



OSA Masquerading as Failure to Thrive and Developmental Delay: A Case Report

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Abstract

An 8-month-old girl was admitted to the hospital for failure to thrive with significant developmental delays. Snoring and hypoxia during sleep were noted and she was started on heated high-flow nasal cannula at 21% FiO₂. She continued to have drops in her oxygen saturation down to 68%, in addition to retractions and tracheal tugging during sleep. Adenoidectomy was performed, removing 100% obstructing adenoids, but snoring and oxygen desaturations while sleeping continued. Polysomnography revealed severe obstructive sleep apnea (OSA) (apnea/hypopnea index (AHI) 160.8/hour, 236.6/hour in active sleep), and oxygen saturation nadir was 65%. Other surgeries were thought neither possible nor safe at that time. Auto titrating continuous positive airway pressure (autoCPAP) 4-6 cmH₂O was started and later increased to 4-10 cmH₂O. Oxygen saturation normalized on CPAP. Eleven days after initiation of treatment, the patient was able to hold her head up, reach for and play with toys, sit up and pull to stand with minimal assistance. She had a 1 kilogram weight gain during the hospital stay. By 14 months of age, she was standing with support and trying to speak. By 17 months, she had grown from below the 1st percentile to almost the 50th percentile for BMI. A tonsillectomy and uvulopalatopharyngoplasty were performed at that time resolving the OSA (AHI 0.4/hour, oxygen nadir 91% per PSG), and CPAP was discontinued. This case is interesting for pediatricians because it highlights the importance of having a high index of suspicion for OSA in children with failure to thrive or developmental delay, it points out the role of noninvasive ventilation for OSA treatment in infants, and It highlights the importance of follow up and continued re-evaluation for availability of surgical options in young children with OSA.

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Received Date: 04 Jul 2017

Accepted Date: 30 Sep 2017

Published Date: 02 Oct 2017

Citation:

El Taoum K, Jambhekar S. OSA Masquerading as Failure to Thrive and Developmental Delay: A Case Report. Ann Clin Case Rep. 2017; 2: 1436.

ISSN: 2474-1655

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Abbreviations

ADHD: Attention Deficit Hyperactivity Disorder; AHI: Apnea Hypopnea Index; ALTE: Acute Life Threatening Event; Auto CPAP: Auto Titrating Continuous Positive Airway Pressure; CPAP: Continuous Positive Airway Pressure; FTT: Failure To Thrive; GERD: Gastro Esophageal Reflux Disease; NC: Nasal Cannula; NIV: Non Invasive Ventilation; OSA: Obstructive Sleep Apnea; PSG: Poly Somno Graphy

Table of Content Summary

FTT can result from different etiologies. This article shows an unusual cause of FTT and the challenges to diagnose it and treat it appropriately.

Contributors' Statement Page

Dr El Taoum drafted the initial manuscript and Dr Jambhekar reviewed and revised the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Introduction

Obstructive Sleep Apnea (OSA) is characterized by partial airway obstruction or intermittent complete obstruction that interrupts normal ventilation during sleep [1]. Infants are at high risk of severe OSA compared to older children and adults due to their upper airway anatomy, laryngeal chemoreceptors, and sleep-related breathing patterns [2]. The most common presenting symptoms of OSA in infants are snoring, nocturnal hypoxia, and acute life threatening event (ALTE). In children, OSA is known to be an important risk factor for development of behavioral dysfunction, attention deficit hyperactivity disorder (ADHD), failure to thrive (FTT), and developmental delay if not recognized and treated early [3].

