

# LIPOMA OF HYPOPHARYNX

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## ABSTRACT

*The histologically benign hypopharyngeal lipoma is a potentially fatal tumour because of the risk of upper airway obstruction. It may be asymptomatic or present with symptoms ranging from vague foreign-body sensation to sore throat, dysphagia or dysphonia. The diagnosis may be suggested by indirect or fiberoptic laryngoscopy. Lateral neck soft tissue X-ray and barium swallow may help but CT imaging of the pharynx enables a more precise preoperative diagnosis. Treatment is by surgical excision of the lesion either perorally, endoscopically or via a lateral pharyngotomy. Long-term follow-up is recommended due to the possibility of recurrence and metachronous lesions.*

**Keywords:** lipoma, lipomatous lesion, pharynx, hypopharynx

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## INTRODUCTION

Lipomas are true benign neoplasms of mature adipose tissue. They rank among the most common types of benign soft tissue tumours and they represent 4% to 5% of all benign neoplasms. Lipomas are commonly found in the soft tissue of the neck, trunk, back and extremities, but are relatively uncommon in the upper aerodigestive tract where they predominantly occur in the mouth. The following case report describes a lipomatous lesion of the hypopharynx.

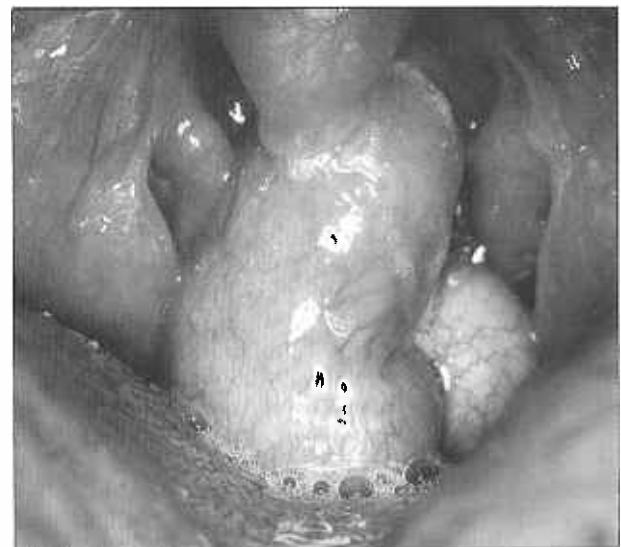
## CASE REPORT

A 44-year-old man presented to the ENT Department, National University Hospital, with a 4-month history of a sensation of something in the throat. During this period he also experienced a few choking attacks which woke him from sleep. He was seen by his family physician several times and treated with antibiotics with persistence of his symptoms. There was no pain, dysphagia, dysphonia or any other head and neck symptoms. He himself noticed a swelling at the back of his throat while trying to clear his throat one week prior to his hospital consultation.

On examination, a smooth, lobulated mass was visible on depressing the tongue (Fig 1). It occupied part of the oro- and hypopharynx and despite indirect and fiberoptic laryngoscopy, the exact origin of the swelling could not be identified. The larynx was partially obscured by the mass.

A lateral soft tissue X-ray of the neck was reported as normal but a CT scan of the hypopharynx revealed a lobulated soft tissue mass arising from the posterior wall of the pharynx. The mass extended from the level of C3 downwards to approximately C5, just above the false cords. It had the attenuation of fat in its central region, enclosed by a rim of soft tissue. There was no evidence of infiltration of the muscles of the posterior pharyngeal wall.

Fig 1 – The mass visible in the patient's oropharynx



An examination under general anaesthesia was carried out. Endotracheal tube was passed without difficulty and direct pharyngo-laryngoscopy was performed. The mass was found to be attached by a broad pedicle to the left posterior pharyngeal wall. The pedicle extended from about 2 cm below the lower pole of the left tonsil to the upper end of the aryepiglottic fold, a total length of about 3.5 cm. Attempted removal of the mass with suspension laryngoscopy was unsuccessful due to limited access. A Boyle Davis gag was then inserted and the mass, with its base, was visualised and removed with scissors and diathermy. The pharyngeal excision site was left unsutured. Post-operative recovery was uneventful.

