

Stage IIIB pulmonary sarcomatoid carcinoma treated with spatially fractionated radiotherapy combined with antiangiogenic therapy and sequential chemo-immunotherapy: A case report

ZIJIAN QIU¹, FENG XUAN², XI MAO¹, PEIPEI YANG¹ and XIAOPING ZHU¹

¹Department of Radiation Oncology, Quzhou People's Hospital, The Quzhou Affiliated Hospital, Wenzhou Medical University, Quzhou, Zhejiang 324000, P.R. China; ²Department of Radiation Oncology, Zhuji Affiliated Hospital of Wenzhou Medical University, Shaoxing, Zhejiang 311800, P.R. China

Received June 10, 2026; Accepted September 4, 2026

DOI: 10.3892/ol.2026.15887

Abstract. Pulmonary sarcomatoid carcinoma (PSC) is a rare subtype of non-small-cell lung cancer, characterized by high invasiveness and treatment resistance. Patients with PSC often exhibit poor treatment outcomes, short median survival and a dismal prognosis, necessitating novel comprehensive treatment strategies to address this clinical challenge. A 64-year-old man with PSC was initially diagnosed with stage cT4N2M0 IIIB disease and was negative for programmed cell death-ligand 1 expression. Owing to the large tumor size and unfavorable performance status, treatment initially involved an induction regimen that combined tomotherapy-based spatially fractionated radiotherapy (SFRT) with recombinant human endostatin injection (Endostar). As the condition of the patient improved, two additional cycles of low-dose albumin-bound paclitaxel were administered as concurrent chemoradiotherapy. Following radiotherapy, the patient received two sequential cycles of systemic therapy with paclitaxel liposome and ivonescimab. This combined regimen demonstrated favorable short-term efficacy with improved performance status. However, the patient developed pneumonia caused by infection with *Pneumocystis jirovecii* and aspergillus, subsequently succumbing to respiratory failure (with an overall survival of

5.5 months). The present case provides a clinical reference for the selection of treatment strategies in locally advanced, large PSC, highlighting the potential of SFRT combined with antiangiogenic therapy as a promising, novel local treatment option. However, the ultimate survival outcome of this patient should serve as a stark warning that heightened vigilance against opportunistic infections is essential during combination therapy in such patients.

Introduction

Pulmonary sarcomatoid carcinoma (PSC) is a rare and aggressive subtype of non-small cell lung cancer (NSCLC), accounting for <1% of all lung cancer cases (1). It is characterized by the presence of carcinomatous and sarcomatoid components, leading to biological behaviors marked by high levels of malignancy, invasiveness and treatment resistance (2). A total of ~70% of patients with PSC are diagnosed in the middle to advanced stages (3). Despite surgical intervention, the risk of recurrence and metastasis remains high and patients with advanced PSC have short survival times, resulting in an unfavorable prognosis.

Current treatment strategies for PSC are primarily based on those for NSCLC, with radiotherapy as the core modality for local palliative or curative treatment. However, conventional radiotherapy approaches yield poor efficacy or are associated with rapid resistance in PSC (4). This is especially true in patients with locally advanced tumors and a large tumor burden, in whom achieving rapid downstaging and creating opportunities for systemic treatment remain challenging. Currently, high-quality, large-scale clinical studies to guide the updating of treatment guidelines are lacking. Therefore, the clinical management of PSC presents a notable challenge, and the exploration of novel, comprehensive treatment strategies capable of overcoming therapeutic resistance is key.

In the present case report, a case is described of locally advanced PSC treated with tomotherapy-based spatially fractionated radiotherapy (SFRT) combined with Endostar antiangiogenic therapy for local treatment, followed by

Correspondence to: Dr Xiaoping Zhu, Department of Radiation Oncology, Quzhou People's Hospital, The Quzhou Affiliated Hospital, Wenzhou Medical University, 100 Minjiang Avenue, Quzhou, Zhejiang 324000, P.R. China
E-mail: 25229661@qq.com

Abbreviations: CT, computed tomography; GTV, gross tumor volume; NSCLC, non-small cell lung cancer; PD-L1, programmed cell death-ligand 1; PSC, pulmonary sarcomatoid carcinoma; SFRT, spatially fractionated radiotherapy

Key words: tomotherapy, spatially fractionated radiotherapy, endostatin, pulmonary sarcomatoid carcinoma, case report

sequential chemo-immunotherapy. Although this approach achieved favorable local control, the patient succumbed to an opportunistic infection. The present case report aims to provide a preliminary demonstration of the clinical implementation process, acute adverse reactions and short-term efficacy of this innovative combination therapy and offer a novel therapeutic perspective for this challenging disease. The present case report also provides preliminary evidence and practical experience for future large-scale clinical trials on PSC treatment.

Case report

The patient was a 64-year-old Chinese man who had undergone transcatheter aortic valve replacement in 2023 and had been diagnosed with type 2 diabetes for >1 year, managed with metformin tablets. The patient reported a history of smoking, with a smoking index of 900 (30 cigarettes daily over 30 years) and social drinking.

In June 2025, the patient was admitted to Quzhou People's Hospital, (Quzhou, China), presented with a 2-day history of worsening chest tightness and fatigue, accompanied by mild coughing with thin, white sputum. The patient denied the presence of other symptoms such as chest pain, hemoptysis or syncope. Upon presentation, the patient had a body temperature of 38.1°C (100.6°F) and a blood pressure of 74/50 mmHg, with an electrocardiogram showing atrial flutter with a 2:1 conduction ratio. A CT scan was performed, revealing a mass (9.7x13.3 cm) in the right upper lobe, with obstruction of the right main bronchus and upper lobe (Fig. 1A). The patient was diagnosed with a space-occupying lesion in the right lung (lung cancer suspected) and admitted for emergency treatment.

