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Pulmonary artery intimal sarcoma misdiagnosed as pulmonary embolism: MDM2 amplification as the diagnostic anchor in an unresectable tumor—a case report

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Case Report

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Abstract

Background

Pulmonary artery intimal sarcoma (PAIS) is a rare and aggressive vascular malignancy whose clinical and radiological features overlap with those of pulmonary thromboembolism (PTE); most patients are initially anticoagulated, and no standard systemic therapy exists for unresectable disease. Amplification of MDM2 is the defining genomic event, yet it is usually treated as a supplementary finding rather than as a diagnostic test.

Case presentation

A 51-year-old man without thromboembolic risk factors presented with cough, sputum production, and chest discomfort. Contrast-enhanced computed tomography (CT) showed filling defects extending from the main pulmonary artery into both branches together with a 5.0 × 2.7 cm left upper lobe mass, and he received rivaroxaban until hemoptysis occurred. 18F-fluorodeoxyglucose (FDG) positron emission tomography (PET)/CT showed concordant hypermetabolism of the intravascular and parenchymal lesions. Two biopsies, including an expert review, narrowed the differential only to intimal sarcoma versus sarcomatoid carcinoma; next-generation sequencing then identified MDM2 amplification with CDKN2A deletion, a low tumor mutational burden (1.95 mutations/Mb), and negative PD-L1 expression, establishing MDM2-driven PAIS. Because the tumor was unresectable, he received six cycles of sintilimab, docetaxel, and cisplatin, two further cycles of sintilimab and docetaxel, consolidative intensity-modulated radiotherapy (60 Gy in 25 fractions), and maintenance sintilimab monotherapy. The best response was stable disease with a 27.9% reduction in the sum of target-lesion diameters, and progression-free survival reached 12.1 months without grade ≥3 immune-related adverse events.

Conclusions

When morphology and immunohistochemistry are indeterminate, MDM2 amplification can be the decisive diagnostic test rather than a supplementary finding, and it should be sought before empirical treatment is committed. Chemotherapy-primed PD-1 blockade followed by consolidative radiotherapy produced durable disease control in an immunologically cold tumor; prospective registries are needed to test this sequence.

Background

Pulmonary artery intimal sarcoma (PAIS) is an exceedingly rare and aggressive malignant mesenchymal tumor arising from the intimal layer of the pulmonary artery, and intimal sarcoma is its predominant histological subtype. Fewer than 400 cases have been reported in the English literature since the first description in 1923.(1, 2) Because the tumor grows along the vessel lumen, its clinical and radiological features overlap with those of pulmonary thromboembolism (PTE) and chronic thromboembolic pulmonary hypertension; a substantial proportion of patients are therefore anticoagulated or thrombolysed before the correct diagnosis is made, which exposes them to hemorrhagic complications and delays definitive treatment.(2, 3) Radical resection with negative margins remains the only potentially curative option, but most patients present with unresectable, locally advanced or metastatic disease.(3, 4)

Histological diagnosis is difficult. The tumor is usually a poorly differentiated spindle or pleomorphic cell neoplasm expressing vimentin with negative or only focal lineage markers, a phenotype that overlaps with pulmonary sarcomatoid carcinoma, and viable tumor is often covered by organizing thrombus so that small biopsies yield little diagnostic tissue.(5, 6) Amplification of the 12q13-15 region, involving MDM2 and frequently CDK4, is the defining genomic event of intimal sarcoma.(6, 7) In practice, however, MDM2 testing tends to be ordered as an adjunct to histology while the differential diagnosis is still open, and treatment may be started before the result is available.

No standard systemic regimen exists for unresectable PAIS. Anthracycline- and gemcitabine-based regimens extrapolated from soft tissue sarcoma have produced heterogeneous results, and anti-angiogenic agents carry a bleeding risk that is particularly relevant in a tumor growing inside the pulmonary artery.(8, 9) Immune checkpoint inhibitors are of interest because intimal sarcoma shows features of adaptive immune resistance, yet most of these tumors are PD-L1-negative with a low tumor mutational burden and single-agent activity in sarcoma is limited.(10, 11) Whether chemotherapy priming can make such a tumor responsive to PD-1 blockade, and whether consolidative radiotherapy adds durable local control, has not been examined in PAIS.

We report a patient with unresectable PAIS in whom next-generation sequencing (NGS) resolved a diagnosis that two biopsies had left open, and in whom a molecularly informed, non-surgical sequence produced sustained disease control. We also tabulate reported cases with their molecular features, treatment, and outcome so that this observation can be read against an explicit literature frame. This case is reported in accordance with the CARE reporting guidelines.(12) The completed CARE checklist is provided as Additional file 1.

