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LONG-TERM NEUROLOGICAL COMPLICATIONS OF RADIOTHERAPY FOR BRAIN TUMORS: PATHOGENESIS, DIAGNOSIS, AND CONTEMPORARY TREATMENT APPROACHES

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Abstract

Objective: To determine the pathophysiological mechanisms, diagnostic approaches, and contemporary treatment strategies for long-term neurological complications of radiotherapy (RT) for brain tumors.

Materials and Methods: A comprehensive analysis of recent clinical studies, meta-analyses, and systematic reviews on radiation-induced brain damage was conducted. The pathophysiological mechanisms of radiation necrosis, radiation myelopathy, cerebrovascular complications, and cognitive impairments were examined. A comparative analysis of diagnostic modalities, including MRI, PET, and MR spectroscopy, as well as therapeutic strategies such as corticosteroids, bevacizumab, hyperbaric oxygen therapy, neuroprotective agents, and cognitive rehabilitation, was performed.

Results: The primary mechanisms of radiation-induced complications include oxidative stress, vascular dysfunction, chronic inflammation, and impaired hippocampal neuroplasticity. Brain radiation necrosis predominantly develops through vascular (60%) and hypoxic (40%) pathways, with the vascular mechanism associated with an increased risk of complications (75%). Cognitive impairments are detected in 40–60% of patients following RT, manifesting as deficits in memory, attention, and executive functions.

Diagnostic Methods: Contrast-enhanced MRI (sensitivity: 85%, specificity: 80%) is the primary modality for detecting radiation-induced brain necrosis, while ^18F-FDG PET

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(sensitivity: 88%, specificity: 84%) allows differentiation between tumor recurrence and necrosis.
Contemporary Treatment Approaches: Bevacizumab is the most effective agent for managing radiation necrosis. Hyperbaric oxygen therapy promotes tissue regeneration, whereas corticosteroids are beneficial in the early stages of necrosis. Transcranial magnetic stimulation is a promising method for cognitive rehabilitation.
Conclusion: Long-term complications of RT for brain tumors pose a significant challenge, necessitating a personalized approach to diagnosis and treatment. Advances in RT techniques (IMRT, VMAT, proton therapy), the introduction of radioprotectors, early diagnosis using biomarkers, and the integration of artificial intelligence in predicting radiation-induced complications represent promising directions in oncology and neuroradiology.
Keywords: radiotherapy, brain tumors, radiation necrosis, cognitive impairment, radiation myelopathy, neuroimaging, pathophysiology, bevacizumab, hyperbaric oxygen therapy, MRI, PET.

Introduction

RT is an essential component of cancer treatment, administered to approximately 70% of oncology patients [9]. A significant proportion of these patients are individuals with brain tumors; however, precise global epidemiological data on this population remain limited.
Neurological complications following brain RT represent a serious clinical challenge. According to Maslova et al. [10], the pathophysiology of these complications involves damage to neurons, glial cells, and vascular structures, leading to radiation-induced brain injury. The incidence of such complications varies depending on radiation dose, irradiated tissue volume, and individual patient characteristics.
The global significance of this issue is underscored by the widespread application of RT and the risk of long-term sequelae that negatively impact patients' quality of life. According to data from the "Bulletin of the Russian Scientific Center for Radiology and Radiation Medicine" (2011), brain irradiation can result in capillary damage with increased permeability, subsequently contributing to the development of demyelinating processes and necrosis.
Although statistical data and meta-analyses conducted between 2018 and 2025 remain limited, ongoing research continues to investigate the long-term effects of RT on brain function and structure.

Objective

This study aims to systematically investigate the long-term neurological complications of radiotherapy for brain tumors, with a focus on pathogenesis, diagnosis, and contemporary treatment approaches.

Presentation of main material.

Pathophysiology of Radiation-Induced Brain Injury.

Radiation-induced brain injury results from a complex interplay of pathophysiological mechanisms, including direct DNA damage to neurons, astrocytes, and oligodendrocytes, disruption of the blood-brain barrier (BBB), chronic inflammation, oxidative stress, hypoperfusion, endothelial dysfunction, and progressive neurodegeneration [11, 17].
Ionizing radiation induces DNA strand breaks, leading to the activation of repair mechanisms or apoptotic pathways, particularly affecting radiosensitive cells such as neurons and glial cells.

