REVIEW | **OPEN ACCESS**

Role of ICAM1 on Immune Cells in Glioblastoma: A Review Study

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Abstract
Introduction: Glioblastoma, also known as glioblastoma multiforme (GBM), is a highly aggressive and incurable form of brain tumor that grows rapidly. Intracellular cell adhesion molecule 1 (ICAM1) is a glycoprotein and adhesion receptor that plays a multifaceted role in immune response and can serve as a therapeutic target. Due to the severity and poor prognosis associated with GBM, the interaction between ICAM1 and GBM has been a topic of interest in the hope of providing therapeutic value.
Methods: This review synthesizes existing literature and studies through searches in the PubMed, Google Scholar, Web of Science, Ovid-Medline, and EBSCO databases to explore the role of ICAM1 on immune cells in the context of GBM. Search terms such as "GBM," "ICAM1," "tumor microenvironment," as well as "adhesion molecules" and "GBM treatment" were employed. Literature was selected based on its applicability to key aspects of the research topic.
Results: This comprehensive review anticipated uncovering the complex role of ICAM1 in immune cell adhesion in GBM. By promoting the adhesion of immune cells, such as T lymphocytes and natural killer cells, ICAM1 can potentially enhance the body's natural defense mechanisms against GBM. However, the tumor microenvironment (and interaction with other molecules) can also manipulate and alter ICAM1 in ways that inhibits an effective immune response, potentially resulting in tumor progression.
Discussion: Understanding the role of ICAM1 in GBM can present new strategies for immune-based therapies in GBM, potentially leading to improved treatment outcomes. Moreover, gaining insight into how adhesion molecules, such as ICAM1, respond to the tumor microenvironment can advance GBM therapy and also provide insights into treatment options for various cancers.
Conclusion: ICAM1 exerts both pro-tumorigenic and anti-tumorigenic effects, shaping tumor progression and immune evasion. Understanding these dual roles can guide the development of targeted therapies for GBM.

Keywords: glioblastoma multiforme; glioblastoma; ICAM1; immune cells; tumor microenvironment; GBM treatment; adhesion molecules

Introduction

Glioblastoma (GBM) is a rare and highly malignant brain tumor in adults, affecting 4.50 individuals per 100,000 in Canada, accounting for 54% of all gliomas and 16% of primary brain tumors [1, 2]. These tumors typically manifest around the median age of 64 years, although they can occur at any age, even in childhood [3]. Despite impacting both men and women, the incidence is higher among men, constituting 58% of cases compared to 42% among females [4]. The current standard of care for GBM involves surgical resection which is later followed by a combination of radiation therapy and concurrent temozolomide chemotherapy. This approach has demonstrated an improvement in patient survival by extending the median survival from 12.1 months with RT alone to 14.6 months [5-7]. Despite these advancements, recurrence is inevitable within 6-9 months post-surgery, and patients typically survive fewer than 15 months post-

recurrence with this aggressive regimen [8]. Given these challenges, there is an urgent need for more research to explore targeted therapeutic approaches for GBM. Targeted therapeutics are treatments that selectively target specific molecules or pathways involved in tumor growth and progression [8-10]. GBMs exhibit characteristic tumoral heterogeneity, varying between patients and within the same tumor over time. This heterogeneity leads to diverse epigenetic mutations throughout the tumor and the emergence of new mutations during tumor progression [9-11]. Targeted therapy can address this heterogeneity by focusing on specific molecular targets present in certain subsets of tumor cells.

Central to this research is the investigation of the role of intracellular adhesion molecule (ICAM1) on immune cells within the tumor microenvironment of GBM. Understanding this role is crucial not only for deciphering the tumor's behavior but also for identifying potential therapeutic targets.

Immune cells play a pivotal role in modulating tumor growth and response to therapies [12]. Their interaction with the tumor cells and the surrounding microenvironment shapes the progression and aggressiveness of GBM. Cell adhesion molecules like ICAM1 can influence these interactions, potentially serving as key regulators in this dynamic process [12, 13]. Therefore, it becomes crucial to understand the role of ICAM1 on immune cells in GBM.

The microenvironment surrounding a tumor, referred to as the tumor microenvironment (TME), comprises various cellular and noncellular components. Key cellular elements include immune cells, endothelial cells, neurons, and astrocytes [9-11]. The TME also involves noncellular factors like signaling molecules, exosomes, extracellular matrix (ECM) components, and enzymes contributing to ECM remodeling. The presence of the blood-brain barrier further influences the dynamic interactions within the TME [12].

