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# *Research on the Treatment of Neuro - Oncological Tumors with Targeted Drugs*

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***Abstract:*** Gliomas have a relatively high incidence and a remarkable degree of malignancy. Traditional treatment methods often have adverse effects on the central nervous system during their application. Targeted drugs, as a cutting - edge treatment approach, can precisely act on specific targets of tumor cells and imped the growth, proliferation, survival, and metastasis processes of tumor cells. This article elaborates on the classification of neuro - oncological tumors and targeted drugs and deeply analyzes the mechanism of targeted drugs in the treatment of neuro - oncological tumors from aspects such as inhibiting tumor growth, blocking angiogenesis, regulating iron metabolism, and reducing drug resistance. Targeted drugs have significant advantages in combination therapy. However, currently, this field faces problems such as insufficient target diversity, tumor heterogeneity and drug resistance, toxicity to normal tissues, and high costs. Looking ahead, research on targeted therapy for neuro - oncological tumors should focus on developing new targets, optimizing combination therapy regimens, overcoming drug resistance, and innovating drug delivery systems, providing more solid theoretical basis and practical guidance for the clinical diagnosis and treatment of neuro - oncological tumors.

***Keywords:*** Targeted drugs, gliomas, treatment mechanism.

## 1. Introduction

Neuro - oncological tumors refer to tumors that occur in the nervous system, which includes tumors found in the central nervous system, and peripheral nerves. In recent years, gliomas, as the most common tumors that are primary in the cranial cavity, are malignant brain tumors, accounting for 81%. Although such diseases are relatively uncommon in clinical practice, they can lead to a notable death rate [1]. Among the histological types of gliomas, glioblastoma is the most common, accounting for approximately 45% of all gliomas, and the survival rate of patients within five years is only 5% [1]. Due to its high degree of malignancy and poor prognosis, it has always been a key area of continuous in - depth exploration in the medical field.

Although current cancer treatment methods are diverse, covering traditional treatment methods such as the chemotherapy, radiotherapy, and surgery, it is worth noting that while these traditional treatment methods play a role in combating cancer, they are very likely to cause many adverse effects on the central nervous system. During chemotherapy, chemotherapeutic drugs act on the whole body through the bloodstream. While killing tumor cells, they may also damage nerve cells, glial cells, etc. in the central nervous system, causing adverse reactions such as cognitive impairment and peripheral

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neuropathy. Radiotherapy uses high - energy rays to precisely irradiate the tumor site. However, during the process of the rays penetrating tissues, they inevitably affect the surrounding normal central nervous system tissues, resulting in complications such as radiation brain injury and spinal cord injury. Although surgical treatment may directly remove the tumor tissue, situations such as traction and bleeding during the surgical operation may also cause varying degrees of damage to the structure and function of the central nervous system, thus affecting the recovery of the patient's nerve function.

Compared with traditional treatment, targeted drugs have gradually come into the spotlight, which can precisely act on specific molecular targets of tumor cells, with minor side effects and obvious advantages in efficacy. Currently, targeted drugs are widely used in the treatment of neuro - oncological tumors and are one of the emerging methods. It has the potential to become a more mature technology and a preferred treatment option for patients suffering from neuro - oncological tumors. Given the relatively limited number of relevant academic references in the field of neuro - oncological tumors at present, this article studies the targeted treatment methods in the field of neuro - oncological tumors to further improve the targeted treatment technology, optimize the treatment plan, and provide more theoretical foundation for the diagnosis and therapeutic method of neuro - oncological tumors in the future.

## 2. Classification of neuro - oncological tumors

The classification of neuro - oncological tumors is a complex and evolving field, the classification is mainly based on factors such as the origin of the tumor cell type and the location of the tumor. From the perspective of the type of origin cells, it includes gliomas (which can be further divided into astrocytomas, oligodendrogliomas, etc.), central neurocytomas, embryonic tumors such as medulloblastomas, as well as meningiomas, schwannomas, pituitary adenomas, etc. When classified according to the location of the tumor, it can be divided into intracranial tumors, tumors in the spinal cord, and tumors in the peripheral nerve.

