MANAGEMENT OF LARYNGO-TRACHEAL STENOSIS: COMPARISON BETWEEN RESECTION ANASTOMOSIS AND AUGMENTATION BY COSTAL CARTILAGE

A SYSTEMATIC REVIEW SUBMITTED FOR PARTIAL FULFILLMENT OF MASTER DEGREE IN OTORHINOLARYNGOLOGY

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List of abbreviations

ACS	Anterior cricoid split
CTR	Criscotracheal resection
GERD	Gastro-esophageal reflux disease
ICU	Intensive care unit
LTS	Laryngotracheal stenosis
Rev Man 5	Review Manager 5.0.5
SGS	Subglottic stenosis
WG	Wagner granulomatous

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Aim of the work

This is a systematic review study to assess the outcome measures (decannulation) of laryngotracheal stenosis treated by resection anastomosis versus augmentation by costal cartilage.

Introduction

Subglottic stenosis (SGS) is narrowing of subglottic airway which is housed in the cricoid cartilage of larynx **(Cotton, 2000).** The subglottic space is the narrowest area of the airway. It is a complete non-expandable and non-pliable ring**,** unlike the trachea**,** which has a posterior membranous section**,** and the supraglottic part of the larynx which has a posterior muscular section **(McClay, 2004).**

Frequency, of SGS is approximately 15% of patients whom are intubated for more than 10 days. Ninety percent of acquired stenosis in infants and children are due to endotracheal intubation, Tracheal stenosis affects 4-13% of adults and occurs in 1-8% of neonates who have had prolonged intubation **(Mark et al., 2009).** Primary tracheal tumors are rare causes of tracheal stenosis. Recurrent respiratory papillomatosis most commonly involves the larynx. Some authors consider these papillomas the most common benign laryngeal tumors. Sarcoidosis involves the larynx in 5% of affected patients and the trachea in 1% of cases; however, this disease remains a diagnosis of exclusion. Laryngeal or tracheal involvement occurs in 50-70% of patients who have relapsing polychondritis. In cases of Wegener granulomatosis, tracheobronchial involvement occurs in 10-20% of the affected patients **(Judy, 2009).**

SGS may be congenital or acquired. It is classified as soft tissue stenosis, hard (cartilaginous) stenosis or combined **(Zalzal, 1999).** The congenital form results from cricoid cartilage malformation as congenital overgrowth circumferentially affecting the cricoid cartilage, inward luminal thickening or incomplete canalization (associated with laryngeal webs) **(Dedo et al., 1984)**. Acquired SGS occurs after manipulation or insult to the airway with endotracheal intubation being the most common

cause in ICU patients. Other underlying causes of acquired SGS are trauma to the anterior neck from vehicular accident.Infection as syphilis, TB and scleroma **(McClay, 2004).**

Despite measures introduced to decrease the frequency and consequences as in intensive care unit management, and modifications in endotracheal, tracheostomy tube designs, and safety belts in motor vehicles. Subglottic stenosis remains a formidable problem facing the otolaryngologist-head and neck surgeon **(Agrawal et al, 2007).**

The management of SGS constitutes a challenge for the otolaryngologist-head and neck surgeon. An adequate airway can be difficult to achieve and efforts to improve the glottis airway can have an effect on glottis function, such as airway protection and vocalization so, the ideal procedure should restore normal glottis function and entail minimal morbidity and this dictates a flexible, and individualized approach and requires the physician to choose among a number of options **(Inglis et al., 2003).**

Review of literature

ETIOLOGY& PATHOPHYSIOLOGY OF SUBGLOTTIC AND UPPER TRACHEAL STENOSIS

SGS may be congenital or acquired. The congenital form result from cricoid cartilage malformation as congenital small ring, circumferential overgrowth of cricoid cartilage, inward luminal thickening or incomplete canalization (associated with laryngeal web) **(Dedo et al., 1984).** Congenital stenosis causes include laryngomalacia, congenital laryngeal web, congenital subglottic stenosis, vocal cord paralysis, and laryngeal cleft **(Myer et al., 1994)**. Acquired SGS may result from many diseases. The following are the most common causes of acquired subglottic and upper tracheal stenosis are Endotracheal Intubation, External Blunt Trauma, Post Tracheostomy, Scleroma and other granulomas, Gastro-esophageal reflux disease (GERD), and Idiopathic subglottic stenosis. We will discuss these causes in more details.

A-Endotracheal Intubation

Laryngotracheal injury after prolonged endotracheal intubation has become a definite entity and very difficult problem after their recovery. Whited 1984 documented a 14% incidence of laryngeal stenosis in patients intubated for more than 10 days. Santos et al., in 1989 and Walner, in 1997 reported approximately 90% of acquired chronic SGS in neonates; infants and children occurs secondary to prolonged endotracheal intubation **(McClay, 2004)**.

Pathophysiology

Injury to the laryngeal mucosa, underlying soft tissue, perichondrium or cartilage caused by an endotracheal tube is more common than generally realized; surface injury occurs even during brief intubation. Most injuries, superficial irritation or minor ulceration heal quickly when the tube is removed. More severe injuries are related principally to diameter of endotracheal tube or to the duration of intubation. They result in edema, ulceration and necrosis **(Abbasidezfouli, et al., 2007)**. This necrosis is a consequence of ischemia resulting from pressure from the tube or the cuff exceeding the capillary pressure of the thin mucosa of the airway. Consequently, the normal mucociliary flow is disrupted which lead to infection in the perichondrium and then extend into the cartilage. The cartilage may weaken and collapse, manifesting as tracheomalacia and healing of the involved segment proceed by secondary intention. This involves three temporally overlapping stages: an inflammatory stage, a proliferative stage and stage of contraction and remodeling. The inflammatory stage begins with the initial injury. It involves active vascular retraction and reconstruction followed by vasodilation mediated by prostaglandins. Platelets adhere to the exposed collagen forming a haemostatic plug followed by migration of different types of active mediators **(Quinn and Ryan, 2002).**