Case presentation

Initial presentation and outside evaluation

A 51-year-old man with no established risk factors for venous thromboembolism presented in March 2025 with several weeks of cough, sputum production, and chest discomfort. Contrast-enhanced computed tomography (CT) at a local hospital (March 8, 2025) showed heterogeneous low-attenuation filling defects in the main pulmonary artery and both branches, together with a 5.0 × 2.7 cm mass with spiculated margins in the left upper lobe and a smaller nodule in the left lower lobe. The relationship between the parenchymal and intravascular lesions was not recognized, and he was managed as PTE; rivaroxaban was started and stopped promptly after hemoptysis. Transthoracic echocardiography showed pulmonary hypertension. 18F-fluorodeoxyglucose (FDG) positron emission tomography (PET)/CT (March 11, 2025) showed a hypermetabolic upper lobe mass (50 × 27 mm; maximum standardized uptake value [SUVmax] 6.4), a lower lobe nodule (19.5 × 14.0 mm; SUVmax 2.1), and increased uptake within the arterial filling defects (SUVmax 5.6), raising the possibility of a primary vascular malignancy.

Diagnostic assessment

Because sampling the intravascular mass itself was considered to carry an unacceptable risk of hemorrhage, the biopsy targeted the parenchymal lesion. A CT-guided biopsy of the lung mass (March 12, 2025) showed a

spindle and polygonal cell neoplasm with hyperchromatic nuclei and focal necrosis. Immunohistochemistry was focal for epithelial membrane antigen (EMA) and weak for smooth muscle actin (SMA); pan-cytokeratin, CAM5.2, p63, TTF-1, CK5/6, CD34, S-100, CD31, and desmin were negative, BRG1 and INI1 were retained, and MDM2 was equivocal. PD-L1 (22C3) was negative (tumor proportion score < 1%, combined positive score 0). Because the specimen was exhausted, the slides were sent for expert review (April 3, 2025), which confirmed cytokeratin negativity but could narrow the differential only to sarcomatoid carcinoma versus a primary mesenchymal malignancy.

He was admitted on April 21, 2025. Repeat CT showed persistent filling defects extending from the main pulmonary artery into both branches, a 5.6 × 2.9 cm left hilar mass, and a 1.5 × 1.2 cm left lower lobe nodule (Fig. 1A). Blood counts, coagulation studies including D-dimer, cardiac biomarkers, and tumor markers were normal. A second CT-guided biopsy (April 23, 2025) showed strong diffuse nuclear MDM2 expression, a Ki-67 index of approximately 10%, retained BRG1/SMARCA4, and negative lineage markers (Fig. 2). NGS (May 19, 2025) identified MDM2 amplification and CDKN2A deletion with a tumor mutational burden of 1.95 mutations/Mb, establishing MDM2-driven PAIS, excluding sarcomatoid carcinoma, and defining an immunologically cold phenotype.

Multidisciplinary decision and treatment

At a multidisciplinary team meeting on May 30, 2025 (radiology, pathology, thoracic surgery, radiation oncology, and medical oncology), R0 resection was considered unfeasible because the main and both pulmonary arteries were involved and parenchymal lesions were present. Because no standard first-line regimen exists, the regimen was designed on three considerations. First, the poorly differentiated spindle cell morphology overlapped with pulmonary sarcomatoid tumors, which supported a platinum-based rather than an anthracycline-based backbone. Second, a PD-L1-negative, TMB-low profile predicted little benefit from single-agent PD-1 blockade, so docetaxel and cisplatin were given concurrently with the intention of inducing immunogenic cell death and antigen release rather than as sequential cytotoxic therapy. Third, anti-angiogenic tyrosine kinase inhibitors were deferred because of the risk of hemoptysis from a tumor within the pulmonary artery, and MDM2 and CDK4/6 inhibitors were reserved for progression. Consolidative radiotherapy was planned for the residual plateau after induction.

From June 6 to November 29, 2025, the patient received six cycles of sintilimab 200 mg on day 1, docetaxel 100 mg on day 1, and cisplatin 60 mg on days 1–2 every three weeks. Cisplatin was then stopped because of cumulative toxicity, and he received two further cycles of sintilimab plus docetaxel. Intensity-modulated radiotherapy (60 Gy in 25 fractions of 2.4 Gy) was delivered to the pulmonary arterial and parenchymal lesions, and maintenance sintilimab was started in February 2026.