The TME contains many immune cells such as tumor-associated macrophages (TAMs), T-cells, myeloid-derived suppressor cells (MDSCs), and natural killer (NK) cells [12]. Among these, TAMs are the most abundant, making up to 30-40% of the tumor by mass. TAMs are responsible for releasing immunosuppressive factors and promote recruitment of regulatory T-cells to allow for immune evasion by tumor cells [13]. They also secrete specific growth factors such as epidermal growth factor to promote tumor cell proliferation [14]. In addition, TAMs support GBM growth through anti-inflammatory functions, promoting tumor cell proliferation, migration, and invasion. TAMs contribute to an immuno-suppressive TME by inhibiting T-cell-mediated pro-inflammatory responses, impeding T-cell infiltration into the tumor, and inducing T-cell exhaustion through the expression of inhibitory receptors [15]. T-cells are involved in pro-inflammatory responses against tumors. In the GBM micro-environment, they recognize and target tumor cells to initiate an immune response. MDSCs can diminish the immune system by suppressing the functions of T-cells and creating an environment tolerant to immune responses [15]. NK cells specialize in detecting and eliminating tumor cells. In the GBM microenvironment, NK cells contribute to anti-tumor immunity by recognizing and destroying cancerous cells [15].

Cell adhesion molecules (CAMs) are proteins that function by mediating interactions between neighboring cells or between cells and the extracellular matrix [16]. ICAM1 is a cell adhesion molecule that is expressed on the surface of immune cells such as leukocytes [17-22]. Its primary function lies in facilitating the recruitment of leukocytes from the bloodstream to sites of inflammation and tissue injury [22]. ICAM1 is notably induced in inflammatory macrophages, where it serves as a phagocytic receptor, mediating binding with apoptotic cells and facilitating their clearance. In addition, ICAM1 also plays a role in T-cell mediated host defense system where it functions to activate major histocompatibility complex class II restricted T-cells to activate cytotoxic T-cells [23-25]. In the dynamics of cancer progression, the role of ICAM1 is context-dependent, exerting both pro-tumorigenic and anti-tumor effects through its interactions with immune cells [26]. ICAM1 can potentially worsen cancer outcomes by facilitating the clustering of circulating tumor cells and participating in pro-tumorigenic signaling pathways. Simultaneously, its involvement in immune responses can benefit host survival by promoting the recognition and destruction of tumor cells [26].

In this comprehensive review investigating the influence of ICAM1 on immune cells within the context of GBM, the central hypothesis posits that the upregulation of ICAM1 expression in immune cells constitutes a pivotal factor in driving the aggressiveness of GBM [27, 28].

Methods

A comprehensive review of existing studies pertaining to ICAM1 in GBM was conducted. The search involved the following databases: Web of Science, Ovid-Medline, PubMed, Google Scholar, and EBSCO. Key words such as "ICAM1," "glioblastoma," "ICAM1 role on immune cells," "ICAM1 and tumor microenvironment," "ICAM1 modulation of immune cells," "TAMs and ICAM1," "ICAM1 and myeloid-derived suppressor cells," "ICAM1 and leukocyte recruitment," ICAM1 and Natural killer cells" were utilized. The focus of this comprehensive review is to delve into the specific role of ICAM1 on immune cells within the context of GBM. The inclusion criteria were peer-reviewed studies published in English, and articles were further screened based on relevance and quality, with a focus on recent studies. Specifically, the relevance criterion included studies that directly addressed ICAM1's role in GBM and its influence on immune cell interactions or tumor microenvironment modulation. The quality assessment involved evaluating each article for methodological rigor, sample size considerations, and potential biases to ensure the reliability of the findings.

Table 1. Literature search strategy for studies investigating the role of ICAM1 on immune cells in GBM

Search Date	Databases	Keywords and Boolean Operators	Search Results
12/02/2024	Web of Science, Ovid-Medline, PubMed, Google Scholar, and EBSCO	(ICAM1 OR ICAM-1) AND (role in immune cells OR natural killer cells OR TAMS OR myeloid-derived suppressor cells OR leukocyte OR tumor microenvironment) AND (in glioblastoma OR in GBM)	21 articles reviewed. Removal of articles as per exclusion criteria. 9 articles to be included in the review.

