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Research Paper

Laryngotracheal stenosis in burn patients requiring mechanical ventilation

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KEYWORDS
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Abstract *Objective:* To identify the incidence of laryngotracheal stenosis (LTS) in burn patients requiring mechanical ventilation at a regional academic burn center.
Methods: A retrospective review of all burn patients requiring endotracheal intubation or tracheostomy for airway management between 2003 and 2009 was performed. A group of trauma patients requiring similar airway instrumentation during the same period of time was used as a control.
Results: None of the trauma patients and 2 of the burn patients developed LTS. Both presented with stridor and were diagnosed within 2—5 weeks after extubation. One patient underwent successful carbon dioxide laser radial incision and dilation and continues to do well. The other patient failed endoscopic treatment and required T-tube placement. The incidence of LTS in burn patients requiring mechanical ventilation was 2.98% overall and 4.76% among those with inhalational injury.
Conclusions: Patients become symptomatic within weeks of the initial injury. Treatment is challenging and multiple surgical procedures are often required. A larger study is necessary to determine if the incidence is higher among burn patients.

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Introduction

Acquired laryngotracheal stenosis (LTS) continues to be a formidable challenge for the otolaryngologist. Its etiology has changed over the years from infectious and traumatic, to primarily iatrogenic as a result of mechanical ventilation.[1,2] The incidence of LTS ranges from 1% to 10% after intubation but increases to 19% in critically ill patients requiring mechanical ventilation.[3–8]

It is generally accepted that iatrogenic LTS begins with mucosal damage by pressure from a translaryngeal or transtracheal tube. This leads to disruption of the microcirculation within the mucosa resulting in ischemia.[9–11] Superficial injury, in which the integrity of basement membrane is preserved, usually recovers completely in a matter of weeks. Deeper damage, however, takes longer to heal, and the normal respiratory epithelium is often replaced with squamous epithelium which impairs tracheal ciliary transport.[12] A breached mucosal barrier, exposed cartilage and impaired ciliary motility all increase the risk of infection and scar formation.[3,4]

Inhalational injury accompanies at least one third of all major burn accidents. The thermal injury to the airway is magnified by a chemical burn from the products of incomplete combustion.[13,14] Together, these two insults cause mucosal sloughing and tracheobronchitis, which in turn results in scarring.

As a large proportion of burn victims require mechanical ventilation at some point during their hospital course, it is often difficult to separate out the two etiologies. Previous reports have suggested that post-intubation LTS tends to be more severe after an inhalation injury.[15] The incidence of LTS in burn patients with inhalational injury requiring endotracheal intubation or tracheostomy has been reported to be between 5% and 24%.[16–18]

We present a series of 67 consecutive patients admitted to our burn unit who required mechanical ventilation and compare them to a similar group of non-burn patients to determine the incidence of acquired laryngotracheal stenosis in burn patients overall and in those with inhalational injury.

Materials and methods

After institutional review board approval was obtained, a retrospective review of all burn patients requiring endotracheal intubation or tracheostomy for airway management at Temple University Hospital, a Level 1 trauma center and a regional burn center, between 2003 and 2009 was performed. A group of non-burn trauma patients requiring endotracheal intubation or tracheostomy during the same time period was used as a control.

Demographic information, past medical and surgical history, mechanism and extent of burn or trauma injury was collected from the inpatient records. In addition, the method and duration of airway management, bronchoscopic findings, time to decannulation and to diagnosis of LTS was recorded. The incidence of LTS was calculated and compared for both groups. Statistical analysis to compare the difference between the two groups was performed using a Student *t*-test.

For patients diagnosed with LTS, the time to presentation, presenting signs and symptoms, management, and follow-up were compiled from the inpatient and outpatient record.

Results

Demographics

One hundred-fourteen burn patients who required mechanical ventilation between 2003 and 2009 were identified. Of these, 47 expired and were excluded. The remaining 67 patients were included in the analysis. In addition, 72 mechanically ventilated non-burn trauma patients were used as a control group.

The mean age was 50 years (range 14–90 years) for the burn patients and 38 years (range 14–87 years) years for the trauma group. Males dominated both groups comprising 58.2% of the burn patients and 86.1% of the trauma patients [Table 1]. The groups were not significantly different in terms of their past medical, surgical, or social history.

Injury

The mechanisms of burn were house fire (60%), gasoline explosion (14%), oxygen explosion (10%), motor vehicle accidents (10%), and electrical burn (6%) [Fig. 1]. The average body surface area burned was 17% (1%–68%). Eleven patients (16%) exhibited inhalation injury only. Of those who suffered cutaneous burns, 28 (49%) had face involvement. The mechanisms of trauma were multiple with motor vehicle accidents, gunshot wounds and falls comprising the majority [Fig. 2].

