Summary

Prognostic and predictive markers of the head and neck tumors

Introduction. Head and neck squamous cell carcinoma (HNSCC) is aggressive tumor with unfavorable prognosis despite the improved therapy in the last decades. HNSCC with its clinicopathological and biological heterogeneity remains difficult to be clinically managed. Therefore, a better understanding its molecular pathobiology is required. The aim of our studies was to analyze the expression profile and possible prognostic and predictive role of selected matrix metalloproteinases (MMP), microRNAs (miR) and proteins p16, p53 and galectin-3 in HNSCC with respect to tumor location (oropharynx, larynx, hypopharynx) and clinicopathological parameters.

Material and Methods. Three studies were performed (1. study: prospective, 46 patients, analysis of preoperative serum levels of MMP-1, MMP-2 and MMP-9 by multiplex method MAGPIX (Luminex, TX, USA); 2. study: retrospective, 58 patients, immunohistochemical analysis of p16, p53 and galectin-3; 3. study: retrospective, 51 patients, analysis of tissue let-7a, miR-21, miR-34a, miR-200c, and miR-375 by real-time quantitative PCR method.

Results. Statistically significant differences in serum levels of MMPs between cancers of different locations were not found. Significant correlations were confirmed between p16 positivity and oropharyngeal cancer, MMP1 and p16 positivity, and recurrence and smoking. Compared to non-neoplastic tissue, miR-21, miR-200c, miR-34a were up-regulated and miR-375 was down-regulated in tumors of all sites. Significant differences of let-7a, miR-200c, miR-34a levels between oropharyngeal and laryngeal cancers were found ($p<0.05$). MiR-34a significantly correlated with oropharyngeal origin ($p=0.0284$) and p16 positivity ($p=0.0218$) and this upregulation has not been so far described.

Conclusions. MMP-1,-2,-9 and deregulated miR-21, miR-200c, and miR-375 can be considered as factor regulating pathobiology, progression and spreading of HNSCC irrespective to tumor location. Let-7a and miR-34a expression support hypothesis on site-specific cancerogenesis in HNSCC.