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JSM Dentistry

Short Communication

Rapid Maxillary Expansion and Childhood Obstructive Sleep Apnea Syndrome

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Submitted: 10 August 2013

Accepted: 10 August 2013

Published: 12 August 2013

Abstract

Obstructive sleep apnea syndrome (OSAS) is a common condition in children and can result in serious neurocognitive, cardiovascular and metabolic complications if left untreated. Recently, the association between OSAS and maxillofacial malformation, and malocclusion has attracted much attention. Rapid maxillary expansion (RME), a commonly used orthodontic procedure to achieve a skeletal expansion of the upper jaw, has shown promise to be an alternative treatment of childhood OSAS. Several case series studies on this subject were reviewed and discussed. Pediatric dentists and orthodontists can play an active role in the interdisciplinary management of OSAS by assessing the condition of the maxillary arch to consider RME therapy.

Obstructive sleep apnea syndrome (OSAS) is a common condition in children and can result in serious neurocognitive, cardiovascular and metabolic complications if left undetected and untreated [1]. According to a report by American Academy of Pediatrics, depends on the population studied, the prevalence of OSAS is in the range of 1% to 5% [2]. The sequelae of OSAS include excessive daytime sleepiness, poor school performance, learning disability, attention deficit, hyperactivity, behavior problems, cardiovascular abnormalities and metabolic disorders [1,2]. Therefore, early diagnosis and treatment of pediatric OSAS is beneficial in improving a child's long-term cognitive development, social interaction, academic achievement, cardiovascular health and overall wellbeing.

Increased upper airway resistance during sleep in children with OSAS is most likely due to a combination of soft tissue hypertrophy, craniofacial dysmorphology, neuromuscular weakness or obesity [1]. The most common cause of pediatric OSAS is adenotonsillar hypertrophy [3]. Recently, the association between OSAS and maxillofacial malformation, and malocclusion has triggered more interest. Most common facial anatomic abnormalities associated with airway obstruction include nasal septum deviation, narrow maxillary arch, and retro-micrognathia [1]. "Long face syndrome" seen in children with mouth-breathing habits is also associated with OSAS [1].

In recently years, rapid maxillary expansion (RME) has been used in treating OSAS in certain pediatric populations [4-7]. RME is an orthodontic procedure commonly used to achieve a skeletal expansion of the upper jaw. RME widens the maxillary arch and increases the volumetric space of the nasal space by applying force to the midpalatal suture; also indirectly expands oropharyngeal space by modifying the resting posture of the tongue [8]. Researchers have long noticed the improvement of nasal resistance after RME [9], however its usage on OSAS treatment has only been studied recently. One of the first reports on the treatment of pediatric OSAS using RME was published in 2004 by Pirelli et al. [4]. 31 young patients with OSAS who have normal BMI, maxillary constriction and no adenotonsillar hypertrophy were included in this study. Rapid expansion was finished within in 10-20 days and the device was kept in place for retention for 6-12 months. ENT evaluation and polysomnography were performed at 3 time points including 1) before the orthodontic therapy (T0); (2) 4-6 weeks (T1) after therapy, with the device still in place; and (3) 4 months after the end of orthodontic treatment (T2). At T0, the mean apnea-hypopnea index (AHI) was 12.2 events/hour. At T2, all children had an AHI<1 event/hour in spite of the differences in their baseline AHI. Increase of nasal pyriform opening was also noted. In addition, the anterior rhinometry indicated normal nasal resistance at T2 in all patients [4]. The same group conducted another study with similar design and same inclusion criteria but larger sample size [5]. The results obtained from 60 patients showed that the decrease of AHI from T0 to T2 is significant. CT images before and after RME therapy confirmed the widening of nasal fossa and the septum release. These changes increased the patency of the upper airway, thus restoring a normal nasal airflow with disappearance of obstructive sleep-disordered breathing [5]. Evidently, RME can be effective in relieving pediatric OSA symptoms. However, only children without enlarged adenoids and tonsils were recruited for these studies [4,5]. Patients with different degree of tonsillar

