

Nasal Obstruction in Newborns

Sharon H. Gnagi, MD^a, Scott A. Schraff, MD^{b,*}

KEYWORDS

- Nasal obstruction • Choanal atresia • Pyriform aperture stenosis
- Nasolacrimal duct cyst • Rhinitis • Nasal dermoid • Glioma • Encephalocele

KEY POINTS

- Nasal obstruction in newborns can range in severity from a mild irritant to a life-threatening situation with potentially devastating consequences including respiratory distress and failure to thrive.
- The differential diagnosis for nasal obstruction of the newborn is vast, requiring a thorough history, physical examination, nasal endoscopy, and imaging for accurate diagnosis and treatment.
- The most common etiology of nasal obstruction is simple inflammation of the nasal mucosa, which may be managed conservatively.
- Several etiologies of nasal obstruction may warrant further evaluation and genetic workup to diagnose associated conditions.

INTRODUCTION

Newborn infants are obligate nasal breathers for the first several months of life, with more than 50% of infants desaturating if nasally obstructed.¹ Anatomically, their entire tongue length is in contact with the hard and soft palate, and the epiglottis is superior to the soft palate, causing difficulty with oral breathing.² This allows for concomitant respiration with oral intake while the infant is learning to mouth breathe in the first 4 to 6 weeks after birth. Therefore, nasal obstruction may lead to serious consequences in the neonate, including respiratory distress or failure to thrive. Thus, nasal obstruction is an important clinical entity to recognize, effectively diagnose, and treat.

EVALUATION

History

Appropriate evaluation of nasal obstruction in newborns requires a thorough neonatal history. Signs and symptoms consistent with nasal obstruction may be described by

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^a Department of Otolaryngology, Mayo Clinic Arizona, 5777 East Mayo Boulevard, Phoenix, AZ 85054, USA; ^b Department of Otolaryngology, Phoenix Children's Hospital, 1919 East Thomas Road, Phoenix, AZ 85006, USA

* Corresponding author.

E-mail address: schraffs@hotmail.com

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the parents, family, or care providers (**Box 1**). Maternal history is equally important, as maternal medical conditions, drug ingestion, and sexually transmitted diseases can be etiologies of nasal obstruction in the newborn. Familial genetic disorders, and/or prenatally diagnosed conditions should also be noted. Birth history including prematurity, length of labor, presentation, and trauma during delivery (eg, use of forceps) can implicate a potential iatrogenic cause of nasal obstruction.

Additionally, timing and onset of symptoms can provide clues as to the etiology of nasal obstruction. While bilateral nasal obstruction often presents in the neonatal period, unilateral nasal obstruction may not present until much later in life, with chronic nasal drainage, skin irritation, and congestion. Intermittent respiratory distress at birth may be associated with a ball-valving obstruction or intermittent nasal congestion associated with the physiologic nasal cycle.³ Thus, Apgar scores and need for resuscitation and/or intubation at birth may help delineate the severity and anatomic extent of the obstruction.

Physical Examination

The first task when assessing the newborn with nasal obstruction is to determine the degree of respiratory difficulty and establish a safe airway. Tachypnea, nasal flaring, substernal and costal retractions, and irritability suggest respiratory distress. Lethargy and cyanosis are more concerning and suggest respiratory fatigue, impending failure, and need for an immediate intervention.

If an adequate airway exists, the physical examination begins with external inspection for a gross deformity, asymmetry, or pit. Then, nasal patency should then be assessed. Anterior rhinoscopy with an otoscope may also help visualize anterior stenosis, masses, or obstructive mucus. Application of a decongestant may enhance the

Box 1 Signs and symptoms of nasal obstruction
Stuffy nose
Rhinorrhea
Mucus
Stertor
Snoring/snorting
External deformity
Nasal flaring
Chest Retractions
Cyanosis (± cyclical nature)
Feeding difficulties
Hyponasal cry
Failure to thrive
Dyspnea/apnea
Aerophagia with abdominal distention
Difficulty sleeping
Epiphora

examination and enable the provider to compare the congested and decongested extent of obstruction to distinguish between anatomic obstruction and mucosal edema.⁴ Alternative methods of assessing patency include placing a mirror or spoon under the nare and visualizing condensation, administering nasal saline and observing bubbles, or closing 1 nare and the mouth and auscultating for air movement.⁵ Another option includes gently passing a small (5 or 6 French) catheter through the nose into the nasopharynx to confirm an open communication. Obstruction at the anterior inlet may suggest pyriform aperture stenosis, while obstruction posteriorly (approximately 32 mm) may suggest choanal atresia.⁶ Visualizing or palpating the tube through the mouth confirms that the tube is not coiled in the nose to prevent misdiagnosis. In infants with craniofacial abnormalities or visible nasal masses, care must be taken when attempting to pass a nasal catheter, as these may be associated with skull base defects and risk intracranial passage of the catheter.⁷ Additional studies to assess nasal patency include the use of a tympanometer placed at the nare to confirm or exclude a closed cavity.⁸

Nasal Endoscopy

Nasal endoscopy is typically the next step to determine the location or causality of the obstruction. A flexible endoscopic nasal examination is a simple and minimally invasive diagnostic procedure that can be performed in the office or at bedside with no sedation by trained specialists. The examination poses essentially no risk and causes minimal distress to the patient and family. In the rare instance that an adequate endoscopic nasal examination cannot be completed at the bedside, further evaluation in the operating room may be warranted. If a nasal mass is identified on endoscopy, it should be assumed to have intracranial extent until proven otherwise. Therefore, no biopsy of a mass should be performed until appropriate imaging has been undertaken.⁹

