

Research progress on SBRT combined with ICIs in the treatment of solid tumors

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Abstract. Stereotactic Body Radiotherapy (SBRT) is a precise radiotherapy technique characterized by the delivery of high-dose radiation to limited tumor targets within a small number of fractions, and has been shown to achieve effective local tumor control in several solid tumors. These factors, particularly an immunosuppressive Tumor Microenvironment (TME), may limit the therapeutic efficacy of PD-1 inhibitors. The combination of SBRT and PD-1 blockade has shown potential benefits in improving tumor response, local control, and progression-free survival in selected patients; however, systemic antitumor responses remain inconsistent and need further validation. Mechanistically, this strategy is biologically plausible because SBRT can induce tumor cell death, facilitate tumor antigen release, activate dendritic cells, and promote cytotoxic T-cell infiltration. However, this combined strategy is still not fully standardized because existing studies differ in tumor type, radiation dose, fractionation schedule, treatment sequence, irradiated target selection, and biomarker evaluation. This review provides an overview of the underlying mechanisms, clinical evidence, safety profiles, current limitations, and future perspectives regarding the combination of SBRT and Immune Checkpoint Inhibitors (ICIs) in solid tumor therapy.

Keywords: Stereotactic Body Radiotherapy, Immune Checkpoint Inhibitors, PD-1/PD-L1 blockade, immunogenic cell death, Tumor Microenvironment

1. Introduction

Solid tumors remain a major clinical challenge, especially in advanced or metastatic settings, where durable disease control is still difficult to achieve [1]. In this context, combining local ablative therapy with immune checkpoint blockade has gradually become an important research direction. A randomized phase II trial in oligometastatic solid tumors evaluated SBRT with systemic pembrolizumab and found that this strategy was feasible, although further optimization was still needed [2]. Consistently, a systematic review of metastatic cancers reported limited response rates with PD-1/PD-L1 inhibitor monotherapy in many tumor types, supporting the rationale for combined treatment approaches [3]. Evidence from specific tumor settings has further suggested the potential value of this approach. In unresectable hepatocellular carcinoma, a multicenter propensity score-matched study indicated that SBRT plus PD-1 inhibitors may prolong progression-free survival compared with SBRT alone [4]. Related reviews have also proposed that radiotherapy may enhance the efficacy of ICIs by promoting tumor antigen presentation, strengthening immune-mediated antitumor

responses, and remodeling the TME [5]. In Non-Small Cell Lung Cancer (NSCLC), this combination has attracted increasing attention because SBRT may improve tumor immunogenicity, increase immune cell infiltration, and create a TME that is more favorable to immune checkpoint blockade [6]. For patients with resectable NSCLC, this neoadjuvant strategy has also been proposed to enhance systemic antitumor immunity and reduce recurrence risk associated with micrometastases [7]. Clinical observations in other malignancies provide additional but still preliminary support. In a case report of metastatic triple-negative breast cancer, this combined regimen was associated with partial responses in both irradiated and non-irradiated lesions [8]. A phase Ib study in patients with refractory solid tumors showed that SBRT combined with cadonilimab, a bispecific antibody targeting PD-1 and CTLA-4, had manageable toxicity and showed early antitumor activity in a heavily pretreated population [9]. In stage IV NSCLC, this strategy may be particularly relevant for oligometastatic disease; however, the optimal patient population, treatment sequence, dose-fractionation pattern, and safety profile still require further validation [10].

2. Biological rationale for combining SBRT with PD-1 inhibitors

2.1. Direct tumor killing and antigen release

SBRT can exert direct tumoricidal effects through the induction of DNA damage, especially DNA double-strand breaks [3, 6]. Radiation-mediated tumor cell injury can promote the release of tumor-associated antigens and damage-associated molecular patterns, which together contribute to the initiation of antitumor immune responses [3, 5]. Released tumor antigens provide substrates for antigen presentation, while damage-associated signals promote dendritic-cell activation, together contributing to the initiation of T-cell-mediated antitumor immunity [3, 5]. Compared with conventional radiotherapy, SBRT delivers higher doses per fraction and may provide stronger immune modulation under selected dose-fractionation schedules, although immune activation induced by radiation does not increase in a strictly dose-dependent linear manner [6, 7].