Physical examination on admission indicated Eastern Cooperative Oncology Group Performance Status (5) score of 3 and decreased breath sounds in the right lung. Laboratory test results (performed June 2025) included squamous cell carcinoma antigen (3.95 ng/ml; normal reference range, 0-1.5 ng/ml), CEA (136.82 ng/ml; normal reference range, 0-5.0 ng/ml) and CA 19-9 (70.79 U/ml; normal reference range, 0-37.0 U/ml). Positron emission tomography (PET)-CT (performed June 2025) revealed a space-occupying lesion in the right upper lobe with markedly increased fluorodeoxyglucose uptake [maximum standardized uptake value (SUV_{max})=13.19], suggestive of lung cancer, and slightly elevated fluorodeoxyglucose uptake (SUV_{max}=3.28) in the mediastinal 2R and 4R lymph nodes (Fig. 2). Bronchoscopic examination (performed June 2025) and treatment revealed a neoplasm covered with necrotic material in the right main bronchus, which largely obstructed the lumen. An intraluminal tumor was resected using a snare (Fig. 3A). Follow-up contrast-enhanced chest CT performed following bronchoscopic treatment showed that the right main bronchus and middle and lower lobe bronchi were patent (Fig. 1B). Hematoxylin-Eosin staining was performed by pathologists after tumor tissue sections according to standard procedures (Fig. 3B). The results demonstrated a malignant tumor with extensive necrosis, showing scattered areas of atypical squamous epithelium locally (based on tissue from endoscopic resection of the right main bronchus). Meanwhile, immunohistochemical staining was performed by pathologists using the EnVision system (6), and the immunohistochemistry

results were as follows: Insulinoma-associated protein 1 (-), CD34 (vascular +), leukocyte common antigen (-), S-100 (-), vimentin (+), SWI/SNF related BAF chromatin remodeling complex subunit B1 (SMARCB1; +), SMARCA4/Brg1 (+), cytokeratin 5/6 (partial +), P40 (focal +), Ki-67 (+; 80%), napsin A (-), thyroid transcription factor-1 [TTF-1 (focal +)], cytokeratin 7 (partial +), cytokeratin (partial +), anaplastic lymphoma kinase (-) and programmed cell death-ligand 1 (PD-L1) tumor proportion score=0 (Fig. 3C-R). All antibodies used for immunohistochemistry were ready-to-use antibody reagents. Insulinoma-associated protein 1 antibody reagent (cat. no. GT246802), CD45 antibody reagent (cat. no. GM070104), S100 antibody reagent (cat. no. GZ031104), vimentin antibody reagent (cat. no. GM072504), integrase interactor-1 antibody reagent (cat. no. GT225704), SMARCA4/Brg1 antibody reagent (cat. no. GT233302), Ki-67 antibody reagent (cat. no. GT209404), were from Shanghai Gene Tech. CD34 antibody reagent (cat. no. BFM-0456-6), cytokeratin 5/6 antibody reagent (cat. no. BFM-0482-6), P40 antibody reagent (cat. no. BFM-0062-6), napsin A antibody reagent (cat. no. BFM-0499-6), TTF-1 antibody reagent (cat. no. BFM-0379-6), cytokeratin 7 antibody reagent (cat. no. BFM-0604-6), cytokeratin antibody reagent (cat. no. BFM-0016-6), were from Hangzhou Baiyin Biotech. Anaplastic lymphoma kinase antibody reagent (cat. no. GA785) was from Agilent Tech, and PD-L1 (cat. no. 8.17.0069.02) antibody reagent was from Xiamen AmoyDx. High-throughput sequencing of tumor tissue was performed at Hangzhou Buping Medical Laboratory (www.bphealth.com). Genetic testing showed positive findings for tumor protein 53, phosphatase and tension homolog deleted on chromosome 10, AT-rich interaction domain 1A and cyclin-dependent kinase inhibitor 2A mutations, the tumor was microsatellite stable and negative for PD-L1 (tumor proportion score <1%). This result indicated that there were currently no approved or recommended targeted drugs for the patient.

The results of immunohistochemistry were key for differential diagnosis. The positive expression of epithelial marker cytokeratin was the core basis for diagnosis (7) (Fig. 3C); meanwhile, the sarcomatoid component often expresses vimentin (Fig. 3D), which is a marker of mesenchymal origin (8). The high Ki-67 index (Fig. 3E) reflects malignant biological behavior (9). These three indicators supported the pathological diagnosis of sarcomatoid carcinoma. TTF-1 was positive (Fig. 3F), suggesting that the tumor originated from the lung or thyroid. However, PET-CT indicated that only the lung had tumor lesions, which ruled out the possibility of tumor metastasis from other sites to the lung. Therefore, the final diagnosis was PSC. The pathological result was formally reported after discussion and review by professional pathologists.

The patient was diagnosed with NSCLC of the right lung (sarcomatoid carcinoma; cT4N2M0 stage IIIB; PD-L1⁻). Considering the high tumor burden and unfavorable performance status score of 3 of the patient, tomotherapy-based SFRT was initiated in June 2025 (Fig. 4A). According to internationally recognized expert recommendations (10), an individualized SFRT plan was formulated for the patient. Target volume delineation was based on PET-CT, with the right lung tumor defined as the gross tumor volume (GTV).

