Opinion

iMedPub Journals
www.imedpub.com

Medical and Clinical Reviews
ISSN 2471-299X

2023
Vol.9 No.1:001

Review of the Role of Platelets in the Process of Malignant Tumor Metastasis

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Received date: December 30, 2022, Manuscript No. IPMCR-23-15799; **Editor assigned date:** January 02, 2023, PreQC No. IPMCR-23-15799(PQ); **Reviewed date:** January 16, 2023, QC No. IPMCR-23-15799; **Revised date:** January 23, 2023, Manuscript No. IPMCR-23-15799(R); **Published date:** January 30, 2023, DOI: 10.36648/2471-299X.9.1.1

Citation: Dong S (2023) Review of the Role of Platelets in the Process of Malignant Tumor Metastasis. Med Clin Rev Vol: 9 No: 1: 001

Description

The hypothesis that platelets facilitate tumor metastasis has gained widespread acceptance in the medical community and is supported by a substantial body of research data thanks to the rapid advancement of science and technology and numerous studies. However, the intricacies of the underlying mechanisms remain a mystery, making platelet-tumor interaction research a hot topic in oncology in recent years. Trousseau's syndrome was given the name association between platelets and cancer in 1865. Later, Billroth accidentally discovered a connection between platelets and tumor cells during autopsies. He made the observation that platelet thrombosis, which was comparable to the armor of tumor cells, frequently took place in conjunction with tumor metastasis. After that, he made the bold statement; Platelets and the process of tumor metastasis are closely linked. However, researchers like Gasic and Li did not confirm this conjecture until more than half a century later. Gasic and co. 6 administered anti-platelet antibody plasma to mice, added a tumor cell suspension to the *in vivo* model, and observed the metastasis of tumor cells to create an *in vivo* thrombocytopenia model.

In the mouse model of thrombocytopenia, the results showed that cancer cell metastasis was significantly reduced. Li and co. 7 injected tumor cells and platelets into mice, and the results showed that mice infused with platelets had significantly more tumor metastases than mice infused with only tumor cells. The aforementioned studies laid the groundwork for subsequent research on the connection between tumor cells, platelets, and metastasis. They provided preliminary evidence that platelets are involved in the process of hematogenous metastasis of malignant tumors. Anti-tumor drugs that target platelets have become a hot topic in scientific research due to their role in hematogenous metastasis of cancerous tumors. Aspirin, clopidogrel, and ticagrelor all inhibit platelets, according to pharmaceutical studies. However, while these medications inhibit platelets, they also damage many of their functions. Liposomal nanoparticles containing the tumor-homing pentapeptide CREKA (Cys-Arg-Glu-Lys-Ala) have been developed to avoid the adverse effects of direct drug infusion in patients in order to balance the benefits and drawbacks of these medications.

Initiation of the External Coagulation Process

The functions of non-tumor-related platelets, such as hemostasis and coagulation, will not be disrupted by liposomal nanoparticles' ability to deliver platelet-inhibiting drugs to tumor tissues. Successful hematogenous metastasis of tumors depends on the interaction between tumor cells and platelets. Platelets and tumor cells communicate *via* a complicated two-way pathway. Platelet activation is the result of interactions between tumor cells and platelets, which results in the release of factors that help tumor cells survive and grow. Platelets are activated in a variety of ways by tumor cells. For instance, they are able to directly secrete thrombin, which is the most potent agonist for activating platelets. Thrombin regulates platelets *via* feedback from the protein kinase receptor, triggering a platelet aggregation waterfall reaction. Additionally, tumor cells are capable of secreting a number of tissue factors in order to activate platelets. This initiates the external coagulation process, encourages thrombosis, and then protects tumor cells. In addition, tumor cells can indirectly activate platelets by releasing the metabolite ADP, which can cause platelet activation. Through a variety of mechanisms, platelets activated by tumor cells can help tumor cells survive and invade. However, recent research has demonstrated that platelets also inhibit tumor growth and metastasis. By preventing the cell cycle, platelets, for instance, prevent tumor cells from multiplying. By binding to its receptor CXCR3B, Platelet Factor (PF4) inhibits tumor metastasis by promoting apoptosis and vascular degeneration. Through interactions between platelets and tumor cells, platelets aid in tumor metastasis. Platelets are triggered by tumor cells, which in turn can trigger tumor cells' systematic distant metastasis. Platelets have been shown to aid in tumor cell immune escape, protect tumor cells from the influence of blood flow shear force, improve tumor cells' resistance to anoikis and apoptosis, promote vascular remodeling, and assist tumor cells in entering the blood circulation and spreading. As a result, one important step is to investigate how platelets aid in tumor metastasis. Natural killer (NK) cells, T cells, and Dendritic Cells (DCs) are examples of peripheral immune cells that monitor and kill cancer cells that circulate in the blood, respectively. Perforin and granzyme are released by NK cells when they encounter tumor cells, triggering apoptosis.