Imaging

There are multiple imaging modalities available for assessing nasal obstruction and the upper airway of newborns. Historically, plain radiographs with radiopaque contrast in the nasal cavity have been used to assess for obstruction. With the advent of more sophisticated imaging technology, plain films are rarely employed because of poor sensitivity and specificity. Computed tomography CT scans allow the best bony definition, and are typically the test of choice to assess choanal atresia and pyriform aperture stenosis. Suctioning of the nose before CT scanning is helpful to clear secretions that may be confused with a soft tissue or membranous obstruction.¹⁰ Magnetic resonance imaging (MRI) is a better choice to evaluate nasal masses to delineate intracranial involvement and extent. MRI also avoids radiation exposure in infants and may be preferentially selected over CT for this reason.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for nasal obstruction in newborns is vast (**Box 2**). A thorough evaluation includes careful consideration of each potential etiology, and acknowledgment that more than 1 etiology may coexist.

Congenital Etiologies

There are countless congenital malformations secondary to aberrant embryogenesis of both the internal and external nose potentially causing nasal obstruction. These include but are not limited to: midfacial hypoplasia, craniosynostosis, arhinia

Box 2 Differential diagnosis of neonatal nasal obstruction	
<i>Congenital</i> • Choanal atresia • Congenital nasal pyriform aperture stenosis • Midfacial hypoplasia • Nasolacrimal duct cysts • Midline nasal masses ◦ Nasal dermoid ◦ Glioma ◦ Encephalocele/meningocele ◦ Thornwaldt cyst	<i>Foreign body</i>
<i>Neoplasms</i> • Teratoma • Hamartoma • Hemangioma • Lymphangioma • Lipoma • Neurofibroma • Rhabdomyosarcoma • Lymphoma	<i>Traumatic/iatrogenic</i> • Septal dislocation • Septal hematoma • Nasal tip depression • Rhinitis medicamentosa • Instrumentation: suction trauma, nasogastric tube, CPAP, nasal prongs
<i>Infectious</i> • Upper respiratory infection • Respiratory syncytial virus • STDs ◦ Chlamydia ◦ Gonorrhea ◦ Syphilis	<i>Inflammatory</i> • Allergic rhinitis (cow's milk, soy) • Gastroesophageal reflux • Recurrent emesis • Idiopathic
	<i>Metabolic</i> • Hypothyroidism
	<i>Maternal</i> • Estrogenic stimuli • Drug ingestion (methimazole, methyl dopa, opiates, tricyclic antidepressants, propranolol)
	<i>Associated syndromes</i> • Cystic fibrosis • Kartagener • CHARGE • Apert • Crouzon • Treacher-Collins • Fetal alcohol syndrome • Down

(complete absence of the nose), nasal hypoplasia (congenitally absent nasal bones), complete or partial nasal duplication, single centrally placed nostril, supernumerary teeth in the nose, Thornwaldt cyst, nasopharyngeal stenosis (incomplete separation of the soft palate and posterior pharyngeal wall), and others. Of these numerous congenital causalities, the most common and clinically significant will be discussed.

Choanal atresia

Normally, the nasal cavity is connected to the remainder of the airway via the nasopharynx. Choanal atresia is the failure of the nasal cavity to connect to the naso- and oropharynx. Theoretically, this results from alterations in embryogenesis with persistence of the buccopharyngeal membrane or failure of the oronasal membrane

to rupture, although no theory has been proven.² It occurs in approximately 1 of 5000 to 8000 live births and is twice as common in girls.¹¹ Unilateral choanal atresia is more common than bilateral involvement, accounting for 65% to 75% of cases. Comparatively, bilateral choanal atresia has more serious clinical implications and is associated with other congenital abnormalities in 50% of patients.⁶ Although most readily associated with CHARGE syndrome (**Box 3**), it is also seen with polydactyly, Crouzon syndrome, craniosynostosis, microencephaly, meningocele, facial asymmetry, cleft palate, hypertelorism, and nasal and auricular deformities.²

Nasal endoscopy demonstrates a blind sac with a lack of communication from the nasal cavity to the nasopharynx (**Fig. 1**). CT is the radiographic imaging method of choice and reveals narrowing of the posterior nasal cavity, medialization of the lateral nasal wall, and thickening of the vomer; it may be classified as bony, membranous, or mixed (**Fig. 2**).¹² Because of its association with multiple other anomalies, a genetic evaluation and thorough workup of possible associated conditions should be completed.

Treatment for choanal atresia requires surgical intervention. The timing of intervention is largely dependent on bilateral versus unilateral involvement. Newborns with bilateral choanal atresia typically present with respiratory distress at birth and may require intubation to establish an airway. In bilateral cases, surgical correction is typically performed in the first week of life and has been described via transnasal puncture, transpalatal and endoscopic transnasal approaches plus or minus postoperative stenting, mitomycin C application, and laser use.⁷ In unilateral cases, treatment is typically delayed until just before school attendance to allow growth and development of the midface, while stopping embarrassing nasal drainage and resultant skin irritation, and relieving nasal obstruction before interaction with peers.²

