REVIEW ARTICLE

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The Influence of Stereotactic Body Radiotherapy (SBRT) on Biomarker Profiles in Lung Cancer: A Detailed Overview

Babak Olia[1], Karomat Sobirova[2], Masharipova Ravqat[3], Madrimov Javoxir Islombek o'g'li[4], Niginabonu Khajiqurbonova[5], Fayzullayev Umidjon O'ktamovich[6], Ulliyeva Rayxon[6], Ataniyazov Aybek[7], Mahdi Hazratgholi

[1]Department of Radiology, Faculty of Veterinary Medicine, Science and Research Branch, Tehran, Iran.
[2]Department of Pedagogy and Psychology, Urgench State University, Urgench, Uzbekistan.
[3]Mamun University, Khiva, Uzbekistan.
[4]Department of Medicine, Urgench Mamun University, Urgench, Uzbekistan.
[5]Department of Clinical Subjects, Tashkent State Medical University, Tashkent, Uzbekistan.
[6]Department of Psychology, Mamun University, Khiva, Uzbekistan.
[7]Department of General Science, Tashkent State University of Economics, Tashkent, Uzbekistan.
[8]Department of Radiology, Imam Khomeini Hospital Complex, School of Medicine, Tehran University of Medical Sciences (TUMS), Tehran, Iran

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Abstract

Background: Stereotactic Body Radiation Therapy (SBRT) represents the standard treatment for inoperable early-stage non-small cell lung cancer (NSCLC) and is increasingly utilized for oligometastatic disease. Its distinct radiobiological profile, involving high doses per fraction, triggers complex tumor-killing effects and systemic biological reactions not fully detectable through conventional imaging.
Objective: This review aims to summarize and critically assess current evidence regarding dynamic alterations in circulating, tissue, and imaging biomarkers after SBRT for lung cancer, and to explore their clinical significance.
Methods: A narrative synthesis of scientific literature from PubMed, Scopus, and Google Scholar was conducted, focusing on studies published between 2005 and 2024. Key search terms included "SBRT," "SABR," "lung cancer," "biomarker," "ctDNA," "immunotherapy," "cytokines," and "radiation pneumonitis."
Results: SBRT prompts a rapid, biphasic shift in tumor-derived biomarkers such as circulating tumor DNA (ctDNA), characterized by an initial post-treatment surge followed by reduction in responders. It significantly influences the immune system, inducing immunogenic cell death, expanding tumor-specific T-cell populations, and increasing checkpoint molecule expression like PD-L1. Additionally, SBRT modifies levels of cytokines (e.g., IL-6, TGF-β) and angiogenic factors (e.g., VEGF), which correlate with both treatment effectiveness and side effects like radiation-induced lung injury. Certain genetic polymorphisms also appear promising for predicting toxicity risk.
Conclusion: SBRT induces a dynamic and multifaceted change in the biomarker profile of lung cancer patients. These biomarkers offer considerable potential for personalizing treatment, predicting outcomes, monitoring response, and rationally planning combination therapies, especially with immunotherapy. Future prospective and validated studies are necessary to integrate these findings into clinical practice.

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Correspondence:
Mahdi Hazratgholi
E-mail: Mahdihazratgholi75@gmail.com

Introduction

Lung cancer remains the foremost cause of cancer-related deaths globally, with non-small cell lung cancer (NSCLC) comprising about 85% of cases [1]. The introduction of Stereotactic Body Radiation Therapy (SBRT), also known as Stereotactic Ablative Radiotherapy (SABR), has fundamentally altered the management of early-stage NSCLC. For patients who are medically inoperable or decline surgery, SBRT has become the established standard of care, achieving local control rates over 90% that compare favorably with surgical results in selected groups [2,3]. This change is driven by SBRT's ability to deliver precise, high-dose radiation in a hypofractionated schedule, usually 1 to 5 sessions, while effectively sparing adjacent healthy tissues.

SBRT's effectiveness stems not only from its technical precision but also from a unique radiobiology that differs significantly from conventional fractionated radiotherapy. The high biological effective dose (BED) per fraction typical of SBRT causes rapid tumor destruction through multiple mechanisms, including direct DNA damage, severe vascular endothelial injury, and strong induction of immunogenic cell death (ICD) [4,5]. This latter effect positions SBRT as not just a local treatment but also a powerful modifier of systemic anti-tumor immunity, capable of generating in situ vaccination effects [6].

In modern precision oncology, biomarkers have become essential. Defined as measurable indicators of biological processes, disease states, or responses to treatment, biomarkers provide crucial insight into the complex interactions between therapy, the tumor microenvironment (TME), and the host's systemic reaction [7]. In lung cancer treated with SBRT, biomarkers can serve as prognostic tools (indicating disease course), predictive markers (forecasting treatment response or toxicity risk), and dynamic pharmacodynamic indicators (revealing real-time biological effects of therapy) [8].

