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RHINOLOGY: NASAL OBSTRUCTION (JV NAYAK, SECTION EDITOR)

Neonatal Nasal Obstruction

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Abstract

Purpose of Review Neonates are obligate nasal breathers. As a result, they are uniquely suspectable to nasal obstruction, particularly when bilateral, and can present with life-threatening respiratory distress, feeding concerns, or noisy breathing. The differential diagnosis for pediatric nasal obstruction is broad; nasal obstruction in the post-natal period can manifest in different levels of the nasal passageway and vary in laterality and severity.

Recent Finding Clinicians must be able to quickly identify signs and symptoms, develop an appropriate differential diagnosis, and plan treatment to manage neonatal nasal obstruction. This review will use the newest literature to highlight common causes of neonatal nasal obstruction and discuss the epidemiology, presentation, associated problems, workup, management, and review controversial topics in neonatal obstruction.

Summary Neonatal nasal obstruction can be congenital; or secondary to neurogenic/neoplastic diseases, infectious processes, foreign bodies, inflammatory processes, or maternal factors; or can be indicative of underlying syndromic diseases.

Keywords Neonatal nasal obstruction · Choanal atresia · Congenital pyriform aperture stenosis · Rhinitis of newborn · Neurogenic nasal masses

Introduction

Neonates are obligate nasal breathers due to the elevated position of the larynx at birth. Oral breathing is difficult for neonates because the tongue is in contact with their hard and soft palate, and the epiglottis is superior and near to the soft palate [1]. The unique anatomy of the neonate pharynx is advantageous because it allows air to flow from the nose to the glottis while simultaneously allowing milk to travel from the oral cavity to the esophagus via the pyriform sinuses [2••]. This allows newborns to breathe and feed simultaneously in the first months of life. Consequently, neonatal nasal obstruction can be highly detrimental to an infant's breathing, sucking, growth, and craniofacial development, and, in some cases, can be life-threatening.

This article is part of the Topical collection on *RHINOLOGY: Nasal Obstruction*

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History and Physical Exam

A careful history and physical examination are crucial for neonates with nasal obstruction and can help identify the etiology and assess the severity. A detailed clinical history should be obtained about the infant's prenatal history, birth history, and family history.

On physical examination, clinicians should assess the breathing for warning signs of increased respiratory effort such as tachypnea, nasal flaring, grunting, intercostal or subcostal retraction, and cyanosis that improves with crying [2••]. Furthermore, clinicians should assess the patient for signs of failure to thrive and observe the patient feeding to assess for signs of poor feeding such as distress or cyanosis, frequent pauses, and assess for syndromic features.

Diagnosis

Anterior rhinoscopy and rigid or flexible nasal endoscopy are cost-effective and minimally invasive procedures that can be used to aid in the diagnosis of neonates with nasal obstruction. When imaging is indicated, computed tomography (CT) or magnetic resonance imaging (MRI) can be helpful depending on the etiology.

When to Refer

Neonates with nasal obstruction should be referred to an otolaryngologist when the symptoms begin to affect breathing, sucking, growth, and development, when abnormal nasal pathology is noted on imaging, or if treatment is indicated. Next, we will introduce and discuss classic pathologies that result in nasal obstruction in the newborn.

Congenital Pyriform Aperture stenosis

Epidemiology

The incidence of congenital nasal pyriform aperture stenosis CNPAS is unknown due to its rarity. However, it has been reported to occur at a frequency of about one-fifth to one-third that of choanal atresia [3].

Clinical Presentation

CNPAS is a rare and potentially lethal cause of nasal obstruction in neonates [4]. The pear-sharped pyriform aperture is the narrowest and most anterior opening of the bony nasal airways, and forms the lateral border of each nostril (Fig. 1). Any decrease in the cross-sectional area of the nasal pyriform aperture could lead to significant changes in airway resistance and nasal airway obstruction [5]. It is hypothesized that CNPAS results from an overgrowth of the maxillary ossification at the nasal process during the fourth months of gestation [6].

Symptom severity in CNPAS varies depending on the degree of obstruction, ranging from non-specific symptoms such as episodes of tachypnea, poor feeding, nasal congestion, cyanosis, inspiratory stridor, sternal retraction, and thoracic asymmetry to acidosis, hypoxemia, and a life-threatening airway emergency [5, 7].