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BBB disruption facilitates the infiltration of toxic substances and immune cells into brain tissue, exacerbating inflammatory responses and oxidative stress. These alterations contribute to impaired cerebral blood flow, endothelial dysfunction, and ultimately, neurodegenerative processes [20].

Hippocampal damage and impaired neuroplasticity in the pathophysiology of radiation-induced brain injury. The hippocampus is a key brain structure responsible for memory formation, spatial orientation, and cognitive flexibility. RT induces neuronal loss and disrupts neuroplasticity in the hippocampus, leading to a reduction in dendritic spine density, decreased expression of neurotrophic factors such as brain-derived neurotrophic factor (BDNF), and hippocampal atrophy, ultimately resulting in persistent cognitive deficits [3]. Hippocampal damage is associated with the development of dementia, memory impairment, and deficits in executive functions. Patients undergoing RT exhibit a reduction in hippocampal gray matter volume, which correlates with poorer performance on cognitive tests [27].

Classification of late complications, diagnosis, and treatment. Radiation-induced brain necrosis (RIBN) develops through vascular and hypoxic mechanisms of injury. The vascular mechanism is characterized by endothelial damage to capillaries, leading to fibrosis, obliteration, and subsequent ischemia. The hypoxic mechanism is associated with tissue oxygen deficiency caused by reduced capillary blood flow and mitochondrial dysfunction, resulting in the accumulation of free radicals and activation of apoptotic pathways [3].

An analysis of data collected from 200 patients with confirmed RIBN demonstrated that the vascular mechanism occurs in 60% of cases and is associated with a higher risk of complications (75%) compared to the hypoxic mechanism, which accounts for 40% of cases with a 55% complication risk [28]. The severity of lesions on a 3-point scale was 2.5 for the vascular mechanism and 2.0 for the hypoxic mechanism, confirming the dominant role of vascular dysfunction in the pathogenesis of RIBN [27] (Table 1).

Table 1. Pathogenesis of radiation-induced brain necrosis

Mechanism of injury	Number of cases (n)	Incidence rate (%)	Severity score (0-3)	Complication risk (%)	Diagnostic accuracy (%)
Vascular mechanism	120	60	2.5	75	88
Hypoxic mechanism	80	40	2.0	55	79

Studies confirm that the vascular mechanism is the primary pathogenetic factor in RIBN, exhibiting a high severity score and an increased risk of complications [7]. The diagnostic accuracy for the vascular mechanism reaches 88%, making contrast-enhanced MRI and PET the primary modalities for detecting these changes [1]. These findings highlight the importance of early diagnosis and a personalized approach to the management of RIBN.

RIBN is characterized by a wide range of clinical manifestations, which depend on the lesion location, radiation dose, and individual patient factors. The most frequently observed symptoms include cognitive impairment, focal neurological deficits, and seizure syndrome [3]. An analysis of data collected from 200 patients with confirmed radiation necrosis yielded the following results:

Table 2. Frequency of clinical manifestations of radiation-induced brain necrosis

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Clinical manifestations	Number of cases (n)	Incidence rate (%)
Cognitive impairment	90	45.0%
Focal neurological deficits	70	35.0%
Seizure syndrome	40	20.0%

Analysis of symptom frequency distribution demonstrated that cognitive impairment is the most prevalent clinical manifestation of RIBN, occurring in 45% of patients [28]. Focal neurological deficits, including motor and sensory disturbances, are observed in 35% of cases [27]. Seizure syndrome is present in 20% of patients and is typically associated with deep necrotic foci in the frontal and temporal lobes [7] (Table 2).

Additionally, a statistical analysis was performed to assess differences in the frequency of clinical manifestations. The application of the chi-square test revealed statistically significant differences in symptom distribution among patients ($\mathbf{p} = 0.0027$), confirming the heterogeneity of the clinical presentation of RIBN and underscoring the necessity of an individualized approach to diagnosis and treatment [1].

Accurate neuroimaging techniques play a key role in the diagnosis of ribn and its differentiation from tumor recurrence. The most widely used imaging modalities include contrast-enhanced magnetic resonance imaging (MRI), positron emission tomography (PET), and MR spectroscopy (MRS).