Gross pathology revealed a pedunculated bilobed mass measuring 3.5x3.0x1.5 cm (Fig 2). It was covered by normal looking mucosa. The cut surface showed two nodules. The smaller and more superficial was soft and had a bright yellow colour. The lower deeper nodule was firm and fresh yellowish pink in colour.

Sections from the smaller nodule showed a circumscribed lesion composed of sheets of benign adipocytes as found in simple lipomas. A covering of normal non-keratinizing stratified squamous epithelium was present. Sections from the larger nodule showed nests of benign adipocytes intermingled with fibromyxoid tissue containing numerous thick and thin-walled blood vessels, consistent with angiofibrolipoma.

The patient was followed up at three, six and twelve months post-operatively. Clinical examination with fiberoptic endoscopy revealed no recurrence.

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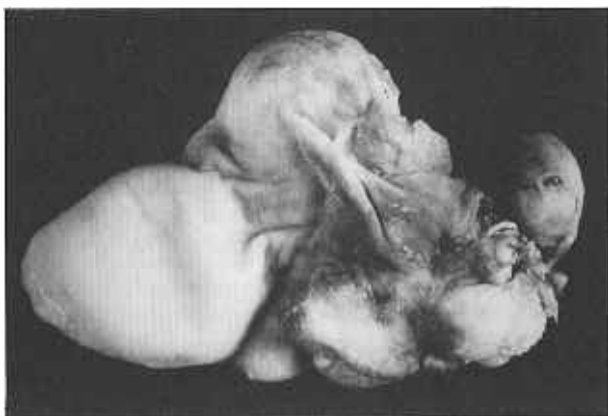
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**Fig 2 – The excised swelling after fixation in formaldehyde**



## DISCUSSION

Lipomas are uncommon in the upper aerodigestive region, despite an abundance of adipose tissue in the submucosa of the tongue, floor of the mouth and buccal area. However, if upper aerodigestive lipomas do occur, they are most frequently found in these areas.

Lipomas account for 4% to 5% of all benign tumours<sup>(1)</sup> but make up only 1% to 2.2% of benign oral tumours. Uncommon head and neck locations for lipoma include the maxillary antrum<sup>(2)</sup>, parotid<sup>(3)</sup>, nasopharynx<sup>(4)</sup>, larynx<sup>(5)</sup>, retropharynx<sup>(6)</sup> and sternocleidomastoid muscle<sup>(7)</sup>. In our case, the lipoma occurred in the hypopharyngeal wall, which is also an unusual site for lipomas. Lipomas in this region had also been reported by other authors<sup>(6,8-10)</sup>.

Most hypopharyngeal lipomas are solitary but synchronous multiple lipomas may occur<sup>(8,9)</sup>. There has been speculation of the potential for malignant change but so far no case of liposarcomatous transformation has been reported. It is the opinion of Batsakis<sup>(11)</sup> that malignant forms of lipomatous tumours arise de novo and not from pre-existing lipomas.

Hypopharyngeal lipomas are generally pedunculated. The size varies from 2-3 cm in diameter up to 15-20 cm in length. Microscopically the lesions have the characteristic appearance of a lipoma, as found in the present case, being composed of univacuolated fat cells without atypia. A qualifying prefix of "fibro" may be used for lipomas possessing an unusually prominent connective tissue component. If the vascularity of the lesion is much greater than that of a simple lipoma, the designation "angiolipoma" is appropriate<sup>(11)</sup>. The latter form is, however, unusual in the head and neck. It is usually subcutaneous and occurs in extremities and trunk<sup>(12)</sup>.

Because of the potential prolapse into the oesophagus or trachea, patients may be either asymptomatic or complain of dysphagia, a foreign body sensation, a change in quality of voice, or have sudden transient attacks of dyspnoea<sup>(8)</sup>. The tumour may be seen through the mouth as in our example. Sleep apnoea secondary to airway compression has also been reported<sup>(13)</sup>. In extreme cases, death occurred from aspiration of pedunculated tumour<sup>(14)</sup>. Our patient's symptoms did suggest few episodes of temporary airway obstruction but he did manage to cough the tumour out of the laryngeal inlet to relieve the obstruction.