Associated Problems

CNPAS can be isolated or associated with midline craniofacial abnormalities such as holoprosencephaly [8, 9]. A single central maxillary incisor has been strongly linked with CNPAS [10].

Workup

CNPAS should be suspected in newborns with narrowed nasal inlets that are not amenable to passing a 5 fr catheter [11]. CNPAS can present in a similar fashion to the more common choanal atresia and can be distinguished by performing a fine-cut facial or sinus CT with axial and coronal sections from the palate to the orbit [4]. Characteristic findings on CT are a bony overgrowth of the nasal processes with resultant narrowing of nasal passages anteriorly. Some studies have described using three-dimensional volumetric reconstructions to better evaluate CNPAS and assist with surgical planning [12]. In patients with associated craniofacial abnormalities indicative of holoprosencephaly, MRI and chromosomal analysis should be considered to assess for hypothalamic and associated brain abnormalities [4].

Management

Once the diagnosis of CNPAS is confirmed, management is generally dictated by the degree of airway stenosis, the symptom complex, and the infant's overall prognosis. Initial management of CNPAS involves establishing a secure airway by McGovern nipple placement or endotracheal intubation and monitoring in the intensive care unit until the cause and severity of CNPAS are established [7]. Patients with mild cases can be managed conservatively in the short term with nasal decongestants and humidification. In a long-term

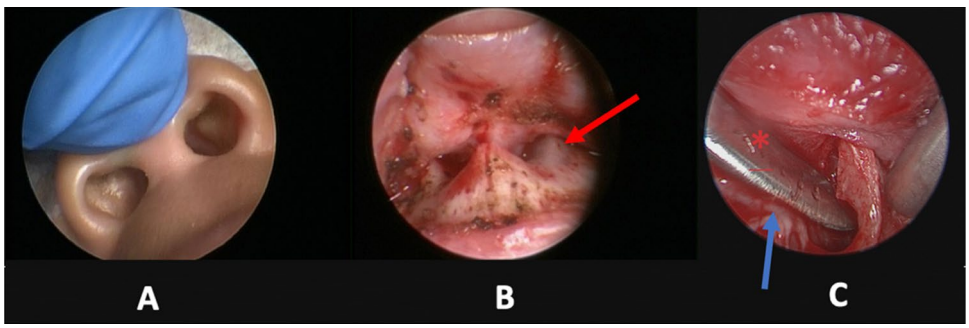

Fig. 1 **a** External view of obstruction due to CNPAS. **b** Red arrow demonstrates thickened left piriform edge bone in CNPAS that is operated on in the adjacent right picture. **c** Instrument (labeled with asterisk) passed medial to thick left piriform edge bone in CNPAS, now partially removed with drill. Nasal septum (blue arrow) is on the left side of the image, deep to the instrument.

analysis of two patients with CNPAS who were managed expectantly, it was found that patients' airways were likely to improve with growth, typically within 6 months of birth [13].

Stenting is sometimes used as a means of conservative management; however, it is not widely advocated due to the risk of mucosal and cartilaginous pressure necrosis and risk of occlusion due to the smaller lumen present in the nasal stents [14]. Surgery is usually the most definitive therapy in infants with CNPAS who continue to be symptomatic with conservative management or in infants with moderate to severe stenosis. The surgical procedure of choice involves pyriform aperture enlargement through an endo-oral sublabial approach to reshape and widen the stenotic area [7]. The timing of surgery varies depending on the degree of stenosis and how the infant responds to conservative management.

When to Refer

Referral to a geneticist and endocrinologist is recommended if the patient is suspected of having comorbid craniofacial abnormalities such as holoprosencephaly.

Rhinitis of Newborn

Epidemiology

Neonatal rhinitis is one of the most common causes of nasal obstruction in newborns [15]. A retrospective analysis of 20 patients noted a strong seasonal variation, with most infants presenting in the fall and winter months [16].