Airway management

The vast majority of patients in both groups were intubated before arrival to the hospital with 19 patients (28.4%) intubated in the field and an additional 24 patients (35.8%) intubated at an outside institution. The remaining patients were successfully intubated in the Emergency Department, except for one burn victim who required an emergency cricothyrotomy. The mean duration of intubation was 8.8 days (range 1–13 days) for the burn group, and 5.8 days (range 1–16 days) for the trauma group. Twenty-one (31.3%) burn patients underwent bronchoscopy within 10 days of admission and were found to have bronchoscopic findings of inhalational injury.

Forty-six (68.6%) burn victims and 22 (30.6%) trauma patients required tracheotomy. The pre-tracheotomy intubation period was 11.4 days (range 1–19 days) for the burn group and 13.5 days (range 1–23 days) for the trauma group. After tracheotomy, 56.7% of burn patients and 66.7% of trauma patients were decannulated prior to discharge or during outpatient follow-up. The mean time to

Table 1 Demographics of burn and trauma groups.

Group	Mean age, years (range)	Males	Females
Burn	50 (14–90)	39	28
Trauma	38 (14–87)	62	10

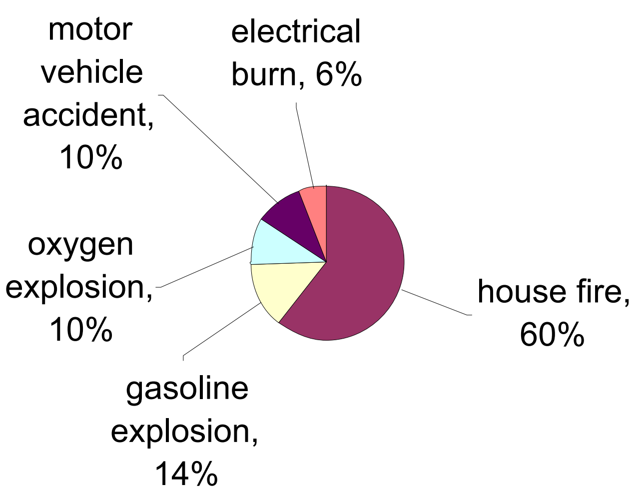

Fig. 1 Mechanism of burn injury.

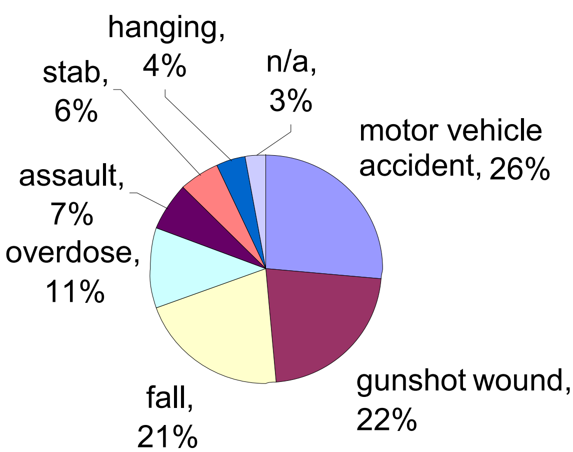

Fig. 2 Mechanism of trauma injury.

Table 2 Airway management of burn and trauma groups.

Item	Burn	Trauma
Duration of intubation, days (range)	8.8 (1–13)	5.8 (1–16)
Duration of intubation prior to tracheotomy, days (range)	11.4 (1–19)	13.5 (1–23)
Percentage requiring tracheotomy (%)	68.6	30.6
Percentage decannulated (%)	56.7	66.7
Time to decannulation, days (range)	31.3 (10–64)	30.9 (16–56)

decannulation was 31.3 days (10–64 days) for the burn group and 30.9 days (range 16–56 days) for the trauma group (Table 2).

Follow up

The follow-up rate among burn patients was 89.6%, but only 52.7% for the trauma patients. The average length of follow-up was 31.9 months (range 10–59 months) for the burn group and 40.6 months (0.3–96 months) for the trauma group.

Laryngotracheal stenosis

Overall, 2 (2.98%) burn patients and none (0%) of the trauma patients developed LTS. One (4.76%) of the inhalational injury patients developed LTS. However, given the low incidence of this complication in our patient population, the difference between the burn and trauma groups was not statistically significant.

