The Journal of Laryngology & Otology
September 2002, Vol. **116,** pp. 733–735

Acquired idiopathic laryngomalacia treated by laser aryepiglottoplasty

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Abstract
Laryngomalacia is the most common paediatric airway problem presenting as stridor in the neonate.[1] This congenital anomaly is thought to be caused by inward inspiratory collapse of the supraglottic larynx due to a prolapsed, tall and tubular epiglottis with flaccid aryepiglottic folds. The natural history of this condition usually results in spontaneous resolution by the second year of life. Although acquired cases of adult laryngomalacia have been reported, a search of the literature has yet to show any cases of idiopathic laryngomalacia. We present two cases of idiopathic acquired laryngomalacia in adults.

Key words: Larynx; Stridor; Surgical Treatment, Operative; Laser Surgery

Case reports

Case 1

A 73-year-old previously fit lady was referred from the chest physicians to the ENT department with a three-month history of dysphonia and stridor. There had been no previous upper respiratory tract infection and she denied symptoms of gastro-oesophageal reflux. Bronchoscopy and computed tomography (CT) scan had been carried out by the respiratory team and proved unremarkable. Fibre-optic laryngoscopy in the ENT clinic revealed oedematous mucosa overlying the aryepiglottic folds (Figure 1), with prolapse of the aryepiglottic folds on inspiration causing stridor. At microlaryngoscopy, performed with spontanous respirations, inspiratory supraglottic collapse could easily be seen. It was noted that the epiglottis was of normal shape. Laser aryepiglottoplasty using the CO_2 laser was carried out (Figure 2 and 3) with an uneventful post-

Fig. 1
Laryngoscopic view showing inspiratory collapse of the oedematous mucosa overlying arytenoids (black arrows).

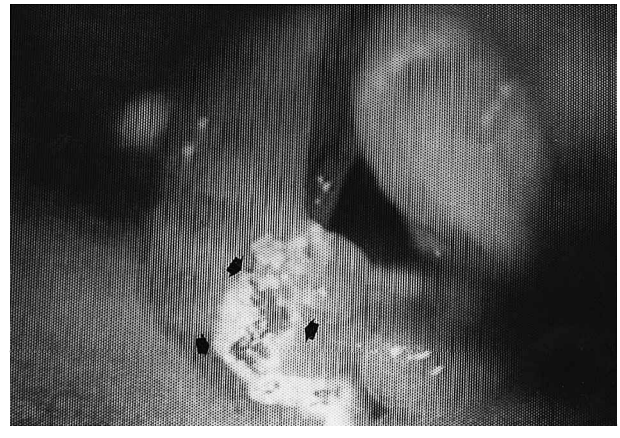

Fig. 2
Per-operative view showing laser application to left aryepiglottic fold and mucosa overlying artenoids (black arrows).

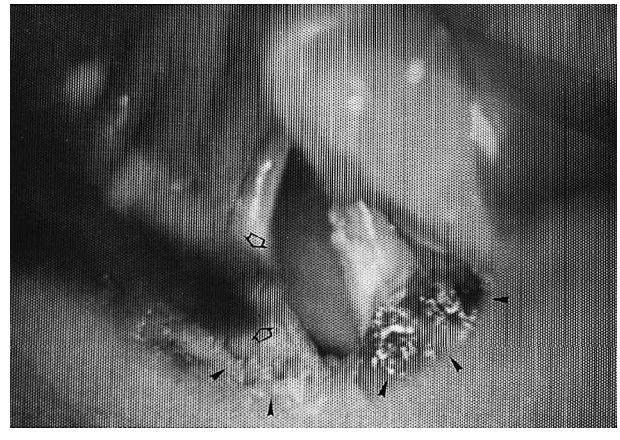

Fig. 3
Post-operative view following aryepiglottoplasty showing sucker tip (open arrows), treated areas (black arrows) and an improved airway.

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Accepted for publication: 5 March 2002

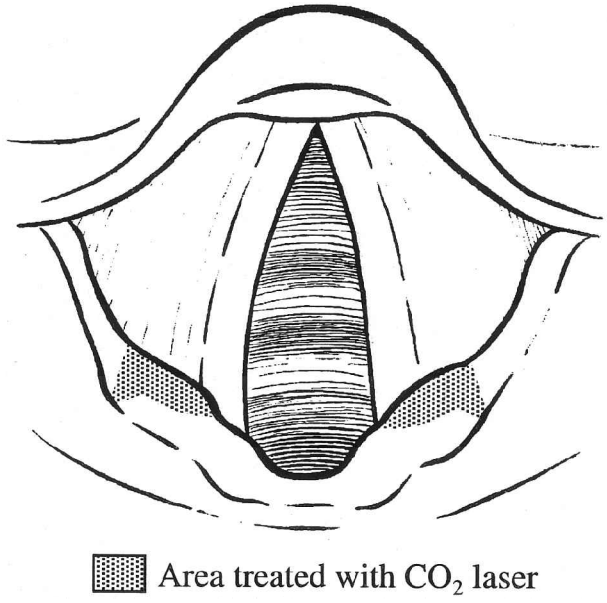

FIG. 4
Diagram demonstrating area targeted by laser.