Congenital nasal pyriform aperture stenosis

Congenital nasal pyriform aperture stenosis (CNPAS) is an uncommon etiology of nasal obstruction resulting from bony overgrowth of the nasal process of the maxilla.¹³ The pyriform aperture is a pear-shaped bony inlet comprising the most anterior and narrowest bony portion of the nasal airway; therefore, any overgrowth causes a decrease in cross-sectional area with resultant exponential increase in airway resistance and associated obstruction.¹⁴ Anterior rhinoscopy reveals a narrowed anterior nasal passage with bony thickening medially, typically affecting bilateral nares (**Fig. 3**). Nasal endoscopy may not be able to be performed secondary to the small anterior passage. CT is typically the imaging method of choice and confirms the diagnosis if the pyriform aperture measures less than 11 mm at the level of the inferior meatus (**Fig. 4**).¹⁵

Box 3

CHARGE association

C: Colobomas

H: Heart abnormalities

A: Choanal atresia

R: Growth or mental retardation

G: Genitourinary anomalies

E: Ear abnormalities



Fig. 1. Nasal endoscopy revealing membranous occlusion of the posterior nasal cavity consistent with choanal atresia.

CNPAS may occur as an isolated anomaly or in association with absence of the anterior pituitary, diabetes insipidus, submucous cleft palate, and hypoplastic maxillary sinuses. Oral examination may reveal an absent upper labial frenulum and a prominent central mega incisor (**Fig. 5**). It may manifest as part of the holoprosencephaly

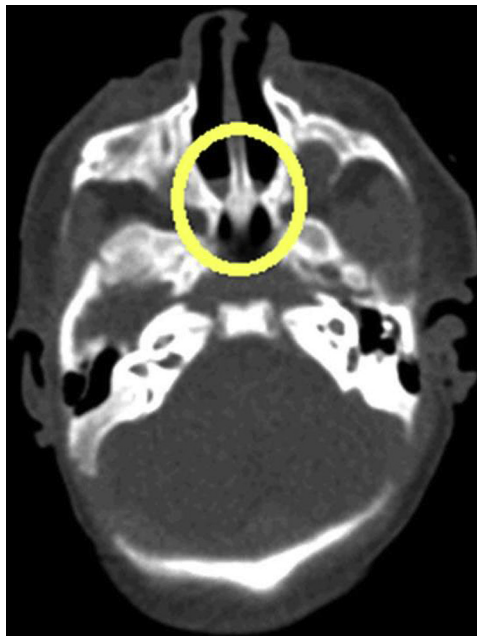


Fig. 2. Axial CT showing atretic choanae. Circle encompasses atresia at posterior nasal vault. Note the champagne flute appearance of the nasal caults characteristic for choanal atresia. (From Shah UK, Daniero JJ, Clary MS, et al. Low birth weight choanal atresia repair using image guidance. *Int J Pediatr Otorhinolaryngol* 2011;75:1339; with permission.)

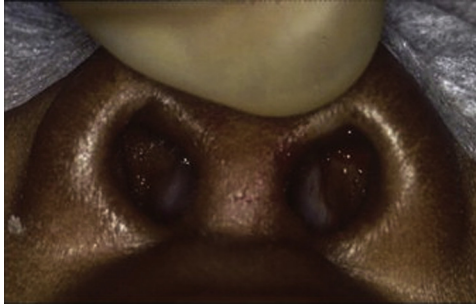


Fig. 3. Congenital nasal pyriform aperture stenosis, as seen on anterior rhinoscopy. (From Elluru RG, Wootten CT. Congenital malformations of the nose. In: Flint PW, Haughey BH, Lund VJ, et al, editors. Cummings otolaryngology: head & neck surgery. 5th edition. Philadelphia: Mosby; 2010. p. 2693; with permission.)

sequence (HPE), characterized by failure of the prosencephalon (forebrain) to divide into bilateral cerebral hemispheres.^{14,16,17} Suspicion of HPE warrants chromosomal analysis, genetic consultation, and possibly an endocrinology workup and electrolyte evaluation. Further imaging of the brain may be required with MRI.

Management is dependent on the severity of symptomatology, but may require surgical intervention to drill and widen the pyriform aperture (**Fig. 6**). This is typically performed via a sublabial approach and may require nasal stents left up to a month postoperatively to prevent restenosis.

Nasolacrimal duct cysts

Nasolacrimal duct obstruction is a common congenital abnormality and occurs in approximately 30% of neonates.¹⁸ In most cases, the obstruction is asymptomatic

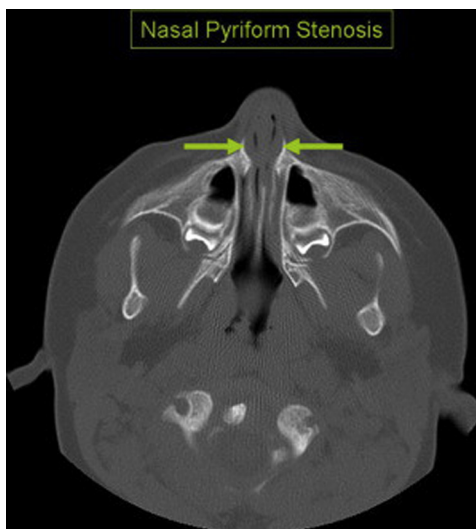


Fig. 4. CTscan of a child who has CNPAS. Note the inward bowing of the nasal processes of the maxillary bone with the pyriform aperture measuring 7 mm (arrows). (From Tate JR, Sykes J. Congenital nasal pyriform aperture stenosis. Otolaryngol Clin North Am 2009;42:523; with permission.)