Patient 1 is a 14 year-old male who was found in cardiopulmonary arrest after a lightning strike. He was intubated in the field with a #8 endotracheal tube. He suffered 32% total body surface area burn not involving the face. The patient was successfully extubated 9 days later. On post-extubation day 33, he presented with gradually progressive stridor. Flexible bronchoscopic evaluation demonstrated approximately 70% upper tracheal stenosis [Fig. 3]. The patient underwent bronchoscopy with CO_2 laser incision and dilation of the stenotic segment with excellent relief of his symptoms. He has remained symptom free.

Patient 2 is a 34 year-old female who was evacuated from a burning car. She was intubated in the emergency department with a #6 endotracheal tube. She suffered a 29% total body surface area burn, with head and face involvement. Flexible bronchoscopic evaluation via the endotracheal tube demonstrated mild edema with soot. She remained intubated for 7 days. She developed tachypnea on post-extubation day 16 and was re-intubated with a #6 endotracheal tube. After self-extubation 3 days later, she developed stridor. Bronchoscopic evaluation at that time demonstrated a 90% stenotic segment 4 cm in length, starting 3 cm below the true vocal folds. The patient failed bronchoscopy with CO_2 laser incision, dilation and Mitomycin C application. She underwent placement of a #14 T-tube [Hood Laboratories, Pembroke, MA]. She has failed two additional endoscopic procedures, refused resection, and remains T-tube dependent [Fig. 4].

Discussion

Laryngotracheal stenosis is a relatively uncommon problem among intubated patients, with an incidence ranging from 1% to 19%. Yang et al[18] reported the bronchoscopic presence of LTS in 0.37% of all their burn patients and in 5.49%

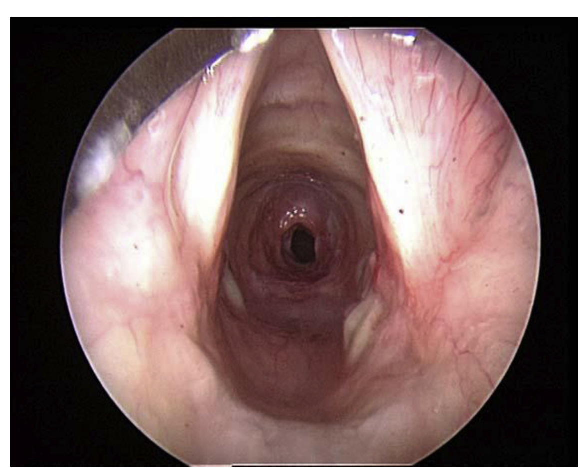

Fig. 3 Patient 1 endoscopic view of upper tracheal stenosis.

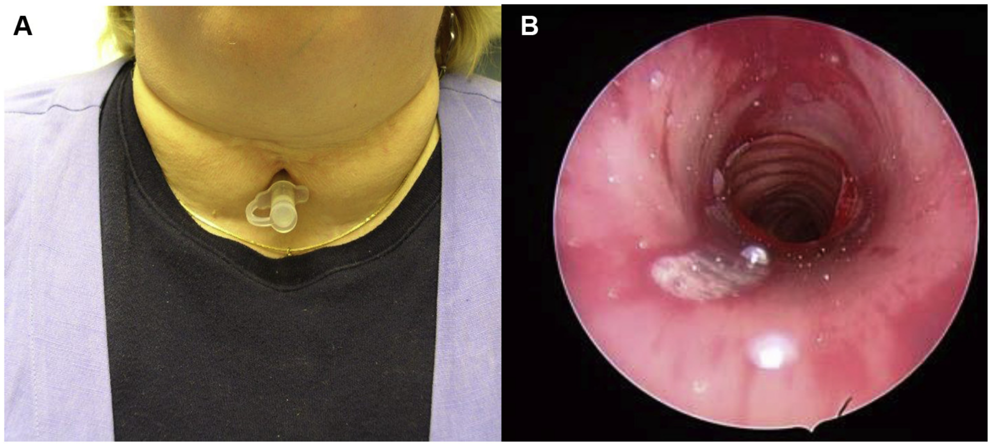

Fig. 4 **A:** External view of T-tube in neck **B:** Endoscopic view inside T-tube.

of those clinically suspected with inhalational injury. Kim et al[19] reported a 12% incidence of LTS in the inhalational injury group. In our series, we found a similar incidence of 2.98% among the overall group, and 4.76% among those with inhalational injury.

