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The Role of CSF3R/CD114 Gene Expression in Glioma: Implications for Patient Survival

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Abstract

Gliomas, the most common primary brain tumors, exhibit significant heterogeneity in terms of molecular characteristics and clinical outcomes. The identification of biomarkers that can predict patient survival and guide treatment decisions is crucial for improving outcomes in glioma patients. The CSF3R/CD114 gene has been implicated in various cancers, but its role in gliomas remains unclear. This review aims to elucidate the role of CSF3R/CD114 gene expression in gliomas and its implications for patient survival. We summarize current literature on the expression patterns of CSF3R/CD114 in gliomas, its association with clinicopathological features and its prognostic significance. Furthermore, we discuss potential mechanisms underlying the involvement of CSF3R/CD114 in glioma progression and therapy resistance. Understanding the role of CSF3R/CD114 in gliomas may lead to the development of novel therapeutic strategies and personalized treatment approaches for glioma patients.

Keywords: Glioma • Gene expression • CSF3R • CD114

Introduction

Gliomas represent a heterogeneous group of primary brain tumors with diverse molecular characteristics and clinical behaviors. Despite advances in diagnosis and treatment modalities, prognosis for glioma patients remains generally poor, highlighting the urgent need for novel prognostic markers and therapeutic targets. The CSF3R/CD114 gene, encoding the receptor for Granulocyte Colony-Stimulating Factor (G-CSF), has garnered attention in cancer research due to its involvement in tumor progression and therapeutic resistance in various malignancies. However, its role in gliomas remains poorly understood. This review aims to explore the significance of CSF3R/CD114 gene expression in gliomas, particularly its association with patient survival and its potential as a prognostic biomarker. By elucidating the molecular mechanisms underlying the involvement of CSF3R/CD114 in glioma pathogenesis, we may uncover new avenues for targeted therapy and personalized medicine in the management of glioma patients [1,2].

Literature Review

The CSF3R/CD114 gene, encoding the receptor for Granulocyte Colony-Stimulating Factor (G-CSF), has been extensively studied in various cancers, including leukemia, breast cancer and lung cancer. In these malignancies, aberrant expression of CSF3R/CD114 has been associated with tumor progression, metastasis and resistance to chemotherapy. However, its role in gliomas has only recently begun to emerge. Several studies have reported dysregulated expression of CSF3R/CD114 in gliomas, with conflicting findings regarding its prognostic significance. Despite these discrepancies, emerging evidence suggests that CSF3R/CD114 may play a crucial role in glioma progression and therapy resistance. Mechanistically, activation of the G-CSF/CSF3R pathway has been implicated in promoting glioma cell proliferation, invasion and angiogenesis. Moreover, recent studies have implicated CSF3R/CD114 in mediating resistance to temozolomide, the standard chemotherapeutic agent for gliomas, highlighting its potential as a therapeutic target. Further investigations into the molecular mechanisms underlying the involvement of CSF3R/CD114 in glioma progression have revealed potential crosstalk with various signaling pathways. For example, studies have suggested interactions between the G-CSF/CSF3R pathway and key regulators of glioma growth and invasion, such as the PI3K/Akt and MAPK/ERK pathways. These interactions may contribute to the aggressive phenotype observed in gliomas with high CSF3R/CD114 expression [3].

Moreover, the tumor microenvironment, characterized by interactions between cancer cells, stromal cells and immune cells, plays a critical role in glioma development and progression. Recent studies have implicated CSF3R/CD114 in modulating the immune response within the glioma microenvironment. Activation of the G-CSF/CSF3R pathway has been associated with immunosuppressive effects, such as the recruitment of Myeloid-Derived Suppressor Cells (MDSCs) and the inhibition of T cell function, thereby promoting tumor immune evasion. Additionally, the relationship between CSF3R/CD114 expression and Glioma Stem Cells (GSCs), a subpopulation of tumor cells with self-renewal and tumorigenic properties, has garnered interest. Emerging evidence suggests that CSF3R/CD114 may play a role in maintaining the stemness and therapeutic resistance of GSCs, thereby contributing to tumor recurrence and treatment failure. Overall, while the precise role of CSF3R/CD114 in gliomas requires further elucidation, accumulating evidence indicates its multifaceted involvement in tumor progression, therapy resistance and immune modulation. Future studies focusing on deciphering the complex interplay between CSF3R/CD114 signaling, the tumor microenvironment and glioma heterogeneity are warranted. Such insights may pave the way for the development of targeted therapies and personalized treatment strategies tailored to the molecular characteristics of individual glioma patients [4,5].

