Interdepartmental Case Report

Pediatric Obstructive Sleep Apnea

Kitirat Ungkanont, M.D.*
Suwannee Suraseranivongse, M.D.**
Duangmanee Laohaprasitiporn, M.D.***
Pakit Vichyanond, M.D.***

Abstract : **Obstructive sleep apnea in children is most commonly caused by adenotonsillar hypertrophy. Chronic longstanding hypoxia at night can lead to cardiovascular complications such as pulmonary hypertension and cor pulmonale.**

We reported a case of a four-year-old boy who had suffered from adenotonsillar hypertrophy and obstructive sleep disturbance for 3 years, resulting in right ventricular hypertrophy. Adenotonsillectomy was performed. Pre-operative awareness of potential complications, good co-operation between pediatric cardiologist and pediatric anesthesiologist with post-operative monitoring in intensive care unit were essential for the success of surgical treatment.

เรื่องย่อ : **ภาวะการหายใจลำบากในเด็ก : การรักษาและการป้องกันโรคแทรกซ้อน**

กิติรัตน์ อังกานนท์ พ.บ.*, สุวรรณี สุรเศรณีวงศ์ พ.บ., ดวงมณี เลาหประสิทธิพร พ.บ.***, ปกิต วิชยานนท์ พ.บ.*****

**ภาควิชาโสต นาสิก ลาริงซ์วิทยา, **ภาควิชาวิสัญญีวิทยา, ***ภาควิชากุมารเวชศาสตร์, คณะแพทยศาสตร์ศิริราชพยาบาล, มหาวิทยาลัยมหิดล, กรุงเทพมหานคร 10700.*

สารศิริราช 2544; 53: 750-753.

รายงานเด็กชายไทยอายุ 4 ปี มีภาวะการหายใจลำบากเวลานอนเนื่องจากต่อมทอนซิลและต่อมอดีนอยด์โตจากการอักเสบเรื้อรังมา 3 ปี ทำให้เกิดโรคแทรกซ้อนคือหัวใจด้านขวาโต การประสานงานระหว่างแพทย์หัวใจและวิสัญญีแพทย์ เพื่อเตรียมการก่อนการผ่าตัดและป้องกันภาวะแทรกซ้อนหลังการผ่าตัด ทำให้การผ่าตัดต่อมทอนซิลและต่อมอดีนอยด์เป็นผลสำเร็จ ไม่มีภาวะแทรกซ้อน

Key words: obstructive sleep apnea, adenotonsillar hypertrophy

---

**Department of Otolaryngology, **Department of Anesthesiology, ***Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok 10700.*
From Interdepartmental Conference, July 13th, 2001.

INTRODUCTION

Obstructive sleep apnea (OSA) is a well-recognized disease in adult patients with extreme obesity. In children, the most common cause of OSA is adenotonsillar hypertrophy but the pathophysiology of pediatric OSA and its consequences have just been established after the introduction of polysomnography. The effect of OSA on the systemic and pulmonary circulations leads to cardiac and pulmonary complications of which we need to be aware and monitor closely if the patient is undergoing corrective surgery. We reported a case of pediatric OSA which demonstrated the effect of chronic upper airway obstruction on cardiopulmonary circulation.

CASE REPORT

A four-year-old boy had snoring and obstructive sleep disturbance for 3 years. The history of respiratory obstruction began in 1998 when he was one year and nine months old, with symptoms of rhinorrhea, nasal obstruction, mouth breathing and restless sleep. He got better with antibiotics and nasal saline instillation. Relapses occurred frequently as he received 9 courses of antibiotics in 8 months. In 1999, he received 13 courses of antibiotics in a year and started to have apnea at night and mouth breathing even in the daytime. Overnight oxygen saturation monitoring did not show significant desaturation, allergy skin test was positive to eggwhite only. Adenotonsillectomy was recommended but the patient was lost to follow up and came back in early 2000 with repeated upper respiratory tract infections and sleep apnea. Surgery was recommended twice and the parents finally agreed in March 2001.

Pre-operative physical examination and investigations were done. Chest x-ray revealed cardiomegaly, an electrocardiogram also showed possible right ventricular hypertrophy (RVH) so we referred him to the pediatric cardiology and pediatric anesthesiology unit. On preoperative anesthetic assessment on the ward, the patient looked active and healthy. No sign of edema, dyspnea, airway obstruction or respiratory tract infection was noted. No arrhythmia was noted. Echocardiogram showed no signs of pulmonary hypertension so surgery proceeded as planned.

