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Laryngeal Web: A Cause of Difficult Endotracheal Intubation

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Laryngeal webs constitute about 3 per cent of all congenital anomalies of the larynx.[1] This percentage evidently refers only to symptomatic cases, some of which may require corrective surgery. The incidence of asymptomatic laryngeal webs is not known. Batsakis states that laryngeal webs may also be found as coincidental findings in adults.[2] The only available report concerning a coincidentally discovered laryngeal web relates to endotracheal intubation difficulties during surgical repair of a tracheoesophageal fistula in a 2-day-old female infant.[3] Two previous similar reports dealt with unsuspected congenital stenosis of the trachea[4] or subglottis.[5]

In asymptomatic patients the presence of an unsuspected laryngeal web may be a problem if endotracheal intubation is indicated. The paucity of such reports in the anesthesia literature prompted us to present two patients with unsuspected subglottic webs discovered during difficult endotracheal intubation. These two patients were identified within a period of 3 months; we assume that the true incidence of asymptomatic laryngeal webs is probably greater than appreciated.

REPORTS OF TWO CASES

Patient 1. A 45-year-old woman was admitted with symptoms of an acute abdomen, fever (39°C) and hypotension (systolic 70 mmHg by palpation). She was given crystalloids and antibiotics intravenously, and prepared for an emergency laparotomy. Past medical history revealed that she was crippled by presently inactive juvenile rheumatoid arthritis severely affecting, among other joints, hands, neck, and temporo-mandibular joints. Past surgical history includes two cesarean sections, the last one 20 years ago. Both were performed under general anesthesia apparently without complication. She underwent hand surgery in 1978 for repair of a rheumatoid deformity.

On arrival in the operating room she had a blood pressure of 100/50–120/60 mmHg, a heart rate of 120–140 per min, a respiratory rate of 20 to 26 per min, and an oral temperature of 35.5°C. The central venous pressure was 24 cmH_2O; hemoglobin was 10.8 g/dl, fasting blood glucose 171 mg/dl, blood urea nitrogen 33 mg/dl, creatinine 3.5 mg/dl, and serum electrolytes were within normal limits. Chest was clear to auscultation. She was anuric in spite of intravenous crystalloid and furosemide administration.

Difficulty of endotracheal intubation was anticipated because of restricted mouth opening and neck extension. The necessity of awake endotracheal intubation prior to general anesthesia was explained to the patient. Laryngoscopy was done with some difficulty revealing normal vocal cord movement; however, a 6-mm ID endotracheal tube could not be passed below the level of the vocal cords. An emergency fiberoptic nasopharyngoscopy verified movement of the vocal cords and demonstrated the presence of a subglottic laryngeal web. The glottic inlet was approximately 5 mm. A tracheotomy was performed under local anesthesia and general anesthesia was then induced and maintained with intravenous narcotics and pancuronium. Cholecystectomy was performed uneventfully. On the 14th hospital day tomograms showed no evidence of laryngeal lesion; however, upon repeat nasopharyngoscopy the laryngeal web was again visualized (fig. 1). The tracheotomy tube was removed on the 19th hospital day; the flow volume loop appeared normal. The patient was discharged from the hospital four days later.

Postoperative review of the patient's old record shows that the hand surgery she had in 1978 was performed under halothane anesthesia via a mask, following unsuccessful attempts to intubate the trachea orally and nasally. Failure to intubate the trachea was attributed at that time to the moderately severe temporomandibular joint fixation. Ventilation was maintained without difficulty with use of both a nasal and an oral airway.

Patient 2. A 63-year-old man was scheduled for elective cystoscopy and possible transurethral prostatectomy. The patient had a history of moderate to severe hypertension and was medicated with alpha-methyldopa and diuretics at irregular intervals. Past surgical history includes right femoropopliteal bypass under general anesthesia with endotracheal intubation in 1972, and transurethral prostatectomy under spinal anesthesia in 1978. In the operating room, arterial blood pressure was 160/100 mmHg, pulse rate 70 per min, and regular, respiratory rate 20 per min. General anesthesia was induced with thiamylal, and succinylcholine was given to facilitate endotracheal intubation; an 8-mm ID endotracheal tube was inserted. The cuff was inflated up to 15 cc of air but an audible leak was still present. Another 8-mm ID endotracheal tube was inserted but resistance was noted when attempts were made to advance the tube. Nevertheless, the cuff was inflated until the leak disappeared with 7cc of air. Breath sounds were heard bilaterally. Anesthesia was maintained with nitrous oxide and oxygen supplemented with 2 per cent enflurane. Ventilation was controlled with the help of pancuronium: tidal volume was 800 cc, frequency of ventilation 12 per min, and airway pressure 20 cmH_2O. However, due to increase of the blood pressure to 180/120 mmHg after 30 minutes under anesthesia, the prostatectomy was cancelled; only cystoscopy was performed.

