

CASE REPORT

A RARE CAUSE OF INTERMITTENT DYSPHAGIA: GIANT FIBROVASCULAR POLYP OF THE PROXIMAL ESOPHAGUS

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ABSTRACT

Fibrovascular polyps account for only 0.5-1% of all benign esophageal tumors and causes intermittent dysphagia. The patient was a 63-year-old gentleman with gradually progressive intermittent dysphagia of 40 days duration. Investigations revealed a submucosal tumor of the proximal esophagus causing luminal compromise. Excision was performed through a cervical esophagotomy and specimen was subject to histopathological examination. Postoperative recovery was uneventful and he was completely relieved of his symptoms.

KEY WORDS: *Dysphagia. Proximal esophagus. Fibrovascular polyp.*

INTRODUCTION

Fibrovascular polyps (FVP) are rare benign intraluminal submucosal tumor-like lesions, characterized by the development of pedunculated, intraluminal masses, which can exhibit enormous intraluminal growth. Based on the varying amounts of fibrous, vascular and adipose tissue, they can be classified as: lipoma, fibroma, fibrolipoma, or fibroepithelial polyps. More recently, they have all been grouped together as FVP (World Health Organization).¹ The FVP is the most common intraluminal non-epithelial tumor, which accounts for 0.5-1% of all benign oesophageal tumors.² These lesions are composed of loose or dense fibrous tissue, adipose tissue, and vascular structures and are covered by normal squamous epithelium. The most common location is the upper third of the oesophagus, near the cricopharyngeus. Dysphagia, vomiting, weight loss and respiratory symptoms are the most frequent complaints. However, long pedunculated lesions can regurgitate into the pharynx or mouth and cause death from asphyxiation if the larynx is occluded.³ There are about 30 case reports in the literature in the last 38 years, the largest study being a case series of 16 FVPs.⁴ We present a case of FVP, causing intermittent dysphagia.

CASE REPORT

The patient was a 63-year-old gentleman, whose main complaint was dysphagia for solids of 40 days duration. The dysphagia was intermittent to start with, and then became progressive. He also had significant loss of weight. There was no haemetemesis or melena. Carcinoma of the oesophagus was the clinical diagnosis. Endoscopy revealed a smooth submucosal mass occluding the esophageal lumen at cm from the incisors. The scope could not be passed beyond this. Magnetic Resonance Imaging (MRI) showed a smooth and elongated intraluminal mass with a length of 8.5cm occupying the upper third of the esophagus (Figure 1). Routine blood and

urine investigations were carried out, serum sodium level was 125 mmol/l, chloride level was 95 mmol/l, potassium level was 2.5 mmol/l, and haemoglobin level was 8 gm%. Based on these findings, the diagnosis was now more in favour of a benign oesophageal tumor. The patient was prepared for surgery. A left Kocher's incision was made in the skin crease; the platysma and deep cervical fascia were incised. The cervical oesophagus was identified and hooked out after dissecting away the left lobe of the thyroid gland. The oesophagotomy was achieved by a vertical incision, 3.5 cm long. An atraumatic forceps was used to carefully deliver the lesion through the oesophagotomy wound (Figure 2). It was quite difficult to do this due to the large size of the tumor. Harmonic shears was used to cut the pedicle close to the base. There was no bleeding from the cut end of the pedicle, so the oesophagus was closed with interrupted 3-0 vicryl sutures. The platysma and deep cervical fascia were closed with interrupted 2-0 vicryl. Skin incision was closed with subcuticular sutures using 3-0 vicryl. Total operating time was 50 minutes. Postoperatively, the patient recovered uneventfully and he was cured of his dysphagia. Oral liquids were allowed 12 hours following surgery and soft solid diet on the 2nd postoperative day (POD). He was discharged on the

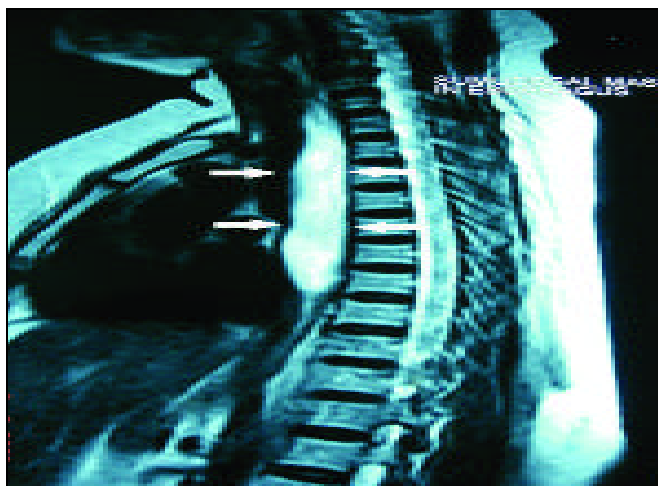


Figure 1: CT scan showing large intraluminal mass (white arrows).

