A 6-month-old infant was born by spontaneous vaginal delivery after an uneventful pregnancy. Maternal serology screening was nonreactive for syphilis, negative for hepatitis B surface antigen, and showed immunity for rubella. Maternal rectovaginal culture grew group B streptococcus, and the mother received 2 doses of antibiotics before delivery. Labor and delivery were uncomplicated, and his birth weight was 3.5 kg. Apgar scores were 8 and 9 at 1 and 5 minutes, respectively. Initial vital signs and physical examination findings were normal. With the first bottle feeding, he regurgitated all the formula through his nostrils. Despite suctioning of the nasopharynx, his airway remained congested, and he had difficulty clearing nasal secretions. Sterile water feeding was attempted, but instant reflux of water occurred through the nostrils.

Findings from complete blood cell count, C-reactive protein, and chest radiograph were normal. He received a sepsis workup and antibiotics for 3 days until blood culture had negative findings. Investigation for nasopharyngeal reflux was initiated with barium esophagram, which showed reflux of contrast from the oropharynx into the nasopharynx and through the nostrils (Figure 1). Some contrast passed down the esophagus into the stomach, and an aspiration episode into the trachea was visualized (Figure 2). The infant underwent direct laryngoscopy, esophagoscopy, and bronchoscopy. No structural anomalies of nasopharynx, larynx, esophagus, or bronchi were present. Computed tomographic scans of the brain, neck, and chest were normal, showing no intrinsic or extrinsic structural obstruction to the esophagus or trachea. A 2-dimensional echocardiogram with Doppler flow study of the heart and major blood vessels did not reveal abnormal vessels or structural anomalies.
Denouement and Discussion

Velopharyngeal Insufficiency Causing Nasopharyngeal Reflux in the Neonate

**Figure 1.** Swallowing study using fluoroscopy shows reflux of contrast from the oropharynx into the nasopharynx and out of the nostrils.

**Figure 2.** Swallowing study using fluoroscopy showing part of the contrast passed down the esophagus to the stomach and a silent aspiration episode into the trachea.

Definitive diagnosis of velopharyngeal insufficiency (VPI) was made by fiberoptic and nasopharyngoscopy. Initially, this infant had poor sucking, swallowing, and breathing coordination, and needed to rest between sucks. He was treated with cisapride to enhance esophageal motility, and after treatment for a week with parenteral fluids, he began oral feeding using a “cleft palate” nipple. Gradual improvement in feeding tolerance and reduction in episodes of nasopharyngeal reflux occurred over the next week. He still had occasional regurgitation episodes and was diagnosed with failure to thrive by age 3 months. Follow-up fiberoptic nasopharyngoscopy revealed persistent VPI.

Velopharyngeal insufficiency represents inability to separate the content of the oropharynx from the nasopharynx during feeding and speech by the muscles of the palate and the posterior and lateral pharyngeal wall. It is a relatively uncommon diagnosis in children and has not been reported in the neonate.1 Velopharyngeal insufficiency may be organic or functional, produced by congenital or acquired causes or due to paresis or local disorders. It most frequently occurs as a result of cleft palate, submucous cleft palate (any combination of bifid uvula, muscular diastasis of soft palate, or bony defect of the submucous cleft palate (any combination of bifid uvula, orders. It most frequently occurs as a result of cleft palate, cleft palate and the posterior and lateral pharyngeal wall. It is a relatively uncommon diagnosis in children and has not been reported in the neonate.1 Velopharyngeal insufficiency may be organic or functional, produced by congenital or acquired causes or due to paresis or local disorders. It most frequently occurs as a result of cleft palate, submucous cleft palate (any combination of bifid uvula, muscular diastasis of soft palate, or bony defect of the submucous cleft palate) or hypopharyngeal incompetence.2 Other entities associated with VPI are DiGeorge syndrome, velocardiofacial syndrome, neurofibromatosis, hemifacial microsomia (facial-aureiculo-vertebral malformation complex),3 Chiari malformation,4 myasthenia gravis, histiocytosis (eosinophilic granuloma), Pierre Robin syndrome, Dubowitzy syndrome,4 Kabuki make-up syndrome,5 encephalopathy, peritonsillar abscess, Mobius syndrome, and myotonic dystrophy.6 Familial cases of VPI with autosomal dominant inheritance have been reported.7 Velopharyngeal insufficiency may be complicated by speech hypernasality, snoring,8 Eustachian tube dysfunction, and chronic otitis media with conductive hearing loss and paranasal sinus infections.9 Velopharyngeal insufficiency may be diagnosed by multiview videofluoroscopy10 but the gold standard for diagnosis is flexible fiberoptic nasal endoscopy.11 Rapid magnetic resonance imaging is a costly but sensitive method to evaluate VPI.12 Treatment modalities include speech therapy for minimal VPI and dental prosthesis for cases in which surgery is contraindicated. The mainstay of treatment for posterior pharyngeal wall augmentation includes using materials such as paraffin, fascia, autologous cartilage, and Teflon (DuPont, Wilmington, Del) paste.13 If these measures fail, a push-back palatoplasty can be attempted. Other surgical procedures used are sphincter pharyngoplasty and palatopharyngoplasty (velopharyngeal flap).14 Obstructive sleep apnea has been reported as a complication of pharyngeal flap surgery.

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