The differential diagnosis of hypopharyngeal mass lesions is listed in Table I. Lipomas of the larynx and valleculae have similar symptomatology and appearance to those of the hypopharynx<sup>(15)</sup>. Benign pedunculated tumours of the oesophagus, like hypopharyngeal lipomas, may produce vague symptoms and may present as masses protruding from the mouth<sup>(16)</sup>.

Microscopically the well differentiated liposarcoma may be misinterpreted as a lipoma. Ten out of 26 well-differentiated liposarcomas reported by Kindblom<sup>(17)</sup> were primarily diagnosed as benign lipomas or fibrolipomas and the true nature of the tumours only became evident when the patients experienced one

**Table I – Differential diagnosis of hypopharyngeal lesions**

Benign:	lipomatous lesion (lipoma, fibrolipoma, angiofibrolipoma) fibroma angiofibroma leiomyoma papilloma adenoma rhinosporidiosis
Malignant:	pseudosarcoma synovial sarcoma paraganglioma

or more recurrences<sup>(18)</sup>. In this aspect, our case needs to be followed up long term to exclude this possibility.

Lateral neck soft tissue X-ray, barium swallow and tomograms may be of diagnostic assistance. CT scanning is of more help in making a correct diagnosis by determining the tissue density of any suspicious hypopharyngeal mass and in delineating the relationship to its surrounding structures<sup>(16)</sup>, as illustrated in Fig 3. Endoscopy is unreliable for identifying the true gross pathology and biopsy is often equivocal, misleading or unsatisfactory<sup>(9)</sup>.

**Fig 3 - CT scan demonstrating the tumour arising from the hypopharynx. The mass has the attenuation of fat in its central region, enclosed by a rim of soft tissue.**



Peroral, endoscopic or lateral pharyngotomy excision of a hypopharyngeal lipoma is the treatment of choice and is usually curative. Intubation may be difficult in some cases<sup>(19)</sup>.

Recurrences were found in three of the cases reviewed by Mansson<sup>(8)</sup>, one from non-radical extirpation and two cases were metachronous lipomas. Long-term follow-up of patients is recommended to prevent airway symptoms that may occur from unrecognised metachronous lesions<sup>(8)</sup>.

Our patient conforms to case reports of hypopharyngeal lipoma reported by others. We treated our patient with careful local excision of the lesion including its base perorally with suspension laryngoscopy. It has been twelve months since the patient completed treatment. The patient is on regular and long-term follow-up in the clinic. With no evidence of recurrence so far.

## REFERENCES

1. Wakely C, Sommerville P. Lipomas. *Lancet* 1952; ii: 995-9.
2. Sibemagel CE. Lipoma of the maxillary antrum. *Laryngoscope* 1938; 48: 427.
3. Watt AE, Perzik SL. Lipomatous lesions of the parotid area. *Arch Otolaryngol* 1976; 102: 230-2.
4. Grybauskas VT, Shugar MA. Nasopharyngeal lipoma. *Laryngoscope* 1983; 93: 363-4.
5. O'Callaghan MB, Emko P, Perl T. Lipoma of the larynx imaged by unconventional radiographic methods. *Laryngol Otol* 1981; 95: 1159-63.
6. Toppozada HH, Shehata MA, Maher AL. Lipoma of the pharynx. *Laryngol Otol* 1973; 87: 787-93.
7. Mattel SP, Persky MS. Infiltrating lipoma of the sternocleidomastoid muscle. *Laryngoscope* 1983; 93: 205-7.
8. Mansson I, Wilske J, Kindblom LG. Lipoma of the hypopharynx. A case report and a review of the literature. *Laryngol Otol* 1978; 92: 1037-43.
9. Jesberg N. Fibrolipoma of the pyriform sinuses. *Laryngoscope* 1982; 92: 1157-9.
10. Nash M, Harrison T, Lucentre FE. Submucosal hypopharyngeal lipoma. *Ear Nose Throat* 1989; 68: 465-8.
11. Batsakis JG, ed. Soft tissue tumours of the head and neck: unusual forms. In: *Tumours of the head and neck*. 2 ed. Baltimore: W B Saunders Co. 1979: 360-4.
12. Howard WR, Helwig FB. Angiolipoma. *Arch Dermatol* 1960; 82: 126-33.
13. Koopman CF Jr, Feld RA, Coulthard SW. Sleep apnoea syndrome associated with a neck mass. *Otolaryngol Head Neck Surg* 1981; 89: 949-52.
14. Penfold JB. Lipoma of the hypopharynx. *Br Med J* 1952; 1: 1286.
15. Goldstein MA. Lipoma of the maxillary antrum. *Laryngoscope* 1915; 25: 142-4.
16. Liliequist B, Wiberg A. Pedunculated tumours of the oesophagus - Two cases of lipoma. *Acta Radiologica* 1974; 15: 383-92.
17. Kindblom LG, Angervall L, Svendsen P. Liposarcoma. A clinicopathologic radiographic and prognostic study. *Acta Pathologica et Microbiologica Scandinavica* 1975; 83A (Suppl 253): 71-92.
18. Kindblom LG, Angervall L, Jarlstedt J. Liposarcoma of the neck. *Cancer* 1978; 42: 744-80.
19. Imperatori CJ. Fibrolipoma of the larynx: report of a case. *Laryngoscope* 1933; 43: 940-4.

