IMMUNOHISTOCHEMICAL STUDIES IN THE DIAGNOSIS OF PROSTATE CANCER

Khairullaev Ravshan Chorikulovich
Bukhara State Medical Institute

The last decade has been marked by the search for new methods of diagnosing prostate cancer that can provide a more accurate assessment of its outcomes, thereby minimizing negative effects and maximizing the positive impact of existing treatment methods. The most active area of scientific research has become the study of associated molecular biological markers characterizing the processes of proliferation, apoptosis and neoangiogenesis of tumor tissue.

In this regard, the following markers were tested: Apoptosis markers (Bcl-2, Bax); Proliferation markers (Ki67); Enzymes (COX-2, ALDH1A1, MMP9); Mutation or expression of p53, p16 and p27. The study of apoptosis markers Bcl-2 and Bax is one of the promising areas in immunohistochemical studies of prostate cancer. The Bcl-2 protein is responsible for inhibiting apoptosis provoked by the action of p53 and other factors (the administration of cytostatics). The synthesis of this protein is encoded by the gene of the same name, located on the long arm of chromosome 18, which, in the case of its hyperexpression, begins to act as an oncogene. In turn, Bax is a protein antagonist for Bcl-2, and is responsible for inducing apoptosis. Normally, Bcl-2 is expressed only by cells of the basal layer of the epithelium. In the available literature, there are a number of works devoted to assessing the effectiveness of Bcl-2 and Bax in diagnosing prostate cancer.

Another promising area of immunohistochemical studies in prostate cancer is the study of the expression of p53, a tumor suppressor gene. P53 is localized on the short arm of chromosome 17, participating in the regulation of proliferation, apoptosis and angiogenesis. This gene is responsible for the synthesis of the corresponding protein, which suppresses the synthesis of fetal growth factor and induces the synthesis of the IGFBP3 protein, which binds insulin-like growth factor

I and II, as well as the synthesis of p21, which is responsible for the repair of damaged DNA. Mutation of this gene leads to uncontrolled cell proliferation and inhibition of apoptosis, which may be associated with a high metastatic potential of the tumor and the development of hormone-resistant cancer.

Studies of the expression of P16 (also known as cyclin-dependent kinase inhibitor 2A) are another promising direction in the diagnosis of prostate cancer. P16 is a tumor suppressor protein, which in humans is encoded by the CDKN2A gene, located on chromosome 9. P16 plays an important role in cell cycle regulation by slowing down the transition of cells from G1 phase to S phase and therefore acts as a tumor suppressor. On the one hand, hypermethylation, mutation or deletion of p16 leads to downregulation of the gene and can lead to cancer through cell cycle dysregulation. Conversely, activation of p16 via ROS pathway, DNA damage or aging leads to accumulation of p16 in tissues and is involved in cell aging.

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