’ still presents difficulties, particularly if the foreign body is radiolucent</td>
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<tr>
<td>• the chronic presence of a foreign body in the oesophagus may lead to severe and unsuspected complications a long time after ingestion</td>
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**Box 2**


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**Increasing need for replacement therapy in long-standing Addison’s disease**

L Ranganath, S R Gould

A diagnosis of Addison’s disease was made in a woman at the age of 23 years. She was commenced on replacement therapy with cortisol acetate 25 mg and fludrocortisone 50 μg in the morning and cortisol acetate 25 mg in the evening; her adrenal insufficiency was well controlled on this replacement therapy. Since the age of 46 years increasing doses of mineralocorticoid and glucocorticoid were administered to control her symptoms. At the age of 52 years she was taking hydrocortisone 40 mg and fludrocortisone 200 μg in the morning and hydrocortisone 20 mg in the evening. Despite this increase in dose of glucocorticoid and mineralocorticoid she felt tired and unwell. She was a keen horsewoman and had always been underweight. She had become menopausal at the age of 28 years and had been put on oestrogen replacement therapy with Prempak C (0.625 mg). Despite the increasing dose of hydrocortisone she remained thin (weight 48.6 kg, Body Mass Index 17.8 kg/m²). Her blood pressure was 130/80 mmHg in the recumbent posture and there was no postural drop.

**Questions**

1 Why is the dose of replacement steroids increasing?
2 What further investigations would you perform?
3 What is the diagnosis in this woman?
4 What tests would you perform in following this patient up?