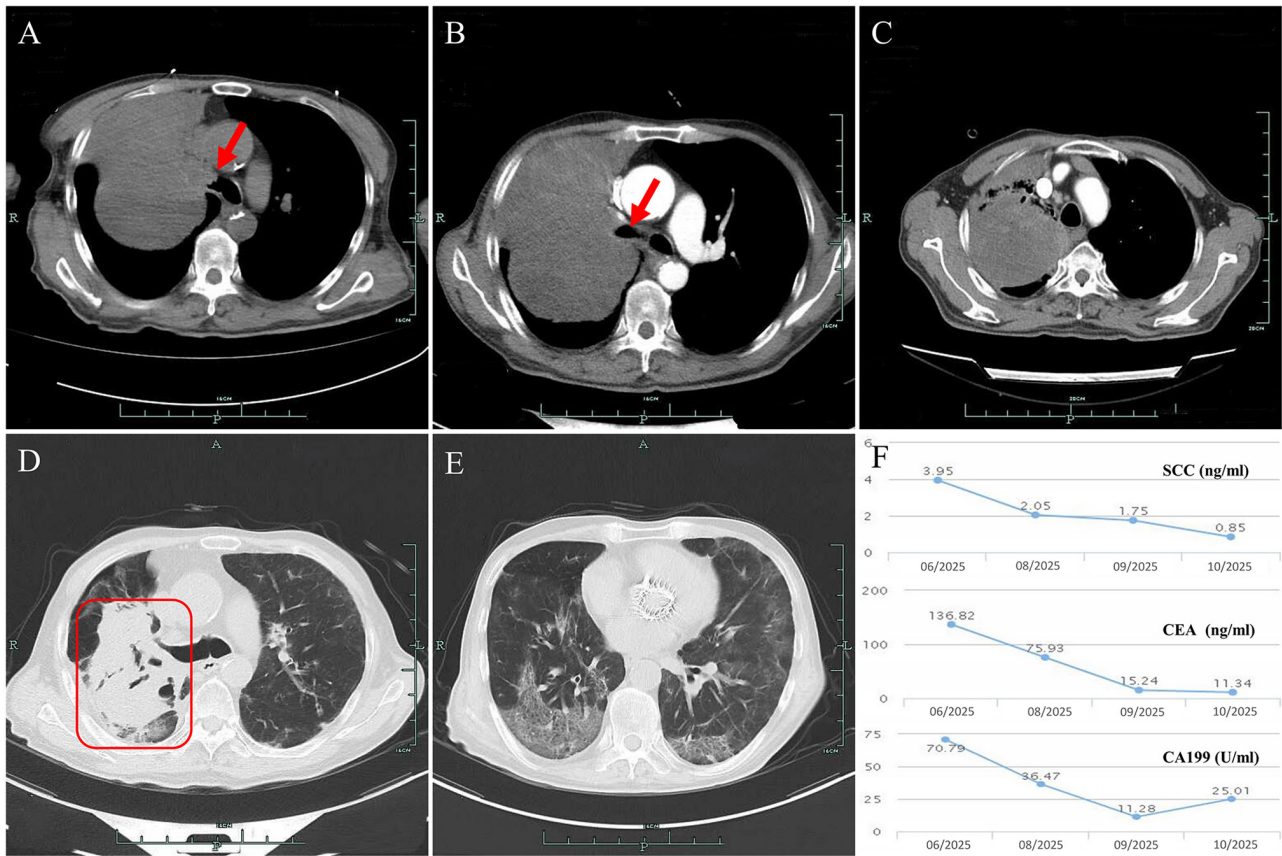


Figure 1. Evolution of the imaging findings and tumor markers of the present patient. (A) CT scan at initial diagnosis reveals a large space-occupying lesion (9.7x13.3 cm) in the right lung, and the red arrow indicates the right bronchial obstruction (June 2025). (B) CT following bronchoscopic intervention presenting airway recanalization, as indicated by the red arrow (June 2025). (C) CT following 10 fractions sessions indicating slight tumor shrinkage (9.3x12.8 cm; July 2025). (D) CT indicating continued tumor shrinkage to 7.8x10.0 cm, as indicated by the red box (October 2025). (E) CT scan indicating multiple infiltrative lesions in the lungs (October 2025). (F) Trends in tumor markers; normal ranges for SCC (range, 0-1.5 ng/ml), CEA (range, 0-5.0 ng/ml) and CA 19-9 (range, 0-37.0 U/ml). SCC, squamous cell carcinoma antigen.