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Received September 25, 2006; accepted November 23, 2006.

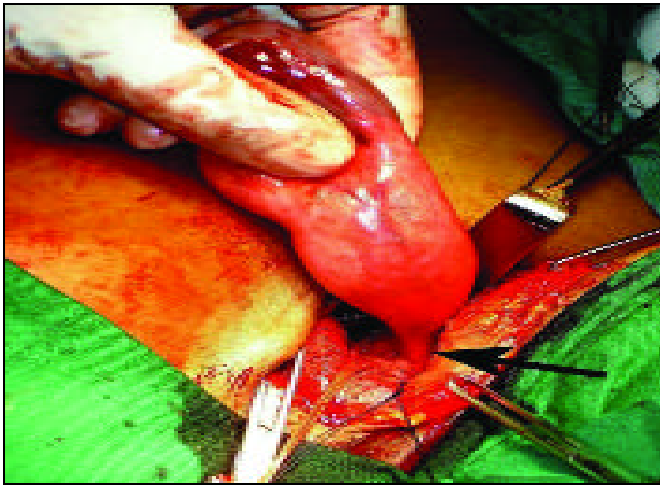


Figure 2: Giant tumor delivered out through an oesophagotomy; black arrow - pedicle.

3rd POD. The patient was reviewed 10 days, 60 days and 12 months following surgery. Endoscopies were done during the second and third follow-up. There were no symptoms, stricture or recurrence.

The length of the tumor was 8.5 cm and histopathology revealed a polypoidal lesion covered by oesophageal mucosa, composed of lymphocytes and plasma cells interspersed with fibroblasts and blood vessels. There was no hyperplasia of mucosal epithelium. The final diagnosis was fibrovascular polyp of the proximal oesophagus.

DISCUSSION

Symptoms occur only when the polyp reaches a large size. Symptoms include dysphagia, a mass in the throat and regurgitation of the polyp into the mouth with its disappearance on swallowing. Asphyxiation can result from impaction of the polyp in the glottis and is the most feared complication.⁵ Unless regurgitated, the presence of a FVP can be difficult to diagnose, and upto 30% of patients may die without a correct diagnosis.⁶ FVPs can sometimes be identified at chest radiography by the presence of a right-sided superior mediastinal mass, anterior tracheal bowing, or both. The lesions usually appear at oesophagography as smooth, expansile, intraluminal masses that arise in the cervical oesophagus and extend into the thoracic oesophagus. Although most FVPs have a site of attachment in the cervical oesophagus, barium studies often fail to demonstrate a proximal pedicle. Accurate diagnosis is best established with endoscopy. But, it may be totally missed at endoscopy because the polyp is covered by normal mucosa and can be easily displaced.⁷ FVPs containing abundant adipose tissue may appear at CT scan as soft-tissue-attenuated lesions (abundant fibrovascular tissue) with a paucity of fat that expand the lumen of the oesophagus.⁸ Surgical excision is the definitive treatment done through an oesophagotomy where direct control of feeding vessel is easily accomplished. Most of the smaller upper oesophageal FVPs can be removed via

endoscopy.⁹ For the larger lesions, surgical, rather than endoscopic removal, is the treatment of choice as bleeding is difficult to control. The stalk must be completely excised or recurrence is possible. These lesions probably originate in loose submucosal tissue of the cervical oesophagus, gradually elongating over a period of years as they are dragged into the middle or distal third of the esophagus by oesophageal peristalsis until the intraluminal portion of the lesion becomes massive. The length and size of the lumen dictates the size and shape of the polyp, as we saw in our patient. Regardless of the size of the polyp, its proximal end is almost always attached to the cervical oesophagus by a discrete pedicle, which was obviously seen in our patient. Malignant degeneration of FVPs is thought to be extremely rare.¹⁰ In conclusion, removal of these lesions is usually recommended because of the progressive and eventually debilitating nature of the symptoms and the small but known risk of asphyxiation and sudden death. Because FVPs almost always arise in the cervical oesophagus, a cervical vertical oesophagostomy appears to be the approach of choice.

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