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MORPHOLOGICAL RECONSTRUCTION OF THE HEMOMICROCIRCULATORY BED OF THE MASTICATORY MUSCLE IN DIABETES MELLITUS UNDER FOLLOWING A SINGLE STRESS IMPACT

МОРФОЛОГІЧНА ПЕРЕБУДОВА ГЕМОМІКРОЦИРКУЛЯТОРНОГО РУСЛА ЖУВАЛЬНОГО М'ЯЗА ПРИ ЦУКРОВОМУ ДІАБЕТІ ЗА УМОВИ РАЗОВОГО ВЛИВУ СТРЕСОВОГО ФАКТОРА

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The masticatory muscles possess a complex anatomical structure that reflects their function and the functional characteristics of the masticatory apparatus. As is well established, masticatory loads represent one of the principal mechanical factors in the morphogenesis of the oral cavity organs and the maxillofacial region [1]. Maintaining myodynamic balance between antagonist and synergist muscles provides the conditions for the normal development of the dentoalveolar system in general, as well as for the formation of various occlusal patterns [2–4]. The predominance of functional activity between the masseter and temporalis muscle pairs during mastication (masseteric or temporal type of mastication) to some extent determines the growth direction of the mandible [5–7]. It is well known that skeletal muscles represent one of the major tissues regulating carbohydrate metabolism through the activation of the insulin-sensitive glucose transporter (GLUT-4), while impaired glucose uptake by the muscles may lead to insulin resistance and the development of type 2 diabetes mellitus

[8–9]. Moreover, in cases of uncontrolled diabetes mellitus, one of the complications is diabetic myopathy, which markedly reduces the quality of life of affected patients.

Based on the above-mentioned, the goal of our study is the investigation how streptozotocin-induced diabetes mellitus (SIDM) and stress affected of morphological changes in the hemomicrocirculatory bed (HMCB) of masticatory muscle.

Material and Methods. The study used 20 adult white laboratory rats (body weight 180-200 g), which were equally divided into 4 groups: group 1 – rats with simulated SIDM and immobilization stress (IS), group 2 – rats with SIDM, group 3 – rats with IS, group 4 – control animals. In groups 1 and 2, SIDM was simulated by a single intraperitoneal injection of streptozotocin "SIGMA" (USA), which was diluted in 0.1 M citrate buffer with a pH of 4.5 (at the rate of 6 mg per 100 g of body weight). In groups 1 and 3, immobilization stress was simulated by placing the animals in a closed plastic container for 5 hours a day. In group 1, SDM was simulated and starting from the 14^{th} day of the experiment IS was simulated on a once-only basis. The material was collected on the 14th day from the start of the experiment. Groups 1 and 2 included rats with glucose levels of at least 13 mmol/L. Histological, electron microscopic and statistical research methods were used. Morphometry was performed using ImageJ version 1.47t. Statistical analysis was performed using the statistical package Stat.Soft.Inc; Tulsa, OK, USA; Statistica 12.

Result. After a single exposure to stress and metabolic alterations in rats with streptozotocin-induced diabetes mellitus (groups 1–3), arteriolar spasm was observed, as evidenced by a significant reduction in their area by 14%–12%–9%, due to a decrease in lumen size by 41%–40%–30%, respectively, compared with the control (in all cases, $p < 0.05$). Such remodeling of the afferent segment of the haemomicrocirculatory bed (HMCB) results in a reduction of its flow capacity. No quantitative changes in capillaries and venules were detected. However, the lumina of capillaries and venules in groups 1 and 2 were filled with formed blood elements and frequently contained erythrocyte sludge.

At the ultrastructural level, in the microvessels of the HMCB of the masseter muscle in groups 1 and 3, most arterioles displayed a slit-like lumen due to spasm. In the endothelial cells of capillaries in groups 1–3, karyopyknosis and an increased number of micropinocytotic vesicles in the cytoplasm were observed, suggesting enhanced transendothelial exchange. In the peripheral zones of some capillary endothelial cells, medium-sized vacuoles and slight protrusions of the luminal plasmalemmal surface into the capillary lumen were evident. Notably, in certain endothelial cells, the electron density of the cytoplasm was reduced, while in others it was

increased, with abundant micropinocytotic vesicles, indicating activation of transendothelial transport.

A distinctive feature of HMCB remodeling in the masseter muscle of groups 1 and 2 was the presence of haemorrheological disturbances in all vessel types, including erythrocyte aggregation, adhesion of erythrocytes and platelets to the luminal surface of endotheliocytes, microthrombus formation, and development of micro- and macroclasmotic changes. In some pericytes of these groups, signs of vacuolar degeneration were observed.

Conclusions. Streptozotocin-induced diabetes mellitus combined with stress leads to impaired microcirculation in the masseter muscle due to reduced flow capacity of the afferent portion of the HMCB. The most pronounced alterations were recorded in group 1 rats with comorbid pathology (SIDM + IS). In these animals, arteriolar spasm was accompanied by severe haemorrheological disturbances in capillaries and venules, further compromising blood supply to the masseter muscle and resulting in hypoxia.

Bibliography:

1. Koshkin O.Y. Features of structural reorganization of neuro-muscular junctions of masticatory and temporal muscles in the postnatal period of ontogenesis.. *Art of Medicine*. 2020. № 3. P. 106–112.

2. Saldarriaga A., Rojas-Gualdrón D., Restrepo M., Santos-Pinto L,. Jeremias F. Dental fluorosis severity in children 8–12 years old and associated factors. *Acta Odontol Latinoam.* 2021. Vol. 34, № 2. P. 156–165.

3. Banasr F. H., Alammari M. R. A novel bio-sensor for registration of biting force in occlusally reactive single mandibular implant overdenture. *Open Journal of Stomatology.* 2013. № 3. P. 370–378.

4. Cui G., Zhang H., Ma C., Zhang X., Shao H. Experimental study on optimization of pipe jacking mud mixture ratio based on MICP technology. *Sci Rep.* 2024. Vol. 14, 12. P. 31459.

5. Sharma N. K., Yadav B. S., Hirani M. S., Dhiman N. K., Singh A. K., Tripathi R. Electromyographic Assessment of Masticatory Muscles & their Asymmetries in Adult Indian Population. *J Maxillofac Oral Surg.* 2024. Vol. 23, № 1. P. 197–203.

6. Nagarajan A., Sinha S.K. Masseter Vestibular evoked myogenic potentials: A new tool to assess the vestibulomasseteric reflex pathway. *J Otol.* 2024. Vol.19, № 1. P. 46–54.

7. Wong Z. Y., Ou K. Q., Ingels KJAO., van Heerbeek N., Pouwels S. Variation and distribution in temporalis muscle innervation: A systematic review and meta-analysis of anatomical studies. *JPRAS Open*. 2024. Vol. 43, №1. P. 122–132.

8. Merz K. E., Thurmond D. C. Role of Skeletal Muscle in Insulin Resistance and Glucose Uptake. *Compr Physiol*. 2020. Vol. 10, № 3. P. 785.

9. DeFronzo R. A., Tripathy D. Skeletal Muscle Insulin Resistance Is the Primary Defect in Type 2 Diabetes. *Diabetes Care.* 2009. Vol. 1, № 32(suppl_2). P. 157–163.