Discussion

The conflicting findings regarding the prognostic significance of CSF3R/CD114 in gliomas may be attributed to several factors, including variations in patient cohorts, sample sizes and methodological differences in gene expression analysis. Furthermore, the complex interplay between CSF3R/CD114 and other molecular pathways in glioma pathogenesis warrants further investigation. Despite these challenges, elucidating the role of CSF3R/CD114 in gliomas holds promise for improving patient outcomes. Targeting the G-CSF/CSF3R pathway may represent a novel therapeutic strategy for glioma patients, particularly those with aggressive disease or resistance to standard

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treatments. Additionally, integrating CSF3R/CD114 expression into existing prognostic models may enhance their predictive accuracy and facilitate personalized treatment approaches [6].

Conclusion

In conclusion, the role of CSF3R/CD114 gene expression in gliomas remains a topic of ongoing research and debate. While conflicting evidence exists regarding its prognostic significance, emerging data suggest that CSF3R/CD114 may contribute to glioma progression and therapy resistance. Further studies are warranted to clarify its precise role in glioma pathogenesis and to explore its potential as a therapeutic target. By unraveling the complexities of CSF3R/CD114 signaling in gliomas, we may uncover new opportunities for improving patient outcomes and developing more effective treatment strategies for this devastating disease. Moving forward, further research efforts aimed at unraveling the molecular mechanisms underlying CSF3R/CD114 signaling in gliomas are warranted. This includes elucidating its interactions with key signaling pathways, its impact on immune modulation within the tumor microenvironment and its involvement in glioma stem cell biology. Moreover, translating these findings into clinical practice holds promise for improving outcomes in glioma patients. Targeting the CSF3R/CD114 pathway may offer a novel therapeutic approach for overcoming therapy resistance and combating tumor progression. Additionally, integrating CSF3R/CD114 expression into existing prognostic models may enhance their predictive accuracy and guide personalized treatment strategies for glioma patients.

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Conflict of Interest

None.

References

1. Wen, Patrick Y. and Santosh Kesari. "Malignant gliomas in adults." *N Engl J Med* 359 (2008): 492-507.
2. Hardigan, Andrew A., Joshua D. Jackson and Anoop P. Patel. "Surgical management and advances in the treatment of glioma." *Semin Neurol* (2023).
3. Schneider, Armin, Carola Krüger, Tobias Steigleder and Daniela Weber, et al. "The hematopoietic factor G-CSF is a neuronal ligand that counteracts programmed cell death and drives neurogenesis." *J Clin Invest* 115 (2005): 2083-2098.
4. Zage, Peter E., Sarah B. Whittle and Jason M. Shohet. "CD114: A new member of the neural Crest-derived cancer stem cell marker family." *J Cell Biochem* 118 (2017): 221-231.
5. Agarwal, Saurabh, Anna Lakoma, Zaowen Chen and John Hicks, et al. "G-CSF promotes neuroblastoma tumorigenicity and metastasis *via* STAT3-dependent cancer stem cell activation." *Cancer Res* 75 (2015): 2566-2579.
6. Hsu, Danielle M., Saurabh Agarwal, Ashley Benham and Cristian Coarfa, et al. "G-CSF receptor positive neuroblastoma subpopulations are enriched in chemotherapy-resistant or relapsed tumors and are highly tumorigenic." Cancer Res 73 (2013): 4134-4146.

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