Thyroid function in epileptic children using carbamazepine, primidone, phenobarbital and valproic acid

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PAO-1

Two cases of Fanconi-Bickel syndrome - first report from China

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Background: Fanconi-Bickel syndrome (FBS) is a rare autosomal recessive disorder of carbohydrate metabolism caused by mutations in Glut2. To date there is no case has been reported from China.
Objective and hypotheses: To summarize the clinical characteristics of FBS.
Methods: We reported the first two cases of FBS in China. We summarized the clinical characteristics of FBS by reviewing the 2 cases and published literature.
Results: The both cases presented similar manifestations as reported, including severe short stature, hypoglycemia, hepatomegaly secondary to glycogen accumulation, severe glycouremia secondary to proximal renal tubular dysfunction. And more points may help to differentiate FBS and type I glycogen storage disease(GSD I) including glucose intolerance with normal lactic acid and uric acid, possible and slightly glucose response to glucagon stimulation without accumulation of lactic acid, severe symptoms of hypophosphatemia and rickets, and metabolic acidosis caused by type II renal tubular acidosis. After receiving symptomatic treatment both children presented catch-up growth.
Conclusions: FBS is a rare inherited disease caused by mutations in Glut2. It should be carefully differentiated from GSD I and diabetes mellitus in clinical practice. Symptomatic treatment can be helpful.

PAO-2

Final height outcome of boys with central precocious puberty treated with gonadotropin-releasing hormone analogue

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Background: Data on the final height outcome of boys with central precocious puberty treated with gonadotropin-releasing hormoneanalogues (GnRHa) are far less than those in girls.
Objective and hypotheses: To report the final adult height of 20 boys with central precocious puberty treated with gonadotropin-releasing hormone analogue (GnRHa).
Methods: Twenty boys with central precocious puberty treated with GnRHa at a dose of 60~80 μg/kg every 4 weeks for (20.5 +/- 6.7) months. At the beginning of therapy, mean chronological age and bone age was (11.2 +/- 1.0) y and (13.0 +/- 0.4) y, respectively. GnRHa was discontinued when the boys reached the chronological age and bone age of (13.2 +/- 1.1) y and (13.7 +/- 0.6) y, respectively. At the conclusion of the study, all the boys had been followed up for(3.3 +/- 1.5)yrs and had achieved adult height. Comparisons were made among their final adult height (FAH), target height (THt), predicted adult height(PAH)at the start and the end of GnRHa treatment (PAHi and PAHe).
Results: Final height was similar to the target height [(168.6 +/- 5.6)cm versus (167.8 +/- 4.6)cm] with no significant difference from the predicted adult height(PAHi) [(168.6 +/- 5.6)cm versus (169.8 +/- 6.6)cm] based on the Bayley-Pinneau method, using a table for average bone age at the beginning of GnRH analogue therapy. Predicted adult height (PAHe) at discontinuation of GnRHa therapy was significantly higher than predicted adult height at the beginning of GnRH analogue therapy [(172.5 +/- 7.6)cm versus(168.6 +/- 5.6)cm, $P<0.05$]. Ninety percent (90.0%) of the boys reached target height range (FAH>/=THt-1SD). The height gain in comparison with predicted adult height before the start of treatment was (-1.2 +/- 3.3) cm, with the residual growth capacity of (10.6 +/- 4.3)cm.
Conclusions: GnRHa treatment can improve final height into the range of target height in boys with central precocious puberty.

PAO-3

Prevalence of impaired glucose tolerance and insulin resistance among obese children and adolescents