Fig. 5. Child who has CPAS and evidence of a single central megaincisor. (From Tate JR, Sykes J. Congenital nasal pyriform aperture stenosis. *Otolaryngol Clin North Am* 2009;42:523; with permission.)

and resolves spontaneously within the first year of life.¹⁹ However, if there are both a proximal obstruction and a distal obstruction within the duct, a cyst known as a dacryocystocele forms, which may herniate into the nasal cavity and cause obstruction. Distal obstruction typically occurs at the valve of Hasner, which normally ruptures with neonatal respirations at birth.²⁰ Bilateral obstruction occurs in approximately 14% of congenital dacryocystoceles, and a fraction of these protrude into the nasal cavity causing resultant respiratory distress.²¹

Associated findings include epiphora and facial swelling with blue-to-red discoloration inferior to the medial canthus of the eye. Nasal endoscopy reveals a mass protruding below the inferior turbinate (**Fig. 7**). Nasolacrimal duct cysts can be differentiated from midline nasal masses by the ability to bypass them medially on endoscopy.²⁰ Imaging reveals cystic masses projecting into the nasal cavity with superomedial displacement of the inferior turbinate, and may demonstrate dilation of the lacrimal duct and sac (**Fig. 8**). Conservative management including warm compresses and gentle massage may be effective for simple dacryocystoceles without intranasal extension. However, for those with intranasal extension, management normally consists of probing of the duct and/or surgical marsupialization of the intranasal cyst.³

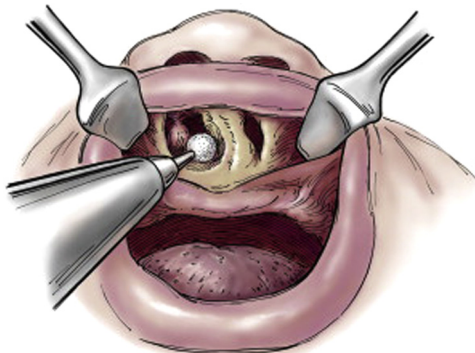


Fig. 6. Sublabial approach to the pyriform aperture. A drill is used to increase the cross-sectional area of the pyriform aperture. (From Tate JR, Sykes J. Congenital nasal pyriform aperture stenosis. *Otolaryngol Clin North Am* 2009;42:522; with permission.)

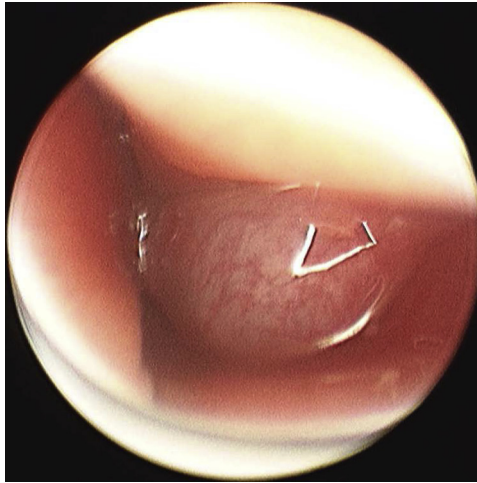


Fig. 7. Endoscopic view of a nasolacrimal duct cyst lateral and inferior to the inferior turbinate resulting in superomedial displacement of the turbinate.

Congenital frontonasal masses

Congenital midline nasal masses may cause nasal obstruction in the newborn and present a diagnostic challenge for the clinician, as they may represent several different underlying pathologies. Although the most common etiologies are nasal dermoid, encephalocele, and glioma, the differential consists of other neoplasms will be discussed (**Fig. 9**). Frontonasal masses are rare and occur in only 1 of every 20,000 to 40,000 births.²² All result from aberrant embryogenesis of the anterior neuropore, typically producing a skull base defect with risk of intracranial connection. Because of this, children presenting with midline nasal masses may be at increased risk of developing meningitis, and a biopsy should not be performed before imaging.

Midline nasal masses may present with an external deformity or may be solely intranasal with no external sign on physical examination. Nasal endoscopy can help delineate the extent of intranasal involvement. MRI is the imaging modality of choice for assessing all masses of the frontonasal region (**Fig. 10**). Advantages include multiplanar imaging, distinguishing cartilage, bone, brain, and fluid interfaces; diffusion imaging to detect epidermoid tumors; and the capacity to evaluate the brain for associated cerebral anomalies.²³ Management often requires surgical correction, classically via a combined approach with multiple surgical subspecialties such as otolaryngology, plastic surgery, and neurosurgery.

Nasal dermoid

Nasal dermoids total 1% to 3% of all dermoid cysts and approximately 4% to 12% of head and neck dermoids.²⁴ They result from faulty involution of a neuroectodermal tract through the anterior neuropore, pulling outlying skin internally. This creates a pit, sinus tract, or cyst containing mesodermal and ectodermal components. There is a slight male predominance, and although most cases are sporadic, a familial predisposition has been reported.²⁵ Associated abnormalities such as craniosynostosis, hemifacial microsomia, lacrimal duct cysts, cleft lip/palate, pinna deformity, hydrocephalus, and hypertelorism have been described.²⁶ Intracranial extension has a variable incidence, with 5% to 45% reported in the literature, and resulting intracranial infection is rare.²⁷



Fig. 8. Sequential MRI images demonstrating dilated lacrimal ducts bilaterally terminating in dilated intranasal mucocoeles or dacryocystocoeles. (From Leonard DS, O'Keefe M, Rowley H, et al. Neonatal respiratory distress secondary to bilateral intranasal dacryocystocoeles. *Int J Pediatr Otorhinolaryngol* 2008;72:1875; with permission.)