Additionally, CD8+ T cells inhibit tumor metastasis by secreting Interferon (IFN-) and Tumor Necrosis Factor (TNF-). Additionally, DCs boost the cellular immune response by secreting a variety of cytokines. In order for immune-related cells to accurately identify, eliminate, and clear tumor cells, tumor cells will significantly cause the aforementioned reactions because they are antigens in the tumor immune microenvironment. As a result, tumor cell metastasis necessitates platelet-mediated immune escape of tumor cells; the current platelet-mediated immune escape mechanisms are primarily NK and T cells.

The Selection of Anti-Platelet Medications

Although there is very little adhesion between tumors and vascular endothelial cells, platelet activation causes an increase in the expression of P-selectin on the plasma membrane and platelet surface integrins like IIb3. This causes a sudden increase in adhesion between mucin on the surface of tumor cells and vascular endothelial cells, which greatly aids in the implanting of tumor cells at the meta The incidence of lung metastasis in mice with combined removal of platelets and NK cells was significantly lower than that in mice with only NK cells removed, according to a study on the effect of the combined removal of NK cells and platelets on liver and lung metastases of melanoma and breast cancer cells. Melanoma liver metastases were significantly more common in mice lacking both platelets and NK cells than in mice lacking only NK cells. Through platelet mRNA sequencing, tumor-acting platelets alter the mRNA profile of platelets, making it easier for patients to differentiate between local and distant metastases. Tumor-acting platelets play an important role in the development of tumors. Additionally, this has diagnostic potential as a potential marker for the diagnosis of a variety of cancers and is significantly related to indicators related to the target drug, such as HER2-positive, MET, and EGFR. Clinically, tumor-associated platelet biomarker-based blood tests can not only detect early cancer but also predict recurrence and personalized treatment closely. Liquid biopsy has been studied for breast cancer, pancreatic ductal adenocarcinoma, and liver cancer because it circumvents the limitations of tissue collection. The potential biological uses of a few significant platelet-related molecules that are involved in the entire process of tumor metastasis, invasion, screening, diagnosis, and prognosis evaluation have gradually emerged. However, the specific mechanisms, including how to balance and develop tumor cells, immune cells, platelets, and inflammatory cells, require additional investigation and clarification due to the complexity of the interactions between platelets and tumor cells. What are typical platelet mechanisms of action in various types of metastatic cancer cells? To confirm which mechanism plays a crucial role and which major molecules are involved at various stages of metastasis promotion, additional research is needed. In addition, the selection of anti-platelet medications is a hot topic in the medical community right now. The selection of medications, their dosages, and their safety will be the future focus of significant research and breakthroughs in the scientific field. Platelets not only aid in tumor metastasis, but they also partially hinder it. It is still unclear how this functional duality of platelets works. There is still a need for more research into the specific mechanism of platelets in tumor metastasis.