GROWTH FACTORS AND APOPTOSIS IN SALIVARY GLANDS OF EXPERIMENTAL RABBITS AFTER LONG LASTING LIGATION OF *A. CAROTIS COMMUNIS*

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Introduction: The secretory activity and quantitative morphological changes of salivary glands depends upon blood supply. The secretion demands a satisfactory blood delivery. If blood supply is obstructed, ischemic disease of maxillofacial region might appear.

Aim: The aim of this study was to detect morphological and imunhistochemical changes in salivary glands after simulated ischemia in *a. carotis communis* reservoir, in experimental model of rabbit.

Materials and methods: Ten experimental rabbits were used in this study. In all animals ligature of *a. carotis communis dextra* was performed by silk suturing material under general anesthesia with Diazepam 2% 2mg/kg for induction and 5% Ketamin hydrochloride 15mg/kg for narcosis injected intra venously by help of local anesthesia infiltrated with 2% Lidocain solution. After ligature wound were sutured by Vycril suturing material.

After 28 days rabbits were sacrificed with overdosage of anesthesia, and tissue examples from both (control and experimental) sides of parotid, buccal and submandibular glands were obtained. The examples were fixed in Sol. *Stefanini,* embedded in paraffin blocks, cut in 5 μm thick slices and performed for staining with Nerve growth factor receptors (NGFR, 1:120, DakoCytomatin, Denmark), Vascular endothelial growth factor (VEGF, 1:50, DakoCytomatin, Denmark), Epidermal growth factor receptors 1 (EGFR1, 1:200, DakoCytomatin, Denmark), Fibroblast growth factor receptors 1 (FGFR1, 1:100, Abcam, UK) by use of immunohistochemistry *(Hsu et al., 1981)* and for routine stainig with Hematoxilin and eosin. Also apoptotic changes in salivary glands were detected by TUNEL method (Negoesku et al., 1998).

Results: Buccal glands showed hypertrophy and degeneration of secretory parts, but submandibular and parotic glands demonstrated proliferation of excretory ducts and fibrosis.

Apoptosis affected cell mean number in salivary glands was as following: in buccal - 40±24.66, in submandibular - 51±15.01, in parotic - 43±15.32. VEGF marked few to moderate endotheliocytes in salivary glands, but NGFR stained moderate number of nerves in salivary glands around the excretory ducts. EGFR1 mainly was found in blood vessels of submandibular gland. FGFR1 richly stained excretory ducts in all salivary glands.

Conclusions: Dystrophy raised by carotid artery ligation occurs in salivary glands in way of changed structure of excretory ducts and hypertrophy of secretory parts. Compensation after long lasting regional ischemy are sclerotization of blood vessels, regional proliferation of nerves and fibrosis in salivary glands. EGFR1 and FGFR1 are main growth factors found in ischemic salivary glands. Apoptotic changes rised by ischemia affects secretory and excretory duct cells of small and main salivary glands in approximately equal number.