Abnormal outcome after T & A, case report and literature review

Abstract

Although tonsillectomy and adenoidectomy continues to be one of the most common surgical procedures in children, the incidence of respiratory complications are still significant. We describe a 4-year old child, who developed hypoxic injury after T&A. His procedure was done in outpatient settings and he had no immediate complications after the surgery. He was discharged home, where he continued to be somnolent and when brought back to the hospital he required intubation. He developed pulmonary edema and head CT revealed ischemia of cerebellum and brainstem. He had prolonged hospitalization and he was discharged home with moderate neurologic deficits requiring physical therapy. Criteria for admission of patients after T&A are not well defined and will need to be address, since significant complications can occur.

Keywords: tonsillectomy, adenoidectomy, obstructive sleep apnea, hypoxic injury

Abbreviations: T&A, tonsillectomy and adenoidectomy; OSA, obstructive sleep apnea; OSAS, obstructive apnea syndrome; EVD, external ventriculostomy device; PICU, pediatric intensive care unit; CPAP, continuous positive airway pressure; PSG, polysomnography

Introduction

Tonsillectomy and adenoidectomy (T&A) have been and continue to be one of the most commonly performed surgical procedures for children.1 T&A is known to have been performed for almost 3,000 years, being described in the literature for the first time in the Hindu Sanskrit document Atharva-Veda dating back to about 700BC. Before 1950 most of the tonsillectomies were performed due to infection, today majority of young children <12years are operated for obstructive problems.2 Obstructive sleep apnea (OSA) is a common condition in childhood, affecting approximately 1–3% of children. Obstructive sleep apnea syndrome (OSAS) was first described by Charles Dickens in “The Pickwick Papers”, with the first professional mention of OSAS in the medical literature in the British Medical Journal in 1889. Clinical history and examination findings fail to distinguish clinically significant OSA from benign snoring. Polysomnography is currently the best method available for diagnosis.3 The incidence of respiratory complications after adenotonsillectomy stated in the literature are 5-25%.4 OSA due to adenotonsillar hypertrophy in children can lead to significant cardiopulmonary complications, poor growth and problems with learning and behavior. Systemic hypertension, a frequent complication of adults with OSAS, has been also reported in children.5

Case presentation

We present a child who developed hypoxic injury after T & A. This is a 4-year old Hispanic male, who has been in the US for only 6months. Per report, he had multiple throat infections, for some of them he was hospitalized and received IV antibiotics. Mother also reports “noisy breathing at night”. He had elective T&A done as an outpatient for OSA and h/o infections but he did not have polysomnography. Surgery was without complications, mother reports that he was sleeping post-surgery but was able to go to the bathroom, therefore he was discharged home. He did not receive any pain medications at home but mother reports that he was sleeping for several hours. When she came to check on him, he was unresponsive and cyanotic. She drove him to the Emergency Room. His initial venous blood gas revealed pH 6.8 with mixed respiratory and metabolic acidosis. He was initially placed on CPAP but shortly after admission to PICU was intubated due to upper airway obstruction. CXR showed post obstructive pulmonary edema with quick resolution by the next day. He was alert and in no distress, therefore he was extubated that afternoon. He was sitting on the bed, playing and interactive with his family. About 4hours later he developed acute respiratory distress and rash. Initially it was thought to have allergic reaction and received Epinephrine s.q., steroids and Benadryl. Subsequently he was reintubated due to severe upper airway obstruction and desaturation to 70% on RA. He did not have airway edema but again experienced pulmonary edema apparent from bloody frothy secretions and new CXR findings. On physical exam at that time he had clear breath sounds, good perfusion but unequal pupils. Stat head CT revealed cerebellar ischemia with obstructive hydrocephalus, brainstem ischemia and herniation of uncal and tonsillar regions. He received Mannitol and hypertonic saline was started. Initially did not have any brainstem reflexes but improved after Mannitol. His neurologic exam worsened again 1hour later and he was taken to the OR for emergent EVD placement and posterior fossa decompression. Over the course of next 10days his neurologic status was improving. He was successfully extubated, he was moving all his extremities, he was alert and following commands but unable to speak. He had difficulty swallowing secretions and had aspiration during modified barium swallow test. He continued physical, occupational and speech therapy after discharge.