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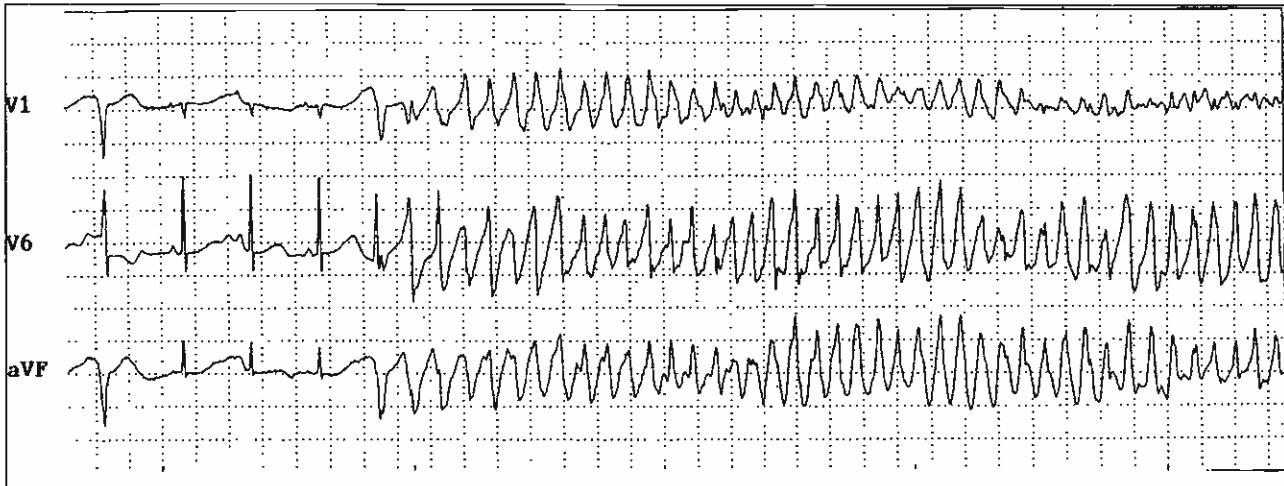
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## ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Long QT syndrome

Fig 2 – ECG rhythm strip showing torsade de pointes



### DISCUSSION

The 12-lead electrocardiogram shows prolonged QT interval. The calculated QTc using Bazett's formula  $QTc = QT(\text{in seconds}) / \text{square root of the RR interval in seconds}$ , was 0.53 seconds. The causes of a prolonged QT interval include acquired long QT syndrome due to antiarrhythmic drugs especially Class I antiarrhythmic drugs (eg quinidine), post myocardial infarction, electrolyte abnormalities due to hypokalemia, hypocalcemia and hypomagnesemia, neurological disorders such as intracranial haemorrhage, hypothyroidism, complete heart block and rarely the congenital long QT syndrome<sup>(1-3)</sup>.

