Nocturnal Enuresis and Obstructive Sleep Apnoea in Two Children

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ABSTRACT

Nocturnal enuresis was a symptom of childhood obstructive sleep apnoea, OSAS. We reported two children with secondary nocturnal enuresis which disappeared after tonsillectomy and adenoidectomy for proven OSAS. Pathogenesis of secondary nocturnal enuresis in OSAS was discussed

Keywords: Child, sleep apnoea, OSAS, enuresis

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INTRODUCTION

Nocturnal enuresis had been described as a symptom of childhood obstructive sleep apnoea, OSAS⁽¹⁾. However, the only controlled study to date of enuresis in children with OSAS did not demonstrate a significant association⁽²⁾. We report here two children with secondary nocturnal enuresis which disappeared after tonsillectomy and adenoidectomy (T&A) for proven OSAS. Secondary enuresis was defined as dryness for at least one year before reappearance of bed wetting.

CASES

YKO was a nine-year-old boy with secondary daily nocturnal enuresis for eight months and nightly snoring for one year before presentation. No apnoea nor daytime somonolence was observed. Physical examination revealed a well-nourished boy. Both tonsils were enlarged, nearly touching mid-line. The rest of examination was normal. Blood for urea, creatinine was normal. Early morning urine osmolarity was 599 mOsm/kg. Mid-stream urine microscopy and culture were normal. Sleep polysomongraphy. PSG, revealed severe obstructive apnoea/hypopnea, 55.3 episodes per hour. Desaturation occurred 61.8 times per hour. T&A was done followed by disappearance of both snoring and nocturnal enuresis within days. On follow-up five months later, he had no complaint of either symptoms.

WPY was a six-year-old girl with secondary nocturnal enuresis, one to two times per month, for two years with history of nightly snoring before presentation. No apnoea nor daytime somnolence was observed. Physical examination revealed a well-nourished girl. Both tonsils were enlarged to mid-line. The rest of examination was normal. Blood for urea, creatinine was normal. Early morning urine osmolarity was 590 mOsm/kg. Mid-stream urine microscopy and culture were normal. Sleep PSG revealed moderate apnoea/hyponoea, 11.3 episodes per hour. T&A was done followed by disappearance of both snoring and nocturnal enuresis within days. She was followed up for one year with no recurrence of either symptom.

DISCUSSION

Nocturnal enuresis is regarded as a symptom of OSAS(1). In a study of 50 children, 18% presented with secondary enuresis(3). However, there is on-going doubt about this association; the only controlled study to date of enuresis in children with OSAS did not demonstrate a significant association⁽²⁾. Carroll⁽⁴⁾ reviewed the subject of childhood OSAS. He asserted that the association remained to be proven in the absence of case-controlled studies. Absence of polysomnographic studies in the published case series that link secondary enuresis and OSAS did not help establish the association⁽⁵⁾. In our two cases, the fact that nocturnal enuresis resolved within days of T&A meant for OSAS lent support to the description of nocturnal enuresis being a symptom of OSAS. It behoves medical practitioners to ask for symptom of snoring when faced with children complaining of secondary nocturnal enuresis. Appropriate diagnosis and treatment of OSAS may relieve the symptoms of secondary nocturnal enuresis. The mechanism of nocturnal enuresis in OSAS probably involves increased production of atrial natriuretic peptide and increased arousal threshold⁽⁶⁾. The increased production of atrial natriuretic peptide was documented in six adults with severe OSAS and the mechanism was attributed to distension of right atrium during obstructed episode when the negative intrathoracic pressure was markedly increased(7).

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