

Expression of P53 Protein in Neoplastic and Non Neoplastic Ovarian Lesions

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Abstract

Background	Ovarian cancer is one of the most common causes of gynecologic neoplasm all over the world.
Objective	The objective is to shed light on the role of p53 protein and patient's age in the pathogenesis of ovarian lesions.
Methods	Paraffin embedded blocks of 62 patients with ovarian lesions were studied. Thirty-five cases of surface epithelial ovarian tumors, (31 cases of invasive surface epithelial ovarian tumors, and 4 cases of borderline intermediate malignancy cases of neoplastic ovarian cystic lesions). In addition, eighteen cases of benign neoplastic ovarian cystic lesions and 9 cases of non- neoplastic functional one were enrolled in this study. All of cases included, were stained with p53 by immunohistochemistry.
Results	Immunohistochemistry for p53 showed that malignant cases were positive for p53 while all benign cases were negative for p53 and the borderline cases were also negative for p53. The non-neoplastic cases were negative for p53. There is a significant statistical difference in P53 expression in malignant cases compared to other groups ($P < 0.001$). A significant difference in mean age of malignant and border line cases in comparison with benign and non-neoplastic cases; ($P < 0.001$).
Conclusion	Protein p53 may play a role in the pathogenesis of malignant ovarian cancer but not in benign lesions. The age of the patient has a role as a risk factor in ovarian lesions.
Keywords	Ovarian lesion, ovarian cancer, p53, immunohistochemistry.

Introduction

Ovarian cancer is one of the most common causes of gynecologic neoplasm and is the fifth cause of cancer mortality in women. The high mortality rate in women with ovarian cancer is due to its detection at advanced stages.

Even though there have been improvements in surgical techniques and treatment options, five-year survival for ovarian cancer still remain at approximately 45% ⁽¹⁾.

Ovarian tumors are heterogenous. Insight into their pathogenesis requires understanding of the mutations involved, overexpression of oncogenes and role of cell cycle regulators. There have been persistent efforts in the investigation of molecular markers in epithelial ovarian tumors, but the results are controversial ⁽²⁾.

Among the most common genetic alterations in human ovarian cancer are p53 mutations. Defects in this tumor suppressor pathway are