On the day of surgery, no sedative was ordered for premedication. EMLA cream was applied on both hands to relieve pain from intravenous cannulation before anesthesia. Standard monitoring:pulse oximetry, electrocardiogram, automated blood pressure monitoring and capnography were attached to the patient. Anaesthesia was induced using inhaled sevoflurane (up to 8%). When the patient was asleep, his airway became obstructed. An oropharyngeal airway was inserted, adequate ventilation was achieved. Then succinylcholine 1 mg/kg was given. He was intubated with moderate difficulty in viewing the larynx using an oral RAE no 5.5 with a stylet inside. Anesthesia was maintained with nitrous oxide, oxygen and isoflurane. Intravenous fentanyl 1µg/kg bolus and 1µg/kg/h were given for analgesia and atracurium 0.5 mg/kg bolus with an incremental dose of 0.2 mg/kg/30 min were given for neuromuscular blockade. Oxygenation and ventilation were adjusted to keep the SaO_2 98-100% and end tidal CO_2 31-35 mmHg. Arterial blood gas showed pH 7.437, pCO_2 32 mmHg, pO_2 258 mmHg, HCO_3^- 21.3 mEq/l, base –1.5 mEq/l. Metoclopramide 0.15 mg/kg IV was given to prevent nausea and vomiting. Adenotonsillectomy was done by dissection and snaring of the tonsils and curettage of the adenoid.The operation was uneventful and last about 1 hour. Then atropine 0.02 mg/kg and neostigmine 0.05 mg/kg IV were given for muscle reversal. After he was fully awake with adequate muscle recovery, extubation was performed in a lateral position. Then, the patient was transferred to the intensive care unit for observation of OSA. Intravenous morphine 0.05 mg/kg bolus and 10 µg/kg/h were given for analgesia.The postoperative course was without complication.

DISCUSSION

Several anesthetic complications of adenotonsillitis with OSA and RVH include difficult airway management, the consequences of OSA[1] and postobstructive pulmonary edema.[2] OSA

induces hypoxemia and hypercarbia which increase pulmonary vascular resistance, followed by pulmonary hypertension, right ventricular hypertrophy, failure and subsequently cor pulmonale.[1]

There are two possible mechanisms of postobstructive pulmonary edema. First, the negative pressure generated during inspiration against an obstructed glottis promotes the transudation of fluid into the alveoli. Second, acute obstruction of the glottis results in hypoxia and a massive sympathetic discharge which increases pulmonary artery pressure and finally promotes pulmonary edema.[3]

In this case, inhalation induction was performed because of predicted difficulty with mask ventilation and intubation because anesthesia could be reversed quickly. An oropharyngeal airway relieves obstruction from the tonsils and adenoids and prevents pharyngeal wall collapse. Equipment to deal with difficult intubation was also prepared. During maintenance of anesthesia, attention was given to prevention of further pulmonary hypertension so the patient was adequately oxygenated and ventilated, kept warm, acid/base status checked and corrected as necessary, and catecholamine release minimized. There was no need for advanced treatment of pulmonary hypertension as inotropes, vasodilators and nitric oxide.[4]

Postoperatively, full awakening was required to avoid OSA. Many centers including ours admit children with OSA who are recovering following adenotonsillectomy to the intensive care unit. This is because of concern that OSA events may occur on the first postoperative night and it may be more severe than preoperative events due to mechanical swelling from surgery or effects of anesthesia, any or all of which tend to obstruct the airway.[5] Moreover, airway obstruction and hypoxia may lead to postobstructive pulmonary edema. Postoperative pain management with narcotics is not recommended in children with OSA for fear of worsening the respiratory status.[5] However, this patient received a morphine infusion because he was closely observed and monitored in the intensive care unit.

The effect of adenotonsillar hypertrophy on cardiopulmonary function results from continuous partial upper airway obstruction which leads to paradoxical respiratory effort with decreased minute volume ventilation, pulmonary ventilation perfusion abnormalities and chronic alveolar hypoventilation. These cause the triad of hypercapnea, hypoxia and respiratory acidosis which leads to pulmonary artery vasoconstriction, increased pulmonary artery pressure and vascular resistance. Sustained pulmonary artery hypertension induces changes in right ventricular morphology, dilation, hypertrophy, eventually followed by cardiac failure. The right ventricular disorder caused by altered cardiopulmonary physiology, excluding secondary change from congenital heart disease, is called cor pulmonale (pulmonary heart disease).[6] In addition, it may cause elevated wedge or left atrial pressure and in some patients, there may also be elevated left ventricular end-diastolic pressure.[7] Recent evidence suggests a multifactorial cascade of pulmonary vasoconstriction and hypertension in response to hypoxia. It has been proposed that the mechanism through which hypoxemia affects the pulmonary vasculature is that pulmonary vascular dilation mediated by endothelial-derived relaxing factors (EDRF) and nitric oxide, were found to be impaired in both acute and chronic hypoxia in vitro.[8-10] Acute vasoconstriction, may occur within minutes and is usually reversible, as found in acute pulmonary circulation obstruction (e.g. thromboembolism) or acute upper airway obstruction (e.g. tonsillar hypertrophy). In contrast, a chronic response to hypoxia leads to structural remodeling of the pulmonary vascular bed with predominant hypertrophy of the tunica muscularis of the medium and small sized pulmonary arteries via many mediator releases. In the latter, the hypertrophy may not be reversible even though the upper obstruction is relieved.[11,12]