Discussion

Reviewing this case the following questions were raised. Should this patient been admitted to the hospital after his T & A? Who is at risk of respiratory compromise and can it be anticipated? It has been apparent that it is difficult to distinguish between snoring and OSA by physical examination. Also the size of the tonsils and adenoids does not correlate with the severity of OSA. The pathophysiology of
OSA in children is not well understood. It is usually related to some combination of decrease upper airway patency, reduced capacity to maintain airway patency and the decreased drive to breath in the face of reduced upper airway patency.\(^6\) Mechanical airway obstruction due to adenotonsillar hypertrophy can lead to cardiopulmonary complications associated with hypercardia, hypoxemia and pulmonary artery vasoconstriction leading to pulmonary hypertension.\(^3\)

The American Academy of Sleep Medicine set criteria for diagnosing pediatric OSA.\(^7\)

i. Snoring and/or obstructed breathing.

ii. At least one of the following: paradoxical breathing, movement arousals, diaphoresis, neck hyperextension, excessive daytime sleepiness, hyperactivity or aggressive behavior, impaired growth, morning headaches, enuresis.

iii. Polysomnography (PSG) shows more than one respiratory event per hour (apnea hypopnea index>1/h).

iv. PSG demonstrates respiratory-related arousals, oxygen desaturation, hypercapnia, or negative esophageal pressure.

v. Conditions are not better explained by another sleep disorder, medical or neurological condition, medical use, or substance abuse.

Children with OSAS had more frequent requirements for medical intervention in the recovery room when compared to children without OSA.\(^8\) PSG is the gold standard for diagnosis of OSAS. But PSG is expensive as standard methods and requires the presence of a technician overnight in the sleep laboratory as well as the set-up and analysis time. Several investigators have sought more cost-effective and less time-intensive alternatives for diagnosing OSAS, including sonography, home video recording, cardio respiratory sleep studies and pulse oximetry.\(^9\) This study also suggests that preoperative nocturnal oximetry could predict which children are most likely to suffer respiratory compromise after adenotonsillectomy. The postoperative respiratory complications are oxygen desaturation, atelectasis, pneumonia, pulmonary edema, pleural effusion, pneumothorax or pneumomediastinum and upper airway obstruction manifested as inspiratory stridor with increased work of breathing.\(^4\) Children with OSAS documented in the results of polysomnography should be admitted for inpatient, overnight monitoring if they are under the age of 3 year or have severe OSAS (apnea-hypopnea index of 10 or more events per hour, oxygen desaturation nadir less than 80%, or both). Also children with following conditions are not candidates for ambulatory surgery: Cardiac complications of OSAS, craniofacial disorders, neuromuscular disorders, cerebral palsy, Down syndrome, failure to thrive, morbid obesity, prematurity, sickle cell disease, central hypoventilation syndromes, genetic/metabolic/storage disease and chronic lung disease.\(^10,11\) Our patient did not fall into any of these categories but sustained significant complication with permanent disability, which brings up a point that new accurate screening tool would be invaluable.

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**Conflict of interest**

The author declares no conflict of interest.

**References**

1. Rosen GM, Muckle RP, Mahowald MW, et al. Postoperative respiratory compromise in children with obstructive sleep apnea syndrome: can it be anticipated? Pediatrics. 1994;93(5):784–788.

2. Hallerrantz E, Ericsson E. Factors influencing the indication for tonsillectomy: a historical overview and current concepts. *ORL J Otorhinolaryngol Relat Spec.* 2013;75(3):184–191.

3. Nixon GM, Kernack AS, Davis GM, et al. Planning adenotonsillectomy in children with obstructive sleep apnea: the role of overnight oximetry. *Pediatrics.* 2004;113(1 pt 1):e19–25.

4. McCarty Statham M, Ellure RG, Buncher R, et al. Adenotonsillectomy for obstructive sleep apnea syndrome in young children. *Arch Otolaryngol Head Neck Surg.* 2006;132(5):476–480.

5. Tatlipinar A, Duman D, Uslu C, et al. The effects of obstructive sleep apnea syndrome due to adenotonsillar hypertrophy on the cardiovascular system in children. *Turk J Pediatr.* 2011;53(4):359–363.

6. Peeke K, Hershberger M, Marriner J. Obstructive sleep apnea syndrome in children. *Pediatric Nursing.* 2006;35(2):489–494.

7. AASM. The international classification of sleep disorders: diagnostic and coding manual. 2nd ed. Illinois: Westchester, USA: Am Academy of Sleep Med; 2005. 58 p.

8. Sanders JC, King MA, Mitchell RB, et al. Perioperative complications of adenotonsillectomy in children with obstructive sleep apnea syndrome. *Anesth Analg.* 2006;103(5):1115–1121.

9. Wilson K, Lachkeeram I, Morielli A, et al. Can assessment of obstructive sleep apnea help predict post–adenotonsillectomy respiratory complications? *Anesth.* 2002;96(2):313–322.

10. Hanna AH, Mason LJP. Challenges in pediatric ambulatory anesthesia. *Curr Opin Anesthesiol.* 2012;25(3):315–320.

11. Marcus CL, Brooks LJ, Draper KA, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome (AAP clinical practice guidelines). *Pediatrics.* 2012;130(3):576–584.