Growth Parameters in Infants with Congenital Adrenal Hyperplasia during the First Year of Life

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Authors declare no conflicts of interest.

Abstract:

- **Background:** Previous reports on growth pattern in children with congenital adrenal hyperplasia (CAH) suggested that the height velocity is not increased during the first year of life, even in untreated patients. Whether decreased height potential during first year of life is caused by inadequate suppression of adrenal androgens, excess steroid therapy or salt wasting itself is a matter of debate.
- **Aims:** To analyze growth pattern in infants with CAH during first year of life and the effect of therapy on their growth parameters.
- **Methods:** Seventy patients with CAH of different variants were subjected to history and clinical examination. Length, weight for length standard deviation scores (SDSs) were calculated at diagnosis and at the end of first year. Laboratory and imaging data were obtained from patients` records.
- **Results:** Length SDS at start of treatment (-0.82±0.1) decreased to -1.06±0.11 at the end of first year (p=0.0001) with adequate weight gain (weight for length SDS -0.41±0.11 at diagnosis versus -0.16±0.13 at the end of first year, p=0.001). Length SDS was significantly reduced from -0.7±0.89 to -1.07±0.9 in salt losing cases (p=0.001) and from -0.78±0.9 to -0.92±0.98 in non- salt losing cases (p=0.031). Better weight gain occurred in salt losing cases. All laboratory parameters decreased while serum sodium rose at the end of first year. The mean hydrocortisone (HC) dose did not differ at diagnosis (14.8±0.4 mg/m^2/day) and at the end of first year (14.98±0.6mg/m^2/day). Patients required lower doses of fludrocortisone (FC) at the end of first year (0.46±0.02mg/m^2/day at

diagnosis versus 0.29±0.01mg/m^2/day at the end of first year, $p<0.001$).

- **Conclusions:** With proper adjustment of therapeutic doses in infants with CAH, linear growth wasn`t accelerated and adequate weight gain occurred at the end of first year.

Table 1. Comparison of clinical, laboratory and therapeutic data at diagnosis and at the end of first year among studied cases (n=70).

	At diagnosis	At end of first year	Z	Mean difference	p
Length SDS	-0.82±0.11	-1.06±0.11	-7.27	-5.13±24.32	**0.0001****
Weight SDS	-0.41±011	-0.16±0.13	-3.26	18.73±25.3	**0.001***
DHEA (µg/dl)	25.88±99	5.9±7.17	-4.373	-55.87±28.13	**0.0001****
Δ4 A (ng/ml)	8.28±5.3	3.42±2.97	-4.88	-57.17±42.47	**0.001***
FT (pg/ml)	9.39±8.14	1.95±3.2	-5.723	-63.57±88.59	**0.0001****
17OHP (ng/ml)	39.45±25.8	12.35±2.13	-5.75	-45.09±10.03	**0.0001****
Na (mEq/L)	134.14±0.86	140.73±0.75	-5.39	5.78±0.89	**0.001***
K (mEq/L)	5.24±0.12	4.7±0.08	4.15*	-8.15±2.66	**0.001***
HC dose (mg/m2/day)	14.77±0.40	14.98±0.6162	-0.474	5.23±4.72	0.636
FC dose (mg/m2/day)	0.46±0.02	0.29±0.01	-5.951	-32.53±3.44	**0.0001*****

Results are expressed as mean±SD and range. SDS: standard deviation score, MPH: mid parental height, *p<0.05, **p<0.01, ***p<0.001, SDS: standard deviation Score, DHEA: dehydroepiandosterone, Δ4 A: Δ4 androstenedione, FT: free testosterone, 17OHP: 17 hydroxy progesterone, Na: sodium, K: potassium, HC: hydrocortisone, FC: fludrocortisone..