Many signs of cor pulmonale can be masked by the primary illness that underlies the pulmonary hypertensive state. Thus, the clinician should maintain a high index of suspicion for the presence of right ventricular dysfunction, despite the possibility that other causes can explain such clinical findings. In the presence of pulmonary hypertension, physical examination may reveal right ventricular enlargement and increased second heart sound (P_2 component). Chest roentgenogram can be a useful marker of cardiac size and can indicate whether right

ventricular dilation is present. Electrocardiogram may show right ventricular hypertrophy (pressure overload) with or without right atrial enlargement. Echocardiography may show evidence of right ventricular hypertension and demonstrate left ventricular diastolic dysfunction by observing the deceleration time which is the only diastolic parameter which is significantly different from the normal population in obstructive sleep apnea syndrome (OSAS).[13] It may be the earliest finding of the left ventricular diastolic function measured. Meywed Hedner, et al[14] also demonstrated an increase in the interventricular septum and left ventricular posterior wall thickness in adult patients with OSAS. In our patient, the only abnormal finding was mild cardiomegaly on chest roentgenogram, which led us to perform adenotonsillectomy. Of all the causes of cor pulmonale, upper airway obstruction is associated with the best prognosis because complete relief of the primary problem is often possible, and if done before chronic right ventricular ultrastructural changes have occurred, cor pulmonale itself may regress.[13,15,16]

REFERENCES

1. Ferrari LR, Vasello SA. Anesthesia for otorhinolaryngology procedures. In: Cote CJ, Todres ID, Ryan JF, Goudsouzian NG, eds. A practice of anesthesia for infants and children, 3rd ed. Philadelphia: W.B. Saunders, 2001: 461-477.
2. Patel CYC. Recurrent pulmonary edema: an uncommon presenting feature of children obstructive sleep apnea hypoventilation syndrome in an otherwise healthy. Clin Pediatr 2001; **40:** 287-290.
3. Yemen TA. How much do we really know about postobstructive pulmonary edema? (Editorial). Paediatr Anaesth 2000; **10:** 459-491.
4. Rich GF. Perioperative management of the patient with pulmonary hypertension and right heart failure. American Society of Anesthesiologists: Annual Meeting Refresher Course Lectures, 2000; **135:** 1-7.
5. Halfaer MA, Wilson MD. Obstructive sleep apnea, control of ventilation and anesthesia in children. Pediatr Clin N Am 1994; **41:** 131-150.
6. Gewitz MH. Cor pulmonale-pulmonary heart disease. In: Allen HD, Clark EB, Gutgesell HP, Driscoll DJ, eds. Moss and Adams' heart disease in infants, children, and adolescents including the fetus and young adult, 6th ed. Vol II. Philadelphia: Lippincott Williams & Wilkins, 2001: 1368-1376.
7. Fakhoury KF, Seilheimer DK. Pulmonology. In: Garson A Jr, Bricker JT, Fisher DJ, Neish SR, eds. The science and practice of pediatric cardiology, 2nd ed. Vol II. Baltimore: Williams & Wilkins, 1998: 2813-2815.
8. Din-Xuan AT, Higgenbottam TW, Clelland CA, et al. Impairment of endothelial-dependent pulmonary artery relaxation in chronic obstructive lung disease. N Engl J Med 1991; **324:** 1539-1547.
9. Liu SF, Crawley DE, Barnes PJ, Evans TW. Endothelium-derived releasing factor inhibits hypoxic pulmonary vasoconstriction in rats. Am Rev Respir Dis 1991; **143:** 32-37.
10. Adnot S, Raffestin B, Addahibi S, et al. Loss of endothelium-dependent relaxant activity in pulmonary circulation of rats exposed to chronic hypoxia. J Clin Invest 1991; **87:** 155-162.
11. Perkin RM, Anas NG. Pulmonary hypertension in pediatric patients. J Pediatr 1984; **105:** 511-522.
12. Brodsky L. Tonsillitis, tonsillectomy and adenoidectomy. In: Bailey JB, Calhoun KH, Deskin RW, eds. Head and neck surgery-Otolaryngology, 2nd ed. Philadelphia: Lipincott-Raven Press, 1998: 1221-1235.
13. GörUr K, Döven O, Unal M, Akkus N, Ozcan C. Preoperative and postoperative cardiac and clinical findings of patients with adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 2001; **59:** 41-46.
14. Hedner J, Ejnell H, Caidahl K. Left ventricular hypertrophy independent of hypertension in patients with obstructive sleep apnea. J Hypertens 1990; **8:** 941-946.
15. Ramakrishna S, Ingle VS, Patel S, et al. Reversible cardiopulmonary changes due to adenotonsillar hypertrophy. Int J Pediatr Otorhinolaryngol 2000; **55:** 203-206.
16. Wiet JG, Bower C, Seibert R, Grieble M. Surgical correction of obstructive sleep apnea in the complicated pediatric patient documented by polisomnography. Int J Pediatr Otorhinolaryngol 1997; **41:** 133-143.