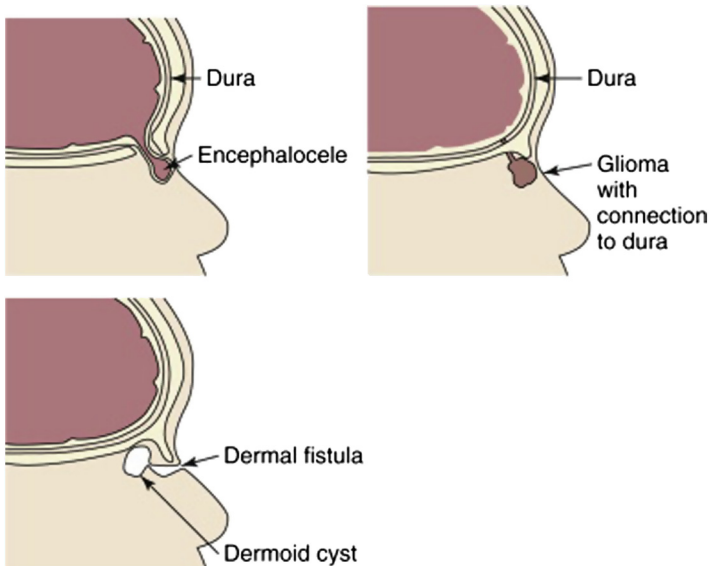


Fig. 9. Schematic view of the common midline nasal masses. (From Elluru RG, Wootten CT. Congenital malformations of the nose. In: Flint PW, Haughey BH, Lund VJ, et al, editors. Cummings otolaryngology: head & neck surgery. 5th edition. Mosby; 2010. p. 2687; with permission.)

These present as a midline mass ranging in location from the base of the columella, along the nasal dorsum, to the nasoglabellar region.²⁸ A sinus opening along this route, with a protruding hair is pathognomonic, and may have discharge or local infection associated with it (Fig. 11).²⁶ Similar to gliomas, they are noncompressible, non-expansile, and do not transilluminate.

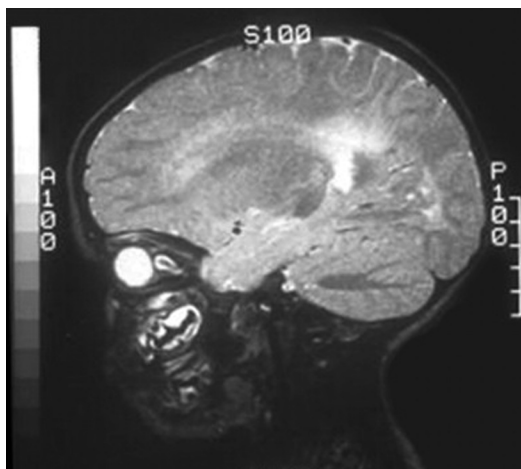


Fig. 10. Sagittal magnetic resonance image of a basal meningocele protruding into the nasopharynx. (From Elluru RG, Wootten CT. Congenital malformations of the nose. In: Flint PW, Haughey BH, Lund VJ, et al, editors. Cummings otolaryngology: head & neck surgery. 5th edition. Mosby; 2010. p. 2688; with permission.)



Fig. 11. Nasal dermoid manifesting as a firm midline nasal swelling associated with a sinus opening. (From Elluru RG, Wootten CT. Congenital malformations of the nose. In: Flint PW, Haughey BH, Lund VJ, et al, editors. Cummings otolaryngology: head & neck surgery. 5th edition. Mosby; 2010. p. 2689; with permission.)

Nasal glioma

Nasal glioma, or more precisely nasal glial heterotopia, consists of isolated extracranial glial tissue which may or may not be connected to the brain by a fibrous stalk. They are rare, with a male-to-female ratio of 3:2, and no familial predisposition.²⁸ Presentation has a 6:3:1 ratio of extranasal:intranasal:combined lesions.⁹ Presentation varies from an overt external mass to subtle findings including telecanthus and a widened nasal bridge. Intranasal lesions are typically seen as pale masses arising from the lateral nasal wall or middle turbinate, but they can arise from the septum. In rare instances, gliomas may extend into the orbit, frontal sinus, oral cavity, or nasopharynx.²⁸

Encephalocele

Encephaloceles are extracranial protrusions of meninges, cerebrospinal fluid, and neural tissue. Meningocele may present similarly without herniation of brain tissue. They are rare with no gender predilection.²⁹ There are several different subclassifications of encephaloceles depending on the location of dehiscence in the skull base, and they may present either as external or internal nasal masses (**Fig. 12**). They are pale, compressible, pulsatile, and transilluminate with light. Compression of the jugular vein with resultant expansion, known as Furstenberg's test, is present. They may also expand with crying or straining. Associated abnormalities are present in 30% to 40% of cases.²⁹

