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Review

Disorders of the neonatal nasal cavity: Fundamentals for practice

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SUMMARY

Neonatal nasal obstruction is a well-known clinical entity. Fortunately, it is rarely life-threatening and usually resolves with conservative management. As with most conditions, a systematic history and thorough physical examination are crucial for correct diagnosis and management. The initial diagnosis may be elusive and require either serial or more in-depth evaluations. Occasionally, examination may reveal structural abnormalities necessitating surgical intervention. Fortunately most of these abnormalities are amenable to surgery; however, a select few are notoriously difficult to treat.

1. Introduction

A long-held misconception pervasive in otolaryngology literature is that the neonate is an obligate nasal breather. The word "obligate" suggests that the neonate has no choice but to breathe through the nose. However, the more fitting terminology is that neonates are "preferential" nasal breathers, since an infant can breathe through the mouth if the nose is obstructed [1–3]. In some conditions, such as that of bilateral choanal atresia or bilateral dacryocystocele, the child has complete nasal obstruction and must breathe through the mouth to survive. In this situation, the patient demonstrates cyclical cyanosis wherein the neonate attempts to breathe through the nose and is unable to do so, resulting in hypercapnia. Thus the neonate cries, opening the mouth to breathe orally and momentarily resolving the cyanosis. This phenomenon demonstrates that the neonate breathes preferentially – but not exclusively – via the nasal cavity, which provides an essential foundation for understanding and treating neonatal nasal disorders.

2. Physiology of the nose

In order to understand neonatal nasal pathology one must also have an understanding of normal nasal physiology. The nasal cavity has several important functions including serving as a conduit for airflow, conditioning inhaled air via heating and humidification, filtration of inhaled particles and microbes, olfaction, and allowing for paranasal sinus drainage [4,5]. Numerous arterioles and arteriovenous anastomoses which drain into venous sinusoids contained in the nasal mucosa act as resistance vessels. Sympathetic fibers innervating these venous sinusoids release norepinephrine leading to a reduction in blood flow and venous return into capacitance vessels. Physiologically, this causes a decrease in nasal congestion. Conversely, acetylcholine released by parasympathetic fibers increases vasodilation and nasal secretions. Local mediators such as neurokinin A, calcitonin gene-related peptide and substance P are released by sensory C-fibers, downregulating sympathetic-mediated vasoconstriction and thus increasing congestion. This interplay of parasympathetic and sympathetic input, as well as the effects of local mediators, results in variable vascular engorgement and congestion of the nasal mucosa [6]. Alterations in the dimensions of the nasal cavity, either secondary to its anatomic structure or due to mucosal engorgement, impact its ability to perform its physiologic functions [7]. Biomechanical studies examining nasal airflow as a function of the three-dimensional shape of the nasal cavity have demonstrated that even minor increases in mucovascular edema – particularly in the internal nasal valve area – may result in significant decreases in airflow [8].

3. Nasal pathophysiology

Nasal obstruction in neonates and infants is generally conceptualized as either an anatomic or non-anatomic issue by an evaluating otolaryngologist. Non-anatomic issues usually manifest as

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mucosal engorgement due to some underlying insult – whether it be allergy, laryngopharyngeal reflux, an infectious etiology, or an adverse effect of a medication. By contrast, anatomic issues in the neonatal population are typically congenital. These pathologies may be categorized by location and laterality. For example, the site of obstruction may be anterior (pyriform), mid-vault, or posterior (choanal), and many of these pathologic entities may be either unilateral or bilateral.

It is imperative for the astute clinician to probe carefully into the pathophysiology of neonatal nasal obstruction. Nasal obstruction is the result of an extremely complex interplay of still poorly defined competing parasympathetic and sympathetic input in the context of any underlying anatomic pathology [8]. As such, care should be taken to address both anatomic and non-anatomic issues that may be present.

4. Clinical presentation

Seldom does the neonate present with complete nasal obstruction, as in the case of bilateral choanal atresia or arhinia. More usually, there is at least some airflow on one side of the nasal cavity. Children with bilateral choanal atresia present with cyclical cyanosis. Specifically, nasal obstruction results in failure to breathe effectively and cyanosis. Next, the child cries, resulting in restoration of oral airflow and resolution of cyanosis. This cycle repeats as the child closes his mouth once again. Placement of an oral airway temporizes the situation, allowing for a more thorough evaluation.