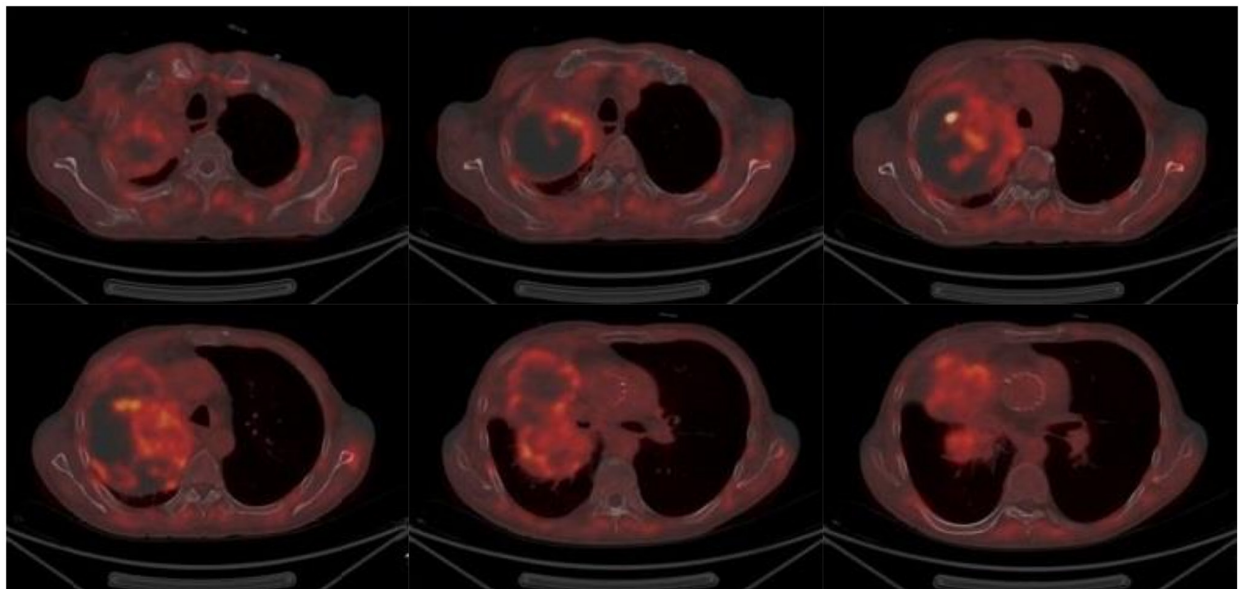


Figure 2. In June 2025, a positron emission tomography-CT scanning sequence demonstrated increased fluorodeoxyglucose uptake in the right lung tumor with necrosis within the lesion.

Within the GTV, voxels of 1 cm in diameter spaced 3 cm apart (GTV1-6) were designated as the lattice. The hypofractionated

radiotherapy dose (GTV: 30 Gy/10 F and GTV1-6: 60 Gy/10 F) was used. Endostar was administered via intravenous infusion

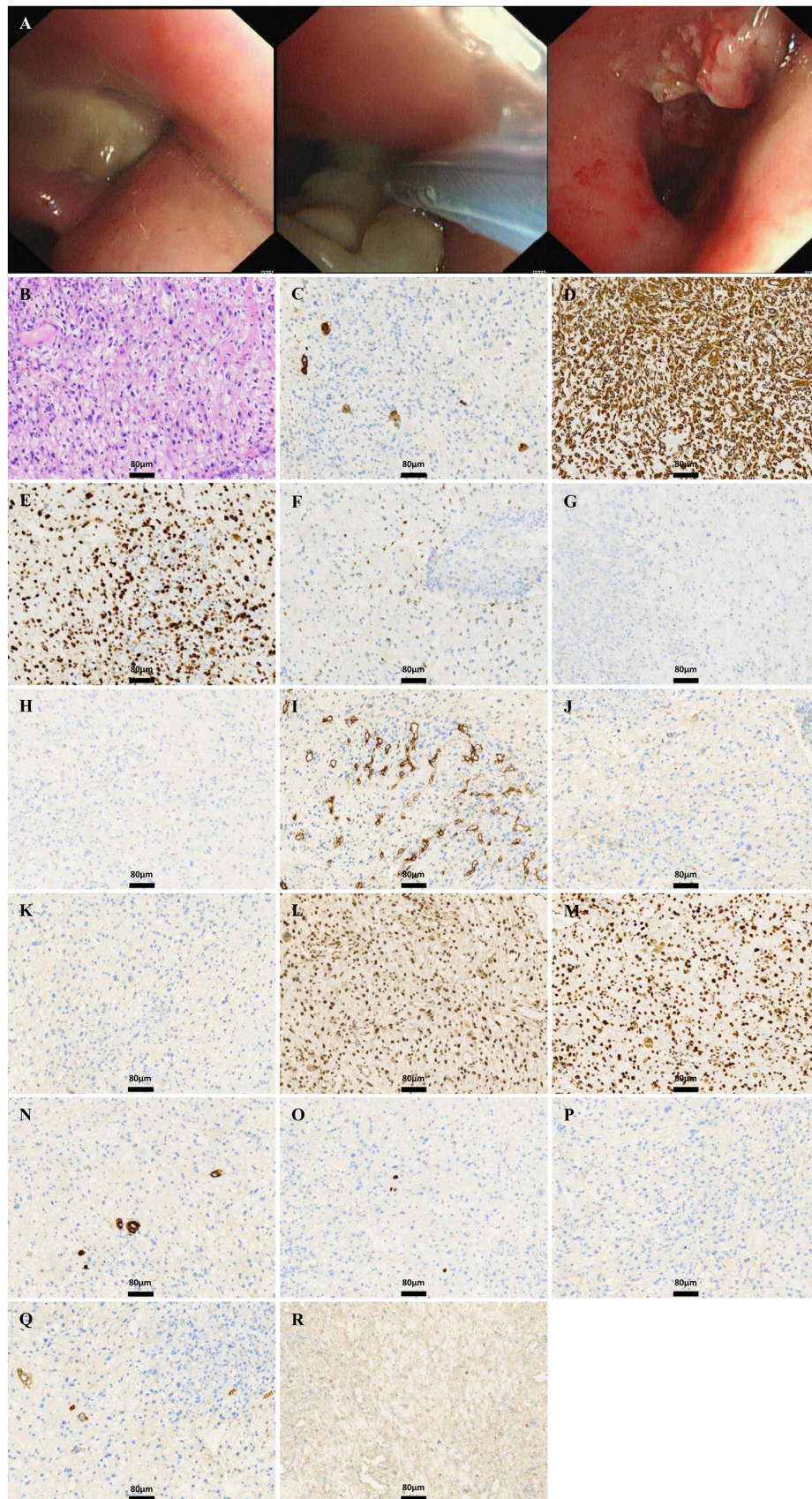


Figure 3. Bronchoscopic treatment and pathology. (A) Bronchoscopic findings and endoscopic resection of intraluminal tumor tissue performed in June 2025. (B) Pathology image (hematoxylin-eosin staining; magnification, x10). (C) Positive expression of cytokeratin. (D) Positive expression of vimentin. (E) High expression of Ki-67. (F) Positive expression of thyroid transcription factor-1. (G) Negative expression of programmed cell death-ligand 1. (H) Negative expression of insulinoma-associated protein 1. (I) Positive expression of CD34 vascular. (J) Negative expression of leukocyte common antigen. (K) Negative expression of S-100. (L) Positive expression of SMARCB1. (M) Positive expression of SMARCA4/Brg1. (N) Positive expression of cytokeratin 5/6. (O) Positive expression of P40. (P) Negative expression of napsin A. (Q) Positive expression of cytokeratin 7. (R) Negative expression of anaplastic lymphoma kinase (scale bars, 80 μ m). SMARC, SWI/SNF related BAF chromatin remodeling complex subunit.

