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Polysomnography in obese children with and without Prader-Willi syndrome

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Background

Sleep abnormalities (excessive daytime sleepiness, sleep apnea) were initially estimated as minor diagnostic criteria of Prader-Willi Syndrome (PWS). However, several studies found a high prevalence of sleep disorders in PWS patients. It is assumed that PWS patients are at a high risk of sleep disordered breathing, such as obstructive sleep apnea (OSA), because of their childhood obesity, associated with muscle hypotonia, leading to upper airway collapse.

Objective

We studied a group of PWS children (genetically confirmed, non-GH-treated) who performed complete sleep studies and compared to a group of non-PWS obese children (OC) matched for sex, age and BMI. All the study patients were in the prepubertal stage.

Subjects and Methods:

All patients underwent overnight polysomnography (Comet, Grass Technology, USA). Data are reported as medians (interquartile range), Manne-Whitney test was used for between-group.

Results:

We didn’t find a statistically significant difference in the prevalence of OSA between obese PWS and non-PWS children (58,8% vs 50%, p=0,64). However, PWS patients were found to have distinctive polysomnographic features – the significantly shortened REM latency vs non-PWS children, suggesting possible dysregulation of sleep-wake cycle. The main polysomnographic parameters of evaluated children are presented in Table 1.

Table 1 – Polysomnography characteristics of PWS and non-PWS obese children

Characteristics	PWS (n=23)	OC (n=8)	p
Age (years)	9,9 [6,9÷13,9]	9,15 [6,85÷13,0]	p=0,98
Males/females	14/9	3/5	p=0,4
Tanner stage	1	1	p=1
BMI SDS	3,16 [2,2÷4,53]	2,9 [2,79-3,4]	p=0,7
Tonsillar hypertrophy	30,4% (n=7)	25% (n=2)	p=0,73
Adenoidal hypertrophy	52,1% (n=12)	62,5% (n=5)	p=0,36
REM latency (min)	81,75 [63,0 ÷ 143,25]	160,5 [125,75 ÷ 205,50]	p=0,01
Apnea-hypopnea index	3,5 [0,6 ÷ 9,2]	1,7 [0,3 ÷ 8,1]	p=0,61
Oxygen desaturation index	5,35 [1,45 ÷ 11,0]	2,8 [0,95 ÷ 17,55]	p=0,74

Conclusion:

There is a high prevalence of OSA in PWS children, but it's not higher than in simply obese pediatric patients. The shortened REM latency in PWS children could suggest the different sleep-wake cycle in this syndrome. Further research is necessary to clarify the mechanism of sleep related disorders in PWS