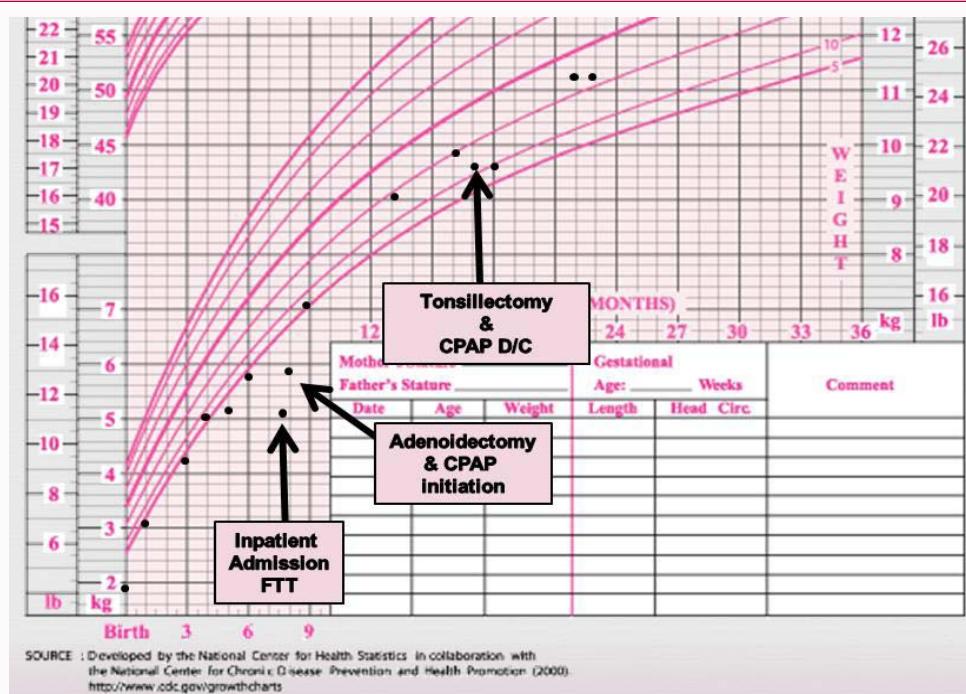


Figure 1: Growth curve before and after intervention.

FTT may result from multiple etiologies. Non organic etiologies are more frequently encountered, but pediatricians should always rule out organic causes first. Some of these causes like malabsorption, formula intolerance, genetic disorders, endocrine disorders, are more common than others like severe OSA with adenotonsillar hypertrophy. OSA with comorbid adenotonsillar hypertrophy is known to increase the risk of FTT in infants and children [4].

Case Presentation

Patient information

This is an 8-month-old, former 34 weeks premature, female who was admitted to the hospital for FTT and severe developmental delay. Her mother reported that she had constant nasal congestion and runny nose that never cleared up. Multiple formula changes had been made with no improvement in her frequent vomiting. Rice cereal was added to her feeds with no improvement. She had presented to the ER for bronchiolitis 2 weeks earlier and was treated with albuterol and antibiotics.

Family history was positive only for mild allergy and constipation. Patient was on no medication, she did not have any known allergy and her immunization was up-to-date.

Physical examination

Patient appeared poorly developed with hypotonia and developmental delay, head lag, and inability to sit up (developmental age 2-3 months, developmental quotient 38%). Her weight was below the 1st percentile for her age, and her height was at the 5th percentile for her age. She was in no acute distress, had nasal congestion, coarse breath sounds bilaterally, with no wheezing or crackles. She did not have micrognathia, cleft lip or cleft palate, or any other craniofacial abnormality. During her first night in the hospital she was found to have snoring and hypoxia during sleep.

Diagnosis

The patient was started on O₂ via nasal cannula (NC) waiting for

her surgery. On day 4 of admission, she had a Micro Laryngoscopy, Bronchoscopy, and Adenoidectomy with final diagnosis of 100% obstructing adenoids and mild tracheobronchomalacia. She continued to have snoring and hypoxia during sleep following surgery. Her symptoms were relieved temporarily by changing her body position during sleep. There was oxygen requirement only during sleep. A comprehensive work-up was obtained including sweat chloride test, upper gastrointestinal series, EKG, chest x-ray, brain MRI, and apnea monitor. Sweat chloride test, EKG and brain MRI were normal. Chest x-ray initially showed peribronchial thickening and scattered atelectasis then normalized 10 days later in a repeat film. The upper gastrointestinal series resulted in normal anatomy with multiple episodes of gastroesophageal reflux disease (GERD) to the mid to upper esophagus. She was treated with nasal steroids and ranitidine. The later was changed to omeprazole with no improvement in symptoms. An initial speech evaluation showed no aspiration but deep level penetration with thin liquids and liquids thickened to nectar consistency. The patient was fed with liquids thickened to honey consistency to decrease the risk of aspiration. Multiple apnea monitor downloads were obtained and showed no central apneas but multiple episodes of desaturation mainly in high 70's to high 80's. The pattern of the tracing suggested the possible presence of increased work of breathing or upper airway obstruction. Due to the limitation of smart monitor in detecting airway obstruction, clinical correlation and additional testing were recommended to rule out upper airway obstruction. As a result, on day 12 of admission, a Polysomnography (PSG) was performed and it showed severe OSA with an Apnea/Hypopnea index (AHI) of 160.8/hr (Normal AHI is less than 2/hr for children), an AHI of 236.6/hr in active sleep, and a minimum SpO₂ of 65%.

