Severe hypothalamic obesity in a girl with craniopharyngioma - case report

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Background

Hypothalamic obesity is a form of obesity syndrome associated with a variety of hypothalamic disorders including intracranial tumors, infections, trauma, vascular problems and hydrocephalus and acquired or congenital functional defects in central energy homeostasis. The pathogenetic mechanisms underlying hypothalamic obesity are multifactorial. Weight gain results from the hypothalamus damage, which leads to excessive appetite and low metabolic rate, multiple pituitary hormonal deficiency, hypomobility and insomnia.

The aim is to present a 20-year-old female who underwent neurosurgical operation in the age of 11 years because of craniopharyngioma and manifested severe obesity thereafter (BMI>50kg/m2).

Patient report

The girl in the age of 10 years was referred to endocrinologic outpatient clinic because of height deceleration and weight gain. In the age of 11 years craniopharyngioma (max. diameter 30mm) was diagnosed. Postsurgically she developed multihormonal pituitary insufficiency and excessive appetite. The girl was on hormonal substitution therapy. With time the disturbed energy balance led to severe obesity with complications like insulin resistance, scoliosis, knee valgus and low self-assessment. The pharmacological treatment with metformin, orlistat, alpha-glucosidase inhibitor and psychological therapy did not bring weight loss. The BMI was critical 52 kg/m2. In the age of 19 years she underwent very unique neurosurgical operation. The stimulator for deep brain stimulation into the nucleus accumbens septi of the brain was installed. The aim of this intervention was to modify the appetite control. The operation was successful. The weight loss was significant - from a total weight of 150kg to 130kg. Since that time the weight is stable. The most important fact is that the self-assessment has been changed. The motivation improved and there is no obsessive thinking about eating.

Tab. 1 Laboratory results of presented patient.

Laboratory tests	11 yrs	15 yrs	18 yrs	Reference range
TSH (µIU/ml)	0.12	0.0	0.11	0.470-4.640
fT4 (ng/dl)	1,26	1,26	0.74	0.71-1.85
Max GH (ng/ml) after onset of sleep	0.03			> 10 ng/ml
Max GH (ng/ml) after glucagone	0.03			> 10 ng/ml
Max GH (ng/ml) after insulin	0.03			> 10 ng/ml
LH (mIU/ml)	0.8	0.0	<0.5	Depending on age and cycle phase
FSH (mIU/ml)	1.0	0.0	<0.37	1.0-8.0
Estradiol	21	6	207	39-189 (follicular phase)
Prolactin (ng/ml)	16.9	17.8	15.41	3.24-29.12
IGF-1 (ng/ml)	280	892	571	Depending on age
ACTH (pg/ml)		9	10.2	10-60
Cortisol (ng/ml) at 7 am	33	11	80	94-260
HbA1c (%)	5,0	5.4	5.6	<6.1
Fasting glucose (mg/dl)	72	100	90	59-101
Fasting insulin (uIU/ml)	9.8	17.3	23.2	< 15
Glucose in OGTT 120'		138	121	<140
Insulin in OGTT 120'		50.0	>300	
Cholesterol (mg/dl)	202	134	152	110-230
Triglycerides (mg/dl)	153	181	163	30,6-105
AlAT (U/l)	53	20	39	1-45

Fig 1. Nucleus accumbens:
-placed in the basal forebrain.
-consists of dopaminergic neurons.
- is a part of reward system which is the main system responsbible for motivation of behavior.

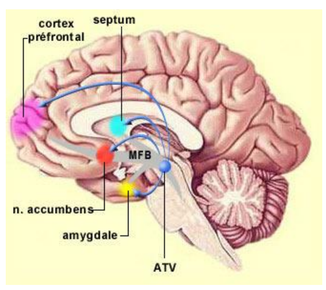


Fig 3. BMI depending on age

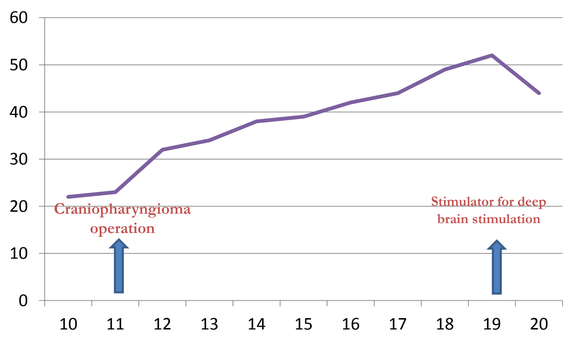
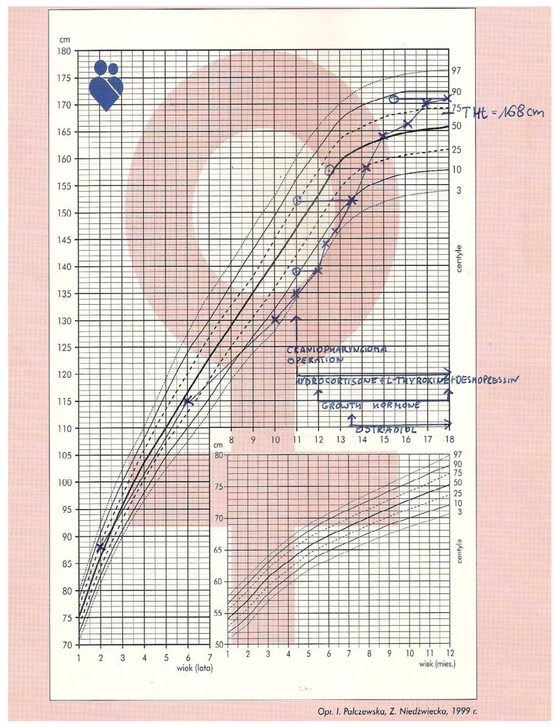


Fig 2. Growth chart of presented patient.



Conclusions

1. **The treatment of hypothalamic obesity is a great challenge.**
2. **The deep brain stimulation gives a chance of better appetite control which seems to be the main problem in the disease.**

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