Not All Postoperative Stridor in Infants Is Due to Endotracheal Tube-Induced Subglottic Edema

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Abstract

A 6-month-old infant presented with clinicoradiological features of a shunt dysfunction. Magnetic resonance imaging brain showed multiple leptomeningeal cysts in the posterior fossa, with the largest in the right cerebellopontine (CP) angle cistern causing compression on the brain stem and fourth ventricle. There was gross hydrocephalus with the malpositioned shunt tube. He underwent shunt revision followed by right retromastoid craniectomy and decompression of the right CP angle cyst. Following extubation, he developed stridor that was diagnosed initially as subglottic edema and treated with humidified oxygen, systemic corticosteroids, and nebulized adrenaline. Failure to resolve the symptoms warranted a video laryngoscopy that revealed right vocal cord palsy (VCP), and he was reintubated. He was started on steroids and got extubated on a nasal continuous positive airway pressure and was gradually weaned off. Intraoperative handling of the vagus nerve while decompressing the cyst led to a right VCP, which was communicated later to the anesthesiologist. Neurological cause and association need to be considered as one of the differentials while managing postoperative stridor after posterior fossa surgery in an infant. Timely communication between the surgeon and anesthesiologist is paramount for reducing morbidity.

Keywords

- infant
- postoperative stridor
- posterior fossa surgery
- vocal cord palsy
- ventriculoperitoneal shunt

Introduction

Children undergoing posterior fossa surgeries are prone to postoperative airway obstruction due to various reasons, and often present with postoperative stridor.[1,2] Airway obstruction can be of supraglottic, glottic, or subglottic origin. Endotracheal tube (ETT)-induced subglottic airway edema (SAE) is the commonest cause of postoperative stridor in an infant with a normal anatomical airway.[3] Though SAE is the commonest cause, one should rule out supraglottic and glottic causes for the stridor and its neurological association, especially in children undergoing posterior fossa surgery.[1–5] Delayed diagnosis increases morbidity and mortality. We report a case of a 6-month-old infant who developed postoperative stridor due to right vocal cord palsy (VCP) after a posterior fossa surgery and its management.

A 6-month-old infant (6.5 kg), born full term, was brought with progressively increasing swelling behind the ear and head circumference. There was no history of feeding difficulty, vomiting, noisy breathing, or extensor posturing. He was diagnosed with neonatal meningitis-induced hydrocephalus at the age of 1 month and underwent a right parietal ventriculoperitoneal (VP) shunt elsewhere. The parents noticed a progressive increase in his head size

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even after the surgery and came to our institution for further management. He had no other congenital anomalies and had a mild developmental delay.

The child was alert, active, and moved all four limbs. The anterior fontanelle was full with a head circumference of 45 cm. The scalp veins were dilated and a sunset sign was present and there was no stridor. Preoperative magnetic resonance imaging brain revealed multiple leptomeningeal cysts in the posterior fossa, with the largest in the right cerebellopontine (CP) angle cistern causing brain stem and fourth ventricular compression resulting in gross hydrocephalus (►**Fig. 1**). The ventricular end of the shunt tube was noted within the frontal parenchyma. He was planned for a shunt revision in a supine position followed by a right retromastoid craniectomy and decompression of the right CP angle cyst in the lateral decubitus position (LDP)