## 3. Classification of targeted drugs and the therapeutic advantages

Targeted drugs can specifically act on specific molecular targets of tumor cells and exert anti - tumor effects by interfering with the growth, proliferation, survival, and metastasis of tumor cells. Currently, targeted drugs are classified according to their mechanism of action, target classification, and drug form:

### 3.1. Mechanism of action of targeted drugs

Analyzed from the perspective of the mechanism of action, anti - tumor drugs cover various types, including the small molecule inhibitors, monoclonal antibodies, and immune checkpoint inhibitors. Small molecule inhibitors include tyrosine kinase inhibitors, proteasome inhibitors, etc. They play a key role in cancer treatment by blocking tyrosine kinase activity or inhibiting proteasome function. Among monoclonal antibody drugs, anti - EGFR antibodies can block its downstream signaling pathway and inhibit tumor cell growth; anti - VEGF antibodies can effectively inhibit tumor angiogenesis and cut off the tumor's nutrient supply; antibody - drug conjugates can greatly reduce drug side effects [2]. In terms of immune checkpoint inhibitors, PD - 1/PD - L1 inhibitors can activate the immune surveillance and killing function of T cells against tumor cells, and CTLA - 4 inhibitors can enhance the attack efficiency of the immune system against tumors (as seen in Table 1).

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Table 1: Classification of targeted drugs according to the mechanism of action

| Type of Anti-tumor drugs | Function | |
|---|---|---|
| Monoclonal Antibodies | Tyrosine Kinase Inhibitors | Can block the activity of tyrosine kinases, inhibit the signal transduction of cell proliferation, and are often used in the treatment of tumors. For example, imatinib is used to treat leukemia. |
| | Proteasome Inhibitors | Inhibit the function of the proteasome, affect the degradation of intracellular proteins, interfere with the growth and survival of tumor cells, and are used in cancer treatment. |
| Monoclonal Antibodies | Anti - EGFR Antibodies | Bind to the epidermal growth factor receptor (EGFR), block its signaling pathway, inhibit the growth of tumor cells, and are used in the treatment of colorectal cancer, etc. |
| | Anti - VEGF Antibodies | Bind to vascular endothelial growth factor (VEGF), inhibit tumor angiogenesis, cut off the nutrient supply of tumors, such as bevacizumab. |
| | Antibody - Drug Conjugates | Link antibodies with cytotoxic drugs. The antibodies target tumor cells, enabling cytotoxic drugs to act precisely on tumors, improving efficacy and reducing side effects. |
| Immune Checkpoint Inhibitors | PD-1/PD-L1 Inhibitors | Block the binding of PD - 1 and PD - L1, restore the immune surveillance and killing function of T cells against tumor cells. |
| | CTLA - 4 Inhibitors | Block the inhibitory signal of CTLA - 4, enhance the activity of T cells, and improve the ability of the immune system to attack tumors. |

### 3.2. Classification by targets

According to the targets, they can be divided into epidermal growth factor receptor inhibitors Epidermal Growth Factor Receptor (EGFR) inhibitors, BRAF inhibitors, vascular endothelial growth factor inhibitors or vascular endothelial growth factor receptor inhibitors (VEGF/VEGFR inhibitors). EGFR inhibitors can effectively inhibit tumor growth and metastasis; BRAF inhibitors can inhibit cancer cell proliferation; VEGF/VEGFR inhibitors aim to cut off the tumor's nutrient supply and suppress tumor growth. (as seen in Table 2).