2.2. Activation of immune pathways

The cGAS-STING pathway provides an important link between radiation-induced cytoplasmic DNA and type I interferon production, which is closely related to dendritic-cell activation [3, 12]. In this process, type I interferon signaling may enhance antigen-presenting function and support the priming of tumor antigen-specific cytotoxic T lymphocytes [3, 12]. SBRT may also trigger immunogenic cell damage and increase immune-related signals such as calreticulin, HMGB1, ATP, and heat-shock proteins [5, 7]. Together, these changes may increase CD8⁺ T-cell infiltration and shift the tumor immune microenvironment toward a more inflammatory state [5, 7].

2.3. Synergy with PD-1 blockade

By restoring the activity of exhausted T cells, PD-1 inhibitors may help maintain antitumor immune responses triggered by radiotherapy [3, 6]. Radiation-induced PD-L1 upregulation in tumor and immune cells also provides a biological explanation for combining SBRT with PD-1/PD-L1 blockade [6]. A recent study further reported that radiotherapy with PD-L1 blockade enhanced immune activity in distant non-irradiated tumors and contributed to the abscopal effect, partly by reshaping immune-cell states within the TME [11]. These findings suggest that this approach may improve local tumor control while also generating systemic antitumor effects beyond the irradiated field [1, 8, 11]. However, regression of non-irradiated lesions remains uncommon

in clinical practice, so the abscopal effect should be interpreted as a potential but unpredictable outcome rather than an expected therapeutic result [3, 8, 11].

3. Clinical evidence in hepatocellular carcinoma

3.1. Supportive evidence from metastatic hepatocellular carcinoma

Hepatocellular carcinoma has emerging clinical and preclinical evidence supporting the combination of radiotherapy, especially SBRT, with PD-1 blockade [1, 4]. In one metastatic hepatocellular carcinoma study, a clinical cohort compared patients receiving IMRT plus PD-1 inhibitors, immunotherapy alone, or no treatment [1]. As shown in Table 1 and Figure 1, the combination therapy significantly prolonged median survival compared with immunotherapy alone or no treatment (17.5 vs. 12.5 vs. 5.2 months, $p < 0.001$) [1]. These findings suggest that radiotherapy combined with immunotherapy may improve survival in metastatic hepatocellular carcinoma; however, the radiotherapy modality used in this clinical cohort was IMRT, not SBRT [1]. To further evaluate whether SBRT could produce similar or enhanced effects, the same study conducted an animal experiment in a mouse model of metastatic hepatocellular carcinoma [1]. Mice were divided into control, anti-PD-1 antibody alone, SBRT alone, and SBRT plus anti-PD-1 antibody groups, with SBRT delivered at 8 Gy $\times$ 3 fractions [1]. As summarized in Table 2, the combination of SBRT and anti-PD-1 antibody achieved superior tumor control at both irradiated and non-irradiated (abscopal) sites, which was associated with increased infiltration of effector CD8⁺ T lymphocytes and enhanced antitumor immune responses [1]. Notably, pathological assessment showed no evident treatment-related injury in major organs, including the liver, spleen, lung, kidney, and heart, after this combined treatment [1].

Table 1. Clinical cohort: survival outcomes by treatment group

Treatment group	RT modality	Patients	Median survival
IMRT+PD-1 inhibitor	IMRT	13	17.5 months
PD-1 inhibitor alone	None	12	12.5 months
No RT or immunotherapy	None	20	5.2 months

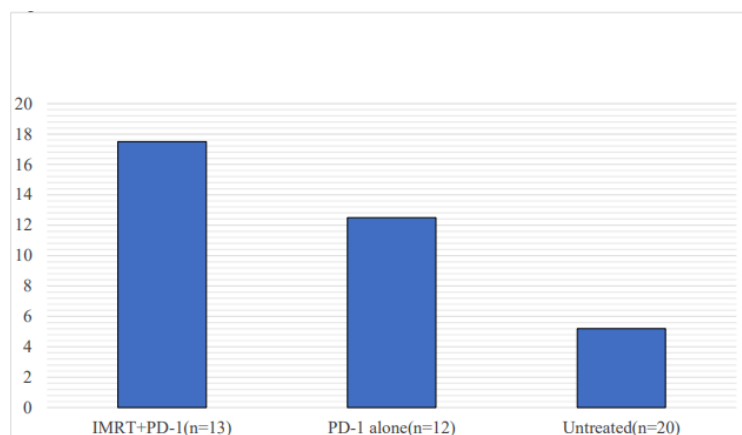


Figure 1. Median overall survival in the clinical cohort

Table 2. Preclinical mouse study: experimental groups and SBRT regimens

Group	SBRT regimen	Key Observation
Control	None	Reference control
Anti-PD-1 antibody	None	Immunotherapy alone
SBRT	8 Gy $\times$ 3 fractions	Local radiotherapy alone
SBRT+anti-PD-1	8 Gy $\times$ 3 fractions	Best local and distant tumor control

