P53 AND P73 EXPRESSION IN OVARIAN NEOPLASMS

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P73 is a new tumor suppressor gene which shares a high degree of structural similarity with the P53 gene and plays an important role in the development of human cancers including ovarian carcinomas. Overexpression of P73 can activate typical P53-responsive genes, and induce apoptosis like P53. The relationships between P53 and P73 expression in ovarian carcinomas have rarely been studied. The aim of the study was the immunohistochemical detection of the expression of P53 and P73 proteins in ovarian neoplasms, taking into account the clinico-pathological parameters. The mutual relationships between the studied markers were also analysed. P53 expression was detected in 54.1% of ovarian carcinomas and was undetectable in benign neoplasms. P73 expression was found in 48.6% of the malignant and 50.0% of the benign ovarian neoplasms. No clear differences between the expression of the two markers and the histological type of the neoplasm, the grade of differentiation of carcinomas and the clinical stage of the disease were found. However, the presence of P73 was observed more frequently in poorly differentiated G3 (77.8%) than in well/moderately-differentiated G1/G2 carcinomas (40.9%). There was no correlation between P73 and P53 overexpression, and co-expression of both markers was only found in 29.7% of the ovarian carcinomas. The coexpression of P53 and P73 was mainly observed in poorly-differentiated carcinomas and in the I/II FIGO stages. Independently of P53 and P73 expression, the progression of the disease was observed more frequently in patients with III/IV than with I/II FIGO stages. The presence of P73 overexpression in benign ovarian neoplasms indicates that P73 changes may play an important role in the development of ovarian neoplasms. However, these data suggest that there is a need for further analysis of the role of P73 in ovarian carcinogenesis. Our data also suggest that the co-expression of P53 and P73 proteins may be associated with biological tumor behaviour.