**CLINICAL VIGNETTE**

# A Case of Stridor

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A 60-year-old male with history of hypertension and hyperlipidemia presents to the Emergency Department with complaint of shortness breath for 2 days. He presented to an outside hospital the day before with a similar presentation and was hospitalized for a day and discharged. He is unable report details of his evaluation and treatment and does not have any discharge paperwork. He denies any pain, recent illness, fever, chills or cough. He also denies any trauma, numbness, weakness to extremities, difficulty speaking or facial asymmetry. There is no prior history of cerebrovascular events or neck surgery. He did acknowledge smoking methamphetamine prior to onset of dyspnea.

Patient presented to triage and was noted to have stridor and difficulty breathing. He was hypoxic to 92% on RA and was started on albuterol and ipratropium nebulization treatment immediately due to concern for wheezing. While receiving treatment, he was able to speak a few words but continued to have stridor with deep inspiratory breaths. Patient was given parenteral dexamethasone and additional testing performed. Laboratory tests were unremarkable. Soft tissue neck X-ray revealed narrowing of the supraglottic airway and possible edema to the epiglottis. Ear Nose and Throat (ENT) physician was consulted and a bedside bronchoscopy was performed and bilateral vocal cord paralysis noted. He was taken to the operating room where an awake emergent tracheostomy was performed. The patient's hospital course did not reveal the etiology of his bilateral vocal cord paralysis.

***Pathophysiology***

Stridor is a high-pitched monophonic sound heard over the anterior neck caused by turbulent flow through narrowed upper airways.[1] Inspiratory stridor indicates a laryngeal obstruction, while expiratory stridor indicates a tracheobronchial obstruction. Biphasic stridor with inspiration and expiration may indicate a fixed obstruction of the glottic or subglottic region such as a foreign body.[2] Patients may additionally note dysphagia, dysphonia, cough, or a sensation of tightness and choking.[3]

Stridor indicates an extrapulmonary pathology, which can be broadly categorized as being infectious, inflammatory, or mechanical in origin.[4] Infectious etiologies of stridor include tonsillitis, epiglottitis, croup and tracheitis. Inflammatory causes include anaphylaxis, angioedema, and caustic inhalation such as cigarette smoke, ammonia or fumes from other chemical cleaners, or construction dust.[5,6] Mechanical causes of stridor generates the broadest differential ranging from extratracheal compression from abscess, tumor, or goiter, to intratracheal or intralaryngeal obstruction such as tracheal stenosis, foreign body, or vocal cord paralysis. In the setting of paralysis, vocal cords may be adducted causing airway obstruction. This can be caused by intubation, neck surgery, or neurologic dysfunction.

***Diagnosis***

On history, the etiology of stridor can be categorized as acute, subacute, or chronic. Acute onset would raise suspicion for foreign body, anaphylaxis, epiglottitis, croup, bacterial tracheitis, or caustic ingestion/inhalation. Subacute onset with prodromal symptoms would indicate a developing infection such as croup, peritonsillar or retropharyngeal abscesses. Chronic stridor is likely to be caused by vocal cord paralysis, spasm, or compressive neoplasm[4] The presence of hives and an allergen exposure would suggest anaphylaxis. Stridor in a neonate or infant is most likely due to congenital anomalies.

Vocal cord paralysis can be caused by structural brain abnormalities such as stroke, hydrocephalus, trauma, or brainstem compression. Among the peripheral causes are motor neuron diseases such as ALS, diabetic neuropathy, or motor end plate disease such as myasthenia gravis. It may also be caused by toxins such as organophosphate poisoning or vinca alkaloid therapy.[7,8]

***Evaluation and Management***

Stridor may present with variable degrees of airway compromise ranging from minimal dyspnea to severe respiratory distress. The first step in evaluating a patient with stridor is to assess their work of breathing, airway patency, and evaluate for signs of hypoxia. Indications of airway obstruction include tachypnea, retractions, trismus, drooling, dysphagia, dysphonia, tripoding, altered mental status and severe fatigue.[4,9] If the patient is thought to be stable but approaching respiratory collapse, then ENT and Anesthesia should be consulted with anticipation of a surgical airway placement in the operating room.

If the patient is unstable with severe respiratory distress, intubation in the ED is necessary. The patient should be maintained seated upright if possible. An experienced provider may use a fiberoptic scope to place an endotracheal tube that is

smaller than would be expected for the patient's age to accommodate for airway edema.[10–12] In case of complete airway loss, a surgical airway kit should be placed at bedside. Other acute measures to bridge the patient to a more definitive airway management include panting and heliox. Panting causes recruitment of the posterior cricoarytenoid muscle, leading to abduction of the vocal cords.[3,13] Heliox is a gaseous mixture of oxygen and helium, which has a lower density than room air, decreasing turbulent air flow and improving oxygenation in patients with partially obstructed airways.[14] Of note, manipulation of the oropharynx for examination or swab should be deferred in the setting of stridor until the airway is secured or the patient is in a controlled setting such as the operating room, as it runs the risk of exacerbating respiratory compromise.[4]

Beyond stabilization, history and exam will help provide the etiology of the stridor and guide treatment. Further evaluation of the patient includes laboratory studies such as a CBC, cultures and a respiratory viral PCR panel from nasopharyngeal swab. Plain films may reveal a foreign body or supraglottic narrowing known as the "steeple sign." Lateral plain films should be evaluated for retropharyngeal space widening, which is suggestive of an abscess, while the "thumbprint sign" raises concern for epiglottitis. Visualization on laryngoscopy or bronchoscopy is the gold standard to assist with diagnosis and treatment.

Treatment varies depending on the etiology of stridor. Beta agonists should be avoided in patients with croup as it can exacerbate airway compromise, and racemic epinephrine and dexamethasone should be administered.[4,15] If infection is suspected, it is important to identify the causative agent to guide antibiotic choice. Bacterial causes of stridor such as bacterial tracheitis or abscess are managed with broad spectrum antibiotics and aspiration and drainage as needed. Anaphylaxis requires treatment with antihistamines, epinephrine and steroids.

In vocal cord paralysis, due to recurrent laryngeal nerve transection, vocal cord function will not recover. However, swallow therapy may be useful. Long-term management prioritizes maintaining an airway through tracheostomy or surgical widening. In the case of neurapraxia, vocal function is expected to recover in 6 months to 1 year, with some evidence that nimodipine may speed the rate of recovery.[16]

***Summary***

When a patient with stridor and respiratory distress presents to the Emergency Department, airway management takes precedence. Immediate consultation with ENT and Anesthesia is crucial to obtain visualization of the larynx with a fiberoptic scope or for transfer to the operating room for a definitive airway. After a secure airway is established, further work up to determine the etiology and appropriate treatment is warranted.

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