3.2. Synergy with PD-1 blockade

A multicenter propensity score-matched study further evaluated this approach in patients with unresectable hepatocellular carcinoma by comparing it with SBRT alone [4]. Before matching, 540 eligible patients were included, of whom 157 received this approach and 383 received SBRT alone. After 1:1 propensity score matching, 314 patients remained, with 157 patients in each group [4]. The main efficacy and safety outcomes are summarized in Table 3.

Table 3. Clinical outcomes of this approach versus SBRT alone in unresectable hepatocellular carcinoma

Outcome	This approach	SBRT alone	Main finding
Patients before matching	157	383	540 eligible patients were included before matching
Patients after matching	157	157	Balanced 1:1 matched cohort
Objective response rate	48.4%	37.6%	Higher with this approach, but not statistically significant, $p = 0.053$
Median progression-free survival	11.0 months	8.5 months	Longer with this approach
1-year progression-free survival rate	69.9%	54.1%	Higher with this approach
Median overall survival	33.9 months	26.3 months	Favored this approach
3-year overall survival rate	41.9%	23.0%	Higher with this approach
Grade 3 or higher adverse events	15.2%	7.0%	More frequent with this approach

Overall, this study suggested that this approach may improve progression-free and overall survival compared with SBRT alone, although it was also associated with a higher incidence of grade 3 or higher adverse events [4].

4. Clinical evidence in NSCLC

4.1. Advanced and metastatic NSCLC

NSCLC represents a key tumor type for evaluating this approach [6, 10]. SBRT has been widely applied in early-stage and oligometastatic NSCLC because it enables focused delivery of high-dose radiation to limited tumor lesions while reducing exposure to adjacent normal tissues [6]. A review of NSCLC suggested that selected SBRT regimens may provide more favorable immune modulation than conventional radiotherapy,

supporting its use as a partner for PD-1/PD-L1 blockade [6]. The main clinical findings are summarized in Table 4.

Table 4. Clinical evidence of SBRT combined with pembrolizumab-based immunotherapy in advanced NSCLC

Study or outcome	Pembrolizumab plus SBRT	Pembrolizumab alone	Pembrolizumab plus conventional radiotherapy	Main finding
PEMBRO-RT: median progression-free survival	6.6 months	1.9 months	Not reported	Numerically longer with the addition of SBRT
PEMBRO-RT: median overall survival	15.9 months	7.6 months	Not reported	Numerically favored the SBRT-containing group
PEMBRO-RT: statistical significance	No statistically significant difference in the overall population	No statistically significant difference in the overall population	Not reported	Suggested possible benefit, but not definitive survival advantage
Out-of-field objective response rate	38%	25%	10%	SBRT may promote stronger systemic antitumor responses than conventional fractionated radiotherapy in selected patients

Overall, these findings suggest that this approach may improve disease control and systemic immune responses in selected patients with advanced NSCLC. However, the available evidence remains insufficient to confirm a definitive survival advantage, and further prospective studies are required to clarify the optimal patient population, radiation schedule, and treatment sequence [6, 10].

4.2. Neoadjuvant NSCLC

In resectable NSCLC, the combination of neoadjuvant approach is being investigated because it may activate systemic antitumor immunity before surgery [7]. This strategy may help eliminate micrometastatic disease and reduce recurrence after surgical resection [7]. One reported neoadjuvant trial showed that adding nivolumab around SBRT reported an event-free survival rate of 77% at 4 years, compared with 53% after SBRT monotherapy [7]. Because this result is summarized from an early clinical study, larger prospective trials are still needed before this approach can become a standard neoadjuvant treatment [7]. Overall, the NSCLC evidence supports further research on this treatment in metastatic, oligometastatic, and neoadjuvant settings [6, 7, 10].