Repeat laryngoscopy performed at termination of surgery revealed

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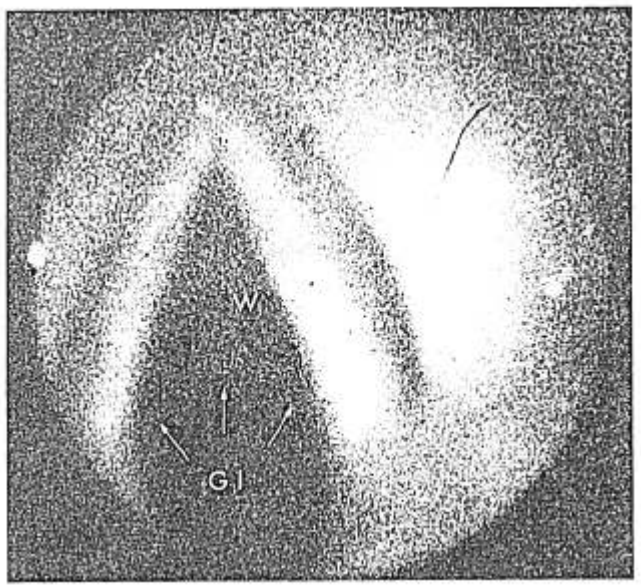

FIG. 1. Patient 1. Anterior subglottic laryngeal web occupying about 60 per cent of the airway. The arrows indicate the edge of the web. W = web; GI = glottic inlet.

the cuff of the endotracheal tube to be partially visible above the vocal cords. Because of the position of the tube and the resistance noted during its insertion, an otolaryngology consult was requested to rule out organic pathology. Fiberoptic nasopharyngoscopy revealed an anterior subglottic web occluding about 60 per cent of the airway. Tomograms of the larynx and flow volume loop were normal.

DISCUSSION

Laryngeal webs consist of thin transparent or thick fibrous membranes[1] and may be congenital or acquired. Congenital webs, usually symptomatic in infancy or early childhood, are the result of incomplete recanalization of the primitive laryngeal airway; their incidence has been estimated at approximately one in 10,000 births.[6] Seventy-five per cent of laryngeal webs are located at the level of the vocal cords, the remainder being either sub or supraglottic.[1] The majority of glottic webs lie anteriorly between the cords; only one to two per cent are posteriorly located. Diagnosis is suggested by clinical symptoms such as stridor, weak cry, and feeding problems, but endoscopic visualization is essential for its correct diagnosis.[1]

Acquired webs or scars may develop as a result of neck trauma and/or injury or inflammation of the mucous membrane and submucosal tissues.[7] There has been a marked increase of laryngo tracheal scars—more so tracheal[8]—in the last 30 years, primarily due to iatrogenic intervention, especially long-term endotracheal intubation for ventilatory assistance.[8-13]

Regardless of their etiology, laryngeal webs or scars may be slight and asymptomatic and may thus be found as a coincidental finding. Conversely, they may be severe and extensive enough to cause respiratory difficulty and abnormalities in phonation; the latter may require corrective surgery. This may include dilation, incision, or CO_2 laser excision for thin membranous webs, but fibrotic webs usually necessitate the use of a Teflon® keel or stent.[7,12]

In these two patients the laryngeal web was an unsuspected and coincidental finding; it created an obstacle to endotracheal intubation, which in the first case was compounded by the rheumatoid sequelae of temporomandibular and cervical spine joints. There is no way of knowing whether the webs were congenital or acquired. Both patients denied any history of trauma to the larynx. The trachea of patient 2 had been intubated before, apparently without complication. It is not known whether the trachea of patient 1 was intubated and perhaps traumatized during the cesarean sections performed more than 20 years ago. The fact that the vocal cords were moving during laryngoscopy argues against a rheumatoid origin of the web.[14] We can only assume that the same laryngeal web was the source of technical problems incurred by the anesthesiologist during the last surgical intervention in 1978.