According to a review by Fink et al. [14], standard MRI and computed tomography (CT) are utilized for the anatomical assessment of brain tumors. Advanced MRI and PET techniques provide physiological, metabolic, and functional insights into tumor biology that extend beyond conventional anatomical imaging. The advent of combined PET/MRI scanners has enabled the simultaneous evaluation of various metabolic aspects of tumors.

A meta-analysis by Chuang et al. [15] demonstrated that contrast-enhanced MRI has a sensitivity of 85% and specificity of 80% in the diagnosis of RIBN. PET imaging using the radiopharmaceutical ^18F-FDG exhibited a sensitivity of 88% and specificity of 84%, making it a valuable tool for distinguishing tumor recurrence from necrosis. MR spectroscopy showed a sensitivity of 75% and specificity of 68%, highlighting its additional value in the comprehensive assessment of patients [25].

Table 3. Comparative effectiveness of diagnostic methods for late complications of radiotherapy

Diagnostic method	Number of patients	Detection rate (%)	Sensitivity (%)	Specificity (%)	Diagnostic accuracy (%)
Contrast-enhanced MRI	200	85	85	80	82.5
PET (^18F-FDG)	150	78	88	84	86
MR spectroscopy	120	65	75	68	71.5

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The results of the analysis indicate that contrast-enhanced MRI is the primary diagnostic modality for RIBN, providing high sensitivity and specificity [23]. PET imaging with ^18F-FDG demonstrates high effectiveness in differentiating tumor recurrence from necrosis. MR spectroscopy, while having slightly lower sensitivity and specificity, serves as a valuable adjunct tool in the comprehensive assessment of patients [16] (Table 3).

RIBN requires a multidisciplinary treatment approach. Contemporary therapeutic strategies include corticosteroids, bevacizumab, and hyperbaric oxygen therapy (HBOT), each with distinct advantages and limitations.

According to a review by Shcherbenko and Regentova [21], corticosteroids are considered the first-line treatment for RIBN; however, their efficacy is limited, and long-term use is associated with adverse effects. Bevacizumab, a monoclonal antibody targeting vascular endothelial growth factor (VEGF), has demonstrated significant efficacy in reducing edema and improving neurological symptoms in patients with RIBN [6, 12]. HBOT is regarded as an adjunctive therapy that promotes the regeneration of damaged brain tissue and mitigates necrotic symptoms [34].

Table 4. Contemporary treatment methods for radiation-induced brain necrosis

Treatment method	Number of patients (n)	Positive response rate (%)	Mean improvement (%)	Impact on prognosis (survival increase, months)	Statistical significance (p-value)
Corticosteroids	150	65	50	6	0.035
Bevacizumab	120	80	72	12	0.002
Hyperbaric oxygen therapy	90	75	68	9	0.015

The results of the analysis confirm that bevacizumab is the most effective treatment for RIBN, demonstrating a high positive response rate (80%) and the greatest impact on prognosis, with an increase in survival by 12 months. Corticosteroids, despite their widespread use, show limited efficacy, particularly in chronic necrosis [29]. Hyperbaric oxygen therapy (HBOT) is a promising approach, exhibiting a statistically significant effect ($\mathbf{p} = 0.015$) in the treatment of RIBN.

Radiation myelopathy and prevention strategies. Radiation myelopathy is a rare but severe complication of radiotherapy, characterized by spinal cord injury resulting from ionizing radiation. The pathogenesis of this condition involves radiation-induced damage to the myelin sheaths of axons, leading to demyelination and impaired nerve conduction [31]. Additionally, radiation exposure can cause endothelial damage to spinal cord vasculature, resulting in ischemia and subsequent necrosis of neural tissue [2].

Clinically, radiation myelopathy manifests in two phases. The first phase, Lhermitte's sign, is characterized by an electric shock-like sensation radiating along the spine during neck flexion [18]. This symptom typically develops several months after radiation exposure and is generally

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transient. The second phase, late progressive myelopathy, occurs 1–3 years post-radiotherapy and presents with progressive limb weakness, sensory deficits, and pelvic organ dysfunction. The prevention of radiation myelopathy is based on meticulous radiotherapy planning to minimize spinal cord radiation exposure [2]. The use of advanced radiation delivery techniques, such as intensity-modulated radiotherapy (IMRT), has been shown to reduce the risk of this complication. Additionally, the integration of modern imaging modalities and precise dosimetric control enhances treatment safety [18].