Intervention

On day 16 of admission, a PSG with oxygen titration was done and showed improved oxygenation but worsening of ventilation and much disrupted sleep with AHI of 160/ hour. After discussion with

ENT and Pulmonary, on day 20, continuous positive airway pressure (CPAP) treatment was initiated and then titrated progressively as patient started tolerating the interface. She had mildly enlarged tonsils, but surgery was postponed due to concern for postoperative complications in view of age of the patient and the severity of OSA.

Course

Patient showed major clinical improvement with no more hypoxia at night. Her weight increased by 1 kilogram in 30 days. She also showed significant developmental gains: she was able to hold her head up independently, reach for and play with toys, sit up with minimal assistance, and pull to stand with minimal assistance (developmental age 6-7 months, developmental quotient 67%), all of which she was not able to do on admission (Figure 1). She was discharged on a CPAP of 10cmH₂O after 30 days in the hospital.

Patient was followed in sleep clinic regularly after discharge. She was seen after 1 month, then every 3 months. Patient was doing better on CPAP and continued to improve her development and weight gain.

At the age of 14 months, she was standing with support and babbling (developmental age 11 months, developmental quotient 78%). At the age of 17 months, she had grown from below the 1st percentile to the 45th percentile for her age, her developmental age was 13-14 months, and developmental quotient 80%.

One year later, she continued to improve but with symptoms of OSA when off the CPAP. Tonsillectomy and uvulopalatopharyngoplasty were performed. After surgery, PSG was repeated. The patient was 2 years old at that time. PSG without intervention showed normal breathing with an AHI of 0.4/hour. Her swallow study was also normal and she had no GERD symptoms. CPAP and reflux medications were stopped and she was allowed to drink liquids of thin consistency. She was then discharged from sleep clinic due to resolution of her OSA (Figure 1).

Discussion

Prematurity, GERD, aspiration, tracheobronchomalacia and laryngomalacia are well known risk factors for OSA and in our patient could be contributing to the severity of her symptoms [5]. Adenotonsillar hypertrophy is a common cause of OSA in children between the ages of 2 and 8 years. In our patient, adenotonsillar hypertrophy lead to OSA at a much younger age and this may be related to the presence of the other contributing causes. Infants with OSA are at increased risk of FTT, and developmental and behavioral compromise.

OSA comorbid adenotonsillar hypertrophy is thought to contribute to FTT by (1) feeding disorder including feeding fatigue [6] (2) increased sleep energy expenditure [7,8] and (3) Reduced Growth Hormone [9,10]. In our patient, it is very likely that the OSA comorbid adenotonsillar hypertrophy also contributed to the dysphagia and may have caused reduced feeding which in turn may have contributed to the FTT.

Adenotonsillectomy alone can lead to major improvement in growth and development in infants with OSA [11]. But infants, like our patient, that have more than one factor leading to their upper airway obstruction will not improve with surgery alone if appropriate work-up and treatment of these factors is not implemented [12,13].

Some patients with OSA do not respond to surgery, and may

require more intervention like non-invasive ventilation (NIV) or occasionally tracheostomy tube placement. Each of these interventions has their own limitations. NIV with mask carries an inherent problem of poor adherence and a risk of facial mal development in children with adequate adherence. Tracheostomy placement has the potential risk of obstruction and asphyxia in the absence of immediate intervention, a risk of increased infections and difficulty with care of the tracheostomy.

NIV is used and studied widely in adults and older children with OSA. The main problem with NIV is the poor adherence with treatment. In our patient, we could successfully use NIV for an infant who was thought to have a greater risk/benefit ratio for surgery at the time until she could grow and undergo successful surgical intervention safely.

Our patient also benefitted significantly from being followed in a sleep clinic on a regular basis where she was re-evaluated for possibility and safety of surgical intervention that was curative and led to discontinuation of NIV.

Conclusion

This case report illustrates several important points. First, it underscores the importance of suspecting OSA in infants presenting with FTT and developmental delay. Second, it highlights the possibility of successful use of NIV for treatment of OSA at a very young age. Third, it proves that a close follow-up, a multidisciplinary approach, and repeated evaluation for alternative treatment plans are imperative for long term management of OSA in infants.

Acknowledgements

We would like to acknowledge the sleep team at Arkansas Children's Hospital for their contribution.

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