Clinical Presentation

Neonatal rhinitis is an idiopathic disorder characterized by a constellation of symptoms that includes noisy breathing, bilateral mucoid rhinorrhea, and unexplained poor feeding in the absence of underlying infectious and structural abnormalities [16]. The exact cause of neonatal rhinitis is unknown. However, the etiology may be multifactorial, involving allergic, drug, hormonal, inflammatory, trauma, and vascular-related factors [17]. Maternal progesterone has been proposed as a possible cause, but no data support this hypothesis to date [18]. Clinical manifestations of neonatal rhinitis are non-specific. However, it can present as nasal obstruction with stertor and feeding difficulties [17]. Examinations typically show symmetric and bilateral mucosal edema and mucoid rhinorrhea. In some cases, neonatal rhinitis may present alongside other causes of nasal obstruction and exacerbate the underlying cause.

Workup

Neonatal rhinitis is diagnosed clinically by excluding structural etiology and infectious etiology. This includes other causes of rhinitis, such as gastroesophageal reflux, cow's milk protein allergy, and hypothyroidism. Additionally, infectious etiology such as upper respiratory tract viruses, chlamydia, and syphilis should be considered. Maternal factors such as estrogen stimulus or medications such as methyldopa, propranolol, and tricyclic antidepressants should also be excluded [2].

Management

Neonatal rhinitis should be managed conservatively with humidification and gentle suction of secretions. Topical steroids and nasal decongestants can be helpful in alleviating symptoms of neonatal rhinitis. Parents should be advised to limit the use of vasoconstrictive agents to 3 days to avoid rhinitis medicamentosa, which could be life-threatening in the neonatal period [19]. Topical steroid-antibiotic combination drops such as otic Ciprodex applied to the nose for 2–3 days often treat the problem while avoiding rebound congestion. Care must be taken to avoid adrenal suppression, however. Patients with rhinitis related to specific ingestion are diagnosed clinically, and treatment consists of diet modification and avoiding offending agent. Patients who do not improve with conservative therapy warrant further evaluation by a pediatric otolaryngologist.

Septal Deformity

Epidemiology

The incidence of nasal septal deformity in neonates is controversial, varying from 0.6 to 31% in the literature [20].

Clinical Presentation

Neonatal septal deformities can be classified into two types: anterior dislocation and combined septal deformity [21]. Anterior dislocations are thought to occur as a result of birth trauma and involve subluxation of the septal cartilage from the maxillary groove and show an external deformity. Combined dislocations are thought to occur due to intrauterine

nasal compression and involve both the cartilaginous anterior and bony posterior septum and may not show an external deformity. Dislocation of the septal cartilage may manifest as an external nasal deformity with nasal discharge and noisy breathing, or, in severe cases, may demonstrate poor feeding and respiratory compromise.

Associated Problems

The neonatal septal deviation may be associated with syndromes or cleft lip/palate deformities. A persistently deviated nasal septum may predispose to sinusitis, epistaxis, Eustachian tube dysfunction, otitis media, upper and lower respiratory tract infections, dental malalignments, and poor general health [20]. Passage of supportive devices such as suction or nasoenteral feeding tubes may also be more challenging.

Workup

The physical exam will show a deviated nasal tip, an angulated columella, and a flattened and asymmetric nasal ala. Diagnosis of nasal septal deviation can be achieved with anterior rhinoscopy, which will show nasal septal asymmetry. Nasal endoscopy can confirm the diagnosis. Differential diagnosis includes a flattened nose, which can present with similar symptoms but would show normal findings on the endonasal exam [1]. Differential diagnosis should also include septal hematoma, which would require incision and drainage to avoid abscess, cartilage necrosis, and nasal deformity.

Management

The management of septal deviation is controversial, and there is no consensus available. The literature suggests that immediate treatment of nasal deformity does not significantly affect the clinical status of the nose at a 7-year follow-up [22]. In patients with significant septal deviation, a closed reduction can be performed at the bedside with various techniques or under general anesthesia. In deciding management, one article recommends taking into account the type of deviation, degree of obstruction, symptoms, extent of cosmetic deformity, and family wishes [21]. In the author's experience, formal septoplasty or septorhinoplasty is not typically performed in an infant, and it is preferred to wait until after adolescent growth is completed due to concern about nasal growth if the aforementioned procedures are performed prematurely.

Nasolacrimal Duct Cyst

Epidemiology

While an estimated 30% of neonates have some degree of distal nasolacrimal duct obstruction at birth, the formation of symptomatic cysts is rare, and most cases of obstruction resolve spontaneously by 9 months of age [23].