Table 2: Classification of targeted drugs according to targets

| The Type of Inhibitors | Function |
|---|---|
| EGFR Inhibitors | EGFR (Epidermal Growth Factor Receptor) plays a crucial role in processes such as the proliferation and survival of tumor cells. Inhibitors can block the EGFR signaling pathway, suppressing tumor growth and metastasis. |

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Table 2: (continued)

| | |
|---|---|
| BRAF Inhibitors | BRAF is a protein involved in cell signaling. Certain mutations can promote tumor development. Inhibitors can block the mutated BRAF signals and inhibit the proliferation of cancer cells. |
| VEGF/VEGFR Inhibitors | VEGF (Vascular Endothelial Growth Factor) and its receptor VEGFR can promote tumor angiogenesis. Inhibitors can inhibit angiogenesis, cut off the tumor's nutrient supply, and suppress its growth. |

### 3.3. Classification by targets classification by drug forms

According to the drug form, anti - tumor drugs can be divided into oral drugs and injectable drugs. Different administration forms have their own characteristics and applicable scenarios. Oral drugs are convenient to use. Patients can take them at home by themselves without having to go to medical institutions frequently, which greatly improves medication compliance. Injectable drugs can be further divided into intravenous injection, intramuscular injection, and other methods. Intravenous injection can make the drug quickly enter the bloodstream and reach the tumor lesion rapidly to exert its efficacy. Many monoclonal antibody drugs often use intravenous injection to ensure that the drug acts precisely and efficiently on tumor cells. Intramuscular injection is relatively more suitable for certain anti - tumor preparations with specific requirements for the absorption of the injection site and a small drug dose.

### 3.4. Therapeutic advantages of targeted drugs

Targeted drugs exhibit excellent characteristics in the field of tumor treatment. Firstly, targeted drugs have high selectivity,which can accurately identify and specifically act on specific molecular targets unique to tumor cells. By virtue of a highly specific molecular interaction mechanism, can minimize unnecessary damage to normal cells, thus significantly reducing non - specific toxicity during the treatment process [3]. In terms of efficacy, due to the precise targeting of the key targets on which the growth, proliferation and survival of tumor cells depend, targeted drugs can often inhibit tumor growth efficiently and remarkably. In many clinical studies and practices, it has been fully proved that it can effectively prolong the survival of patients and significantly improve the prognosis of patients. Compared with traditional chemotherapeutic drugs, targeted drugs have obvious advantages in terms of side effects. The side effects are usually relatively mild, which significantly improves the tolerance of patients during the treatment process, is conducive to protecting the quality of life of patients and ensures the smooth progress of treatment [3]. In addition, targeted drugs show great potential in combination therapy strategies,which can be organically combined with various other treatment methods such as chemotherapy, radiotherapy, and immunotherapy. Through the synergistic effect of different mechanisms of action, the overall anti - tumor effect can be further enhanced, providing a more optimized and comprehensive combination treatment plan for tumor patients.

## 4. Main mechanisms of targeted drugs in the treatment of neuro - oncological tumors

### 4.1. Inhibiting tumor growth

Key signaling pathways of brain glioma stem cells (BTSCs), such as SHH and PDGF, are very crucial to tumor growth and drug resistance [4]. Targeted drugs can inhibit the proliferation of BTSCs, induce apoptosis, and inhibit tumor growth by suppressing these pathways (such as blocking SHH signals)

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[5,6]. Another strategy is to induce the differentiation of BTSCs. Bone morphogenetic proteins (BMPs) can reduce the number of tumor stem cells. However, some tumors are insensitive to BMPs due to the absence of the BMP1 receptor or promoter methylation, and demethylation treatment needs to be combined to improve the efficacy.

### 4.2. Inhibiting angiogenesis

BTSCs rely on the "vascular niche" (a special microenvironment formed around blood vessels, which is composed of various cells and extracellular matrix and plays a key regulatory role in cell survival, proliferation, differentiation, and tissue development, repair, and disease occurrence) to maintain survival. Bevacizumab (an anti - VEGF antibody) can inhibit angiogenesis and reduce BTSCs [7]. Research shows that the 1 - year overall survival rates of the bevacizumab group and the placebo group are 72.4% and 66.3% respectively (P = 0.049); the 2 - year overall survival rates are 33.9% and 30.1% respectively (P = 0.24). At the baseline stage, the bevacizumab group was superior to the placebo group in terms of the duration of maintaining health - related quality of life and functional statusand had a lower demand for glucocorticoids [8].