5. Evidence in other solid tumors

5.1. Oligometastatic solid tumors

A randomized phase II trial enrolled anti-PD-1-pretreated oligometastatic patients and evaluated SBRT at 3×8 Gy combined with pembrolizumab-based multi-agent immunotherapy [2]. In the immediate-treatment arm, patients received intratumoral ipilimumab and avelumab, intravenous pembrolizumab, and intratumoral

myeloid dendritic-cell injection after SBRT [2]. This trial enrolled 13 patients, including patients with NSCLC and melanoma [2]. The 1-year progression-free survival rate was higher in the immediate-treatment arm than in the control arm, at 10% and 0%, respectively [2]. Among patients in the immediate-treatment arm, two achieved partial responses, while one had stable disease as the best overall response [2]. The primary endpoint was not met, indicating that the observed antitumor activity should be interpreted cautiously [2]. This finding is consistent with a recent review of radioimmunotherapy in oligometastatic disease, which emphasized that current outcomes remain heterogeneous and that patient selection, radiation dose-fractionation, and immunotherapy integration require further optimization [13]. Therefore, although this complex SBRT-based multi-agent immunotherapy strategy appears feasible, it does not provide direct evidence for the efficacy of this approach alone [2, 13].

5.2. Metastatic triple-negative breast cancer

In metastatic triple-negative breast cancer, one case report described a patient treated with this regimen together with GM-CSF and thymosin alpha-1 [8]. The patient achieved a partial response in the target lesion and three additional evaluable lesions according to RECIST 1.1 [8]. After two cycles of immunotherapy, the target lesion regressed by 78.97%, while the observed lesions regressed by 56.73% [8]. This case suggests a possible abscopal effect in one selected patient, but its evidentiary weight remains limited because it was based on a single case report [8]. Consistently, a recent systematic review of immunotherapy combined with radiotherapy in breast cancer reported that the available clinical data remain heterogeneous, particularly regarding treatment timing, dose-fractionation, irradiated volume, safety, and efficacy assessment [14].

5.3. Refractory solid tumors

A phase Ib study in patients with refractory solid tumors further evaluated this approach using cadonilimab, a bispecific antibody targeting PD-1 and CTLA-4 [9]. This study included 63 patients with advanced solid tumors who had experienced disease progression after at least two prior lines of systemic therapy [9]. The median number of previous treatment lines was 3.0, and 46.0% of patients had previously received PD-1 or PD-L1 inhibitors [9]. The objective response rate was 23.8%, with a 95% confidence interval of 14.0%–36.2% [9]. Median progression-free survival and overall survival were 7.2 months and 10.0 months, respectively [9]. The local control rates at 6 and 12 months were 98.4% and 93.0%, respectively [9]. In another recent phase II study, cadonilimab-based therapy also showed antitumor activity in patients with recurrent or metastatic nasopharyngeal carcinoma who had failed previous anti-PD-1 treatment [15]. Overall, these results indicate that SBRT combined with PD-1/CTLA-4 bispecific immunotherapy may have preliminary antitumor activity in heavily pretreated patients with refractory solid tumors, although larger controlled studies are still needed to confirm its therapeutic value [9, 15].

6. Safety consideration

The safety of this approach depends on tumor location, irradiated organ, radiation dose, fractionation schedule, baseline organ function, and immune-related toxicity risk [4, 5, 10]. In hepatocellular carcinoma, liver function is especially important because many patients have cirrhosis or impaired hepatic reserve before treatment [5]. Radiation-induced liver disease and immune-related hepatitis should both be considered when radiotherapy is combined with PD-1 blockade in liver cancer [5]. In the HCC animal study, this treatment was well tolerated and did not produce obvious organ damage on pathological examination [1]. In the multicenter study of unresectable HCC, the overall incidence of adverse events was comparable across treatment groups;

however, grade 3 or higher adverse events occurred more frequently in patients receiving this approach [4]. In the tumor-agnostic phase II study, the only grade 3 treatment-related adverse event was iatrogenic pneumothorax [2]. In the metastatic breast cancer case report, the patient experienced a grade 2 skin reaction, which improved following antiallergic therapy [8]. In the phase Ib study of refractory solid tumors, treatment-related adverse events were reported in 38.1% of patients, while grade 3 treatment-related adverse events occurred in 3.2% of patients [9]. The most frequently reported treatment-related adverse events in this study included pain, increased transaminase levels, pneumonia, fatigue, nausea, and fever [9]. Overall, available evidence indicates that this approach has a generally manageable safety profile; however, careful monitoring remains necessary because radiation-related toxicity and immune-related adverse events may overlap [4, 9, 10].

