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-

fancy. The most severe complication of this condition is brain injury due to severe hypoglycemia.

KEYWORDS: hyperinsulinism, hypoglycemia, infancy.

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THE INFLUENCE OF DPP-4 INHIBITORS ON FAT METABOLISM IN TYPE 2 DIABETES PATIENTS

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Background. To evaluate the effect of sitagliptin in combination with metformin on glucose toxicity and lipotoxicity in patients with type 2 diabetes and obesity. **Material and methods.** The study involved 82 patients (55 women, 27 men, mean age 56,1±5,47 years) with obesity, lipid metabolism disorders, who have not reached target levels HbA_{1c} (average HbA_{1c} 8,3±1,6%) after metformin and diet therapy. The first group of patients (42 patients) received co-formulated drug, consisting of sitagliptin 100mg and metformin 2g once a day; the second comparison group (40 patients) received metformin 1.5—2.0 g/day. Dynamics of fasting glycemia, postprandial glycemia, glycated hemoglobin, weight, BMI, WC, WHR, lipid profile (total cholesterol, triglycerides, LDL, HDL, apoβ protein), insulin, proinsulin, leptin, adiponectin, insulin resistance using the index HOMA IR and functional activity of β-cells (by HOMA-β index) was evaluated at baseline and at 6 months of therapy. In addition, MRI was performed to assess visceral fat in all the patients. **Results.** At 6 months patients in both groups demonstrated significant positive changes in the levels of fasting glucose, postprandial glycemia and glycosylated hemoglobin. In group I, HbA_{1c} decreased from 8.3±1.6 to 6.6±1.24% (p<0.01), in group II there was a decrease from 8.35±1.75 to 7.62±1.39% (p<0.01). FPG and late products of glycosylation levels in group I reduced on average by 2.67 and 3.3 mmol/L, correspondingly, in group II by 2.1 and 1.8 mmol/l. No significant differences in the dynamics of total cholesterol, HDL between the groups were found. LDL in group I lowered by 0.7 mmol/l, in group II by 0.3 mmol/l (p<0.05); in group I, TG decreased by 1.33 mmol/l. in group II by 0, 63 mmol/l (p<0.05); in group I IRI reduction was 3.45 mcU/ml in group II 1.63 mcU/ml (p=0.05). Proinsulin level dropped down in group I by 2.93±3.02, in group II by 1.26±1.1, C-peptide level increased by 1.4±1.6 ng/ml, in group II 0.16±0.1 ng/ml, HOMA-β grew up in group I by 23.4 standard units, in group II by 4.8 standard units (p<0.005). The ratio of proinsulin/insulin dropped down in group I by 0.19±0.7, in group II by 0.02±0.2. There were no significant differences between the groups in the dynamics of HOMA IR and both groups demonstrated positive dynamics. Adiponectin levels were different between the groups, there was an increase by 1.9 ng/ml in group I, in group II by 0.49 ng/ml. (p<0.01). Leptin lowered by 7.37 ng/ml in group I, in group II by 1.21 ng/ml (p<0.01). Also groups showed dramatic difference in anthropometric parameters dynamics (p<0,001). Average weight loss was 4.9±3.2 kg in group I, in group II 2.0±0.94 kg correspondingly. BMI in group I decreased by 1.8±1.3, in group II by 0.68±0.3. WC shortened by 6.5±4.7 sm in group I, in group II by 2.42±1.02 sm. WHR in group I decreased from 0.95±0.06 sm to 0.91±0.05 sm, in group II from 0.94±0.03 sm to 0.93±0.03 sm respectively. Also MRI showed a significant reduction of visceral fat area by 20.6±13.5 sm^2 (7.5%) in group I, compared to group II with 5.7±3.75 sm^2 reduction(1.76%; p<0.01), while in the dynamics of the area of the subcutaneous fat there is no reliable dynamics between groups. Episodes of hypoglycemia have not been registered in any of the groups during the treatment. **Conclusion.** The administration of sitagliptin and metformin decreased glucose toxicity and lipotoxicity that generally led to the improvement of glycemic control.

KEYWORDS: DPP-4 inhibitors, type 2 diabetes mellitus, obesity.

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THE IMPACT OF LOCAL NEGATIVE PRESSURE WOUND THERAPY ON TISSUE REPAIR PROCESSES IN PATIENTS WITH DIABETIC FOOT ULCES

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Aim. To evaluate clinical, histological and immunohistochemical effects of NPWT in comparison to standard management in diabetic foot ulcers (DFUs). **Material and methods.** Clinical examination, transcutaneous oxygen monitoring, ulcer biopsies (haemotoxylin-eosin and immunohistochemistry for CD68 (macrophages), MMP-9 and TIMP-1(proteolytic activity), CD31 (vessels) before and after local treatment. **Results.** 42 patients were enrolled (28 men; 14 women) with DFUs after surgical debridement and divided into 2groups. Group1 (n=21) was treated with NPWT (−90—120 mm Hg), group 2 (n=21) was treated with atraumatic dressings for 9±2 days. The groups matched by DM type, age (group 1 60 (52; 64), group 2 60 (57; 72) years), HbA_{1c} in group1 8.8% (7.4; 10.6%), in group 2 8.8% (7.6; 9.7%), severity of microvascular complications, form of diabetic foot (neuropathic — 41, neuroischemic-1(after revascularization)), wound size (group 1 — 25,0 (16,2; 44,5) cm^2, group 2 — 23.5 (12.3; 55.3) cm^2, wound depth (group 1 — 3,3 (1,5; 6,5) cm, group 2 —3.2 (2.4; 5.2) cm), $tcpO_2$ (group 1 46 (38; 52) mm Hg; group 2 — 43 (38; 47) mm Hg; p>0.05). Histologically both groups presented edema, poorly organized extracellular matrix (ECM), small quantity of fibroblast-like cells and severe inflammation (p>0.05). Immunohistochemically: increased staining of