Clinical Presentation

Cannulization of the nasolacrimal duct normally begins proximally at the lacrimal end of the duct and progresses inferiorly [23]. Nasolacrimal duct cysts form due to aberrant cannulization distally and persistent occlusion of the nasolacrimal duct [23] [24•]. The cyst forms due to fluid accumulation in the nasolacrimal duct secondary to an imperforate valve of Hasner and proximal valve–like obstruction at the junction of common canaliculus and lacrimal sac [25]. Neonates with unilateral nasolacrimal duct cyst present with nasal obstruction and epiphora and may also have a cystic mass of bluish coloration in the medial canthal region [25]. Neonates with large bilateral cysts that fill the inferior meatus present with respiratory distress and may need early surgical intervention.

Associated Problems

A retrospective medical review by Lueder et al. [26] found that almost all infantile dacryocystoceles and most cases of infantile acute dacryocystitis are associated with nasolacrimal duct cysts.

Workup

Anterior rhinoscopy or nasal endoscopy with a focus on the inferior meatus can aid in proper diagnosis by showing cystic masses in the inferior meatus [23]. CT scan may help confirm the diagnosis by showing a dilated nasolacrimal duct, intranasal cyst, displaced inferior turbinate, and/or cystic dilation of the lacrimal sac.

Management

Most patients with nasolacrimal duct cysts can be managed conservatively with warm compresses and lacrimal massage [25]. Operative management of nasolacrimal duct cysts consists of transnasal endoscopic marsupialization of the cyst with a microdebrider or curette in neonates with feeding problems, infection, or respiratory obstruction [23].

Ophthalmology should be consulted to evaluate for the need of nasolacrimal duct probing and possible nasolacrimal duct stenting.

Neurogenic Nasal Mass

Epidemiology

Congenital neurogenic frontonasal masses are rare non-neoplastic lesions that occur in one out of every 20,000–40,000 births [27]. The three major types of frontonasal masses are nasal dermoids, nasal gliomas, and encephaloceles. Nasal dermoid cysts are the most common type of nasal midline masses [28]. Nasal gliomas occur sporadically and have 3:2 male predilection [1]. Encephaloceles are more common in Asian populations with an incidence ranging from 1:5000 to 1:6000 [29]. The incidence of encephaloceles is decreasing due to emphasis on prenatal folic acid supplementation in pregnant women [30].

Clinical Presentation

Midline nasal masses occur as a result of maldevelopment of the anterior neuropore. The fonticulus nasofrontalis is the space between the developing nasal base and frontal bone, and eventually fuses with the foramen cecum, ultimately dividing intracranial and extracranial structures. Midline nasal masses can manifest clinically as an external deformity or can be solely intracranial with no apparent signs on physical examination. Large nasal masses can present with nasal obstruction in neonates and are important to consider in the differential diagnosis of neonates with respiratory distress, particularly because biopsy or resection carries a significant risk of CSF leak.

Nasal dermoids form when the neuroectodermal tract fails to involute through the anterior neuropore. This can result in a pit, sinus tract, and/or cyst localized from the nasoglabellar region to the base of the columella. The most common location is the lower third of the nasal bridge. Nasal dermoids contain ectodermal and mesodermal components and may contain various skin appendages including hair follicles, sweat glands, and sebaceous glands [31]. Intracranial extension can occur; however, the incidence reported in the literature varies from 5 to 45% [32]. Patients with nasal dermoid may present at birth with midline nasal dorsum lump and/or pit. Alternatively, patients may present with a slow-growing mass that becomes inflamed or infected in early infancy. Hair from the pit is pathognomonic for nasal dermoid. Nasal dermoid cysts do not transilluminate, are not compressible, and are non-expansile [1].

Nasal gliomas are isolated extracranial lesions containing dysplastic neuroglial tissue which do not contain patent CSF communications with the subarachnoid space and can be located in either intranasal or extranasal compartments. Nasal gliomas can sometimes connect to the brain by a fibrous stalk. It has been reported that 30% of nasal gliomas are intranasal, appearing pale and polypoid in the nasal cavity which can cause nasal congestion and obstruction; 60% are extranasal which appear smooth, firm, and non-compressible, has negative Furstenberg, do not transilluminate, and often found along the nasal dorsum; and 10% are combined and are firm and non-compressible, have no change on crying, and have negative Furstenberg test [31]. Presentation ranges from an external mass to subtitle findings such as telecanthus and widened nasal bridge [1].

