

MORPHOLOGY OF EXPERIMENTAL BREAST CANCER WITH METASTASIS TO THE LUNGS

Hasanov Komiljon Sunnatillaevich

Bukhara State Medical Institute named after Abu Ali Ibn Sina, A. Navoi Street, Bukhara, Uzbekistan.

Abstract. Metastasis is the final stage of malignant progression; theoretically, a tumor of any size has metastatic potential. The biological nature of cancer and the characteristics of the host determine the possibility of further disease progression even after radical treatment. According to several authors, 12 to 30% of all lung and pleural tumors are metastatic. This review focuses on the comparative morphology of experimental breast cancer with lung metastasis.

Keywords: breast cancer, metastasis, lungs, immunohistochemical markers, morphology, morphometry.

Breast cancer has a pronounced metastatic nature, usually spreading to the bones, liver, lungs and brain. The development of breast cancer metastases in a particular organ is a complex process that depends on many factors, the main of which is the molecular type of the tumor. There are 4 subtypes of breast cancer, depending on gene expression, ER, PR, HER2 status and proliferation status, as determined by the Ki-67 proliferative activity index. As the tumor becomes more aggressive, there are luminal subtype A (ER+/PR+), luminal type B (ER+/PR+/HER2–/+/Ki-67+), HER2 overexpressing subtype (ER–/PR–/HER+) and basal-like/triple negative (TN–PR–HER) subtype (ER–/PR). The TN subtype of breast cancer has the highest tendency to metastasize to the lungs (32%), while luminal A/B (21%) and HER2+ (25%) have a lower tendency.

Tumor metastasis typically involves several sequential steps, including the primary tumor's invasion of surrounding tissues, intravasation into the blood and/or lymphatic vessels, formation of circulating tumor cells, extravasation, and subsequent dissemination and transformation of tumor cells with the formation of metastases. Many factors contribute to the development of breast cancer metastases in the lung. A number of genes have been identified that contribute to the formation of micro- and macrometastases in the lung. These include the BMP (bone morphogenetic protein) inhibitor Coco, the differentiation inhibitor proteins, DNA-binding proteins ID1 and ID3; extracellular matrix protein tenascin C, CXCL1 (chemokine (C–X–C motif ligand 1), vascular cell adhesion molecule 1 (VCAM 1), as well as a gene encoding a fine-tuning gene for epithelial-mesenchymal transition and circulating cancer biomarkers (microRNA).

The development of metastases in lung tissue is facilitated by the formation of so-called premetastatic spaces (PMB). Exogenous factors, including smoking and long-term exposure to environmental pollutants, play a major role in these processes. Long-term exposure to pollutants and tobacco smoke negatively affects the innate and adaptive immune systems. Chronic exposure to nicotine promotes the formation of PMB in the lung, signaling transducer and activator of transcription-3 (STAT3), serine/threonine protein kinase B (Akt), and NF- κ B (nuclear factor κ B). In this case, pro-tumor N2 neutrophils promote the colonization and growth of metastases. STAT3 is secreted by the activated lipocalin 2 (LCN2), which stimulates the release of STAT3. LCN2 has been shown to persist for a long time in the lungs of rats exposed to tobacco smoke and in the plasma of smoking patients. Studies also confirm the immunosuppressive effects of cigarette smoke due to decreased natural killer (NK) cell activity, antigen-presenting macrophage activity, decreased dendritic cell numbers, and increased T cell activity. Smoking increases the risk of metastasis by 18% and reduces survival at diagnosis by 33%.

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