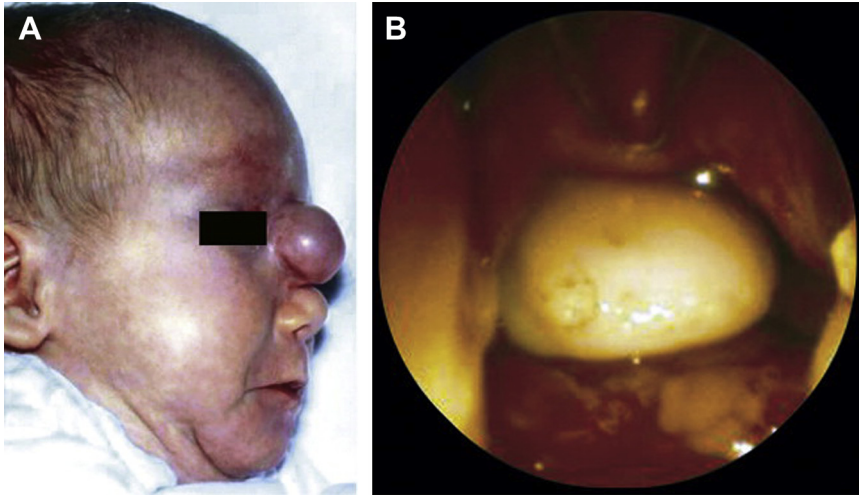


Fig. 12. (A) Sincipital encephalocele. A soft, bluish compressible mass protruding from the glabellar region. (B) Basal encephalocele seen in nasopharynx with a 120° angled telescope. (From Elluru RG, Wootten CT. Congenital malformations of the nose. In: Flint PW, Haughey BH, Lund VJ, et al, editors. Cummings otolaryngology: head & neck surgery. 5th edition. Mosby; 2010. p. 2687–8; with permission.)

Neoplastic Etiologies

In addition to congenital midline nasal masses, congenital neoplasms should be considered, including both benign and malignant forms. Reported prevalence for all congenital tumors is in the range of 1.7 to 13.5 cases per 100,000 live births.³⁰ These may present similarly to the previously mentioned nasal masses both within the nasal cavity and externally. Differential diagnosis of congenital nasal neoplasms includes teratomas, hamartomas, rhabdomyosarcoma, hemangiomas, neurofibromas, epidermoid cysts, lipoma/lipoblastomas, and lymphatic malformations among other rare neoplasms. Radiographic imaging with CT or MRI is recommended to evaluate for extent of the lesion (Fig. 13). Any prenatal ultrasound imaging concerning for a nasal or head and neck mass should alert the clinician to the risk of respiratory distress and elicit appropriate perinatal planning (Fig. 14). The first step in treatment is securing an airway, followed by treatment specific to etiology. This may include consideration of an ex utero intrapartum treatment (EXIT) procedure, in which the fetus is delivered via cesarean section while maintaining the placenta and umbilical cord, allowing an airway to be established by means of intubation or tracheostomy.³⁰

Infectious Etiologies

Viral and bacterial infections may lead to partial nasal obstruction in newborns either independently or coexisting with other causes of nasal obstruction. Infections of the nasolacrimal duct, termed dacryocystitis, may also lead to nasal obstruction with a presentation similar to nasolacrimal duct cysts. Sexually transmitted diseases (STDs) in the mother, such as chlamydia, gonorrhea, and syphilis, are important bacterial pathogens to recognize as possible etiologies of nasal congestion and obstruction requiring prompt treatment with antibiotics. Clinical suspicion based on maternal sexual history, substance abuse, prior STD diagnosis, poor prenatal care, and perinatal STD testing should guide further investigation.

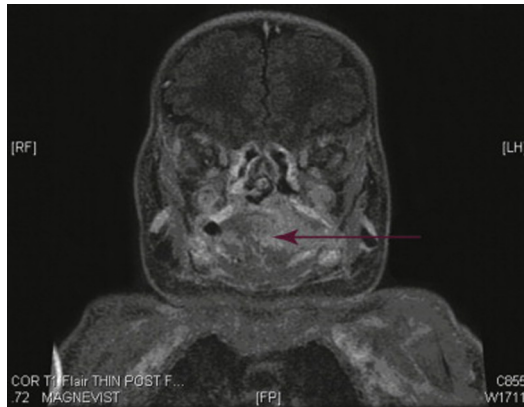


Fig. 13. Nasopharyngeal teratoma on a coronal magnetic resonance image with characteristic heterogeneous signal intensity (arrow). (From Elluru RG, Wootten CT. Congenital malformations of the nose. In: Flint PW, Haughey BH, Lund VJ, et al, editors. Cummings otolaryngology: head & neck surgery. 5th edition. Mosby; 2010. p. 2696; with permission.)

Although chlamydial and gonorrheal infection in infants most commonly present as conjunctivitis, both may also present with nasopharyngitis and rhinitis responsive to oral antibiotics.^{31,32} Conjunctivitis associated with *Chlamydia trachomatis* typically develops 5 to 12 days after birth and can be associated with an afebrile, subacute pneumonia at 1 to 3 months of age.³³ In contrast, conjunctivitis associated with gonorrhea

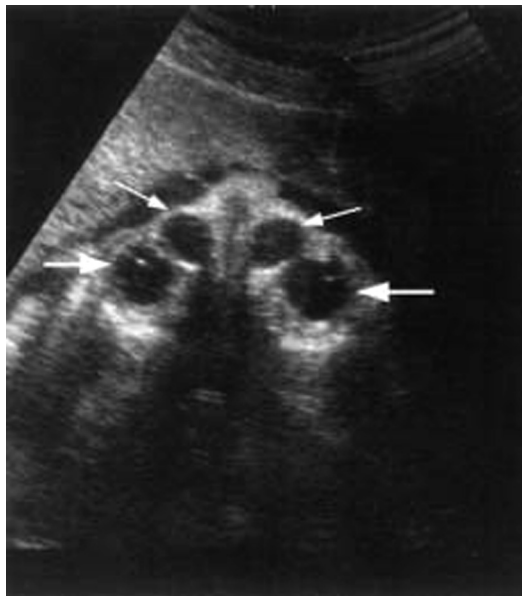


Fig. 14. Anterior coronal view of fetal face demonstrating bilateral dacryocystoceles (small arrows) anterior-medial to the orbits (large arrows). (From Goldberg H, Sebire NJ, Holwell D, et al. Prenatal diagnosis of bilateral dacryocystoceles. Ultrasound Obstet Gynecol 2000;87:448, with permission.)