operative recovery. The aryepiglottic folds on both sides were targeted, in order to lengthen the antero-posterior dimensions of the laryngeal inlet, in addition to the redundant mucosal tissue overyling the arytenoids (Figure 4). Laryngoscopy at one month follow-up revealed a normal healthy larynx and her voice and stridor had markedly improved. She remains symptom-free 12 months following surgery.

Case 2

A 65-year-old man was referred by the anaesthetic department following failed extubation. His past medical history was unremarkable. Flexible laryngoscopy showed inspiratory collapse of the supraglottic larynx at the level of the aryepiglottic folds with the characteristic appearance of infantile laryngomalacia. Laser aryepiglottoplasty was performed and he made an uneventful recovery. He remains fit and well two years after treatment.

Discussion

Congenital stridor was first described by the French authors, Rilliet and Barthez in 1853.[2] Various cases were reported after this, but the term 'laryngomalacia' was not used until 1942.[3] This was derived from the Greek word 'malakia' – meaning morbid softening of part of an organ. However, no intrinsic laryngeal pathology is thought to exist and the laryngeal cartilages are histologically normal. The aetiology of congenital laryngomalacia has not been fully elucidated but it is thought to be multi-factoral. The relative dimensions of the paediatric larynx are significantly less than those of the adult that further compounds the problem. This is why laryngomalacia resolves as the child grows. The diagnosis is made on laryngoscopy with the patient breathing spontaneously.[4] The consistent features seen are listed in Table I.[5] Congenital laryngomalacia has been associated with gastro-oesophageal reflux disease (GORD) that has been identified as a posssible coexisting factor.[6]

Adult acquired laryngomalacia is very rare. It has been described following severe neurological trauma either following head injury[7] or cerebrovascular accident.[8] The resulting central nervous system damage is thought to cause loss of laryngeal motor tone with impairment of co-ordination.

TABLE I
FEATURES OF LARYNGOMALACIA SEEN AT LARYNGOSCOPY

Tall tubular, in-rolled 'Ω shaped' epiglottis which tends to collapse on inspiration
Short flaccid aryepiglottic folds which prolapse medially on inspiration,
Prominent arytenoid cartilages covered with loose oedematous mucosa.
Deep supraglottic larynx.

Adapted from Cinnamond 1997[5]

Laryngomalacia has been seen in patients undergoing major oral surgery.[9,10] Resection of the elevator muscles of the hyoid bone during floor of mouth resection was thought to be the main cause of the flaccid epiglottis tilting posteriorly against the posterior pharyngeal wall. This has been successfully treated with hyoid suspension techniques.[11] Although congenital laryngomalacia is thought to be associated with normal laryngeal cartilages, acquired laryngomalacia may arise in patients with connective tissue disorders. A case on 'non-organic' airway obstruction has been described in a patient with Ehlers-Danlos syndrome, which is a disorder of weak and lax connective tissues, this may have been a case of undiagnosed laryngomalacia.[12]

The mainstay treatment for laryngomalacia that fails to resolve conservatively is surgery. Surgical approaches include excision of the redundant mucosa on the arytenoids,[13] division of the aryepiglottic folds to lengthen the laryngeal inlet in the anteroposterior axis[14] and resection of the aryepiglottis folds followed by glossoepiglottopexy.[15] Common to all these procedures is the need to preserve a mucosal bridge between the resected areas in order to prevent inter-arytenoid scarring. These procedures may be carried out with microscissors and microforceps[2] or using the carbon dioxide laser.[4]

Both our cases were unusual as they presented relatively late in life, no aetiological factor could be found in either case. The laryngoscopic findings failed to show any abnormal or infantile epiglottic anatomy. Both cases were managed successfully by laser aryepiglottoplasty.

Conclusion

Acquired idiopathic laryngomalacia is very rare. We present two cases arising late in life successfully treated by laser aryepiglottoplasty. The aetiology of congenital laryngomalacia remains uncertain.

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Mr G. Siou takes responsibility for the integrity of the content of the paper.
Competing interests: None declared