The most common cause of laryngotracheal stenosis reported in the literature is mechanical ventilation. Inhalational injury has also been implicated in the development of LTS. Lund et al[16] reports LTS in 4 of 17 (23.5%) survivors of inhalational injury who required ventilator support. Jones et al[17] found LTS in 6 of 28 patients with inhalational injury who developed major upper airway sequelae after a tracheotomy. Although limited, our data supports this hypothesis, as the patient with inhalational injury presented earlier with symptoms, required a greater number of airway procedures, and remains T-tube dependent.

Intubation causes LTS by inciting mucosal ischemia and subsequent necrosis. While the tube is still in place, the airway is stented open. Upon extubation however, healing by secondary intention begins, which may result in scar formation and stricture over several weeks. Consequently, the patients do not become symptomatic for some time after the initial insult. Both patients in our series who developed LTS presented between 2 and 5 weeks after extubation.

Laryngotracheal stenosis manifests itself primarily through symptoms of airway compromise. Endoscopic evaluation of the airway in usually not performed unless symptoms present. As patients generally do not become symptomatic at rest until the airway lumen decreases to less than 30% of normal, a large proportion of patients with LTS may be undiagnosed.[3] Most published series of LTS are unfortunately biased in this way. In our series, only 31.3% of patients underwent bronchoscopy for evaluation of the airway. Routine endoscopy of patients after extubation would allow a more accurate assessment of the true incidence of the problem.

The goal insurgical management of LTS is to restore a maximally functional airway. Treatment of LTS includes a wide spectrum of procedures from endoscopic incision and dilation, combined with steroid injections or Mitomycin C application, to placement of stents, open resection or reconstruction.[1,2,20–22] No one procedure is applicable in all situations, nor uniformly successful. At our institution we use the carbon dioxide laser incision, dilation, steroid injection, and Mitomycin C application for relatively uncomplicated LTS. For more complicated cases or for patients who fail the above, we proceed to T-tube placement or an open procedure, as in our second patient.

Prevention of laryngotracheal stenosis after mechanical ventilation is the ultimate objective for all patients. Measures such as keeping the cuff pressures below the capillary filling pressure of the tracheal mucosa (20–30 mm Hg, 1 mm Hg = 0.133 kPa) have been shown to be successful.[1,9,12] Grillo et al[23] demonstrated the benefit of using a high-volume, low-pressure cuffs to reduce the incidence of LTS. Ferdinande and Kim also cite endotracheal tube and cuff material, size and design, method of intubation, trauma during insertion and maintenance of the endotracheal tube as other factors that contribute to the pathogenesis of LTS.[19,24] There is also likely a genetic predisposition as well as racial differences in the likelihood of developing laryngotracheal stenosis.[25,26] These have not been specifically studied in burn patients but may be useful in identifying patients at risk, with the goal of potentially decreasing this serious complication in an already severely injured group of patients.

Conclusion

Laryngotracheal stenosis in burn patients requiring mechanical ventilation is rare. It is more common in those with inhalational injury. A high index of suspicion must be maintained as symptoms may present several weeks after the initial injury and intubation. Its treatment continues to be a challenge in the 21st Century. A larger study is necessary to determine if this problem is more common among burn patients.

References

1. Koshkareva Y, Gaughan JP, Soliman AM. Risk factors for adult laryngotracheal stenosis: a review of 74 cases. *Ann Otol Rhinol Laryngol*. 2007;116:206–210. http://www.ncbi.nlm.nih.gov/pubmed/17419525.

