

loss over the 12 months prior to presentation (15 kg). About 10 years before, she had been diagnosed with polymyalgia rheumatica and was treated with corticoids for some periods. Prior to our observation, in the context of headaches, a brain CT was performed revealing a pituitary macroadenoma. Laboratory tests showed hyperkalaemia (6.7 mmol/L) and hyponatremia (129 mmol/L), an elevated ACTH (3353 pg/mL) and low cortisol (9.7 ug/dL). The ACTH stimulation test with tetracosactide was also consistent with primary adrenal insufficiency. Adrenal antibodies were positive and the adrenal CT was normal. There was no evidence of other autoimmune endocrinopathies. Considering the high levels of ACTH and the pituitary lesion, the patient was treated with dexamethasone plus fludrocortisone, leading to clinical improvement and normalization of ACTH. 12 months after, a brain-MRI was done, showing a significant reduction of the pituitary lesion. **Conclusion.** Primary adrenal failure may lead to corticotroph hyperplasia and pituitary adenomas. The aggressive treatment of AD, aiming to achieve a normalization of the ACTH level, can lead to a reduction or remission of the pituitary masses.

KEYWORDS: addison's disease, pituitary adenoma, cortisol.

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PLASMA LEPTIN AND NEUROPEPTIDE Y CONCENTRATION IN PATIENTS WITH IMPAIRED GLUCOSE TOLERANCE

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Background. Leptin, an adipose tissue-derived product of the obesity (OB) gene, is an important regulator of energy metabolism and may be associated with the occurrence of insulin resistance and diabetes in humans. At present the leptin problem in impaired glucose tolerance (IGT) is widely discussed. **Aim.** The aim of the present study was to determine the change of leptin and neuropeptide Y (NPY) levels in patients with IGT. **Material and methods.** 46 patients (20 males, 26 females), mean age $58,2 \pm 13,2$, mean BMI $28,9 \pm 6,47$ kg/m², waist-to-hip ratio (WHR) $0,82 \pm 0,11$. The average fasting plasma glucose (FPG), 2-hour plasma glucose concentrations (2-h PG) following a 75-g oral glucose tolerance test, HbA_{1c}, total cholesterol, triglycerides. Serum leptin and NPY levels were measured by ELISA and results were compared by Statistica 10.0. **Results.** The averages were FPG $7,47 \pm 1,27$ mmol/l, 2-h PG $8,9 \pm 1,2$ mmol/l, HbA_{1c} $6,3 \pm 0,2\%$ total cholesterol $5,6 \pm 0,9$ mmol/l, triglycerides $1,5 \pm 0,7$ mmol/l. In patients with IGT serum leptin levels $22,4 \pm 15,97$ ng/ml, serum NPY levels $0,78 \pm 1,12$ ng/ml. The relationships leptin/NPY $28,7 \pm 14,1$ ng/ml. There was no significant correlation between serum NPY levels and BMI and

WHR, but relationships leptin\NPY correlated with BMI ($r=0.89$; $p<0.05$), HbA_{1c} ($r=0.62$; $p<0.05$), FPG ($r=0.78$; $p<0.05$) and triglycerides ($r=0.74$; $p<0.05$) in patients with IGT. **Conclusions.** These data suggest that the relationships leptin/NPY significant in-creses in patients with impaired glucose tolerance.

KEYWORDS: leptin, neuropeptide Y, impaired glucose tolerance.

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CONGENITAL HYPERINSULINISM IN INFANCY

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Introduction. Congenital hyperinsulinism — is a rare condition, which characterized with inadequate increased insulin secretion and severe hypoglycemia. **Case.** A girl, born at 23.12.16 (1st, delivery by caesarian session at the 35 week of pregnancy). Birth weight 4570 gr., length 55 sm. Due to the presence of severe hypoglycemia (1,4—2,8 mmol/l), prematurity and respiratory insufficiency, she was transferred to the intensive care department. Hormonal evaluation was made: cortisol 197,45 nmol/l (norm 170—720), insulin 321,8 mUE/ml (norm 3—25,5). Sodium and potassium were in normal range. A high glucose levels (13—14 mg/kg/min) and prednisolone (3—6 mg/kg) treatment were given from birth without any effect. After that a child came to the Republican Endocrinological Center in the intensive care department in severe condition due to hypoglycemia. Additional examination of computed tomography was made: diffuse hyperplasia of left adrenal, hepatomegaly, symptoms of spina bifida Th 11,L5—S2. Due to the clinical-laboratory features a clinical diagnosis was made: congenital hyperinsulinism, prematurity (35 weeks gestational age). Intravenous glucose titration (1 g/kg) and octreotide (45 mg/day with increasing 240 mg/day due to the presence of hypoglycemia). Despite this treatment glucose levels were low, so treatment with diazoxide 75 mg/day (instead of octreotide) was started. On this dosage child had severe vomiting (was diazoxide treatment side effect), that's why we lowered dosage to 50 mg/day and gave antireflux food and domperidone to her. Hormonal evaluation: insulin 33,8 mUE/ml (norm 3—25,5), C-peptide 5,05 ng/ml (norm 1,1—4,4). Genetical evaluation: no mutation was determined (50% of congenital hyperinsulinism cases didn't evaluate any mutations due to genetical testing). A child was discharged from the hospital with weight 4850 g, length 57 sm. She takes now diazoxide 50 mg/day, eat antireflux food and need in subsequent observations by pediatric endocrinologist. **Conclusions.** This case demonstrates the importance of timely diagnosis and treatment of congenital hyperinsulinism in early in-