A CASE REPORT OF PITUITARY GIGANTISM OF 27-YEAR-OLD MALE PATIENT IN CHELYABINSK REGION OF RUSSIA

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Pituitary gigantism is a rare disorder. It refers to growth hormone (GH) excess that occurs before fusion of the epiphyseal growth plates. We report a 27-year-old male patient with a pituitary macroadenoma who underwent transsphenoidal surgery in 2004 at the age of 15 with the height up to 215 cm. He had history of visual impairment and severe headache. The patient's condition improved after the surgery but GH and insulin-like growth factor (IGF-I) levels did not normalize; as a consequence, he was referred for postoperative somatostatin analogue injection (30 mg per 28 days) with poor response. He continued to grow. In 2014 his height and body weight were approximately 235 cm and 142 kg, respectively, with a BMI of 25,7 kg/m^2. The concentration of plasma GH and IGF-1 levels maintained a high level, which were 15.67 ng/mL and 408 ng/mL, respectively. Pituitary magnetic resonance imaging (MRI) revealed macroadenoma 33×28×23 mm without negative dynamic compared with 2008 year sizes 23×31×28 mm. The patient suffered from back pain, restriction of movement because of difference in length of the legs. Valgus deformation of knee joints was detected. For this reason he decided to undergo surgery in traumatology department in 11.2014 before he stabilized GH secretion. Osteosynthesis of the left hip with the extension apparatus, osteology of the femur in the distal third (clinoid resection of the femur) were performed. Postoperative period was more then 3 months and patient walks with crutches till nowadays. He has edema of the left ankle experienced over a 1-year period and the huge strained left knee. In this consideration, he had a disability and impossibility to move. He needs more examinations in the National Research Center for Endocrinology. In conclusion all surgical manipulation should be provided after achieving the target level of GH and IGF-I secretion.

KEYWORDS: pituitary gigantism, case report.

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A CASE OF ASSOCIATION OF TYPE 1 DIABETES MELLITUS AND PRECOCIOUS PUBERTY

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Introduction. The processes of intensive growth and puberty are regulated by growth hormone and sex hormones, which are contrinsular. During the pubertal period the metabolic control is getting worse in the most of patients with type 1 diabetes mellitus (T1DM). One of the reasons of bad diabetes control at prepubertal children can be precocious puberty. **Case report.** A 4-year-old girl was diagnosed with T1DM 6 months ago. The daily dose of insulin was 0.5 units/kg. But during the last month glycemic control become worse and she was admitted to hospital for treatment correction. The daily dose of insulin increased to 1 units/kg and glucose levels according to continuous glucose monitoring system (CGMS) were very variable (from 2,9 to17,7 mmol/L). HbA_{1c} level was 10,1%. On examination, girl was found to have Tanner stage 2 breast development. There was no axillary or pubic hair. Her height was above the 90th percentile (6 months ago it was above 50th percentile, familial target height – 50th percentile). Bone age was 4 years and 6 months (Greulich Pyele). There was no thyroid swelling, café au lait spots or any bone abnormality. Her investigations showed normal hemogram, liver and renal functions. Thyroid functions were normal. Her basal hormonal profile was as follows: LH 0,133 mIU/ml, FSH 1,67 mIU/ml. On ultrasonography, increase in ovarian volumes (bilateral) and uterus was found (uterus measured 35×7,8×12,5 mm, OS — 20×10 mm, OD — 27×15 mm). On the GnRH stimulation test, the peak LH and FSH levels were 10 times higher than basal levels, which was compatible with a diagnosis of central precocious puberty. Brain magnetic resonance imaging (MRI) was performed and the organic cause of precocious puberty was excluded. **Conclusion.** Association of T1DM with precocious puberty is rare. But in the case of an unexplained severe course of the disease, this reason must also be considered.

KEYWORDS: diabetes mellitus type 1, case report, precocious puberty.

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PITUITARY MACROADENOMA IN ADDISON'S DISEASE

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Introduction. Long-standing primary failure of pituitary-dependent endocrine glands may lead to hyperplasia of the pituitary cells. Particularly, primary adrenal failure may predispose to corticotroph hyperplasia and in some patients to the development of corticotroph adenoma. We describe a rare case of a pituitary macroadenoma in a patient diagnosed with Addison's disease (AD). **Case report.** A 57-year-old female presented to the endocrinology outpatient department with complaints of weakness, dizziness and easy fatigability, nausea with occasional vomiting and darkening of the skin in the last 3 years. She also noticed progressive weight

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 NEYROPEPTIDE 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±13,2, mean BMI 28,9 ±6,47 kg/m^2, waist-to-hip ratio (WHR) 0,82±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±1,27 mmol/l, 2-h PG 8,9±1,2 mmol/l, HbA_{1c} 6,3±0,2% total cholesterol 5,6±0,9 mmol/l, triglycerides 1,5±0,7 mmol/l. In patients with IGT serum leptin levels 22,4 ±15,97 ng/ml, serum NPY levels 0,78±1,12 ng/ml. The relationships leptin/NPY 28,7±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-creases in patients with impaired glucose tolerance.

KEYWORDS: leptin, neyropeptide 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-