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p53 and bcl-xl - apoptotic markers predicting axillary recurrence following axillary radiotherapy in carcinoma of breast

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Introduction: Management of node-positive axilla from breast cancer remains controversial, axillary radiotherapy, axillary clearance being the two options. Specific molecular markers in axilla and primary tumour that mediate radioresistance could influence choice of treatment.

Aim: To assess the expression of apoptosis proteins as markers for axillary recurrence following axillary radiotherapy.

Methods: Ten patients who developed axillary recurrence following axillary radiotherapy were compared with 10 matched controls who remained disease-free at 5 years. All underwent a wide local excision (WLE)/mastectomy followed by radiotherapy to the breast/chest wall and axilla under similar regime. Immunohistochemical analysis was performed on primary tumour, axillary node samples for p53, anti-apoptotic markers (bcl-2,bcl-xl,mcl-1) and pro-apoptotic markers (bad,bak,bax). Scoring was done by 2 independent observers.

Results: In 5 out of the 10 cases, the primary tumour was positive for p53 as opposed to none in the controls, the difference being statistically significant (p= 0.016,Fisher's exact test). A statistically significant difference was also found in the expression of bcl-xl between the axillary samples of cases and controls (p=0.035, Fisher's exact test). None of the other markers were significantly different between the 2 groups.

Conclusion: p53 mutation in the primary tumour is a significant risk factor for axillary recurrence following axillary radiotherapy. Over-expression of bcl-xl in the axillary sample may also contibute to radioresistance by failing to initiate an appropriate apoptotic response in cells with radiation induced damage.

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p53 and p21WAF1/CIP1 proteins and cells proliferation in ovarian carcinomas

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Now time the selection of therapeutic approach of patients with ovarian carcinoma predominantly is based mainly on morphological characteristics. However the molecular profile of ovarian neoplasm is necessary also for correct prognosis.

Aim: To determine the proliferative activity and the overexpression of supressor genes p53, p21WAF1/CIP1 as possible prognostic factors in ovarian tumors.

Materials and methods: Archives materials from 50 patients (age 17-74) were used. Fourty patients were with serous ovarian carcinomas and 10 patients – with normal ovary obtained from women with hysteromyoma (control group). Tissue sections were immunohistochemically stained using a monoclonal antibodies: for Ki-67 clone MIB, for p53 clone DO-7 and for p21WAF1/CIP1 clone SX118 ("Dako"). The presentage of immunopositive cells was calculated as labeling index (LI).

Results: The low proliferation activity (Ki-67 LIs = 1,0) and negative expression of p53 and p21WAF1/CIP1 were found in normal ovary. There were heterogeneity in Ki-67, p53 and p21WAF1/CIP1 expression in ovary tumors. Ki-67 was detected in 92,5% ovary tumors. The most tumors (90,0%) were highly proliferating (Ki-67 LI > 10,0). Nuclei p53 staining was found in 95,0% tumors (LI = 40,3±0,3; 6,7 œ 72,5) and p21 - 87,5% (LI = 6,8±0,3; 1,5 œ 31,3). p53 and p21WAF1/CIP1 overexpression was found respectively in 72,5% tumors (LI > 30,0) and 12,5% (LI > 15,0). It was observed dependence between Ki-67, p53 and p21 expression and the level of ovarian carcinomas differentiation. Ki-67 and p53 increase from well to poorly differantiated and were equal to 14,0±0,4 and 37,1±0,4, respectively and Ki-67 - 34,9±0,7 and 45,8±0,5, respectively for p53. However, p21 was changed in oposite direction from 9,4±0,8 and 3,4±0,3. It was determined that Ki-67 level was higher in tumors of patients with survival time 5-10 years, than more 10-20 years, p53 expression was nearly similar.

Conclusion: The levels of Ki-67, p53 and p21WAF1/CIP1 expression objectively reflect ovarian tumor cells biological particularities and can be used for prescription of adequate treatment and as independent factors for prognosis.

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Prognostic value of Ki-67, p53, p21(WAF1) and p16 expression in glandular and atypical hyperplasia of endometrium

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Aim: Determination of tissue proliferation activity, expression of tumour supressor genes P53, P21, P16 in patients with glandular, atypical hyperplasia in comparison with high differentiated adenocarcinoma.

Materials and methods: Formalin-fixed scrapes and operative materials were obtained from 67 patients (age 31-69) with glandular and atypical hyperplasia. Tissue sections were immunohistochemically stained for Ki-67 using a monoclonal anti-Ki-67 antibody (clone MIB), p53 antibody (clone DO-7), p21 (clone SX118), p16 (clone DCS-50), DAKO Cytomation.

Results: Analysis of proliferative potential showed significant variation of expression Ki-67 in hyperplastic endometrium (individual variations 1-37,1%), and high differentiated carcinoma (5-45%). Hight proliferation (>10% cells staining) was observed in 50% cases of hyperplastic endometrium and 83,3% cases of carcinoma. Reliable increasing of proliferative activity in comparison with normal endometrium was determined in