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ISSN: 2684-1614
Journal of Tumour Research & Reports

Opinion Article

Massive Causes of Brain Metastasis and its Origin

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DESCRIPTION

Many patients with lung cancer, breast cancer, and melanoma develop brain metastases that are resistant to conventional treatments. The median survival time for untreated patients is 1-2 months, but surgery, radiation therapy, and chemotherapy can extend it to 6 months [1,2]. The outcome of metastasis depends on multiple interactions of the metastatic cells with the host's homeostatic mechanism, which utilizes tumor cell survival and proliferation. The blood-brain barrier is leaky in metastases larger than 0.5 mm in diameter due to the production of vascular endothelial growth factor by metastatic cells. Brain metastases are surrounded and infiltrated by microglia and activated astrocytes. Interaction with astrocytes results in upregulation of several genes in metastatic cells, including some viable genes involved in increasing the resistance of tumor cells to cytotoxic drugs. These results support the importance of the seed and soil hypothesis that successful treatment of brain metastases requires targeting the microenvironment of the organ. The main cause of death from cancer is intractable metastasis. Primary neoplasms and metastases include growth rate, cell surface receptors, antigenicity, immunogenicity, hormone receptors, adhesion molecule profiles, extracellular matrix protein production, susceptibility to cytotoxic agents, angiogenic potential, invasiveness, and contains multiple cell populations with different characteristics such as metastatic potential. This biological heterogeneity is generally due to the genetic instability of tumor cells, especially metastatic cells [3,4]. The biological heterogeneity of cells is also reflected in the generation of organ-specific metastases. The clones selected in the primary neoplasm can cause secondary metastases to specific organ sites such as the brain.

Causes of metastasis

The process of metastasis is highly selective and consists of a series of consecutive interconnected steps that must be completed if clinically relevant lesions occur. When a tumor mass reaches a diameter greater than 1 mm, tumor cells must proliferate at the primary site and induce angiogenesis (establishment of a new vascular system). Tumor cells need to invade the host stroma and into lymphatic or vascular channels [5]. Individual cells or masses of cells can circulate and reach distant organs. This tumor embolus must withstand circulatory immune and non-immune turbulence in order to be retained in the capillary bed of the receiving organ ("niche"). Tumor cells can grow in blood vessels or overflow into organ parenchyma, where they must proliferate to form micrometastases. As the size of these lesions increases, new cells can enter the circulation and cause metastases from metastases.

Origin of brain metastasis

Clinical observations show that brain metastases produced by many solid tumors occur later in the diseases, whether brain metastases are produced by tumor cells secreted by lymph nodes or by visceral metastases. This is an important issue for the etiology of brain metastases. If the spread of metastases causes brain metastases, aggressive and prophylactic resection of lymph node or visceral metastases can reduce the risk of developing fatal brain lesions. On the other hand, if brain metastases are caused by the direct diffusion of special metastases from the primary tumor, prophylactic dissection of extracranial metastases may not be able to prevent the development of brain metastases.

CONCLUSION

Over 40% of patients with lung and breast cancer develop brain metastases. Improved local control and treatment of visceral metastases predicts increased morbidity and mortality from late-diagnosis brain metastases. The median survival time for untreated patients is 1-2 months and can be extended to 6 months with radiation and chemotherapy. Despite improvements in diagnosis, surgical techniques, general patient care, and local and systemic adjuvant therapies, most cancer deaths result from metastases that are resistant to conventional therapies. The process of cancer metastasis is continuous and selective and contains probabilistic elements. Therefore, metastatic growth represents the endpoint of many events in which few tumor cells survive. Primary tumors consist of

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Received: 25-Feb-2022, Manuscript No. JTRR-22-16296; **Editor assigned:** 28-Feb-2022, Pre QC No.JTRR-22-16296 (PQ); **Reviewed:** 15-Mar-2022, QC No JTRR-22-16296; Revised: 21-Mar-2022, Manuscript No.JTRR-22-16296(R); **Published:** 29-Mar-2022, DOI: 10.35248/2684-1614.22.7.156.

Citation: Valiente P (2022) Massive Causes of Brain Metastasis and its Origin. J Tum Res Reports.7:156.

multiple subpopulations of invasive and metastatic cells that can metastasize in processes that depend on the interaction of the tumor cells with the host factor. Metastatic cells are genetically unstable with susceptibility to various karyotypes, growth rates, cell surface properties, antigenicity, immunogenicity, marker enzymes, and various therapeutic agents, resulting in biological heterogeneity. Bring. The recognition that different metastases can originate from multiple progenitor cells contributes to biodiversity and poses additional challenges for therapeutic intervention. Moreover, heterogeneity can develop rapidly, even within a single metastasis of clonal origin. Immunohistochemical and morphological analysis show that the density of blood vessels within experimental metastases in the brain of nude mice or in clinical specimens of human lung cancer brain metastases is lower than that in the brain parenchyma without adjacent tumors.

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