typically manifests 2 to 5 days after birth and may be associated with sepsis, arthritis, meningitis, vaginitis, urethritis, and infection at fetal monitoring sites such as abscesses from scalp electrodes.³³

Congenital syphilis commonly presents with nasal discharge, often referred to as syphilitic snuffles. Initially it is seen as watery discharge, but it becomes progressively thick and purulent, causing nasal obstruction. Differentiating features of syphilis include nonimmune hydrops, jaundice, hepatosplenomegaly, skin rash, and pseudoparalysis of an extremity.³³ It is responsive to standard antibiotic therapy.³⁴

Iatrogenic Etiologies

Trauma during delivery or postnatal care can cause or exacerbate nasal obstruction. Birth trauma affecting the head and neck occurs in 0.82% of births and is associated with vaginal delivery, primiparity, forceps delivery, infants large for gestational age, and male gender.³⁵

Dislocation of the cartilaginous nasal septum from the vomerine groove occurs in 0.6% to 0.93% of newborns and may result in nasal obstruction because of the deviated septum.^{35,36} The exact etiology is debatable; however, it is most commonly attributed to birth trauma, although abnormal intrauterine pressures or other prenatal causes may play a role.³⁷ Physical examination reveals a deviated nasal tip with an angulated columella and flattened, asymmetric nasal ala. The nasal tip flattens easily with light pressure on palpation, and inspection with an otoscope or speculum reveals dislocation of the cartilage toward the narrowed nostril.³⁷ In comparison, a flattened nose is a frequent normal finding in newborns that may also cause transient nasal obstruction, typically resolving within 48 hours. It can be distinguished from septal dislocation by pressing lightly on the nasal tip and meeting resistance. Septal dislocation treatment is controversial and may consist of either observation or bedside manual manipulation of the septum back into the septal groove and columella.

Another iatrogenic etiology of nasal obstruction is nasal vestibular stenosis, resulting from soft tissue damage to the external nares.³⁸ In addition to this, trauma from nasal prongs, continuous positive airway pressure (CPAP), nasogastric feedings tubes, and suction trauma from overvigorous suctioning of secretions can cause clinically significant nasal obstruction and possible synechia formation (**Fig. 15**).^{39,40} Septal hematomas may occur during birth or from the previously mentioned causes and require incision and drainage to avoid cartilage necrosis and subsequent saddle nose deformity.

Inflammatory, Systemic, and Other Etiologies

Neonatal rhinitis, or inflammation of the nasal mucosa, is the most common cause of nasal congestion and partial obstruction.³⁹ Infants present with typical symptoms of nasal obstruction, and physical examination reveals mucosal edema and secretions. This can occur alone, as a manifestation of another condition, or in tandem with other etiologies of nasal obstruction, thereby exacerbating the underlying cause.

Mucus production varies from 0.1 to 0.3 mg/kg/d; therefore, mucus itself may cause significant nasal occlusion independently. Deficiencies of mucociliary clearance such as Kartagener syndrome or cystic fibrosis are especially prone to mucoid obstruction. While rhinitis with obstruction has been reported with cow's milk allergy/intolerance⁴¹ or hypothyroidism,⁴² more common associations would be gastroesophageal reflux disease (GERD), inferior turbinate hypertrophy, or adenoid enlargement. Maternal etiologies include estrogenic stimuli (similar to rhinitis of pregnancy)⁴³ or maternal drug ingestion, such as methyl dopa,⁴⁴ methimazole,⁴⁵ tricyclic antidepressants, narcotics,

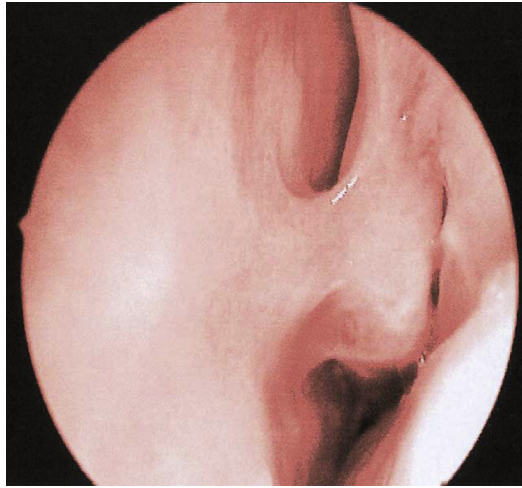


Fig. 15. Naso-endoscopy revealing synechia on the right side from CPAP catheters. (*Adapted from DeRoew A, Landsberg R, Fishman G, et al. Neonatal iatrogenic nasal obstruction. Int J Pediatr Otorhinolaryngol 2004;68:615; with permission.*)

or antihypertensive medications such as propranolol.³⁹ Most neonatal rhinitis cases are idiopathic in nature.

