

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} $8.3 \pm 1.6\%$) 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, apoB 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 $6.6 \pm 1.24\%$ ($p < 0.01$), in group II there was a decrease from 8.35 ± 1.75 to $7.62 \pm 1.39\%$ ($p < 0.01$). 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 cm in group I, in group II by 2.42 ± 1.02 cm. WHR in group I decreased from 0.95 ± 0.06 to 0.91 ± 0.05 , in group II from 0.94 ± 0.03 to 0.93 ± 0.03 respectively. Also MRI showed a significant reduction of visceral fat area by 20.6 ± 13.5 cm² (7.5%) in group I, compared to group II with 5.7 ± 3.75 cm² 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 (haematoxylin-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 2 groups. Group 1 ($n = 21$) 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 group 1 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) cm², group 2 — 23.5 (12.3; 55.3) cm², wound depth (group 1 — 3.3 (1.5; 6.5) cm, group 2 — 3.2 (2.4; 5.2) cm), tcpO₂ (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