The frequency of obstructive sleep apnea in children with hypothalamic and exogenous obesity

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INTRODUCTION

Hypothalamus takes part in sleep regulation and appetite control. Sleep dysregulation associated with severe obesity can be a feature of hypothalamic damage as well as exogenous obesity.

AIM

To investigate the hypothalamic damage on sleep regulation and its relation to metabolic parameters.

METHODS AND MATERIALS

14 hypothalamic (4 males) obese children as study group (10 craniopharyngiomas, 2 suprasellar non-glial tumors, 1 septo-optic dysplasia and 1 patient with damaged hypothalamus and hydrocephalus due to sequela of meningoencephalitis), and 30 exogenous (11 males) obese children as control group in prospective, cross sectional, case control study. Oxalogical findings and pubertal status were evaluated. Mallampati scores and scoring of sleepiness were assessed. All patients underwent full-night polysomnography (PSG) for assessment of OSA. Fasting blood glucose, insulin, lipid profile, hsCRP, TNFα were measured.

RESULTS

The apnea-hypopnea index (AHI) of hypothalamic obesity group was higher than that of exogenous obesity group in PSG. After adjusting for age, sex and BMI SDS; the odds of OSA increased 4.4-fold for hypothalamic obese subjects in multivariant analysis. Risk of OSA is significantly increased in hypothalamic obesity in comparison to exogenous obesity. OSA (AHI score) was not correlated to hypertension, insulin resistance, dyslipidemia, levels of inflammatory markers.

Table 1. Clinical findings

	Hypothalamic (n=14)	Exogenous (n=30)	p
Age	13,3 ± 4,44	13,07 ± 3,16	0,842[a]
Prepubertal	3 (%21)	7 (%23)	0,865[b]
Pubertal	11 (%78)	23 (%76)	
Weight (kg)	62,88 ± 30,89	89,6 ± 25,84	**0,005[a]**
Height (cm)	138,7 ± 20,61	159,8 ± 12,7	**0,005[a]**
BMI	30,82 ± 8,69	32,25 ± 8,38	**0,023[b]**
BMI SDS	2,07 ± 0,50	2,39 ± 0,34	**0,041[b]**

Table 2. Polysomnography findings

	Hypothalamic (n=14)	Exogenous (n=30)	p
AHI	10,7 ± 10,34	4,27 ± 5,45	**0,015[b]**
Sleep efficiency (%)	64,48 ± 21,49	79,08 ± 14,13	**0,041[b]**
Arousal index	14,46 ± 6,61	10,87 ± 3,72	0,075[a]
Minumum spO2 (%)	84,86 ± 8,39	89,1 ± 5,84	**0,046[b]**
Desaturation time (min)	16,04 ± 45,65	1,32 ± 3,88	**0,034[b]**
Desaturation index	3,98 ± 11,46	0,18 ± 0,67	0,063[b]

Table 3. Sleepiness scoring

	Hypothalamic (n=14)	Exogenous (n=30)	p
Daytime sleepiness	5 (%35,7)	10 (%33,3)	0,87[a]
Snoring	8 (%57,1)	21 (%70)	0,402[b]
Napping	0	2 (%6,7)	
Attention deficit	4 (%28,6)	3 (%10)	0,184[a]
Disturbed sleep	9 (%64,3)	12 (%40)	0,133[c]
Total score	1,5 ± 1,29 (0 – 4)	2,0 ± 0,89 (0 – 3)	0,547[d]

Table 4. Metabolic and inflamatory markers

	Hypothalamic (n=14)	Exogenous (n=30)	p
HbA1c (%)	5,54 ± 0,73	5,23 ± 0,41	0,075[b]
FBG (mg/dl)	86,9 ± 12,74	93,17 ± 10,72	0,106[b]
Insulin (µIU/ml)	29,94 ± 53,19	35,43 ± 35,52	0,087[b]
Dyslipidemia	8 (%47,1)	9 (%52,9)	0,165[b]
hsCRP (pg/ml)	635,96 ± 377,47	786,48 ± 374,5	0,246[b]
TNFα (pg/ml)	25,51 ± 32,99	28,42 ± 50,97	0,840[b]

(a-T test, b-Mann Whitney U test, c-Pearson's Chi-squared test with Yates' continuity correction)

CONCLUSIONS

- **Frequency and severity of OSA is higher in hypothalamic obesity group in comparison to exogenous obesity.**
- **Inflamation (CRP, TNFα), hyperinsulinism or Mallampati scores are not enough to explain increased risk of OSA in both groups.**
- **Polysomnography should be a part of routine investigation in hypothalamic obesity, even without any complaint suggesting a sleep disorder.**