The proliferative phase lasts 10-14 days. It begins with re-epithelization, this process of resurfacing the defect begins at about 12 hours after injury, the epithelial cells 1-2 mm from the wound edge undergo phenotypic changes and the cell replication rate increase 17 fold. The next part of proliferative phase is neovascularization in which blood vessels regeneration begins with the migration of endothelial cells; macrophages secrete angiogenic factors in response to high lactate levels

and low wound oxygen levels. The results of endothelial migration is capillary bud formation, collagen deposition begins when fibroblast enter wound at 48-72 hours post injury. The collection of fibroblast, inflammatory cells and capillary buds is referred to as granulation tissue persists until epithelial resurfacing is complete **(Duynstee. et al., 2002).**

The final phase of wound healing is the wound contraction and remodeling phase. Wound contraction begins at 6-7 days after injury and is maximal for 10 days. Wound contraction eventually decreases the defect by 40-60%. Skin flaps and grafts can reduce the amount of wound contraction by 50-70%. Myofibroblast, a modified fibroblast, provides the contractile force required for contraction, they have many characteristics of smooth muscle cells and are distributed throughout the wound. Wound collagen levels reach maximum at 2-3 weeks after injury, however, tissue strength is still only 5-10% of that of unwounded skin. The wound neo-matrix is gradually replaced over 6-12 months by stronger interwoven cartilage as Type I collagen displaces Type III collagen. Remodeling results in scar resulting in SGS **(Quinn, 2007)**.

Predisposing factors in development of subglottic stenosis after prolonged intubation:

1. Duration of Intubation

The degree of injury is related to the duration of intubation. Intubation of 12 hours or less produces shallow ulcers, loss of epithelium and compression of mucosal capillary beds. Between 12 and 48 hours there are deep ulcers, deeper and broader subglottic ulceration and exposed inner perichondrium. After 96 hours of intubation there is excavation of laryngeal cartilage and after 120 hours there is cartilage necrosis **(Spector, 2000)**

There is some agreement that approximately 7 days is reasonable time to decide to continue adult intubation or tracheostomy; a decision assisted by endoscopic assessment under general anesthesia **(Benjamin, 1998).**

2. Size of the tube

Oversized tubes produce early mucosal damage and underlying cartilage ulceration and necrosis **(Spector, 2000).** Suggestions have been made that the upper limit of the inside diameter of the tube is 8.0 mm in males and 7.0 mm in females. In practice, the size of the tube should be individualized to the patients **(Benjamin, 1998)**.

3. Cuff pressure

Cuff pressure is thought to be a major factor in laryngotracheal damage. There is a direct correlation between the degree of airway damage and the severity of respiratory failure, mainly because a need for high peak expiratory pressure levels requires higher cuff pressure **(Alessi et al., 1989 and Abbasidezfouli, et al, 2007)**.

Generally it's recommended that a small air leak should be present following intubation that ventilation is at less than 20 mmH_2o. Cuffed tubes that are inflated further increase the endoluminal tube size. Inflation of the tube cuff to internal pressures above 20-30mmH_2o will occlude the mucosal and sub mucosal circulation in the subglottic region. This will cause devascularization with ischemic necrosis of the soft tissues and cartilages of the trachea and cricoid. The latter will predispose to subglottic stenosis. The intermittent reduction of a double cuff or the use of low pressure cuffs will ameliorate the risk **(Spector, 2000)**.

4. Type of the tube

Silastic and polyvinyl chloride are considered the safest materials for prolonged intubation, as they are less irritating , softer and easily compressed. Ethylene oxide sterilization of polyvinyl chloride tubes produces a troublesome toxic residue that causes chemical irritation **(Santos et al., 1989 and Quinn 2007)**.

5. Shearing Motion of the Tube

It causes abrasive traumatic action on the mucosa in the area of the tube tissue interface in the posterior larynx especially in restless patients, patients on respirators and with oro-tracheal intubation. The interarytenoid space serves as a fulcrum for see-saw action of the endotracheal tube significant injury in this site results in disturbed laryngeal physiology for both airway and sphincteric functions **(Sittel et al, 2008)**.

6. State of Larynx

A normal larynx is less prone to intubation trauma than an abnormal larynx. For example, changes are more likely in croup because of the acutely inflamed, edematous, narrowed subglottis **(Benjamin, 1998)**.

7. Nasogastric Tube

The presence of a feeding nasogastric tube aggravates trauma by promoting gastroesophageal reflux, predisposing to aspiration and causing pressure necrosis and ulceration in the postcricoid region **(Duynstee et al., 2002)**.

8. Repeated and Traumatic Intubations

Repeated intubations or traumatic intubations predisposes to early mucosal and cartilaginous damage especially if the tube size is large. The aseptic injured mucosa due to compression with a large tube is further damaged by mechanical trauma, leading to greater laryngeal damage **(Spector, 2000).**

9. Bacterial Super-infection

Bacterial adhesion and infection can be detected within 24 hours. Care should be taken to minimize infection when a tracheotomy is performed below a larynx that is already the site of prolonged intubation trauma. Subsequent stomal contamination may delay healing and predispose to scar formation **(Schamal et al, 2003)**.

10.Poor General Health

Toxic states, anemia, hypotension, hypoxemia, liver failure, renal or heart failure, pulmonary infection and altered levels of consciousness are associated with poor tissue perfusion; hypoxia and ultimately more sever changes from intubation trauma **(Duynstee et al., 2002)**.