E-mail: m.welling@erasmusmc.nl

Dextroamphetamine treatment in children with hypothalamic obesity

Van Schaik J[1,3]*, Welling MS[2]*, de Groot CJ[2], Abawi O[2], Burghard M[3], Kleinendorst L[4], Van der Voorn B[2], Van Haelst MM[4], Oude Ophuis B[3], Bakker B[3], Tissing WJE[2], Kamp GA[5], Rotteveel J[6], van Schouten-Meeteren AYN[1], van den Akker ELT[2]**, van Santen HM[1,3] ** (* Shared first authorship, ** Shared last authorship)

1 Princess Maxima Center, Utrecht, The Netherlands; 2 Erasmus MC Sophia Children's Hospital, Rotterdam, The Netherlands; 3 Wilhelmina Children's Hospital, University Medical Center Utrecht, The Netherlands; 4 Amsterdam UMC, Amsterdam, The Netherlands; 5 Tergooi Hospitals, Blaricum, the Netherlands; 6 Vrije Universiteit Medical Center, Amsterdam, the Netherlands.

BACKGROUND

Hypothalamic obesity (HO) in children may have severe consequences. Lifestyle intervention is mostly insufficient. Amphetamines are known for their stimulant effect on resting energy expenditure (REE) and suppressing appetite. We present our experiences of dextroamphetamine treatment in children with HO.

PATIENTS AND METHODS

Retrospective analysis at two endocrine pediatric clinics

- Patients with progressive, therapy-resistant acquired, genetic, or congenital HO were treated
- Measurements at start and during treatment: anthropometrics, REE, (hyperphagic) behavior, and side effects

BASELINE CHARACTERISTICS

- 19 patients started dextroamphetamine treatment (mean age of 12.3 years ± 4.0)
- Of 17 patients, BMI SDS could be evaluated
- Mean treatment duration: 19.5 months ±12.9

EFFECTS ON BMI AND REE

See table and figure

EFFECTS ON BEHAVIOR

13 patients (68.4% of n = 19) reported improvement of hyperphagia, energy level, or behavior.

ADVERSE EFFECTS

Two patients developed hypertension during treatment.

5 children had stopped treatment at last moment of FU, because of no effect on weight or adverse side effects

CONCLUSION

In children with HO, adding dextro-amphetamine treatment to supportive lifestyle interventions, may lower or stabilize BMI SDS, reduce hyperphagia, and improve behavior and activity level

FUTURE STUDIES

International multicenter studies are needed to increase sample size, with randomized placebo control design

	Total Group	Responders	Non-responders
Cause of HO	*n* = 12 acquired *n* = 4 genetic *n* = 1 congenital	*n* = 10 acquired *n* = 3 genetic *n* = 1 congenital	*n* = 2 acquired *n* = 1 genetic
ΔBMI SDS at last moment FU	-0.40 ± 0.86	-0.60 ± 0.82	+0.52 ± 0.09
BMI SDS increase per month (1 year before treatment)	+0.02 ± 0.07	+0.02 ± 0.08	+0.01 ± 0.01
BMI SDS increase per month (during 1st year of treatment)	-0.04 ± 0.06 **(*p* = 0.009)**	-0.06 ± 0.06 **(*p* = 0.004)**	+0.02 ± 0.02 **(*p* = 0.29)**
ΔREE, % predicted	+10.9 ± 14.1 (*n* = 13)	+8.9 ± 14.2 (*n* = 11)	+29.0 and +14.6% (*n* = 2)

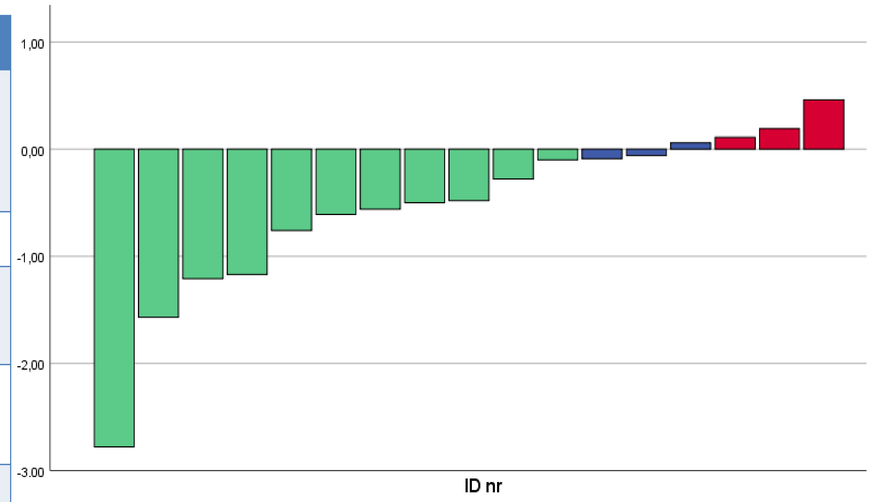

Figure 1. Waterfall plot of differences in BMI SDS between start of treatment and 12 months during treatment

Presented at: ESPE 2021
Fat, metabolism and obesity
Ms Welling
P2-216