

Quick Review: Laryngomalacia & Asthma

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Abstract

One of the most common causes of Stridor in children. LM is a congenital deformity (the most common congenital laryngeal abnormality) causing laxity of the epiglottis and supraglottic aperture with an accompanying weakness of the tracheal wall. This leads to varying degrees of airway collapse during inspiration. Males are affected twice as often as females.

LARYNGOMALACIA (LM)

One of the most common causes of Stridor in children. LM is a congenital deformity (the most common congenital laryngeal abnormality) causing laxity of the epiglottis and supraglottic aperture with an accompanying weakness of the tracheal wall. This leads to varying degrees of airway collapse during inspiration.

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Clinically, LM presents as noisy, harsh inspiratory sounds that may not be very evident during the first 1 - 2 months of life (given that this type of breathing is very common in the first few months of life).

The symptoms can be intermittent but become worse when the baby lies on his or her back. The symptoms can range from noisy breathing, "crowing", hoarseness, dyspnea, and inspiratory retractions (in the supraclavicular, intercostal, or subcostal spaces).

Patients with severe forms can have difficulty in nursing to result in general malnutrition and poor weight gain; the weight gain was not a problem with our pt given his premature birth and the "catching up" that he has done since. The stridor should, classically, slowly disappear as the child grows and develops; with normal maturation, the airway stiffens to take up the "extra slack" in the folds.

LM - DIAGNOSIS

Established by direct laryngoscopy. Differential diagnosis

should include malformations of the laryngeal cartilage or vocal cords, intraluminal webs, generalized severe chondromalacia of the larynx and trachea, tumors of the larynx, mucus retention cysts, thyroglossal duct cysts amongst others.

LM - PROGNOSIS

Fairly good for this condition with the far majority of cases resolving completely

by the age of 18 months. However, there may be some degree of inspiratory obstruction that persists but treatment is usually not required.

LM - TREATMENT

Primarily involves supportive therapy and parental reassurance because this condition resolves spontaneously. Feedings should be slow and careful with attention to possible aspiration.

Most infants seem more comfortable and "less noisy" in a prone position. Severe forms may require intubation but tracheostomy is rare.

ASTHMA

A discussion of a disorder must begin with a definition of that condition; however, with Asthma there is no universally accepted definition ! Asthma may be considered as a "diffuse, obstructive lung disease with (1) hyperactivity of the airways to a variety of stimuli and (2) a high degree of reversibility of the obstructive process, which may occur

either spontaneously or as a result of treatment.”.

This disease carries a variety of other names, including: Reactive Airway Disease, “Wheezy Bronchitis”, “Viral-associated wheezing”, and “Atopic related Asthma”, and “Chronic Desquamating Eosinophilic Bronchitis”. On a more-basic level, the pathophysiology involves two components:

- Bronchoconstriction;
- Inflammation.

There are several interesting epidemiological points involving Asthma. It seems that inheritance is multifactorial: a child with one affected parent has a 25% risk of developing it; with both parents affected, the risk increases to 50%. However, in monozygotic twins it is not universally present. The condition may have an onset at any age but 30% of patients are symptomatic by 1 yr of age, with 80-90% of patients having their first symptoms by the age of 5. The majority of cases have an intermittent and fluctuating course with only brief episodes of respiratory compromise. The incidence and mortality have both increased in the last 2 decades. Specific causes are unknown but high-risk factors behind the mortality include:

- development of sudden, acute, severe asthmatic episodes,
- chronic, steroid-dependent asthma,
- underestimation of the severity of the illness (by pt, family, and physician) leading to a delay in treatment,
- underuse of steroids,
- noncompliance with prescribed treatments,
- family dysfunction and stress,
- severe atopic disease, and
- African-American race

Asthma, characterized by narrowing of the large and small airways, is due to bronchoconstriction (spasm of the bronchial smooth muscle), hypersecretion of Mucus, Mucosal Edema, Cellular Infiltration, and desquamation of epithelial and inflammatory cells. Various triggers initiate the bronchoconstriction and inflammatory response and the

summary steps below can quickly remind us of the pathophysiologic process:

TRIGGERS

Bronchoconstriction
Mucosal Edema
Hypersecretions

THE STEPS

Airway Obstruction
Nonuniform ventilation
Hyperinflation
Atelectasis
Decreased Compliance
V:Q Mismatch
Decreased Surfactant
Increased Work of Breathing
Acidosis

Pulmonary Vasoconstriction
Increased PCO₂
Decreased PO₂

Clinically, onset can be either insidious or acute. When it develops acutely, the airway obstruction is most likely due to exposure to irritants and leads to smooth muscle spasm in the larger airways. Attacks precipitated by viral illnesses (which is likely what is seen in this pt's current episode) are usually slower in onset with gradual and progressive increases in the frequency and severity of cough and wheezing. Nighttime is a common period when exacerbations are seen - given that airway patency decreases at night.

SIGNS & SYMPTOMS

Signs and symptoms include cough (“sounds tight”), wheezing, tachypnea, and dyspnea with prolonged expiration and the use of accessory muscles, cyanosis, hyperinflation of the chest, tachycardia, and pulsus paradoxus (these later signs may not be present until late into an attack).

When the pt is in severe distress, the “cardinal sign” of Asthma (wheezing) may actually not be present - due to such a degree of decreased air entry. Once bronchodilator therapy is initiated and the chest “opens up”, then wheezing should be appreciated. The pt in distress may assume a “tripod position” which increases longitudinal thoracic diameter and allows easier and more-efficient use of the accessory muscles. Abdominal Pain is very common - especially in younger children, and is thought to be due to the use of

abdominal muscles; the liver and possibly, the spleen may become palpable due to hyperinflation of the lungs.

Vomiting is common and may help to temporarily relieve symptoms. Clubbing is a rare finding. Between attacks, the child will usually be symptom free and have no evidence of pulmonary disease!

DIAGNOSIS

The diagnosis is often strongly suggested by recurring episodes of coughing and wheezing (a chronic unremitting cough may be the only symptom!).

Pulmonary Function testing before and after treatment with bronchodilators can help to establish the proper diagnosis; PFT's should be a mainstay in the management of any asthmatic - allowing some objective clue to the severity of the current "reversible obstruction".

The laboratory study most suggestive of asthma is eosinophilia ($> 250 - 400$ cells/mm³). Serum protein and Ig levels are usually normal except that IgE level is increased.

Differential includes other causes of airway obstruction (congenital malformations, foreign bodies), infectious bronchiolitis, CF, hypersensitivity pneumonitis, immunologic deficiency disease, and other rarer conditions (including Tb and fungal infections).

TREATMENT

Treatment includes basic concepts of trying to avoid "triggers", improving air entry, and reducing inflammation.

Pharmacologic therapy is the primary form of treatment and includes:

1. Oxygen at 2 - 3 L/min - indicated during an acute attack
2. Bronchodilator Aerosols (has replaced the use of epinephrine injection, in most cases). Albuterol solution is both safe and effective at a dose of 0.15 mg/kg (maximum 5 mg/kg) at intervals of 20 - 30 minutes until response is adequate.. Nebulization with oxygen prevents hypoxemia.
3. If response is still not adequate, Aminophylline can be given IV (5 mg/kg for 5 - 15 min- which is safe in a pt without a hx of theophylline use) to help in the acute phase of the asthmatic reaction as well as further stabilization of mast cells. It is controversial to add aminophylline when Beta agonist are being used appropriately and at the correct regimens - but it is mentioned in Nelson's as a therapeutic approach.
4. The use of corticosteroids is not an "automatic" in the usual treatment of asthma unless the pt is corticosteroid-dependent or has had steroids in the past as part of the regimen. The use of steroids may shorten the length of recovery and, as long as they are tapered, should not provide serious side effects.

References

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