2. Goldenberg D, Esclamador R, Flint P. Cummings CW. In: *Otolaryngology-Head and Neck Surgery*. 4th ed. 2005: 2103–2128.
3. Wain JC. Postintubation tracheal stenosis. *Chest Surg Clin N Am*. 2003;13:231–246.
4. Kastanos N, Estopá MR, Marín PA, Xaubet MA, Agustí-Vidal A. Laryngotracheal injury due to endotracheal intubation: incidence, evolution, and predisposing factors. A prospective long-term study. *Crit Care Med*. 1983;11:362–367. http://www.ncbi.nlm.nih.gov/pubmed/6839788.
5. Sarper A, Ayten A, Eser I, Ozbudak O, Demircan A. Tracheal stenosis after tracheostomy or intubation. *Clin Invest*. 2005;32: 154–158.
6. Zias N, Chroneou A, Tabba MK, et al. Post tracheostomy and post intubation tracheal stenosis: report of 31 cases and review of the literature. *BMC Pulm Med*. 2008;8:18. http://www.ncbi.nlm.nih.gov/pubmed/18803874.
7. Stauffer JL, Olson DE, Petty TL. Complications and consequences of endotracheal intubation and tracheostomy. *Am J Med*. 1981;70:65–76.
8. Lukáš J, Votruba J, Paska J, Cernohorský S, Lukáš D, Machoň V. Laryngotracheal stenosis in critically ill patients. *Acta Otolaryngol*. 2011;131:91–95. http://www.ncbi.nlm.nih.gov/pubmed/20809886.
9. Nordin U. The trachea and cuff-induced tracheal injury. An experimental study on causative factors and prevention. *Acta Otolaryngol Suppl*. 1977;345:1–71. http://www.ncbi.nlm.nih.gov/pubmed/335778.
10. Hanson B, Hansson P, Nilsson K. Cuff pressure at endotracheal intubation. *Acta Otolaryng*. 1973;75:391–392.
11. Stenqvist O, Bagge U. Cuff pressure and microvascular occlusion in the tracheal mucosa. An intravital microscopic study in the rabbit. *Acta Otolaryngol*. 1979;88:451–454. http://www.ncbi.nlm.nih.gov/pubmed/532620.
12. Nordin U. The regeneration after cuff-induced tracheal injury. An experimental study in rabbits. *Acta Otolaryngol*. 1982;94: 541–555. http://www.ncbi.nlm.nih.gov/pubmed/7180426.
13. Moylan JA, Chan CK. Inhalation injury—an increasing problem. *Ann Surg*. 1978;188:34–37. http://www.ncbi.nlm.nih.gov/pubmed/666374.
14. Heimbach DM, Waeckerle JF. Inhalation injuries. *Ann Emerg Med*. 1988;17(12):1316–1320.
15. Gaissert HA, Lofgren RH, Grillo HC. Upper airway compromise after inhalation injury. Complex strictures of the larynx and trachea and their management. *Ann Surg*. 1993;218:672–678. http://www.ncbi.nlm.nih.gov/pubmed/8239783.
16. Lund T, Goodwin CW, McManus WF, et al. Upper airway sequelae in burn patients requiring endotracheal intubation or tracheostomy. *Ann Surg*. 1985;201:374–382. http://www.ncbi.nlm.nih.gov/pubmed/3883921.
17. Jones WG, Madden M, Finkelstein J, Yurt RW, Goodwin CW. Tracheostomies in burn patients. *Ann Surg*. 1989;209:471–474. http://www.ncbi.nlm.nih.gov/pubmed/2930291.
18. Yang JY, Yang WG, Chang LY, Chuang SS. Symptomatic tracheal stenosis in burns. *Burns*. 1999;25:72–80. http://www.ncbi.nlm.nih.gov/pubmed/10090389.
19. Kim IW, Choi DJ, Kim TH, et al. Laryngotracheal stenosis in burned patients with inhalation injury: analysis of risk factors. *Korean J Otorhinolaryngol-Head Neck Surg*. 2008;51: 1025–1029.
20. George M, Lang F, Pasche P, Monnier P. Surgical management of laryngotracheal stenosis in adults. *Eur Arch Otorhinolaryngol*. 2005;262:609–615. http://www.ncbi.nlm.nih.gov/pubmed/15668812.
21. Abbasi-Dezfouli A, Javaherzadeh M, Keshoofy M, Pejhan S, Daneshvar G. Treatment of postintubation airway stenosis. *Arch Iranian Med*. 2004;7:300–303.
22. Furák J, Szakács L, Nagy A, Rovó L. Multiple costal cartilage graft reconstruction for the treatment of a full-length laryngotracheal stenosis after an inhalation burn. *Interact Cardiovasc Thorac Surg*. 2011;13:453–455. http://www.ncbi.nlm.nih.gov/pubmed/21798888.
23. Grillo HC, Cooper JD, Geffin B, Pontoppidan H. A low-pressure cuff for tracheostomy tube to minimize tracheal injury: a comparative clinical trial. *J Thorac Cardiovasc Surg*. 1971;62: 898–907.
24. Ferdinande P, Kim DO. Prevention of postintubation laryngotracheal stenosis. *Acta Otorhinolaryngol Belg*. 1995;49: 341–346. http://www.ncbi.nlm.nih.gov/pubmed/8525831.
25. Anis MM, Zhao Z, Khurana J, Krynetskiy E, Soliman AM. Translational genomics of acquired laryngotracheal stenosis. *Laryngoscope*. 2014;124:E175–E179. http://www.ncbi.nlm.nih.gov/pubmed/23946168.
26. Anis MM, Krynetskaia N, Zhao Z, Krynetskiy E, AMS S. Determining candidate single nucleotide polymorphisms in acquired laryngotracheal stenosis. *Laryngoscope*. 2018;128:E111–E116. http://www.ncbi.nlm.nih.gov/pubmed/29164621.

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