Nasal encephaloceles are extracranial herniation of meninges, cerebrospinal fluid, and neural tissue through a skull base defect. Subclassifications of nasal encephaloceles include (1) frontoethmoidal (sincipital) encephalocele which presents as a soft blueish compressible mass protruding from glabella and (2) basal encephaloceles which present as a intranasal polypoid mass, often clinically silent, although they can present with respiratory distress in neonates [31]. Nasal encephaloceles usually present at birth as a soft midline nasal mass. Characteristic nasal encephalocele features include being pulsatile and compressible, having bluish coloration, increasing in size while crying, transillumination, and having a positive Furstenberg test—mass expansion or pulsation with compression of the ipsilateral jugular veins. Encephaloceles can be distinguished from gliomas by the presence of ependymal tissue in encephaloceles in some cases. [33].

Neoplasia should be considered in the differential diagnosis when evaluating patients with suspected neurogenic masses. Differential diagnosis should include teratoma, hamartomas, rhabdomyosarcoma, hemangioma, neurofibroma, salivary tumors, and lymphatic malformations.

Associated Problems

Nasal dermoids are associated with other congenital anomalies in up to 40%, including but not limited to abnormalities such as craniosynostosis, hemifacial microsomia, lacrimal duct cysts, cleft lip/palate, pinna deformity, hydrocephalus, and hypertelorism [1]. Encephaloceles are associated with abnormalities in 30–40% of cases [34].

Workup

MRI is the imaging modality of choice for evaluating neurogenic masses as it allows multiplanar imaging, distinguishing the interface among cartilage, bone, brain, and fluid, diffusion imaging to detect epidermoid tumors, and the capacity to evaluate the brain for associated cerebral anomalies [35]. CT may be helpful when evaluating neurogenic

masses for visualizing bony defects of the skull base and identifying the presence of a sinus tract (Fig. 2).

Management

Surgical correction is often required to remove the lesion and close any intracranial communication to decrease the risk of meningitis and cerebrospinal leak. Depending on the extent of the lesion, surgery may be performed in collaboration with an otolaryngologist, plastic surgeon, and neurosurgeon (Fig. 2). Extracranial lesions can be addressed with external rhinoplasty and midline incision or via extended endonasal approaches to the anterior skull base.

Choanal Atresia

Epidemiology

The incidence of choanal atresia is estimated between 1:5000 and 1:7000 live births, with a higher propensity to occur unilaterally (60%) compared to bilaterally 40%, and 2:1 female to male predominance [36, 43].

Clinical Presentation

Choanal atresia is a congenital disorder that occurs when there is failed recanalization of the nasal fossae during fetal development. This results in the anatomical closure of the posterior nasal choanae by soft tissue, bone (i.e., atresia plate), or a combination of both (Fig. 3). A retrospective study reviewing computer tomography and histology specimens in 63 patients showed 29% pure bony atresia and 71% mixed membranous and bony atresia, with no pure membranous atresia [37]. There are several proposed theories as to the pathogenesis including the persistence of the buccopharyngeal membrane, persistence of the nasobuccal membrane of Hochstetter, incomplete resorption of the nasopharyngeal mesoderm, and local misdirection of neural crest cell migration [36].

The presentation of an infant with choanal atresia varies from acutely life-threatening airway obstruction to chronic recurrent rhinosinusitis, depending on whether choanal atresia is unilateral, bilateral, or associated with other coexisting airway abnormalities. Unilateral choanal atresia commonly presents with purulent nasal discharge and obstruction on the affected side and/or a history of chronic rhinosinusitis. Some patients with unilateral choanal atresia are not diagnosed until adulthood due to nonspecificity of symptoms.

Infants with bilateral choanal atresia often present with episodes of acute respiratory distress and cyanosis that are characteristically relieved by crying. Cyanosis tends to recur with rest, termed paradoxical cyanosis [36].

Associated Problems

In a study of 129 patients with choanal atresia and stenosis, 51% of subjects had another congenital anomaly [38]. Interestingly, our experience has even included patients with cleft palate and bilateral choanal atresia in the setting of Treacher-Collins syndrome. Choanal atresia is most often associated with CHARGE syndrome; however, it can also be seen in patients with chromosomal abnormalities, single-gene defects, deformations, and secondary to teratogens [38]. Unilateral cases were more likely to be isolated, while bilateral cases were more likely to be associated with specific disorders/congenital anomalies. A mouse model by Dupe et al. [39] has demonstrated an association between retinoic acid deficiency and choanal atresia. While several case reports have associated prenatal thioamide exposure (methimazole, propylthiouracil, and carbimazole) with CA, a case–control study proposed that a mother's hyperthyroidism may be the causal factor for choanal atresia [43].

