MORPHOFUNCTIONAL CHANGES IN THE ENDOCRINE PANCREAS IN EXPERIMENTAL METABOLIC SYNDROME

Ergashev Sukhrob Tokhir ugli
Tashkent State Medical University
suhrobergashev955@gmail.com

Annotatsiya. *Tadqiqotda 60 ta kalamushda 90 kunlik yuqori yog‘li-fruktozali ratsion bilan chaqirilgan metabolik sindromda oshqozon osti bezining endokrin qismi o‘rganildi. Langergans orolchalarining dastlabki kompensator giperplaziyasi 90-kunga kelib atrofiya, β-hujayralar distrofiyasi va fibroz bilan almashindi.*

Kalit so‘zlar: *metabolik sindrom, Langergans orolchalari, β-hujayralar, morfometriya, fibroz.*

Аннотация. *Изучена эндокринная часть поджелудочной железы 60 крыс при метаболическом синдроме, вызванном 90-дневным высокожировым фруктозным рационом. Начальная компенсаторная гиперплазия островков Лангерганса к 90-м суткам сменялась атрофией, дистрофией β-клеток и фиброзом.*

Ключевые слова: *метаболический синдром, островки Лангерганса, β-клетки, морфометрия, фиброз.*

Abstract. *The endocrine pancreas was studied in 60 rats with metabolic syndrome induced by a 90-day high-fat, high-fructose diet. Early compensatory hyperplasia of the islets of Langerhans was replaced by atrophy, β-cell degeneration and fibrosis by day 90.*

Keywords: *metabolic syndrome, islets of Langerhans, β-cells, morphometry, fibrosis.*

Introduction. Metabolic syndrome (MS) substantially increases the risk of type 2 diabetes mellitus (T2DM), whose prevalence continues to grow worldwide [1]. Insulin resistance and β-cell dysfunction are the key links between MS and T2DM [2, 3], yet morphological data on the endocrine pancreas in the prediabetic stage remain scarce. The aim of this study was to assess the dynamics of morphofunctional changes in the endocrine pancreas in experimental MS.

Materials and methods. Sixty male white rats (180–200 g) were divided into a control group (n = 20) and an experimental group (n = 40). For 90 days, the experimental rats received a diet of 60% standard chow and 20% mutton fat with a 20% fructose solution as drinking water. On days 30, 60 and 90, organometry, histology (hematoxylin and eosin, van Gieson), digital morphometry (QuPath, ImageJ) and biochemical tests (glucose, insulin, lipids, HOMA-IR) were performed. Differences were assessed with Student's t-test ($p < 0.05$).

Results and discussion. By day 90, body weight increased by 22.8%, blood glucose by 2.16 times and LDL by 2.5 times, while insulin reached 27.9 ± 1.3 μU/mL and HOMA-IR exceeded 2.5. Pancreatic weight changed in two phases: it rose on day 30 (0.90 ± 0.07 g versus 0.82 ± 0.06 g, $p < 0.05$) and fell by day 90 (0.70 ± 0.05 g versus 0.86 ± 0.06 g, $p < 0.01$). On day 30, the islets were enlarged owing to compensatory hyperplasia, and capillaries were congested. By day 60, peri-insular fibrosis, arteriolar hyalinosis and perivascular edema had developed. On day 90, β-cells showed degranulation, vacuolization and karyopyknosis, the islet area decreased to 62–74% of the control value, and interstitial connective tissue rose from 8–12% to 24–31%. This sequence is consistent with the concept of stepwise β-cell failure [3–5]. Based on these data, the SEMA web application (certificate No. DGU 59406) was developed for the early prediction of T2DM risk.

Conclusion. Experimental MS causes a biphasic, initially compensatory and then decompensatory, remodeling of the endocrine pancreas. Karyopyknosis and vacuolar degeneration of β-cells, a reduced islet area and stromal fibrosis may serve as morphological criteria for predicting T2DM.

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