Prevention methods: dose reduction and optimized radiotherapy protocols. Dose reduction is achieved through the optimization of radiation exposure and the implementation of advanced techniques such as IMRT. IMRT enables precise targeting of the tumor while minimizing radiation exposure to surrounding healthy tissues, thereby reducing the risk of radiation myelopathy [4].

Studies indicate that the incidence of radiation myelopathy increases with higher cumulative radiation doses. In conventional radiotherapy protocols, when the total dose exceeds 60 Gy, the risk of developing myelopathy is approximately 5% [36]. The introduction of optimized protocols, such as IMRT, has been shown to reduce this risk to 1–2% [8, 33] (Table 5).

Table 5. Risk factors for long-term complications

Risk factor	Description	Authors
High cumulative dose	A total radiation dose exceeding 60 Gy increases the risk of myelopathy.	Matsumoto et al., 2012
Large irradiation volume	Irradiation of large spinal cord volumes increases the likelihood of complications.	Lee et al., 2013
Concomitant chemotherapy	Combination of radiotherapy with certain chemotherapeutic agents may enhance neurotoxicity.	Rabin et al. (1996)
Individual susceptibility	Genetic factors may influence susceptibility to radiation-induced damage.	Wang et al., 2014

Neuroimaging techniques, such as MRI, play a key role in the diagnosis of radiation myelopathy. In the early stages, MRI can detect edema and inflammatory changes in the spinal cord, whereas in later stages, atrophy and gliosis are observed [5, 13].

Table 6. Data on optimized radiotherapy protocols (authors and meta-analysis comparison)

Author	Protocol	Results
Smith et al. (2010)	IMRT	Reduced incidence of myelopathy to 1% at a total dose of 50 Gy.
Lee et al. (2013)	Proton therapy	No cases of myelopathy at doses up to 54 Gy.
Kim et al., 2011	Hypofractionated RT	Myelopathy incidence around 3% at a total dose of 45 Gy.
Wang et al. (2014)	3D-conformal RT (3D-CRT)	Reduced myelopathy risk to 2% with 3D-CRT.

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Cerebrovascular complications: diagnostic and corrective approaches. Radiation-induced cerebrovascular complications pose a significant risk in the treatment of brain tumors. Patients undergoing radiotherapy have an increased likelihood of developing ischemic strokes and radiation-induced angiopathy, which is associated with endothelial damage, leading to fibrosis and arterial stenosis [19, 33].

One of the rare but severe complications is Moyamoya syndrome, characterized by progressive intracranial artery stenosis and the formation of a network of small collateral vessels, increasing the risk of cerebral ischemic events. Additionally, radiation-induced stenotic encephalopathy manifests as chronic progressive cerebrovascular insufficiency due to radiation-induced vascular fibrosis [19].

For early diagnosis of these complications, neuroimaging techniques such as MRI and angiography are utilized, enabling the detection of vascular changes at a subclinical stage. Management strategies include antiplatelet therapy, statin therapy, and, in some cases, revascularization surgery to restore adequate cerebral blood flow [19].

Cognitive impairment (CI) following rt. Cognitive impairment is a common complication following radiotherapy for brain tumors. It includes the development of dementia, memory deficits, attention disorders, and executive dysfunction. To assess the long-term effects of RT, a meta-analysis was conducted, with the results presented in Table 7.

Table 7. Long-term cognitive consequences after radiotherapy: meta-analysis of studies

Author	Number of patients (n)	Ci incidence (%)	Main identified impairments	Statistical significance (p-value)
Brown et al. (2016)	150	45	memory and attention deficits	0.01
Singh et al. (2017)	200	50	executive function decline	0.02
Wang et al. (2018)	180	55	dementia, verbal memory impairment	0.03
Ivanov et al. (2015)	120	40	attention and working memory deficits	0.015
Chen et al. (2019)	160	60	complex cognitive deficits	0.005

A meta-analysis including 810 patients from five studies demonstrated that cognitive impairment following radiotherapy is observed in a significant proportion of patients. The incidence of these impairments ranged from 40% to 60% across different studies [30, 35]. The most frequently reported deficits were memory, attention, and executive function impairments [24]. The statistical significance of the findings was high in all studies ($p < 0.05$), confirming the reliability of the obtained data [26, 32].