7. Optimization of treatment strategy

7.1. Dose and fractionation

The optimal SBRT dose and fractionation for immune activation remain uncertain [3, 6]. Preclinical evidence suggests that hypofractionated regimens such as 8 Gy $\times$ 3 fractions may activate immune pathways more effectively than some single high-dose regimens, although this finding has not been fully validated in clinical settings [3]. For NSCLC, reported SBRT regimens in combination trials vary widely, including 24 Gy in 3 fractions, 30 Gy in 3 fractions, 50 Gy in 4 fractions, and 30 to 60 Gy in 3 to 5 fractions [6, 10]. This variation makes it difficult to determine a universal optimal SBRT schedule for this treatment [6, 10].

7.2. Treatment timing

The timing of this approach also remains unresolved [3, 7]. SBRT before immunotherapy may help release tumor antigens and prime immune responses [3, 10]. Concurrent or near-concurrent treatment may theoretically help maintain T-cell activation and strengthen systemic antitumor immunity, but the optimal sequence remains uncertain [6, 7]. However, different studies use different treatment intervals, and the best sequence has not been clearly established [6, 10].

7.3. Irradiated target selection

The number and location of irradiated lesions are important issues in metastatic disease [3, 10]. Irradiating only one lesion may be insufficient in patients with multiple heterogeneous metastases [3]. Multisite irradiation may increase antigen release and immune stimulation, but it may also increase toxicity risk [3, 6]. Future studies should clarify whether single-site SBRT or multisite SBRT is more suitable for different metastatic patterns [3, 10].

7.4. Biomarker-guided patient selection

Patient selection should be improved through biomarkers such as PD-L1 expression, tumor mutation burden, mismatch repair status, microsatellite instability, circulating tumor DNA, and immune-cell infiltration [5, 6]. Dynamic immune biomarkers before and after SBRT may help identify patients who develop favorable immune activation after radiation [6, 7]. To date, validated biomarkers for selecting patients most likely to benefit from this treatment remain lacking [6, 10].

8. Current limitation and future direction

Most current studies are early-phase trials, retrospective analyses, reviews, or case reports [2, 4, 8]. The available studies include different tumor types, treatment lines, radiation doses, target volumes, PD-1 inhibitors, and combination partners [2, 6, 9]. This heterogeneity makes it difficult to compare results across studies and prevents a single standard treatment regimen from being recommended [3, 6, 10]. The evidence for hepatocellular carcinoma is encouraging, but liver function and radiation-induced liver disease remain important clinical concerns [4, 5]. The evidence for NSCLC is relatively broad, but many trials remain small and the best treatment sequence has not been determined [6, 7, 10]. The evidence for breast cancer and refractory solid tumors remains preliminary and exploratory [8, 9]. Future studies should include larger prospective randomized controlled trials to confirm survival benefit and define suitable patient populations [4, 9, 10]. Future research should also focus on optimal SBRT dose, treatment sequence, irradiated target selection, biomarker-guided treatment, and long-term toxicity monitoring [3, 6, 10].

9. Conclusion

This approach is a promising investigational strategy for solid tumors because it may integrate precise local tumor ablation with systemic immune modulation. Mechanistically, SBRT may reshape antitumor immunity by triggering immunogenic tumor cell damage, which facilitates antigen presentation and subsequently promotes cytotoxic T-cell recruitment into the tumor site. PD-1 blockade can enhance and maintain radiation-induced antitumor immunity by restoring exhausted T-cell function. Clinical evidence indicates potential benefit in HCC and NSCLC, but data from metastatic TNBC and refractory solid tumors are still limited and exploratory. Across selected studies, frequently reported signals include improved local control, increased response rate, prolonged progression-free survival in some cohorts, and occasional out-of-field responses. However, the current evidence is not sufficient to establish a universal standard regimen for all solid tumors. For clinical practice, this treatment should be regarded as a developing individualized strategy rather than a fully standardized treatment approach.

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