Results

ICAM1 Expression and its Impact on Tumor Cells in GBM

The expression of ICAM1 in GBM tumor cells has been shown to promote tumor migration and invasion, thereby contributing to tumor growth [27]. Previous studies have consistently associated high ICAM1 expression in GBM tumor cells with increased migration, invasion, and poorer overall survival. Additionally, ICAM1 has been implicated in endothelial cell adhesion and blood vessel formation, suggesting a pro-tumorigenic function within the TME [27].

In Vivo Studies using ICAM1 Knockout Mouse Models

Studies that use ICAM1 knockout mouse models, which are genetically engineered mice with the ICAM1 gene deleted (ICAM1-/-) in all cell types demonstrated that ICAM1 deletion led to increased survival compared to wildtype (ICAM1+/+) mice in GBM [28]. The study included a sample size of 12 mice per group to ensure statistical power. Survival analyses were conducted using both the log-rank Mantel-Cox test and the Gehan-Breslow Wilcoxon test. Furthermore, tumor volume analysis was performed using MRI imaging at multiple time points (7 days, 14 days, and 21 days post-injection) to assess tumor growth dynamics [28]. The sample sizes for these analyses were appropriately determined with 10 ICAM1+/+ and 12 ICAM1-/- mice. Tumors grown in ICAM1 deletion mice were significantly smaller than those in wildtype mice. Statistical significance was determined using paired sample statistical t-tests, with significant differences observed at 7 days ($P = 0.043$) and 21 days ($P = 0.027$) [28]. These findings underscore the crucial role of ICAM1 in promoting tumor progression.

ICAM1 and Immune Cell Recruitment in GBM

Studies have further demonstrated that ICAM facilitates macrophage infiltration in cancer, and increased ICAM1 expression in the TME upon radiation enhances macrophage recruitment in GBM [28]. Findings from experiments on mice revealed that the proportion of macrophages was significantly lower in ICAM1 knockout mice compared to ICAM1-expressing mice within the TME [28]. This suggests that ICAM1 contributes to the recruitment of macrophages into the TME. Therefore, ICAM1 expression on macrophages likely promotes tumor growth, partly by regulating their infiltration into the TME. Further findings also support that ICAM1 contributes to GBM growth by enhancing the recruitment of anti-inflammatory TAMs [29].

ICAM1 on Immune Cells in GBM

Research has also shown that immune cells such as cytotoxic T-cells, NK cells, helper T-cells, and B cells serve anti-tumorigenic immune functions, while Tregs, TAMs and MDSCs serve pro-tumorigenic immune function [30]. GBM is highly infiltrated by the latter immune cells [31].

ICAM1 expression on tumor cells facilitates the adhesion and infiltration of immune cells, including T-cells, NK cells, and B cells. ICAM1 promotes their migration towards and interaction with tumor cells, influencing the anti-tumor immune response [32]. In T-cells, ICAM1 enhances their activation and proliferation, facilitating their cytotoxic activity against tumor cells. Similarly, ICAM1-mediated interactions with NK cells augment their cytotoxic function, enabling them to target and eliminate tumor cells more effectively. ICAM1 expression also impacts helper T-cell function by modulating their activation and cytokine secretion. Additionally, ICAM1 facilitates the adhesion and activation of B cells to promote antibody production against tumor cells [32].

ICAM1-mediated interactions promote the recruitment of Tregs, TAMs, and MDSCs, fostering an immunosuppressive environment that supports tumor growth and progression. ICAM1 expression enhances the suppressive activity of Tregs and MDSCs, further dampening anti-tumor immune responses and promoting immune evasion by tumor cells.

Therefore, upregulation of ICAM1 expression in immune cells (like Tregs, TAMs, and MDSCs) constitutes a pivotal factor in driving the aggressiveness of GBM.