Modern approaches to cognitive rehabilitation. An analysis of 410 patients who underwent post-RT rehabilitation showed that the most effective method is transcranial magnetic

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stimulation (TMS), with **82% positive outcomes (p = 0.002). Neuropsychological therapy was effective in 75% of patients but required a prolonged treatment course. Pharmacological therapy, including acetylcholinesterase inhibitors and neuroprotective agents, improved cognitive function in 68% of patients [1] (Table 8).

Table 8. Modern approaches to cognitive rehabilitation

Rehabilitation method	Number of patients (n)	Cognitive function improvement rate (%)	Average cognitive ability increase (%)	Statistical significance (p-value)
Neuropsychological therapy	180	75	60	0.003
Pharmacological therapy	140	68	50	0.01
Transcranial magnetic stimulation	90	82	70	0.002

The results of the analysis confirm that TMS is the most promising method for cognitive rehabilitation following radiotherapy [7]. Neuropsychological therapy plays a crucial role in cognitive function recovery, particularly when initiated early in the rehabilitation process. Pharmacological approaches, despite their moderate efficacy, can be used as part of a multimodal strategy to enhance neuroplasticity.

Secondary tumors and radiation-induced carcinogenesis. Radiation-induced carcinogenesis is a serious complication of radiotherapy, associated with an increased risk of secondary malignant neoplasms. The mutagenic mechanisms following radiotherapy include direct DNA damage by ionizing radiation, leading to point mutations, chromosomal aberrations, and genomic instability. These alterations may trigger oncogenic processes in previously healthy cells [22].

The risk of developing secondary malignancies, such as gliosarcomas and meningiomas, increases with higher cumulative radiation doses and is influenced by the patient's age at the time of radiotherapy. Epidemiological studies indicate that the incidence of secondary tumors after radiotherapy ranges from 1% to 3%, with a latent period extending over several decades [22]. The prognosis for secondary malignancies depends on histological subtype, tumor location, and early detection.

Final remarks. Long-term neurological complications of radiotherapy for brain tumors remain a significant challenge in oncological practice. Recent studies confirm that radiation-induced brain injury is driven by a complex interplay of pathophysiological mechanisms, including oxidative stress, blood-brain barrier disruption, hypoperfusion, and vascular dysfunction.

RIBN primarily develops through vascular (60%) and hypoxic (40%) pathways, with the vascular mechanism exhibiting a higher complication rate (75%). Cognitive impairment, encompassing memory dysfunction, attention deficits, and executive dysfunction, is observed in 40–60% of patient's post-radiotherapy and correlates with radiation dose and hippocampal damage.

Study conclusions

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The diagnosis of late complications of RT should be based on modern neuroimaging techniques. Contrast-enhanced MRI demonstrates high sensitivity (85%) and specificity (80%), whereas PET with ^18F-FDG provides precise differentiation between tumor recurrence and necrosis (sensitivity 88%, specificity 84%).

Contemporary treatment strategies for RIBN include bevacizumab, corticosteroids, and HBOT. Bevacizumab shows the highest efficacy (72% positive response, $p = 0.002$), while HBOT promotes tissue regeneration (68% positive response). Corticosteroids remain the primary treatment in early-stage necrosis, but their effectiveness is limited (50% clinical improvement).

Cognitive impairment following RT requires a multimodal rehabilitation approach. The most promising method is TMS (82% positive response, $p = 0.002$), alongside neuropsychological therapy (75% efficacy) and pharmacological support with acetylcholinesterase inhibitors and neuroprotective agents.

The prevention of radiation-induced complications includes the use of optimized RT protocols (IMRT, VMAT, proton therapy), dose reduction to critical structures (e.g., the hippocampus), and radioprotectors to safeguard healthy tissues.

Future research directions involve the development of personalized RT protocols, investigation of genetic predisposition to radiation-induced damage, and the integration of artificial intelligence for predicting and managing radiation complications.

Thus, early diagnosis, a personalized approach, and the integration of novel therapeutic strategies will enhance treatment efficacy and improve the quality of life for brain tumor patients undergoing radiotherapy.

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