5. Diagnosis

The preliminary examination is focused on the evaluation of the patient's ability to breathe through the nose, and identification of a potential unilateral or bilateral nasal obstruction. Stertor, a low-pitched, non-musical, inspiratory sound due to turbulent airflow and vibration of nasopharyngeal and/or oropharyngeal soft tissue is often appreciated. Although a 6 Fr catheter is often threaded through each nasal cavity to establish patency, this maneuver is often erroneously interpreted secondary to the catheter coiling in the nasal cavity or bouncing against the inferior turbinate. An alternative is the mirror test, in which nasal patency is established when fog appears on a mirror positioned in front of each naris. Rigid telescope endoscopy, despite its superior optic properties, is not practicable in the neonate. Rather, flexible fiberoptic endoscopy is warranted when patency is unclear and will help identify and visualize the location and amount of obstruction. Pyriform, mid-nasal, or posterior nasal stenoses, nasal septal deviations, or masses in the nasal cavity or nasopharynx may be visualized with flexible fiberoptic endoscopy.

In the context of neonatal nasal obstruction, assessment of breathing and feeding co-ordination is crucial. This feeding assessment in afflicted patients may include a clinical swallow therapy evaluation, modified barium swallow (MBS), and/or fiberoptic endoscopic evaluation of swallowing (FEES). The necessity for surgical correction or management may be hastened if the neonate's nasal obstruction results in poor or discoordinated feeding with associated choking.

Imaging investigations are critical adjuvant studies in the work-up of nasal obstruction secondary to anatomic factors. Computed tomography (CT) is most informative for diagnosing bony abnormalities as seen in pyriform aperture stenosis and choanal atresia, and is essential for appropriate surgical planning. By contrast, magnetic resonance imaging (MRI) offers superior soft tissue definition of masses in the nasal cavity or nasopharynx. MRI can determine the degree of intracranial extension of nasal dermoids, tumors, and encephaloceles. The advent of antenatal fetal MRI has been extremely valuable in the early detection of masses that can cause postnatal respiratory obstruction. Numerous case reports have demonstrated the utility of fetal MRI in diagnosing a variety of nasal masses suggested by fetal ultrasonography as well as allowing for surgical planning and prompt surgical intervention [9].

6. Differential diagnosis and management

Typically a diagnosis is readily apparent after obtaining a history, performing a thorough physical examination with appropriate endoscopic exams, and acquiring appropriate radiographic imaging studies. Treatment of anatomic pathology is typically surgical whereas treatment of non-anatomic pathology is aimed at addressing the underlying insult manifesting as mucosal edema.

6.1. Trauma during birth

Birth-related trauma is an etiology of neonatal nasal pathology [10]. The incidence of structural birth trauma – more specifically external nasal deformity or significant nasal septal deviation – has been reported as ~1–2%. Yet, in clinical experience, it appears much less prevalent secondary to the lack of symptomatology in most patients. Several mechanisms have been proposed with respect to the origin of these nasoseptal deformities. Prospective studies aimed at correlating intrauterine and parturition factors with nasal septal deformities have suggested the existence of two distinct types of deformities with distinct mechanisms. Isolated anterior septal deviations are thought to be the result of trauma in the birth canal and are more likely to be associated with external nasal deformity. Combined anterior and posterior septal deviations are thought to be the result of transmitted intrauterine forces during fetal skull molding. Statistical analysis demonstrated an increased incidence of septal deformity in complicated or difficult deliveries [11]. However, the multitude of factors associated with acquired nasoseptal trauma highlights the importance of not diagnosing all neonatal nasoseptal deformities due to iatrogenic birth trauma, when the mechanism may be positioning of the head in the birth canal during a relatively uneventful passage. Minor nasal deviations often normalize within several weeks postnatally. Rarely, the septum is so severely misaligned that a closed reduction in the first week after birth is necessary for normal nasal breathing.

6.2. Rhinitis in the neonate

Neonatal rhinitis is a condition unique to the newborn that manifests as clear or mucoid rhinorrhea with nasal mucosal edema in the afebrile newborn associated with stertor, poor feeding and respiratory distress. The paramount distinction is the lack of evidence suggestive of an infectious or anatomic etiology. Despite its relatively high incidence, neonatal rhinitis remains a somewhat poorly understood disease process with an unclear mechanism. Current literature has proposed a conservative empiric treatment regimen based on concurrent initiation of a topical decongestant (i.e. oxymetazoline or neosynephrine) and topical nasal steroid in addition to nasal saline drops and bulb suctioning. The decongestant is maintained for only three or four days. Retrospective study has shown that the vast majority of cases will respond to this treatment algorithm, with more in-depth evaluation being warranted in those cases that fail to respond within the expected timeframe [5].

6.3. Medication-induced rhinitis

Placental blood flow can transfer medications and drug metabolites from maternal to neonatal circulation, resulting in