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Background: Obesity is one of the most important nutritional disorders in the world which has an obvious relationship with the incidence of metabolic diseases. Obesity prevalence has increased among children and adolescents during recent decades, leading to a rise in Type 2diabetes mellitus (DM II) prevalence in these two age brackets. Hence, the aim of this study was to assess impaired glucose tolerance and insulin resistance, and gather metabolic findings in obese children and adolescents.
Methods: We studied 110 obese children and adolescents (body mass index. 95th percentile for age and gender) 4–18 years of age referred to the endocrine clinic of the Children's Hospital at Tabriz University in a descriptive cross-sectional study. Fasting glucose, insulin, and lipid profile in all subjects were determined. Oral glucose tolerance test after eating 1.75 g/kg glucose was performed. Homeostatic model assessment was used to estimate insulin resistance.
Results: Impaired glucose tolerance and insulin resistance prevalence in 68 obese adolescents was 14.7% and 31.8%, respectively. Impaired glucose tolerance and insulin resistance was not seen in 23.8% of 42 obese children. No case of DM II was seen. There was a significant statistical difference in glucose ($P = 0.003$) and insulin (P , 0.001) level at minute 120 in individuals with impaired glucose tolerance compared to obese children and adolescents without impaired glucose tolerance. Rate of insulin resistance in patients with impaired glucose tolerance was greater and had a significant statistical difference ($P = 0.03$).
Conclusions: Obesity has a close relationship with increased risk of impaired glucose tolerance and insulin resistance in children and adolescents. Oral glucose tolerance test, unlike fasting glucose test, is a benefit test to predict impaired glucose tolerance. With prompt identification and treatment of obese children with impaired glucose tolerance, we can prevent it from progression towards DM II.

PAO-4

Thyroid function in epileptic children using carbamazepine, primidone, phenobarbital and valproic acid

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Introduction: In this study, we investigated the changes of serum levels of Free T4 and T3, T3 resin uptake (T3RU) and TSH in epileptic children during chronic treatment with anti-epileptic drugs (carbamazepine, Primidone, phenobarbital and valproate) and 3 months later than prescription.
Material and method: This study consisted of four case-series comparisons, was accomplished on 115 (in 4 same groups) epileptic children who were involved 37 girls and 78 boys with ages between 2 months up to 15 years (mean: 62.06 ± 44.97 months), who were taking either phenobarbital (n=29), PRM(n=28), CBZ (n=29), or VPA (n=29) at least for 3 months were evaluated T3, T3 resin uptake (T3RU), T4 and thyroid-stimulating hormone (TSH) levels in start and end of study.
Results: All patients were in euthyroid state, there were no clinical findings or laboratory results of hypothyroidism. In collation with thyroid hormones before of prescription in all bundles (Phenobarbital, CBZ, VPA and primidone), there was no significant distinctions in serum FT3, FT4, T3ru and TSH levels. No statistically meaningful relation were found between thyroid function and thyroid hormons levels variants and among AEDs receiving time and thyroid function and thyroid hormons levels, in any of 4 groups ($P > 0.05$).
Conclusions: Thyroid function should be evaluated intermittently in epileptic children using AEDs specially in long term prescriptions.

PAO-5

Influence of birth brain size on newborn serum insulin-like growth factor-I: role of birth size beyond the presence of intrauterine growth retardation and of preterm birth

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Background: The deviation from the proportionality between brain size and body size at birth has been related to fetal-neonatal stress.
Objective and hypotheses: We evaluated the possibility that preterm birth (PT) and intrauterine growth retardation (SGA) do not completely explain the birth size - related predictor role of birth brain weigth (BRW) on serum Insulin-like Growth Factor-I (IG1) in the human newborn (NWB).
Methods: 78 NWBs 1) free of diabetes mellitus (DM)/life-threatening disease, 2) free of mother with DM and 3) with the availability of all of the following variables were included in the study: gender (SEX), birth gestational age in completed weeks (GA), birth head circumference in cm and birth body weight in gr (resp HC and BW), IG1 measured in ug/dL at one of the first 5 postnatal days (x), 5 days after x (y) and 10 days after x (z) by radioimmunoassay, as well as postnatal age in completed days at x (PNA)(PT definition: GA≤36; SGA definition: BW<10.th centile for GA and SEX; males, n=43; GA range=28-42; PT, n=46; SGA, n=20). BRW was calculated in gr according to the formula " $BRW = 0.037 \times HC^{2.57}$ " (McLennan JE, 1983; Lindley AA, 2000). An estimate of birth body size not represented by brain was obtained by subtracting BRW from BW (BW minus BRW, NBBW). Natural log-transformed IG1 (resp. IG1x-ln, IG1y-ln and IG1z-ln) resulted near-normally distributed. SEX, PT and SGA were dichotomized.
Results: Table 1 shows t value (t), partial correlation coefficient (r) and significance level (p) of partial correlations of BRW with IG1x-ln, IG1y-ln and IG1z-ln, and Multiple Linear Regression (MLR) whole model R2 and p regarding MLR models bearing IG1x-ln, IG1y-ln or IG1z-ln as outcome and, as predictors, either 1) BRW, SEX, PT, SGA and PNA (Table 1A), or 2) BRW, SEX, PT, NBBW and PNA (Table 1B).