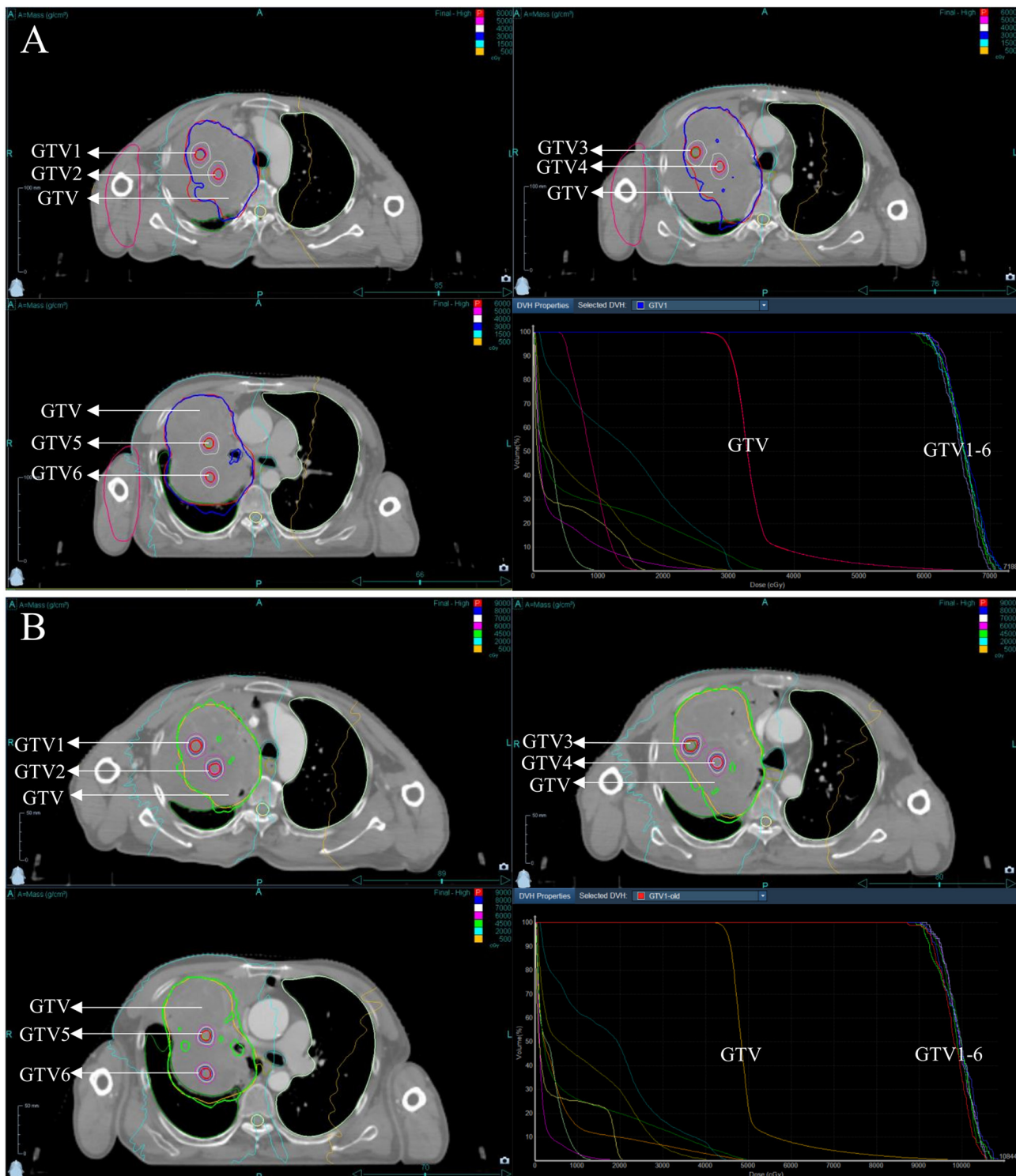


Figure 4. Spatially fractionated radiotherapy plan. (A) Right lung tumor is designated as the GTV. Within the GTV, voxels of 1 cm in diameter spaced 3 cm apart (GTV1-6) are delineated as the lattice. The radiation dose was GTV: 30 Gy/10 F and GTV1-6: 60 Gy/10 F. (B) In July 2025, five additional fractions were added to the original radiotherapy plan; the radiation doses were GTV: 15 Gy/5 F and GTV1-6: 30 Gy/5 F. GTV, gross tumor volume.

at 30 mg on days 1-7. After 10 fractions, follow-up chest CT indicated that the right upper lung mass (9.3x12.8 cm) had shrunk slightly (Fig. 1C), and the performance status of the patient score had recovered to 1. The limits of the organ at risk remained within the tolerable range and the dose was raised as high as possible for local control. In July 2025, five additional fractions were added to the original radiotherapy plan, adjusting the total dose to GTV: 45 Gy/15 F, with the lattice

region receiving 90 Gy/15 F (Fig. 4B). The peak-to-valley dose ratio of the SFRT plan was 2.9 and the SFRT target volume/GTV ratio was 0.5%. The organs at risk were tolerable and total dose values as follows: V5 of the right lung was 31.7% (limit value: <60%), V20 was 9.7% (limit value: <30%), V30 was 6.4% (limit value: <20%), the mean dose to the right lung and heart was 925 cGy (limit value: <1,500 cGy) and 195 cGy (limit value: <800 cGy), respectively, and the maximum doses

to the esophagus and spinal cord were 4,443 cGy and 2204 cGy (limit value: <4,500 cGy), respectively. Additionally, 100 mg albumin-bound paclitaxel was administered twice weekly as concurrent chemotherapy. Following the completion of this treatment phase, the patient was discharged without incident.