After inducing with sevoflurane, standard American Society of Anesthesiologists (ASA) monitors were attached. The intravenous (IV) access was established, and the anesthetic plane was deepened using propofol and fentanyl. He was paralyzed with atracurium and intubated with a 4.5 mm uncuffed ETT in a single attempt by the senior anesthesiologist without undue resistance. An ultrasonography-guided 5.5F femoral venous catheter was placed for blood, fluid, and vasopressor administration. A 24-G arterial line was inserted for continuous monitoring of invasive blood pressure. Anesthesia was maintained with total intravenous anesthesia (TIVA) using propofol. The child underwent right-sided shunt revision in the supine position and then was repositioned in the LDP. Right retromastoid suboccipital craniotomy and decompression of CP angle cysts were done that lasted for 4 hours with a blood loss of 130 mL. Since the baseline hemoglobin was 9.3 gm/dL, 100 mL of blood was transfused intraoperatively. Dexamethasone (0.75 mg) was given to prevent airway edema. At the end of surgery the residual neuromuscular blockade was reversed with neostigmine and glycopyrrolate. The ETT was removed after meeting the extubation criteria (adequate tidal volume >5 mL/kg, respiratory rate 25/min, stable hemodynamics, purposeful movement of all four limbs, facial grimace, eye-opening). Soon after extubation, the child developed stridor and was initially treated with propofol and positive pressure ventilation using a face mask. Initially, we felt that this could be due to SAE and was treated with adrenaline nebulization (0.3 mL 1/1,000 adrenaline in 3 mL of 0.9% normal saline) with propofol sedation for effective nebulization. Every time the child was awake, the stridor got worse, and it did not get better even after administering the second dose of adrenaline nebulization after 30 minutes. Hence, we decided to do a video laryngoscopy (VL)-assisted fiberoptic to rule out supraglottic and glottic causes for stridor. The child was sedated with propofol and fentanyl and was maintained on spontaneous breathing. Since the VL revealed right VCP, fiberoptic was deferred and the finding was communicated to the operating surgeon (►**Fig. 2**). The child was reintubated with 4 mm uncuffed ETT without resistance and shifted to pediatric intensive care unit on propofol sedation. During this period, the saturation was maintained between 99 and 100% with a nebulization mask alternating with Jackson Rees modification of Ayre's T-piece circuit for providing positive pressure. The child had hypertension and tachycardia because of adrenaline nebulization. While discussing the finding with the operating surgeon, he revealed that there was the handling of the right vagus nerve while dissecting the cyst wall and the possibility of right vagal nerve paresis. Postoperatively child was treated with IV dexamethasone and got extubated after 30 hours, on a nasal continuous positive airway pressure device and weaned off.

Managing stridor in an infant is a challenging task. There is no proposed algorithm for the management of postoperative stridor in an infant. Agitation and crying can aggravate airway edema and prevent the effective delivery of nebulized adrenaline; hence, mild sedation is mandatory. Humidified oxygen therapy, systemic corticosteroid administration

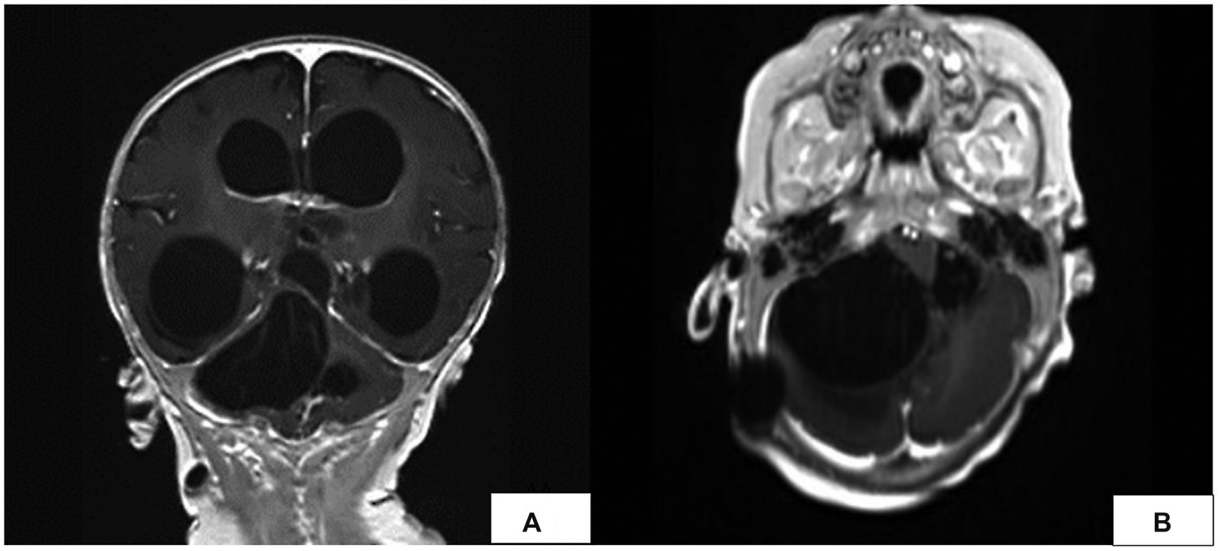

Fig. 1 T1-weighted-postcontrast coronal (**A**) and axial (**B**) magnetic resonance imaging brain shows a large cerebrospinal fluid-containing cyst at the right cerebellopontine angle causing effacement of the fourth ventricle resulting in severe hydrocephalus.