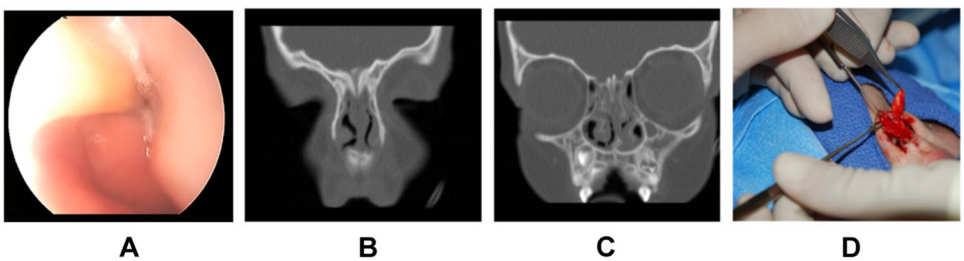

Fig. 2 **a** Endoscopic view of an obstructing nasolacrimal duct cyst (NDC) on the right side of the nose causing the inferior turbinate (IT) to be pushed superior medially. **b** Coronal CT showing deep fonticulus frontalis in patient with nasal dermoid with intracranial extension. **c** Coronal CT showing bifid crista galli in patient with nasal dermoid. **d** Open rhinoplasty approach to nasal dermoid excision. The photograph shows an excised nasal dermoid tumor with crista galli at tip of instrument. Legend: IT, inferior turbinate. NS, nasal septum. NDC, nasolacrimal duct cyst.

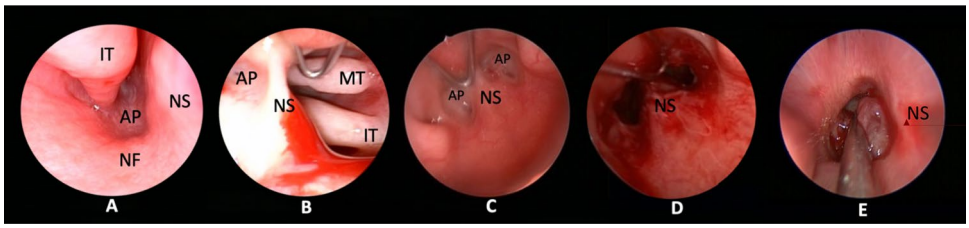

Fig. 3 **a** Endoscopic view of unrepaired choanal atresia. **b** Transoral 120° endoscope view of unrepaired unilateral choanal atresia—note contrast between patent and atretic sides. **c** Transoral 120° telescope view of bilateral choanal atresia. **d** Transoral 120° telescope view of nasopharynx and patent choanae after endoscopic choanal atresia repair with dilators, drill, Kerrison Rongeurs, and backbiter. **e** Endonasal endoscopic view of nasopharyngeal teratoma requiring EXIT-to-airway followed by endoscopic resection. Image courtesy of Douglas R. Sidell, MD. Legend: IT, inferior turbinate. MT, middle turbinate. NF, nasal floor. NS, nasal septum. AP, atresia plate.

Workup

Initial clinical evaluation includes the introduction of a 6Fr suction catheter or small nasogastric tube via the nostrils, methylene blue dye test, cotton wisp test, and laryngeal mirror test [43]. Obstruction that is 3–3.5 cm from the alar rim in the neonate is characteristic of choanal atresia. Flexible nasal endoscopy in patients with proper preparation is the preferred method of diagnosis of choanal atresia as it allows for direct visualization of the point of obstruction in the nasal passage and confirms the presence of an atretic plate in the choana. Non-contrast CT of sinuses with fine cuts allows for definitive evaluation. As with nasal endoscopy, patients undergoing CT scans should receive proper nasal preparation.