Subsequently, in August and September 2025, two sequential cycles of systemic therapy were administered using 210 mg of paclitaxel liposome and 1,000 mg of ivonescimab. Imaging revealed continued tumor shrinkage to 7.8x10.0 cm (Fig. 1D), accompanied by decreased tumor markers (Fig. 1F). The therapeutic effect was evaluated as ‘partial response’ according to the RECIST criteria (11); local control was achieved.

In October 2025, the patient developed a fever, cough and sputum production. Chest CT revealed multiple infiltrates in the lungs (Fig. 1E). Pathogen testing showed positive nucleic acid detection for *Pneumocystis jirovecii* and *aspergillus*, suggesting pulmonary infection; however, the possibility of immune-related pneumonia could not be ruled out. A combination regimen of meropenem 1.0 g intravenously every 8 h, compound sulfamethoxazole tablets 1.44 g orally every 6 h, voriconazole tablets 200 mg orally twice daily and methylprednisolone 80 mg intravenously once daily was administered. After 10 days, the pulmonary inflammation decreased and the antibiotic was de-escalated to piperacillin-tazobactam 4.5 g intravenously every 8 h, methylprednisolone was reduced to 40 mg once daily, and compound sulfamethoxazole tablets and voriconazole tablets were continued at the original dose. In November 2025, the patient's pulmonary inflammatory lesions increased again and nintedanib capsules 100 mg orally every 12 h were added. Subsequently, sputum test showed *aspergillus* nucleic acid positive, alveolar lavage fluid galactomannan test 8.38 $\mu\text{g/l}$ (positive), fungal glucan 677.29 pg/ml (positive) and *Pseudomonas aeruginosa* positive. Despite aggressive antimicrobial therapy, the condition of the patient continued to deteriorate. In November 2025, the patient died of respiratory failure (with an overall survival of 5.5 months). Detailed antitumor and pneumonia treatments are presented in Table I.

Discussion

PSC often presents as a large, irregular mass that closely adjoins key organs such as the spinal cord, heart and major blood vessels (12), posing a dilemma in conventional radiotherapy. Increasing the dose to enhance local control elevates the risk of severe radiation injury, whereas reducing the dose may lead to treatment failure, thereby presenting difficulties in striking a balance between treatment risks and benefits. Therefore, there is an urgent clinical need to explore innovative radiotherapy techniques that can effectively improve local tumor control rates while maintaining safety.

SFRT is an innovative radiotherapy technique that uses advanced imaging guidance to achieve high-dose coverage of tumors via an alternating ‘peak-and-valley’ (similar to a three-dimensional lattice) dose distribution pattern (13). SFRT safely increases the local tumor dose and more effectively eliminates tumors via complex radiobiological effects such as vascular normalization, abscopal effects and enhanced immunogenicity (14), making it particularly suitable for large tumors (15).

Regarding biology, hypoxia is a key factor contributing to radiotherapy resistance (16). Endostar is a recombinant human

endostatin that suppresses tumor angiogenesis. By exploiting the ‘vascular normalization’ window, it temporarily improves blood perfusion and oxygenation within the tumor (17).

In the present case of PSC, the following was observed: i) The tumor was invasive, presenting as a large mass with necrosis. It was already locally advanced as initially diagnosed and carried co-mutations in tumor suppressor genes, such as tumor protein 53; ii) PD-L1 expression was negative, suggesting a poor response to monotherapy with immune checkpoint inhibitors; and iii) due to airway obstruction caused by the large tumor and poor general condition, the patient was unable to tolerate standard-dose chemoradiotherapy.

Therefore, the present case report proposes an innovative integrated treatment approach for refractory PSC, involving an induction regimen combining tomotherapy-based SFRT with Endostar antiangiogenic therapy. This strategy aims to achieve more effective tumor targeting by leveraging the dual synergistic effects of physics (SFRT to overcome dose limitations) and biology (Endostar to improve the microenvironment), while harnessing the ‘*in situ* vaccine’ effect to create favorable conditions for the efficacy of subsequent immunotherapy (18). After the actual treatment, it was observed this induction therapy achieved the effect of shrinking the tumor and improving the performance status score of the patient, thereby creating an opportunity for the subsequent treatment.

Subsequent sequential treatment with ivonescimab (simultaneously targeting programmed death-1 and vascular endothelial growth factor) combined with chemotherapy showed a certain extent of antitumor activity in PSC cases negative for PD-L1 and without clinically notable mutations. This was reflected in the decline of tumor markers and the continuous regression of lesions on imaging. This antitumor effect may be related to non-PD-L1-dependent mechanisms such as modulating T-cell function and enhancing effector-cell infiltration (19).

However, the final outcome of this case may suggest that this treatment approach still faces certain challenges. The patient developed pulmonary inflammation following two cycles of chemoradiotherapy. Considering the imaging characteristics of multiple infiltrates in both lungs outside the field of radiotherapy, radiation pneumonitis was temporarily ruled out, but the possibility of immune-related pneumonitis could not be completely excluded. The etiological results suggested that there was a pulmonary infection, and a pneumonia treatment plan combined with glucocorticoids and antibiotics was initiated. The pneumonia was well controlled in the short term, but it eventually developed into a fatal mixed-opportunistic infection, potentially associated with the following: i) The advanced age of the patient and history of diabetes and heart valve replacement surgery, which may have compromised baseline immune function; ii) the use of immune checkpoint inhibitors, leading to T-cell exhaustion or abnormal activation, thereby disrupting the body's immune surveillance against opportunistic pathogens; and iii) the tumor and comprehensive treatment regimen themselves, which may have led to systemic immune dysregulation.