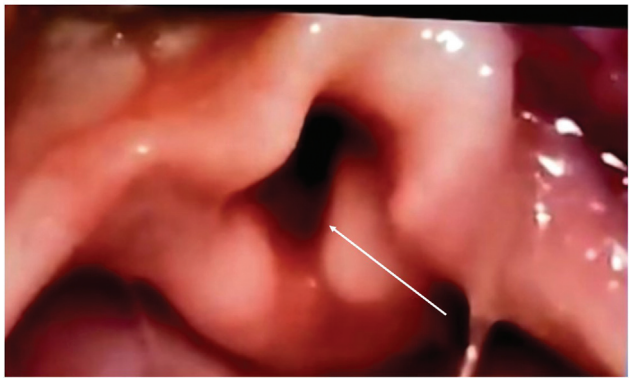

Fig. 2 Video laryngoscopy view of the vocal cord during inspiration shows right vocal cord palsy (arrow indicates the right vocal cord in the paramedian position).

especially dexamethasone and racemic adrenaline, and albuterol nebulization are given to treat airway edema.[6–9] Since this child had already received dexamethasone intraoperatively, adrenaline nebulization was given that failed to improve the symptoms. Failure to improve the severity of stridor with two doses of adrenaline nebulization alerted us to the possibility of VCP. If the possibility of right VCP due to intraoperative handling of IX/X nerves could have been communicated to the anesthesiologist at the end of the surgery, vocal cord assessment could have been done before extubation. In that scenario, the anesthesiologist could have either continued the ventilation or the reintubation could have been done early. Also, adrenaline nebulization induced an increase in heart rate and blood pressure during the immediate postoperative period that could have been avoided.

The head position (head down and extreme neck flexion), duration of mechanical ventilation, large-size ETT, recent laryngotracheal bronchitis, repeated traumatic intubation attempts, and presence of preoperative stridor (neurological causes such as raised intracranial pressure (ICP), Arnold Chiari malformation with occipitoencephalocele, hydrocephalus) are some of the risk factors for the development of postoperative stridor in infants.[2–5] As reintubation adds to morbidity, one should take all measures to prevent reintubation.

Ventilatory management is the key in children with raised ICP. Since the child's head was away from the anesthesiologist and the child was positioned in the LDP, managing the ventilation will be challenging if there is a peritubal leak. Hence, we chose 4.5 mm ETT instead of 4.0 mm ETT. While introducing the 4.5 mm ETT during the initial intubation, there was no resistence noted; even during reintubation with 4 mm ETT, there was no resistence felt at the subglottic level which ruled out the presence of subglottic edema.

Postoperative stridor is a common complication in an infant and small children. Though subglottic edema is a common cause of postoperative stridor in infants, neurological cause and association need to be considered as one of the differentials while managing postoperative stridor especially, after posterior fossa surgery. Timely communication between the surgeon and anesthesiologist is paramount for reducing morbidity.

Conflict of Interest
None declared.

References

1 Bhagat H, Bloria SD, Kapil S, Kaloria N. Airway considerations in pediatric neurosurgical patients. Indian J Neurosurg. 2020;9(03): 179–182
2 Saigal D, Ganjoo P, Sharma MU, Singh D. Managing tracheal extubation in infants with stridor and congenital neuraxial anomalies. J Pediatr Neurosci 2016;11(04):335–337
3 Khemani RG, Hotz J, Morzov R, et al. Evaluating risk factors for pediatric post-extubation upper airway obstruction using a physiology-based tool. Am J Respir Crit Care Med 2016;193 (02):198–209
4 Gupta R, Williams A, Vetrivel M, Singh G. Stridor in children: is airway always the cause? J Pediatr Neurosci 2014;9(03):270–272
5 Nascimento MS, Prado C, Troster EJ, Valério N, Alith MB, Almeida JF. Risk factors for post-extubation stridor in children: the role of orotracheal cannula. Einstein (Sao Paulo) 2015;13(02):226–231
6 Khemani RG, Randolph A, Markovitz B. Corticosteroids for the prevention and treatment of post-extubation stridor in neonates, children and adults. Cochrane Database Syst Rev 2009;(03): CD001000
7 Anene O, Meert KL, Uy H, Simpson P, Sarnaik AP. Dexamethasone for the prevention of postextubation airway obstruction: a prospective, randomized, double-blind, placebo-controlled trial. Crit Care Med 1996;24(10):1666–1669
8 Soler M, Raszynski A, Kandrotas RJ, Sussmane JB, Aznavorian R, Wolfsdorf J. Fewer interventions in the immediate post-extubation management of pediatric intensive care unit patients: safety and cost containment. J Crit Care 1997;12(04):173–176
9 Lukkassen IMA, Hassing MBF, Markhorst DG. Dexamethasone reduces reintubation rate due to postextubation stridor in a high-risk paediatric population. Acta Paediatr 2006;95(01): 74–76