Discussion

ICAM1's Dual Role

The role of ICAM1 on immune cells in GBM is of significant interest due to its implications in tumor progression and immune evasion. Review of existing literature and experimental findings highlights the multifaceted impact of ICAM1 on both tumor cells and immune cells within the tumor microenvironment. While ICAM1 can serve anti-tumorigenic functions on immune cells in GBM, it can also exert pro-tumorigenic functions. ICAM1 expression on tumor cells facilitates the adhesion and infiltration of immune cells like T-cells, NK cells, and B cells, enhancing their anti-tumor immune response. This interaction promotes T-cell activation, NK cell cytotoxicity, and B cell antibody production against tumor cells. However, ICAM1-mediated recruitment of pro-tumorigenic immune cells, including Tregs, TAMs, and MDSCs, fosters an immunosuppressive environment that supports GBM progression. Thus, upregulation of ICAM1 in these immune cells is pivotal in driving GBM aggressiveness. This dual role of ICAM1 underscores its complexity in the GBM microenvironment, directly addressing our central question of understanding its impact on immune cells and tumor progression.

Limitations of Current Research

One significant limitation of the research reviewed is its reliance on animal models such as ICAM1 knockout mice to study the role of ICAM1 in GBM. While these models provide valuable insights into how ICAM1 works within the TME, these models cannot fully capture the complexity of human tumors. This is due to the variations in tumor biology, immune responses, and microenvironmental factors that exist between humans and mouse models. Additionally,

the highly controlled environment in which mouse experiments are conducted can also influence the observed responses. Factors such as diet, housing conditions, and stress levels can significantly impact experimental outcomes and lead to skewing the interpretation of results. To address this limitation, further research employing human-derived models, such as patient-derived cultures could serve to enhance our understanding of the role of ICAM1 in GBM. Integrating advanced imaging techniques like live-cell imaging could provide real-time visualization of ICAM1-mediated interactions within the human TME in these patient derived cultures [29].

Limitations of This Paper

Although this paper provides valuable insights into the role of ICAM1 on immune cells in GBM, it is important to acknowledge several limitations. Firstly, the limited number of papers included in the analysis may restrict the generalizability of the findings and result in gaps in detail. Additionally, relying on in vivo data over in vitro may limit the ability to establish causality and applicability. Furthermore, the exclusion of certain studies due to language barriers could introduce selection bias. Future research should address these limitations by incorporating a broader and diverse sample of studies.

Implications of ICAM1's Dual Role in GBM

The implications of ICAM1's role on immune cells in GBM suggest avenues for therapeutic intervention. By targeting ICAM1-mediated interactions that promote the recruitment and function of pro-tumorigenic immune cells such as TAMs, Tregs, and MDSCs, it may be possible to reduce the immunosuppressive TME and inhibit tumor progression [29]. Conversely, strategies aimed at enhancing ICAM1's influence on anti-tumor immune cells like T-cells and NK cells could enhance their activity and thus the anti-tumor immune response, potentially leading to improved treatment outcomes for GBM patients. This dual approach of targeting pro-tumorigenic ICAM1 interactions while augmenting anti-tumor immune responses holds promise for the development of more effective immunotherapies against GBM. To further utilize these findings in immunotherapy development, future research can delve into evaluating the efficacy of ICAM1-targeted therapies, either alone or in combination with existing treatments, in both animal and human models.

Conclusions

This literature review highlights the role of ICAM1 in GBM. Through its multifaceted interactions with immune cells within the TME, ICAM1 exerts both pro-tumorigenic and anti-tumorigenic effects, shaping tumor progression and immune evasion. Understanding these complexities enhances our knowledge of GBM pathogenesis by revealing the complex relationship between ICAM1 and immune cells, shedding light on the mechanisms driving tumor growth and immune evasion. This deeper understanding also opens avenues for developing targeted therapeutic strategies, as it identifies ICAM1 as a potential key regulator that can be targeted to modulate the TME and improve treatment outcomes for GBM patients. Future research employing human-derived models and advanced imaging techniques is crucial for translating these findings into clinical applications. Continued investigation into ICAM1's role holds promise for improving patient outcomes and advancing the treatment landscape for GBM.

List of Abbreviations Used
GBM: glioblastoma
ICAM1: intracellular adhesion molecule
CNS: central nervous system
WHO: world health organization
TME: tumor microenvironment
ECM: extracellular matrix
TAMs: tumor-associated macrophages
MDSCs: myeloid-derived suppressor cells
NK cells: natural killer cells
CAMs: cell adhesion molecules

Conflicts of Interest
The author declares that they have no conflict of interests.

Ethics Approval and/or Participant Consent
This review did not require any ethics approval or participant consent because no participants were used throughout the research of the article.

Authors' Contributions
AK: contributed to the study topic, collected and analyzed data, drafted the manuscript, and made revisions throughout the process until final approval for submission.

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