The immune function of the patient was not monitored, including the measurement of the CD4⁺ T-cell count, and subsequent failure to use prophylactic antibiotics during the treatment period. These were the limitations in the present

Table I. Timeline of clinical events.

Time point	Clinical events
June, 2025 (day 1)	The patient was diagnosed with PSC, cT4N2M0 stage IIIB, with lung tumor size of 9.7x13.3 cm.
June to July, 2025 (days 12-22)	SFRT was administered to the right lung tumor, one radiotherapy session per day, daily dose: GTV 3 Gy and GTV1-6 6 Gy. Total course of 10 days (excluding weekends): Total dose of the first stage radiotherapy: GTV 30 Gy/10F and GTV1-6 60 Gy/10F.
June to July, 2025 (days 12-18)	Endostar 30 mg was administered once daily by intravenous infusion.
July, 2025 (day 22)	CT showed the lung tumor reduced to 9.3x12.8 cm; performance status score had recovered. Albumin-bound paclitaxel 100 mg was administered once by intravenous infusion.
July to July, 2025 (days 24-28)	Five additional fractions added to the original radiotherapy plan, adjusting the total dose to GTV: 45 Gy/15F, with the lattice region receiving 90 Gy/15F.
July, 2025 (day 28)	Albumin-bound paclitaxel 100 mg was administered once by intravenous infusion.
August, 2025 (day 56)	CT indicated the lung tumor had reduced to 9.0x11.8 cm.
August, 2025 (day 58)	Cycle 1 of paclitaxel liposome 210 mg and ivonescimab 1,000 mg was administered by intravenous infusion.
September, 2025 (day 80)	Cycle 2 of paclitaxel liposome 210 mg and ivonescimab 1,000 mg was administered by intravenous infusion.
October, 2025 (day 123)	CT showed multiple infiltrates in the lungs; the tumor continued to reduce to 7.8x10.0 cm. Meropenem 1 g administered every 8 h and methylprednisolone 80 mg administered once daily.
October, 2025 (day 126)	Sputum test: <i>Pneumocystis jirovecii</i> and aspergillus nucleic acid were positive. Compound sulfamethoxazole tablets 1.44 g were taken orally every 6 h and voriconazole tablets 200 mg were taken orally twice daily.
October, 2025 (day 133)	Reduction in pulmonary inflammatory lesions. Piperacillin-tazobactam 4.5 g was administered every 8 h; methylprednisolone was reduced to 40 mg once a day. Compound sulfamethoxazole tablets and voriconazole tablets continued orally.
November, 2025 (day 145)	The tumor size remained stable, but the pulmonary inflammatory lesions increased. Nintedanib capsules 100 mg were taken orally every 12 h.
November, 2025 (day 150)	Sputum test showed positive aspergillus nucleic acid. Alveolar lavage fluid galactomannan test 8.38 ug/l (positive), fungal glucan 677.29 pg/ml (positive) and <i>Pseudomonas aeruginosa</i> positive.
November, 2025 (day 154)	The inflammatory lesions in the lungs continued to increase. The condition of the patient deteriorated further and respiratory failure occurred. A ventilator was used for assisted breathing.
November, 2025 (day 160)	The patient died.

PSC, pulmonary sarcomatoid carcinoma; SFRT, spatially fractionated radiotherapy; GTV, gross tumor volume.

case. Although PSC itself carries an unfavorable prognosis, the direct cause of mortality in the present patient was respiratory failure due to severe pneumonia rather than the progression of the tumor. As a result, it is suggested that patients with lung cancer with underlying diseases who are undergoing combined treatments such as radiotherapy, chemotherapy and immunotherapy should be prophylactically administered antibiotics (20) while monitoring their immune function status, in order to prevent pneumocystis pneumonia and aspergillosis.

In conclusion, for locally advanced, PD-L1⁺ PSC, a treatment regimen centered on SFRT combined with antiangiogenic therapy may be an effective induction treatment, thereby creating an opportunity for subsequent systemic therapy. However, during chemotherapy, radiotherapy and immunotherapy, vigilance must be maintained against opportunistic

infections. High-risk patients with underlying conditions and undergoing combined treatments, in whom aggressive prophylaxis, monitoring and early intervention are essential, require particular attention. The present case provides a reference for personalized treatment decisions; however, more clinical data are needed to validate the efficacy and safety of this treatment model.

Acknowledgements

Not applicable.

Funding

No funding was received.

Availability of data and materials

The data generated in the present study may be requested from the corresponding author. The high-throughput sequencing datasets (SRX35321793) are available from the NCBI Sequence Read Archive (<https://www.ncbi.nlm.nih.gov/sra/PRJNA1531924>).

Authors' contributions

ZQ was responsible for the study conception, investigation, writing the original draft, and reviewing and editing. FX was responsible for data analysis, writing the original draft, and reviewing and editing. XM and PY were responsible for investigation, resources and writing the original draft. XZ was responsible for study conception, project administration, and reviewing and editing. All authors read and approved the final manuscript, and are responsible for all aspects of the work. ZQ and XZ confirm the authenticity of all the raw data.

Ethics approval and consent to participate

The present work was approved by the Ethics Committee of Quzhou People's Hospital (Quzhou, China; approval no. 2026-050).

Patient consent for publication

As the patient is deceased, the patient's daughter signed a written consent form for the use of the patient's clinical data, medical images and clinical outcomes for publication.

Competing interests

The authors declare that they have no competing interests.