Table 1.	vs.	A) IG1x-ln	A) IG1y-ln	A) IG1z-ln	//	B) IG1x-ln	B) IG1y-ln	B) IG1z-ln
BRW	r/t/p	2.506/ .283/a	2.812/ .315/b	2.211/ .252a	//	-0.059/ -.007/ns	0.790/ .093/ns	0.301/ .035/ns
	R2/p	.285/c	.459/c	.302/c	//	.375/c	.486/c	.347/c
Signi-ficances:	a, p<.05;	b, p<.01;	c, p<.001;	ns, not significant.				

Conclusions: A direct BRW relation to IG1 was observed in studied NWBs after controls including PT and SGA, which could be in part explained by peripheral, i.e., not BRW-related, birth size.

PAO-6

Influences of birth brain size on the ratio between newborn serum insulin-like growth factor binding protein-2 and -3: role of birth size after controlling for the presence of intrauterine growth retardation and of preterm birth

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Background: The birth brain size - body size ratio has been related to fetal-neonatal stress.
Objective and hypotheses: The birth size - related predictor role of birth brain weigth (BRW) on the ratio between serum Insulin-like Growth Factor Binding Protein-2 and -3 (resp. IB2 and IB3) in the human newborn (NWB) could be not completely explained by preterm birth (PT) and intrauterine growth retardation (SGA).
Methods: 78 NWBs 1) free of diabetes mellitus (DM)/life-threatening disease, 2) free of mother with DM and 3) with all of the following variables available were included in the study: gender (SEX), birth gestational age in completed weeks (GA), birth head circumference in cm and birth body weight in gr (resp. HC and BW), IB2 and chronologically corresponding IB3 measured in ug/dL at one of the first 5 postnatal days (x), 5 days after x (y) and 10 days after x (z) by radioimmunoassay and postnatal age in completed days at x (PNA)(PT definition: GA≤36; SGA definition: BW<10.th centile for GA and SEX; males, n=43; GA range=28-42; PT, n=46; SGA, n=20). BRW in gr was calculated according to the formula " $BRW = 0.037 \times HC^{2.57}$ " (McLennan JE, 1983; Lindley AA, 2000). An estimate of birth body size not represented by brain was obtained by subtracting BRW from BW (BW minus BRW, NBBW). IB2 was divided by the chronologically corresponding IB3 (IB2/IB3). Natural log-transformed IB2/IB3 (resp. IB2/IB3x-ln, y-ln and z-ln) were near-normally distributed. SEX, PT and SGA were dichotomized.
Results: Table 1 shows t value (t), partial correlation coefficient (r) and significance level (p) of partial correlations of BRW with IB2/IB3x-ln, IB2/IB3y-ln and IB2/IB3z-ln, and Multiple Linear Regression (MLR) whole model R2 and p regarding MLR models bearing IB2/IB3x-ln, IB2/IB3y-ln or IB2/IB3z-ln as outcome and, as predictors, either 1) BRW, SEX, PT, SGA and PNA (Table 1A), or 2) BRW, SEX, PT, NBBW and PNA (Table 1B).

Table 1.	vs.	A) IB2/ IB3x-ln	A) IB2/ IB3y-ln	A) IB2/ IB3z-ln	//	B) IB2/ IB3x-ln	B) IB2/ IB3y-ln	B) IB2/ IB3z-ln
BRW	t/r/p	-2.489/- .281/a	-2.483/- .281/a	-2.946/- .328/b	//	-0.733/- .086/ns	.0.665/- .078/ns	-0.397/- .047/ns
	R2/p	380/c	.405/c	.394/c	//	.403/c	.425/c	.463/c
Signi-ficances:	a, p<.025;	b, p<.005;	c, p<.001;	ns, not significant.				

Conclusions: Inverse BRW relations to IB2/IB3 in studied NWBs after controls including PT and SGA could be in part explained by not BRW-related birth size.