Editorial

Role of miRNA in the malignant transformation of oral lichen planus

Recent evidence suggests that a substantial number of molecular changes in oral potentially malignant disorders (OPMDs) and oral cancers are regulated by microRNAs (miRNAs). miRNAs are small noncoding RNAs modulating cellular proliferation, differentiation, and apoptosis. Although protein coding is not a direct function of miRNAs, they act as posttranscriptional regulators controlling the expression of a more than 25% of protein-coding genes in humans. miRNAs bind to its target mRNA's in a sequence-specific manner, thus modulating the translation of hundreds of genes by degrading or inhibiting its target mRNA. Deregulation of a specific miRNAs may lead to an increased expression of an oncogene or tumor suppressor.[1-3] Such miRNAs have been demonstrated in OPMDs and oral cancer, stressing its role in tumour initiation and progression. Their small size and potential for affecting the expression of thousands of genes makes it an ideal biomarker in the diagnoses, treatment and prognosis of OPMD'S and Oral cancer. By defining the regulatory miRNA networks in these lesions, it may be possible to address the controversies pertaining to the molecular biology of such lesions. Studies implicating the association of miRNA signature with OPMD's and oral cancer are limited. Due to the clinical and histological prognostic limitations it is vital to establish a genetic marker prediction the course of OPMDs. miRNAs provides an ideal environment for cancer initiation and progression. Its role in tumorigenesis is due to a synergistic deregulation between multiple miRNAs and their protein-coding counterparts.[1-3] Oral lichen planus (OLP) represents a chronic mucosal disease AND its classification under OPMDs has been a subject of debate for the past few decades.[4] Follow- up studies on the progression of OLP has yielded support in favor of malignant transformation, yet critical molecular data elude its confirmation. Cervigne et al[5] showed significant variation in the miRNA signature differentiating OSCC and progressive leukoplakia from nonprogressive leukoplakia. Study conducted by Gassling et al[6] on miRNA-mRNA networks associated with in OLP confirmed miRNA as a potential modulator in the pathophysiology of OLP. Dang et al[4] compared the miRNA-137 promoter methylation in OLP and oral squamous cell carcinoma with normal oral mucosa. The study group included cases of OLP with no dysplasia. The results showed significant difference between healthy controls and patients with OLP. Further, the promoter methylation of OLP was 35% in comparison to 58.3% in OSCC patients suggesting methylation to be a common event in both these entities. Our current knowledge in understanding the intricate functioning of miRNAs is incomplete. Follow-up studies evaluating miRNA signature in OLP by sequential sampling will aid us in validating its malignant potential. Further, establishing such miRNA signature databases may serve as therapeutic targets in the future.

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