Treatment for neonatal rhinitis includes humidification, gentle suction of secretions, and topical steroids or nasal decongestants. Limitation of vasoconstrictive agents to 3 days in infants is crucial to avoid rhinitis medicamentosa, which could be life threatening secondary to rebound congestion and obstruction in the neonate.⁴⁶ Rhinitis associated with a specific ingestion is diagnosed via history and treated by maternal avoidance or change of formula. No response to conservative management would warrant further evaluation.

Gastroesophageal reflux

Gastric content reflux into the nasal cavity may cause chronic inflammation and subsequent nasal obstruction. The resulting inflammation has been associated with adenoid enlargement⁴⁷ and alteration of mucosal conditions increasing susceptibility to bacterial and/or viral infections, which may further exacerbate nasal obstruction.⁴⁸ Other symptoms in affected infants include episodes of spitting up, irritability, and possibly arching or torticollis. The current gold standards for diagnosing otolaryngologic manifestations of GERD are the dual-channel 24-hour pH or impedance probe.^{49,50}

Airway obstruction has been demonstrated as a contributing factor in the pathogenesis of GERD in animal models, whereby by the mechanical force required to overcome upper airway obstruction during expiration resulted in gastric reflux.^{48,51} In addition to this, aspirated reflux contents with resultant coughing further promotes GERD secondary to increased intra-abdominal pressure. As a result, children with other etiologies of nasal obstruction may subsequently be affected by GERD, which can aggravate the underlying obstruction via mucosal edema and inflammation resulting in a vicious cycle. Therefore, antireflux medications in infants with nasal obstruction are a valid consideration, especially if other symptoms of reflux are present. GERD has also been shown as a complicating factor in choanal atresia repair, further supporting the use of antireflux medications in these patients.⁵² Currently, proton pump

inhibitor use in the newborn is controversial, and some studies have shown no benefit. This may be explained by the fact that even nonacidic reflux has been shown to induce inflammation. In addition to a reflux medication trial, modifications such as propping infants up after feedings and thickening formula may be helpful.³⁹

SYNDROMIC ASSOCIATIONS

Several etiologies of nasal obstruction may present with concurrent abnormalities that may require further assessment, including genetic evaluation. Nasal obstruction has been described in association with Crouzon, CHARGE, Pfeiffer, Apert, Treacher-Collins, Down, and fetal alcohol syndromes, among others.^{39,53–56} Infants with craniofacial synostosis typically have airway obstruction secondary to maxillary hypoplasia in the setting of normal nasopharyngeal and oropharyngeal soft tissue development. As many as 50% of infants with craniofacial anomalies require a tracheostomy at some point in their life.⁵⁷

TREATMENT

Treatment options for nasal obstruction include observation or medical or surgical management, with securing an adequate airway as the first goal of therapy. In newborns, the nasal passages are approximately 50% of the total airway resistance. Over the first 6 months of life, the internal nasal airway doubles in size, often leading to spontaneous resolution of symptoms present at birth.^{43,58} In addition to the increased area of the nasal passage, cervical growth and descent of the larynx during the first months of life facilitate mouth breathing.²

Conservative management for mild symptoms includes discontinuation of vigorous suctioning and introduction of gentle suctioning with swabbing for mucus removal as needed. Humidification with administration of saline drops may be beneficial. Other medications include nasal steroid drops (dexamethasone ophthalmic solution, beclomethasone, or triamcinolone spray), and application of decongestant drops (phenylephrine 0.125% or oxymetazoline 0.025%) twice daily for a maximum of 3 days.^{39,59} Most infants respond quickly to this regimen within 3 to 5 days, and symptoms resolve within 2 to 4 weeks. No response or worsening would require prompt further evaluation. It may be prudent to discharge patients with a cardiopulmonary monitor and instruct parents in airway management and cardiopulmonary resuscitation.

Severe obstruction may necessitate endotracheal intubation or tracheostomy until further surgical intervention depending on the infant's condition and associated anomalies. Respiratory distress may be treated with placement of a nasal trumpet if able to be placed on 1 side, or an oral airway. An endotracheal tube placed transorally into the esophagus opens the mouth, establishes an oral airway, and can be used for feedings. A McGovern nipple (a large nipple with the end cut off) allows infants to mouth breathe between swallows while feeding, and this can be secured as an oropharyngeal airway between feedings.⁶⁰ Many infants have associated feeding problems and uncoordinated swallowing efforts, placing them at risk for failure to thrive and recurrent aspiration.⁶¹ These infants require nutritional and speech pathology assessment and support, and may ultimately require gavage feedings.

Surgical intervention is indicated in infants with respiratory distress, unresponsiveness to conservative measures, or failure to thrive.¹⁴ In patients with a stable respiratory status, surgeons may apply the rule of tens and delay surgery until the child is 10 lbs, 10 weeks of age, and has a hemoglobin of 10.¹⁰ Other cases, such as unilateral choanal atresia, may be repaired later in childhood.

SUMMARY

Severity of nasal obstruction in the newborn ranges from being a nuisance to a life-threatening scenario. It is an important clinical entity for pediatricians to recognize, quickly evaluate, and treat. Causality has a broad differential diagnosis and may be multifactorial in nature. Although the most common etiology is inflammation of the nasal mucosa, other possibilities must be considered when evaluating the infant with nasal obstruction, and further evaluation of associated abnormalities may be warranted. Management may be complex and require a multidisciplinary approach including neonatologists, pediatricians, obstetricians, and pediatric subspecialists, including: otolaryngologists, anesthesiologists, neurosurgeons, plastic surgeons, ophthalmologists, gastroenterologists, nutritionists, speech pathologists, and support services.

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