The absence of symptoms, in spite of a marked decrease of the glottic inlet, is consistent with the observations of Dane and King[15] that symptoms at rest rarely occur until at least 75 per cent of the tracheal diameter has been obliterated. Others reported exertional dyspnea when the diameter reaches ≤ 8 mm,[16] and stridor at rest when it reaches ≤ 5 mm.[17]

In view of the lack of symptoms surgical treatment was not recommended for these patients. However, the patients were advised of the potential problems they could face in the event of future surgery. We believe that in any future major surgery, although cautious intubation with a smaller endotracheal tube may be worth trying, it would certainly be safer to avoid repeated attempts of endotracheal intubation. Especially in patient 1, the probability of a presurgical tracheotomy appears more likely because of her rheumatoid arthritis, but whenever possible, regional anesthesia would be preferred in such patients. A medi-alert identification band or card should be issued to such patients.

These two cases illustrate that asymptomatic laryngeal webs or scars may be an occasional cause of difficult endotracheal intubation. Furthermore, these cases suggest that fiberoptic nasopharyngoscopy should be considered in all difficult intubations if no apparent cause is present and exposure of the larynx is difficult. Flow volume loops may not be sensitive in the detection of laryngotracheal lesions;[8] tomograms may also be negative if the lesions are thin and if they do not alter the diameter of the trachea or subglottis. It is important to maintain vigilance and gentleness in attempting difficult endotra-

cheal intubations; inadvertent rupture of a laryngeal web or scar could lead to recurrence, and possibly formation, of a symptomatic occlusive scar.

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Physostigmine Reversal of Midazolam-induced Sedation

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Midazolam is a new, water-soluble benzodiazepine currently being evaluated for induction of anesthesia. Properties of midazolam include rapid induction of anesthesia, absence of pain or phlebitis following intravenous injection, hemodynamic stability, and anterograde amnesia. Like other benzodiazepines, midazolam causes respiratory depression[1] which may be prolonged,[2] but few other side effects have been reported.

As part of an institutionally approved study on consenting ASA physical status III and IV patients, we observed three cases of prolonged postoperative somnolence following induction of anesthesia with midazolam. In all instances, sedation was rapidly reversed by physostigmine after other measures to awaken the patients had failed.

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Reports of Three Cases

Patient 1. A 60-year-old 52-kg man was scheduled for esophageal dilitation. He had previously undergone bilateral radical neck dissections for laryngeal carcinoma, with no apparent anesthetic complications. He had chronic obstructive airway disease and hypothyroidism which was treated with 0.05 mg thyroxine daily. He had also lost twenty pounds in the last year. Fifty minutes before induction of anesthesia, he received 5.2 mg morphine and 0.21 mg glycopyrrolate, im, for premedication. While breathing oxygen, 13.1 mg midazolam (0.25 mg/kg) iv resulted in loss of response to commands and of voluntary movements, but the eyelid reflex remained active; an additional 2.6 mg (0.05 mg/kg) of midazolam abolished this reflex. Ten min later, the patient moved in response to surgical stimulation, and was given 100 mg thiopental, iv. Twenty-five min after the initial injection of midazolam, surgery ended. In the post-anesthesia room, he remained asleep and unresponsive while breathing 40 per cent oxygen from a T-piece. Ninety min later, he began to open his eyes on command; however, he remained disoriented and lethargic for another ninety min (3 h after induction of anesthesia), at which time 0.2 mg naloxone given iv had little effect on his level of consciousness or degree of orientation. Five min later, 2 mg physostigmine and 0.2 mg glycopyrrolate were given iv, which resulted in a profound increase in the level of consciousness and orientation to place and time. He was then discharged to his room.

Patient 2. A 66-kg, 58-year-old man with mandibular atrophy was scheduled for vestibuloplasty. His medical problems included severe chronic obstructive airway disease requiring treatment with terbutaline and theophylline, mild congestive heart failure requiring digoxin therapy, and previously resected carcinomas of the colon and bladder. Forty-five min before induction of anesthesia, he received 6.6 mg mor-