hypertrophy were included in a study done by Villa et al. [6,7]. At 12 month after RME, The improvement of mean AHI in patients with severe tonsillar hypertrophy was noted (6.2 to 2.3 events/h, p=ns), however the changes was less significant in comparison to those in patients with mild tonsillar hypertrophy (5.6 to 1.0 events/h, p=0.034) [6]. 10 out of 14 children in this study were enrolled in their follow up study to assess the long-term efficacy of the RME [7]. Twenty four months later, changes in the AHI after RME were still observed in 8 out of 10 patients. Even though the value of this study may be limited by its small sample size, the persistent effect of RME is noteworthy. Treatment relapse/failure was observed in one patient with severe tonsil hypertrophy and another patient with significant weight gain [7]. The efficacy of RME alone in children with enlarged tonsils and adenoids is uncertain. Based on these case series studies, RME showed promise as an alternative treatment for pediatric OSAS, particular in children who have maxillary constriction and without enlarged adenoids. However, other than small sample size, another common limitation of these studies is the lack of control group due to ethical reasons. Currently, there is no randomized controlled clinical trial assessing the efficacy of RME therapy on pediatric OSAS. In addition, further research is needed in identifying the patient population who may benefit most from RME and clarifying the ideal timing for RME therapy.

Adenotonsillectomy is still the primary treatment for a child diagnosed with OSAS who has adenotonsillar hypertrophy [2,10]. However, persisting OSA symptoms are not uncommon after surgery [10,11]. Continuous positive airway pressure (CPAP) therapy has been proved to be effective in these situations, but the efficacy of CPAP is limited by poor long-term compliance [2,10]. Guilleminault et al. believed that a partial response to adenotonsillectomy may be associated with facial abnormalities [11]. Therefore post-op RME therapy can be a better choice than CPAP in resolving OSAS in these patients.

Pediatric dentists and orthodontists who perform a comprehensive head-neck exam are in a unique position to identify young patients with increased risk for OSAS and refer them to sleep specialist for further evaluation. With RME becoming a useful treatment option for pediatric OSAS, dentists will play a more active role in the interdisciplinary management of OSAS.

REFERENCES

1. Katz ES, D'Ambrosio CM. Pediatric obstructive sleep apnea syndrome. Clin Chest Med. 2010; 31: 221-34.
2. Marcus CL, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics. 2012; 130: 576-84.
3. Marcus CL. Sleep-disordered breathing in children. Am J Respir Crit Care Med. 2001; 164: 16-30.
4. Pirelli P, Saponara M, Guilleminault C. Rapid maxillary expansion in children with obstructive sleep apnea syndrome. Sleep. 2004; 27: 761-6.
5. Pirelli P, Saponara M, De Rosa C, Fanucci E. Orthodontics and obstructive sleep apnea in children. Med Clin North Am. 2010; 94: 517-29.
6. Villa MP, Malagola C, Pagani J, Montesano M, Rizzoli A, Guilleminault C, et al. Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. Sleep Med. 2007; 8: 128-34.
7. Villa MP, Rizzoli A, Miano S, Malagola C. Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. Sleep Breath. 2011; 15: 179-84.
8. Principato JJ. Upper airway obstruction and craniofacial morphology. Otolaryngol Head Neck Surg. 1991; 104: 881-90.
9. Timms DJ. The effect of rapid maxillary expansion on nasal airway resistance. Br J Orthod. 1986; 13: 221-8.
10. Marcus CL, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics. 2012; 130: 576-84.
11. Guilleminault C, Huang YS, Glamann C, Li K, Chan A. Adenotonsillectomy and obstructive sleep apnea in children: a prospective survey. Otolaryngol Head Neck Surg. 2007; 136: 169-75.

Cite this article

Xu Z (2013) Rapid Maxillary Expansion and Childhood Obstructive Sleep Apnea Syndrome. JSM Dent 1(2): 1010.