References

- Yendamuri S, Caty L, Pine M, Adem S, Bogner P, Miller A, Demmy TL, Groman A and Reid M: Outcomes of sarcomatoid carcinoma of the lung: A surveillance, epidemiology, and end results database analysis. *Surgery* 152: 397-402, 2012.
- Travis WD, Brambilla E, Nicholson AG, Yatabe Y, Austin JHM, Beasley MB, Chirieac LR, Dacic S, Duhig E, Flieder DB, *et al*: The 2015 World health organization classification of lung tumors: Impact of genetic, clinical and radiologic advances since the 2004 classification. *J Thorac Oncol* 10: 1243-1260, 2015.
- Ung M, Rouquette I, Filleron T, Taillandy K, Brouchet L, Bennouna J, Delord JP, Milia J and Mazières J: Characteristics and clinical outcomes of sarcomatoid carcinoma of the lung. *Clin Lung Cancer* 17: 391-397, 2016.
- Wei Y, Wang L, Jin Z, Jia Q, Brcic L, Akaba T and Chu Q: Biological characteristics and clinical treatment of pulmonary sarcomatoid carcinoma: A narrative review. *Transl Lung Cancer Res* 13: 635-653, 2024.
- Oken MM, Creech RH, Tormey DC, Horton J, Davis TE, McFadden ET and Carbone PP: Toxicity and response criteria of the Eastern cooperative oncology group. *Am J Clin Oncol* 5: 649-655, 1982.
- Kämmerer U, Kapp M, Gassel AM, Richter T, Tank C, Dietl J and Ruck P: A new rapid immunohistochemical staining technique using the EnVision antibody complex. *J Histochem Cytochem* 49: 623-630, 2001.
- Nakajima M, Kasai T, Hashimoto H, Iwata Y and Manabe H: Sarcomatoid carcinoma of the lung: A clinicopathologic study of 37 cases. *Cancer* 86: 608-616, 1999.
- Blaukovitsch M, Halbwedl I, Kothmaier H, Gogg-Kammerer M and Popper HH: Sarcomatoid carcinomas of the lung-are these histogenetically heterogeneous tumors? *Virchows Arch* 449: 455-461, 2006.
- Mitchell KG, Parra ER, Nelson DB, Zhang J, Wistuba II, Fujimoto J, Roth JA and Antonoff MB; MD Anderson Lung Cancer Immune Microenvironment Working Group: Tumor cellular proliferation is associated with enhanced immune checkpoint expression in stage I non-small cell lung cancer. *J Thorac Cardiovasc Surg* 158: 911-919.e6, 2019.
- Li H, Mayr NA, Griffin RJ, Zhang H, Pokhrel D, Grams M, Penagaricano J, Chang S, Spraker MB, Kavanaugh J, *et al*: Overview and recommendations for prospective multi-institutional spatially fractionated radiation therapy clinical trials. *Int J Radiat Oncol Biol Phys* 119: 737-749, 2024.
- Eisenhauer EA, Therasse P, Bogaerts J, Schwartz LH, Sargent D, Ford R, Dancey J, Arbuck S, Gwyther S, Mooney M, *et al*: New response evaluation criteria in solid tumours: Revised RECIST guideline (version 1.1). *Eur J Cancer* 45: 228-247, 2009.
- Xu XL, Song W, Sui X, Song L, Wang X, Feng RE and Li Y: Computed tomographic and pathological features of primary pulmonary sarcomatoid carcinoma. *Zhongguo Yi Xue Ke Xue Yuan Xue Bao* 38: 93-98, 2016.
- Seol Y, Lee YK, Kim BJ, Choi KH, Hong JH, Park CB, Kim SH, Park HW, Kim JI, Cheon W, *et al*: Feasibility of optimal vertex size and spacing for lattice radiotherapy implementation using helical tomotherapy. *Front Oncol* 15: 1512064, 2025.
- Billena C and Khan AJ: A current review of spatial fractionation: Back to the future? *Int J Radiat Oncol Biol Phys* 104: 177-187, 2019.
- Li W, Piao M, Zhai L, Zhu Y, Lou F, Chen L and Wang H: Effectiveness and safety of lattice radiotherapy in treating large volume tumors: A systematic review and meta-analysis based on single-arm clinical studies. *Balkan Med J* 42: 311-320, 2025.
- Beckers C, Pruschy M and Vetrugno I: Tumor hypoxia and radiotherapy: A major driver of resistance even for novel radiotherapy modalities. *Semin Cancer Biol* 98: 19-30, 2024.
- Meng MB, Jiang XD, Deng L, Na FF, He JZ, Xue JX, Guo WH, Wen QL, Lan J, Mo XM, *et al*: Enhanced radioresponse with a novel recombinant human endostatin protein via tumor vasculature remodeling: Experimental and clinical evidence. *Radiother Oncol* 106: 130-137, 2013.
- Wang Y, Li Y, Yang Y, Swift M, Zhang Z, Wu S, Sun Y and Yang K: In situ vaccination caused by diverse irradiation-driven cell death programs. *Theranostics* 14: 1147-1167, 2024.
- Hack SP, Zhu AX and Wang Y: Augmenting anticancer immunity through combined targeting of angiogenic and PD-1/PD-L1 pathways: Challenges and opportunities. *Front Immunol* 11: 598877, 2020.
- Classen AY, Henze L, von Lilienfeld-Toal M, Maschmeyer G, Sandherr M, Graeff LD, Alakel N, Christopeit M, Krause SW, Mayer K, *et al*: Primary prophylaxis of bacterial infections and *Pneumocystis jirovecii* pneumonia in patients with hematologic malignancies and solid tumors: 2020 updated guidelines of the Infectious Diseases Working Party of the German Society of Hematology and Medical Oncology (AGIHO/DGHO). *Ann Hematol* 100: 1603-1620, 2021.



Copyright © 2026 Qiu et al. This work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0) License.