Response and follow-up

Serial CT showed progressive regression: after two cycles the upper lobe mass measured 5.0 × 2.2 cm and the nodule 0.9 × 0.6 cm, and after four cycles 4.3 × 1.5 cm and 0.5 × 0.6 cm, with no further change after six cycles (Fig. 1). The sum of target-lesion diameters fell from 68 mm to 49 mm, a 27.9% reduction, and the best

overall response by RECIST 1.1 was stable disease. No grade ≥ 3 hematological or non-hematological toxicity and no immune-related adverse event occurred. Post-radiotherapy imaging showed no further shrinkage and no local failure. At the last follow-up (June 11, 2026) he remained on maintenance therapy with sustained stable disease, no local or distant progression, a progression-free survival of 12.1 months, and a satisfactory quality of life (Fig. 3). Written informed consent for publication was obtained.

Patient perspective

The patient was invited to describe his experience of the diagnostic process and of the treatment he received, and he approved the following statement.

For a long time I went from one hospital to another with a cough that would not settle, and at each visit I was told that I had a blood clot in my lungs and was given medicine to thin my blood. Nobody explained to me why the clot was there, and it was only much later that I understood this was the reason my diagnosis took so long. When I was admitted to this hospital, the doctors explained to me, in words I could follow, that what I had was not a blood clot but a tumor growing inside the pulmonary artery, and why it had to be treated differently. I was anxious when they proposed chemotherapy followed by radiotherapy, but every step was discussed with me and with my family before I agreed to it. More than a year later I am still able to take care of myself, and I am grateful for the care I have received here.

Discussion and conclusions

Key findings

This case yields two observations. First, in a patient whose pulmonary arterial filling defects had been treated as PTE, MDM2 amplification was decisive: it resolved a differential that two biopsies and an expert pathology review had left open, and it did so before systemic treatment was committed. Second, in an unresectable, PD-L1-negative tumor with a low mutational burden, chemotherapy-primed PD-1 blockade followed by consolidative radiotherapy and maintenance immunotherapy produced stable disease with a 27.9% reduction and 12.1 months of progression-free survival without high-grade toxicity.

Comparison with similar research

Imaging offers the first opportunity to separate PAIS from PTE. On CT pulmonary angiography, PAIS typically appears as an expansile, mass-like defect that enhances with contrast, distends the vessel, and produces the “wall eclipsing sign,” whereas thrombus is eccentric and non-enhancing.(13, 14) FDG PET/CT adds discrimination, with an SUVmax cut-off of 3.3 proposed for this purpose, and detects distant disease.(15) The test is not infallible, because tumors with abundant myxoid stroma or necrosis may take up little fluorodeoxyglucose.(16) In our patient the concordant hypermetabolism of the intravascular and parenchymal lesions was the diagnostic turning point and made biopsy rational. Laboratory findings are unhelpful: despite an extensive endovascular tumor burden, D-dimer and coagulation studies were normal, consistent with reports that D-dimer cannot be used to exclude PAIS.(17)

Pathological confirmation is limited by the tumor itself. Viable tissue is often obscured by organizing thrombus, small biopsies are consumed by the immunohistochemical panel, and MDM2 staining may be equivocal, so FISH or NGS is required in a substantial proportion of cases.(6, 7) This sequence was reproduced exactly here: the first biopsy was equivocal for MDM2, the expert review could not subtype the tumor, and only NGS settled the diagnosis. Intimal sarcoma is classified among malignant mesenchymal tumors of uncertain differentiation, and its spindle cells may show myofibroblastic, osteosarcomatous, or rhabdomyoblastic features, none of which is specific; sarcomatoid carcinoma, in contrast, is excluded by the combination of complete cytokeratin negativity on two specimens and MDM2 amplification.(5, 6) The molecular findings also explained the biology, because MDM2 amplification drives p53 degradation while the concurrent CDKN2A deletion removes CDK4/6 inhibition, the two events cooperating to deregulate the cell cycle.(18, 19) Proliferation markers do not discriminate reliably: Ki-67 in PAIS has been reported across a wide range, and the index of 10% in this tumor, although low, is compatible with the diagnosis.(20)

Systemic treatment remains empirical. Complete resection is the strongest prognostic factor, with a median survival of 36 months after complete clearance compared with 11 months after incomplete resection, but it was precluded here.(4) Doxorubicin-based chemotherapy failed in all eight patients of one series of locally advanced disease, and a multicentre analysis of MDM2-positive intimal sarcoma found only limited benefit from anthracyclines, gemcitabine, and pazopanib.(8, 9) Anti-angiogenic therapy has produced contrasting outcomes, with a transient response curtailed by toxicity in one report and a 25-month progression-free survival when combined with chemotherapy and surgery in another.(21, 22) Immunotherapy experience is sparse: single-agent checkpoint blockade has limited activity in soft tissue sarcoma,(11) and one patient with PAIS achieved a partial response lasting 24 months with sintilimab plus chemotherapy and endostatin.(23) Radiotherapy has been used after incomplete resection and for palliation; one MDM2-amplified case remained disease-free 7 months after endarterectomy, adjuvant doxorubicin/ifosfamide, and 60 Gy of intensity-modulated radiotherapy.(24, 25) Table 2 summarizes these reports.

