SDM
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"CELL CYCLE ABERRATION IN AMELOBLASTOMA AND ADENOMATOID ODONTOGENIC TUMOUR: AS EVIDENCED BY THE EXPRESSION OF p53 AND SURVIVIN"

by

Dr. ZULFIN SHAIKH

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Dr. NIRANJAN K C

DEPARTMENT OF ORAL PATHOLOGY & MICROBIOLOGY
S.D.M. COLLEGE OF DENTAL SCIENCES & HOSPITAL
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ABSTRACT

Background and Objective:

Tumour suppressor genes normally act as regulators of cell growth and apoptosis. Inactivation of these genes by mutation and/or loss of heterozygosity (LOH) in both alleles results in tumour development. Evasion from apoptosis by aberrations of apoptotic regulatory factors has been found to cause accumulation of neoplastic cells in various tumours. The aim of this study was to correlate the expression of p53, a tumour suppressor gene involved in cell cycle and Survivin, a bifunctional protein in Ameloblastoma and Adenomatoid odontogenic tumour (AOT).

Methods:

Qualitative and quantitative assessment of p53 and Survivin expression was evaluated in a total of 60 cases which included 10 tooth germs, 25 cases each of histologically diagnosed Ameloblastoma and Adenomatoid odontogenic tumour. The percentage of positive tumour cells was considered for statistical evaluation. Nuclear labelling index for p53 and nuclear, cytoplasmic and combined labelling index for Survivin was obtained from the stained slides.

Results:

p53 and Survivin expression in tooth germs had an average percentage of 24.89% and 29.1% respectively. In Ameloblastoma, the expression of p53 mainly involved the peripheral ameloblast-like cells whereas Survivin was expressed in both peripheral and central stellate reticulum-like cells with average percentage of 75.97% and 78.25% respectively. In AOT, the expression of p53 and Survivin was seen in whorls, duct-like areas and rosettes with an average percentage of 69.87% and 62.94% respectively. A positive correlation was found between p53 and Survivin in tooth germs, Ameloblastoma and AOT **(r=0.793, p=0.006; r=0.251, p=0.226;**

r=0.846, p=0.000 respectively**).** Qualitatively, statistical significant difference was seen in the expression of p53 and Survivin in Ameloblastoma **(p=0.004)** but no significant difference was seen in tooth germ and Adenomatoid odontogenic tumour **(p=0.705 & p=0.059)**. Quantitatively, statistically significant difference was seen in the expression of p53 and Survivin in AOT **(p=0.003)** but not in tooth germ and Ameloblastoma **(p=0.499 & p=0.411).** A significant difference was seen in the percentage of positive cells expressing Survivin between Ameloblastoma and Adenomatoid odontogenic tumour (AOT) but no such significance was observed in the expression of p53 **(p=0.002 and p=0.554** respectively). No significant difference was seen qualitatively in the expression of p53 and Survivin between Ameloblastoma and Adenomatoid odontogenic tumour (AOT) **(p=0.337 & p=0.547** respectively).

Conclusion:

There was no much difference in p53 expression in Ameloblastoma and AOT suggestive of cell cycle aberration in both the odontogenic tumours, but significant difference in Survivin expression in Ameloblastoma and AOT with higher percentage of positive cells in Ameloblastoma may be indicative of an aggressive behaviour of Ameloblastoma. This study suggests an association between p53 and Survivin in the pathogenesis of both the tumours.

Keywords: Survivin; p53; Ameloblastoma; Adenomatoid odontogenic tumour; Immunohistochemical expression