On admission, the patient's episodes of fits were documented to be due to recurrent episodes of torsade de pointes (Fig 2). There was no definite family history of sudden death or of congenital deafness. The ECGs of her 3 daughters were all found to have prolonged QT interval as well. ECG monitoring in the coronary care unit, showed episodes of T wave alternans especially prior to episodes of torsade de pointes.

The patient thus has the diagnostic criteria for the congenital long QT syndrome of the Romano-Ward type<sup>(4)</sup>, which is autosomal dominant in inheritance. It is distinguished from the other type of congenital long QT syndrome, the Jervell Lange Nielsen<sup>(5)</sup> type, by the absence of perceptive deafness and is autosomal recessive in inheritance. The management of these patients involves the use of high dose beta blockers<sup>(1-3)</sup>. This contrasts markedly with the acquired pause-dependent long QT syndrome, which sometimes needs isoprenaline, magnesium or high rate cardiac pacing<sup>(1-3)</sup>. In recalcitrant cases, temporary blockade of the left stellate ganglion may achieve emergency control of the recurrent torsades de pointes and lessen the risk of anaesthesia<sup>(6)</sup>. Patients who do not respond to high dose beta blockers treatment will benefit from left high thoracic sympathectomy<sup>(7)</sup>. In some patients permanent pacing have been

successful in those who have been unsuccessfully treated with both a beta blocker and left cervicothoracic sympathectomy<sup>(8)</sup>. Finally, patients who fail all such treatment, may benefit from the implantation of an implantable defibrillator<sup>(9)</sup>. In our patient, unfortunately, she had sustained anoxic brain damage as a result of the recurrent episodes of torsade de pointes. Her arrhythmia was controlled by a combination of high dose beta blockers and left sympathetic block by scalene block. She had a high left thoracic sympathectomy which controlled her recurrent torsades. She however did not recover from her anoxic brain damage and eventually died of pneumonia. It is important to recognise that patients with recurrent "fits" may be arrhythmic in origin and the long QT syndrome should be excluded<sup>(10)</sup>. Treatment with high dose beta blockers and in selected patients with high thoracic left sympathectomy have been shown to improve the prognosis<sup>(7)</sup>.

### REFERENCES

1. Jackman WM, Friday KJ, Anderson JL, Aliot EM, Clark M, Lazzara R. The long QT syndromes: A critical review, new clinical observations and a unifying hypothesis. *Prog Cardiovasc Dis* 1988; 31: 115-72.
2. Schwartz PJ, Periti M, Malliani A. The long QT syndrome. *Am Heart J* 1975; 89: 378-90.
3. Moss AJ. Prolong QT interval syndromes. *JAMA* 1986; 256: 2985-7.
4. Ward OC. New familial cardiac syndrome in children. *Irish Med J* 1964; 54: 103-6.
5. Jervell A, Lange-Nielsen F. Congenital deaf mutism, functional heart disease with prolongation of the QT interval, and sudden death. *Am Heart J* 1957; 54: 59-68.
6. Yanagida H, Kemi C, Suwa K. The effects of stellate ganglion block on the idiopathic prolongation of the QT interval with cardiac arrhythmia (the Romano-Ward syndrome). *Anes Analg* 1976; 55: 7827-7.
7. Schwartz PJ, Locati EH, Moss AJ, Crampton RS, Trazzi R, Ruberti U. Left cardiac sympathetic denervation in the therapy of the congenital long QT syndrome. *Circulation* 1991; 84: 503-11.
8. Eldar M, Griffin JC, Abbott JA, Benditt D, Bhandari A, Herre JM, et al. Permanent cardiac pacing in patients with the long QT syndrome. *J Am Coll Cardiol* 1987; 10: 600-7.
9. Platia EV, Griffith LS, Watkins L, Mirowski M, Mower MM, Reid PR. Management of the prolonged QT syndrome and recurrent ventricular fibrillation with an implantable automatic cardioverter-defibrillator. *Clin Cardiol* 1985; 8: 490-3.
10. Kenny RA, Sutton R. The prolonged QT interval - a frequently unrecognised abnormality. *Postgrad Med J* 1985; 61: 379-86.