Using p53 to help diagnose ovarian cancer

Our first issue had an exhaustive editorial on the scenario of oncology and its facts in the South-Asian region. We received an overwhelming response for it. It is delightful and makes the team of South Asia Journal very proud to have such a wide acceptance of the journal. This issue of ours has articles that represent true nature of science and are simple yet effective in their approach.

We extend our congratulations to all the contributing authors for their significant contribution to the ever changing field of science. Our best wishes to them that they may continue working toward the betterment of the oncology in the region. The two articles selected for the editorial review were on the basis of their importance and reflecting the ongoing quest of science, i.e., form bench to bedside.

In this issue of SAJC, Choudhury *et al*.[1] demonstrated a need to evaluate the diagnostic accuracy of imprint cytology in ovarian neoplasms and also investigated the biological significance of p53 expression and correlated it with grade and stage of the ovarian tumor. They successfully demonstrated that p53 expression has a diagnostic value and it also has a role in the pathogenesis of ovarian cancers.

The tumor suppressor p53 also known as "guardian of genome" is an important transcription factor which prevents the growth and survival of malignant cells.[2] Various stresses leading to DNA damage, attrition of the telomeres or any other oncogenic assault activates p53. This activation of p53 is important for cell-cycle arrest followed by apoptosis, the programmed cell death. There is strong evidence that p53 mutations represent the most common molecular change in the tissues afflicted with cancerous growth.

The p53 being a transcription factor leads to expression of hundreds of genes. The important ones are apoptotic genes involved in the mitochondrial apoptotic pathways such as BAX, NOXA, PUMA, and APAF1. The others: The genes involved in growth arrest like cyclin dependent kinase CDK -inhibitor protein $p21^{WAF1}$ which mediates G1/S arrest by blocking cyclin E-Cdk2-mediated phosphorylation of Rb. It also stimulates the expression of the genes involved in death receptor pathway like FAS and PIDD.[3]

The observation of mutated p53 and its role in the oncogenesis makes it a lucrative target for anticancer therapy. However, lots of consideration is needed before using the drugs leading to activation of inactivated p53. There have been articles which suggest that unregulated activation of p53 in mouse models have demonstrated premature aging. The article by Tyner *et al*., showed the other side of p53 in which they introduced a mutation which activated p53. These mice exhibited resistance to tumors compared to mice with wild type p53. However, they also displayed premature aging which included shortened life span, osteoporosis, organ atrophy, and diminished stress tolerance. In addition to these limitations, it has been suggested that there are more than 2000 different types of mutations in p53 protein which will be difficult to target with the same drug.[4]

The article by Choudhury *et al*., once again emphasizes the underlying oncogenic pathways in the cancerous tissues and the complex process of oncogenesis. They successfully showed that all the benign tumors were negative for p53 whereas 42% of the malignant tumors were positive. These results are comparable with the established literature. The present study also shows that p53 over-expression did not correlate with the histological grade or tumor stage.

Many articles have been devoted to the relevance of the immunohistological assessment of p53 in tumors and their importance with regard to diagnosis and prognosis. These studies compare the presence of p53 protein in normal as well as in cancerous tissues and once again present the facts that simple techniques like immunohistochemistry can play an important role in diagnosis as well as in prognosis of a particular neoplasm.

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