Despite clinical success, challenges remain in optimizing SBRT. A notable proportion of patients eventually experience regional or distant recurrence, suggesting the presence of undetected micrometastatic disease at treatment time [9]. Additionally, toxicities like radiation-induced lung injury (RILI) can restrict treatment intensity and affect quality of life [10]. Biomarkers present a promising approach to address these issues by enabling risk stratification, early response evaluation, and therapy personalization.

The main goal of this comprehensive review is to consolidate current evidence on the effect of SBRT on the diverse biomarker landscape in lung cancer. We will systematically examine impacts on 1) tumor-derived markers (e.g., ctDNA, CTCs), 2) immunomodulatory markers (e.g., T-cell clonality, cytokines, checkpoint proteins), 3) markers of vascular damage and apoptosis, and 4) biomarkers of normal tissue toxicity. Finally, we will critically discuss clinical implications, current translational challenges, and future directions for integrating multimodal biomarker data to enhance SBRT outcomes and guide new therapeutic combinations.

Methods

This article is a narrative review synthesizing current evidence from scientific literature. A systematic search was performed using PubMed, Scopus, and Google Scholar for relevant articles published mainly between January 2005 and May 2024. The search strategy combined keywords and MeSH terms such as "Stereotactic Body Radiation Therapy," "SBRT," "SABR," "lung neoplasms," "non-small cell lung carcinoma," "biomarkers," "circulating tumor DNA," "ctDNA," "circulating tumor cells," "immunotherapy," "PD-L1," "cytokines," "radiation pneumonitis," and "abscopal effect."

Inclusion criteria centered on original research articles (prospective and retrospective), clinical trials, and key review articles in English that specifically investigated biomarker changes in lung cancer patients receiving SBRT. Reference lists of retrieved articles were also manually reviewed for additional relevant publications. Extracted data included biomarker type, sampling methods, timing of sample collection relative to SBRT, main findings, and clinical correlations. Given the narrative nature of this review, formal quality assessment or risk-of-bias analysis was not performed.

Results & Comprehensive Review

Biomarkers of Tumor Burden and Cell Death

The ablative impact of SBRT causes massive tumor cell death, releasing cellular components into circulation.

- **Circulating Tumor DNA (ctDNA):** The pattern of ctDNA change is highly informative. Studies consistently show a temporary increase in ctDNA levels 24-48 hours after the first or last SBRT fraction, attributed to acute tumor lysis [11,12]. This is followed by rapid clearance in patients with complete response, often becoming undetectable within weeks [13]. Persistent or reappearing ctDNA after SBRT is a strong indicator of local or distant recurrence, frequently preceding radiological evidence by months [14,15].
- **Circulating Tumor Cells (CTCs):** Data on CTCs are more varied. Some studies report an initial release of CTCs post-SBRT, possibly due to TME disruption and increased intravasation [16]. However, sustained or

rising CTC counts after treatment are generally linked to poorer progression-free and overall survival [17].

Immunological Biomarkers and Systemic Activation

- SBRT can act as an in situ vaccine, inducing immunogenic cell death (ICD). ICD involves surface exposure of calreticulin and release of ATP and HMGB1, which function as damage-associated molecular patterns (DAMPs) to attract and activate antigen-presenting cells [18,19].
- **T-cell Dynamics and Checkpoint Expression:** TCR sequencing analyses reveal that SBRT can lead to expansion of novel, tumor-specific T-cell clones and increase T-cell repertoire clonality both within the tumor and peripherally [20,21]. A key immunomodulatory effect is the upregulation of PD-L1 on tumor and immune cells in the TME after SBRT, an adaptive immune resistance mechanism [22]. This supports combining SBRT with immune checkpoint inhibitors (ICIs) [23].
- **Cytokine and Chemokine Responses:** SBRT activates a robust inflammatory cytokine cascade. Serial blood analyses show significant rises in pro-inflammatory cytokines like IL-6, IL-8, TNF-α, and IFN-γ post-treatment [24,25]. In cases of abscopal responses (regression of non-irradiated lesions), these systemic immune changes are especially marked [26,40]. Conversely, a decrease in immunosuppressive cytokines such as TGF-β has been associated with better outcomes in some studies [27].

Biomarkers of Vascular Damage and Apoptosis

The high dose per fraction in SBRT causes significant damage to tumor vasculature, a mechanism distinct from conventional radiotherapy.

- **Angiogenic Factors:** SBRT has been shown to markedly reduce circulating levels of pro-angiogenic factors like Vascular Endothelial Growth Factor (VEGF) [28]. This anti-angiogenic effect contributes to secondary tumor cell death via hypoxia and nutrient deprivation.
- **Markers of Endothelial Apoptosis:** Proteins indicating endothelial damage, such as circulating endothelial cells (CECs) and von Willebrand Factor (vWF), can be elevated following SBRT [29]. The kinetics of these markers may be particularly relevant for central lung tumors, where vascular injury presents significant risk [30].