### 4.3. Regulating iron metabolism

Iron metabolism disorder is one of the important causes of cancer. Tumor cells promote growth and metastasis by increasing iron absorption and reducing iron output [9]. In addition, the accumulation of iron in chronic inflammation sites is also considered one of the causes of malignant tumors. Although iron supplementation may promote tumor development and drug resistance, iron supplementation is still necessary because chemotherapy often causing anemia. Therefore, how to regulate the balance of iron in the body has certain potential value for improving the prognosis of advanced cancer.

Current treatments for gliomas, especially glioblastoma, are limited. For example, temozolomide only slightly improves the survival rate. While research shows that inducing ferroptosis (an iron - dependent form of cell death) can enhance the efficacy of temozolomide and improve the treatment effect of neuroblastomas, meningiomas, and gliomas. For example, during the process of temozolomide exerting its therapeutic effect, its toxic effect has a unique mechanism: this toxicity is generated in an xCT/SLC7a11-dependent manner and is promoted through the ferroptosis mechanism [10]. However, it should be noted that drugs that induce ferroptosis (such as sorafenib) and gene manipulation (such as targeting xCT and GPX4) may have an effect on neuronal function and cognitive ability . Nevertheless, ferroptosis is still regarded as a potential strategy for treating drug - resistant glioblastomas, which provides a new direction for the therapeutic method of advanced cancer.

### 4.4. Reducing drug resistance

Brain glioma stem cells (BTSCs) are resistant to radiotherapy and chemotherapy, which greatly affects the effect of conventional treatment. Research shows that after radiotherapy, the survival rate of CD133+ tumor cells is significantly higher than that of CD133 - cells. The reason may be related to the enhanced DNA repair ability of CD133+ cells [11]. Specifically, when cells are damaged by radiotherapy, checkpoint kinases CHK1/CHK2 are preferentially phosphorylated in CD133+ cells. This phosphorylation activates a series of repair mechanisms to help cells repair damaged DNA. Therefore, targeted blocking of CHK1/CHK2 and interfering with its phosphorylation process is expected to be a potential strategy to eliminate the resistance of CD133 + cells, opening up a new direction for brain glioma treatment.

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In addition, there are other targeted treatment methods for neuro - oncological tumors, such as targeting epigenetic factors, such as HDAC and DNA methyltransferase inhibitors, to reverse the abnormal epigenetic modifications of tumor cells; targeting tumor metabolism, such as IDH1/IDH2 and glutamine metabolism inhibitors, to change tumor metabolism and inhibit growth; and regulating the tumor microenvironment, such as inhibiting tumor - associated macrophages and extracellular matrix components to block tumor invasion and immune escape [3]. These strategies provide new directions for the treatment of neuro - oncological tumors through multi - level precise interventions.

## 5. Conclusion

This article reviews the classification of neuro - oncological tumors and targeted drugs, explores the main mechanisms of action and therapeutic advantages of targeted drugs. The main mechanisms through which it acts involve the inhibition of tumor cell proliferation, the induction of tumor cell apoptosis, the inhibition of tumor angiogenesis, the activation of the immune system, and the targeting of tumor stem cells. Targeted drugs can specifically act on relevant targets of tumor cells. Compared with traditional treatments, they reduce damage to normal cells, improve treatment precision, and provide more treatment options for patients with neuro - oncological tumors, especially those with refractory tumors. At the same time, it also promotes a deeper understanding of the tumorigenesis mechanism in the field of neuro - oncology and provides a theoretical and practical basis for subsequent research.

However, although targeted drugs show great potential in the treatment of neuro - oncological tumors, there are still many challenges. For example, the diversity of targets is limited; tumor heterogeneity and drug resistance may lead to the failure of targeted therapy; some targeted drugs still have toxicity to normal tissues; and the research, development, and production costs of targeted drugs are high, making them expensive. With the further application of targeted drugs in the treatment of neuro - oncological tumors, future research should focus on developing new drug targets, optimizing combination therapy strategies, overcoming drug resistance, and developing new drug delivery systems (such as nanocarrier delivery).

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