Management

Initial management of choanal atresia is generally dictated by the severity of respiratory distress at birth. The goal of initial treatment is aimed at maintaining an adequate oral airway, which can be achieved with a McGovern nipple, which can help achieve an adequate airway while also allowing feeding via a small feeding tube that can be placed through another hole in the nipple. Endotracheal intubation should be considered if McGovern nipple is not successful in maintaining an adequate airway. Infants who cannot undergo early atresia repair due to cardiopulmonary instability and multilevel airway obstruction may require a tracheostomy. Infants with bilateral choanal atresia or CHARGE syndrome are more likely to fail atresia repair due to their more contracted nasopharynx, narrower posterior choanal region, and poor tongue/pharyngeal muscle control. Asher et al. [40] recommend early tracheostomy in patients with CHARGE syndrome due to their propensity to fail early choanal atresia repair and risk of cerebral hypoxia.

Patients with unilateral choanal atresia have relatively lower morbidity, and definitive treatment is often delayed until later in life when anatomy is more favorable, and chances of successful surgical repair are higher. Multiple surgical approaches have been proposed to repair choanal atresia, and in spite of abundant literature, there is no consensus on the recommended surgical technique. However, the transnasal endoscopic repair has become the primary procedure for most pediatric otolaryngology surgeons according to a survey performed by Park et al. [41]. Postoperative stent placement has been traditionally used after choanal atresia repair as a measure to reduce rates of restenosis. However, recent studies, including a review by Bedwell et al. [42] and meta-analysis of 238 cases from 20 studies by Durmaz et al. [44], suggest that there are no significant differences in restenosis rates between stenting and no stenting [42, 43, 44]. Furthermore, post-operative stenting is associated with complications such as discomfort, localized infection and ulceration, and scar or granulation tissue formation [45]. The routine use of mitomycin C to decrease restenosis rate after choanal atresia repair is not supported by recent studies, which demonstrate no significant difference in restenosis rate between the mitomycin group and control group [46, 47, 48].

Nasopharyngeal Mass

Epidemiology

The frequency of Tornwaldt cyst was 3.5% according to the autopsy series, while an MRI series ranged from 1.9 to 5% [49]. Head and neck nasopharyngeal teratomas occur in one of every 40,000 births [50].

Clinical Presentation

Tornwaldt cysts are closed sacs that develop when the pharyngeal bursa or pouch of Luschka becomes occluded secondary to inflammation or surgical trauma [49]. The size of

the cyst can range from 1 to 30 mm, and while most patients are asymptomatic, patients with large cysts can develop post-nasal drip, occipital halitosis, stiffness of cervical muscles, and aural fullness secondary to Eustachian tube dysfunction [49]. Infected cysts can present as a nasopharyngeal mass.

Nasopharyngeal teratomas are a type of germ cell tumor composed of the three embryologic layers that present as firm midline or lateral pharyngeal wall lesions with localized tissue destruction [17]. Teratomas may have a stalk and can be mobile with different positions and manifest as a soft midline neck mass.

Associated Problems

Hydrops fetalis, polyhydramnios, and pulmonary hypoplasia have been associated with teratoma formation [52].

Workup

Patients with Tornwaldt cyst will show fluid attenuating lesions between the longus capitis muscles on CT [49]. Tornwaldt cyst is easily recognizable in MRI as a midline cyst in the nasopharynx with high signal on T2 and T1. In addition to imaging, laboratory studies such as alpha-fetoprotein and beta-hCG tumor markers may be elevated in teratoma. Teratomas manifest as heterogeneous masses with fatty and bony components on MRI. CT can reveal calcification, ossification, or cystic formation.

Management

Nasopharyngeal Tornwaldt cysts causing nasal obstruction can be treated with intranasal, endoscopic approach to marsupialize the cyst [51•]. Nasopharyngeal teratomas require complete surgical excision with endoscopic transnasal approach or transoral resection, although larger tumors may require lateral rhinotomy or transpalatal approach [17].

Conclusion

Neonatal nasal obstruction can range from asymptomatic to life-threatening respiratory distress that requires immediate attention. Physical exam findings can help localize the level and severity of obstruction in the nasopharynx. Imaging is very useful in narrowing the differential diagnosis. Surgery via endoscopic or transoral approach can be curative. Syndromic conditions can manifest as neonatal obstruction and are important to keep in mind when working up a neonate with nasal obstruction.

Declarations

Conflict of Interest The authors declare no competing interests.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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•• Of major importance

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