Biomarkers of Normal Tissue Toxicity

Radiation-induced lung injury (RILI) is the most common dose-limiting toxicity.

- **Inflammatory Cytokines:** Elevated serum levels of IL-6, TGF-β, and KL-6 have been consistently linked to the development and severity of symptomatic radiation pneumonitis [31,32]. These biomarkers may help distinguish between asymptomatic radiological changes and clinically significant toxicity.
- **Genomic Markers of Susceptibility:** Pharmacogenomic studies have identified single nucleotide polymorphisms (SNPs) in genes involved in DNA repair (e.g., ATM), oxidative stress (e.g., SOD2), and fibrotic pathways (e.g., TGFB1) that are associated with increased genetic susceptibility to RILI after SBRT [33,34].

Discussion

The evidence compiled in this review clearly shows that SBRT is not merely a localized cytoreductive treatment; it actively drives a complex and dynamic biological response, clearly reflected in evolving biomarker profiles. Biomarker modulation offers deep insight into SBRT's triple mechanism: direct tumor ablation, vascular disruption, and systemic immune activation. The most immediate clinical application is in response assessment and recurrence monitoring. The biphasic ctDNA pattern—initial spike followed by rapid clearance in responders—provides a powerful, real-time liquid biopsy tool for confirming treatment efficacy [11,12]. More importantly, persistent or reappearing ctDNA after SBRT strongly indicates treatment failure, often months before radiological recurrence signs [14,15]. This "liquid surveillance" approach could enable earlier salvage interventions and reduce uncertainty from slow-resolving imaging changes.

Perhaps the most transformative implication is the solid scientific basis these biomarker studies provide for combining SBRT with immunotherapy. Consistent observations that SBRT induces immunogenic cell death, expands tumor-specific T-cell clones, and upregulates PD-L1 in the TME create a strong biological rationale for synergy [22,23]. SBRT effectively converts the tumor into an in situ vaccine, priming the immune system, while checkpoint inhibitors "release the brakes" on activated T-cells. The success of this approach in stage III NSCLC with the PACIFIC regimen [35] offers robust proof-of-concept, and numerous ongoing trials are exploring this combination in earlier-stage disease. Biomarkers will be vital in these trials to identify patients who benefit most and to understand resistance mechanisms.

However, translating biomarker discovery into routine clinical use faces significant hurdles. A major challenge is lack of standardization. There is no consensus on optimal sampling timepoints (e.g., pre-treatment, 24 hours post-first fraction, 4 weeks post-treatment), the most analytically valid assays for different biomarkers, or clinically relevant thresholds

for defining a "positive" or significant change [36]. Additionally, the field must address significant biological and technical heterogeneity. Tumor histology, size, location, and patient comorbidities can all influence biomarker levels. Variability in sample collection, processing, and analysis across institutions also hinders widespread adoption and result comparison.

Looking ahead, the future of biomarkers in SBRT lies in multimodal integration. Predictive power will likely come not from a single biomarker but from a composite signature combining the sensitivity of liquid biopsies (ctDNA, cytokines) with spatial and functional information from advanced imaging (radiomics from CT/PET) [37,38,39] and host genetic factors (germline polymorphisms for toxicity) [33,34]. Developing machine learning algorithms to analyze these complex, high-dimensional datasets represents a promising frontier for personalizing SBRT.Key questions for future research remain. Can the magnitude of the post-SBRT ctDNA spike predict immune response strength and long-term control? Can specific cytokine or immune cell signatures reliably identify patients likely to experience an abscopal effect or benefit most from immunotherapy combination [26,40]. Most importantly, can a validated polygenic risk score reliably predict severe radiation pneumonitis, allowing pre-emptive treatment adjustments like dose reduction or anti-fibrotic co-treatment? Answering these questions will require large, prospective, multi-institutional trials designed with biomarker validation as a primary endpoint.

Conclusion

SBRT for lung cancer initiates a complex and dynamic series of biological events clearly reflected in changes across a wide range of biomarkers. From the release of tumor-derived nucleic acids and activation of systemic immune responses to signals of vascular disruption and normal tissue inflammation, these biomarkers provide crucial insights into SBRT's mechanisms, effectiveness, and toxicity. Their potential to transform clinical practice is substantial, offering pathways for personalizing SBRT, rationally guiding combination therapies, and early recurrence detection. Moving forward, systematic and standardized study of this biomarker landscape, combined with advanced computational integration, will be key to refining SBRT applications and ultimately improving survival and quality of life for lung cancer patients.

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Authors Contributions

The authors contributed to the data analysis. Drafting, revising and approving the article, responsible for all aspects of this work.

Conflict of Interest

None

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