Explanations of findings

The response should be read for durability rather than magnitude. The best response was stable disease, and the 27.9% reduction fell just short of the partial-response threshold; in a disease whose historical survival is measured in months, however, 12.1 months without progression is a meaningful outcome.(4, 26) Dimensional stability after radiotherapy should not be interpreted as failure, because irradiated mesenchymal tumors often undergo fibrotic stabilization and coagulative necrosis rather than shrinkage; the absence of local failure is the relevant endpoint.(25) The contribution of each component cannot be separated in a single patient. The immunogenic-cell-death rationale that justified concurrent chemotherapy follows the cancer-immunity cycle and remains hypothesis-generating, particularly because no serial immune profiling was performed.(27) The sequence itself was pragmatic: platinum was chosen on morphological grounds, radiotherapy was delivered once residual disease had plateaued and the dose constraints of adjacent heart and lung could be respected, and maintenance was continued because a complete response had not been achieved.

Strengths and limitations

The strengths of this report are the molecularly confirmed diagnosis in a case that two biopsies had left unresolved, complete documentation of the diagnostic sequence, and serial imaging through 12.1 months.

The limitations are those of a single observation: no causal attribution to any component, no immunological or circulating tumor DNA correlates, follow-up too short to describe the eventual pattern of progression, and MDM2 and CDK4/6 inhibitors not yet tested in this patient. The diagnosis also rests on the concordance of imaging with a parenchymal biopsy, because no direct intravascular sampling was performed. NGS and multidisciplinary expertise are not universally available, which limits generalizability.

Implications and actions needed

Three actions follow. A “pulmonary embolism” that does not behave as one, with persistent defects despite anticoagulation, a hypermetabolic intravascular lesion, no thromboembolic risk factors, or an associated parenchymal mass, should prompt tissue diagnosis and molecular profiling rather than repeated anticoagulation. MDM2 amplification should be tested early in spindle cell tumors of the pulmonary vasculature and treated as diagnostic when present, not merely supportive. Treatment decisions should be made by multidisciplinary teams and, where possible, within registries that capture molecular profile, treatment sequence, and outcome; MDM2 antagonists and CDK4/6 inhibitors are rational options at progression,(18, 19) whereas PDGFRA and EGFR coactivation and KIT gains remain unproven targets.(28)

Conclusions

MDM2 amplification can be the decisive diagnostic test in PAIS when histology is indeterminate, and it should be sought before empirical treatment is started. In this patient, chemotherapy-primed PD-1 blockade followed by consolidative radiotherapy and maintenance immunotherapy produced durable disease control in an unresectable, immunologically cold tumor. Because a single observation cannot establish efficacy, multicentre registries and prospective evaluation of chemo-immunotherapy and MDM2/CDK4-6-directed strategies are needed.

Abbreviations

CK-pan pan-cytokeratin

CT computed tomography

EMA epithelial membrane antigen

FDG fluorodeoxyglucose

IHC immunohistochemistry

MDM2 murine double minute 2

NGS next-generation sequencing

OS overall survival

PAIS pulmonary artery intimal sarcoma

PD-1 programmed cell death protein 1

PD-L1 programmed death-ligand 1

PET positron emission tomography

PFS progression-free survival

PTE pulmonary thromboembolism

RECIST Response Evaluation Criteria in Solid Tumors

SMA smooth muscle actin

SUVmax maximum standardized uptake value

TMB tumor mutational burden

VTE venous thromboembolism

Declarations

Ethics approval and consent to participate

All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the Declaration of Helsinki (as revised in 2013). Formal ethics approval was not required for a retrospective, single-patient case report at our institution. Written informed consent was obtained from the patient for participation in his care and for the publication of this case report and the accompanying images, and a copy of the written consent is available for review by the editorial office of this journal.

Consent for publication

Written informed consent for publication of this case report and any accompanying images was obtained from the patient. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Availability of data and materials

All data generated or analysed during this study are included in this published article [and its additional files]. Any additional clinical or molecular details are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Hongmei Mou and Yi Wang contributed equally to this work and share first authorship. Conception and design: Hongmei Mou, Yi Wang, Yajie Zhu; provision of study materials or patients: Yajie Zhu, Xi Xu; collection and assembly of data: Hongmei Mou, Yi Wang, You Wang; data analysis and interpretation: You Wang, Xiaorong Liu, Xi Xu; manuscript writing: all authors; final approval of the manuscript: all authors. All authors read and approved the final manuscript.

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Authors' information

Not applicable.

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30. Clinical. imaging, and laboratory features that help discriminate pulmonary artery intimal sarcoma from pulmonary thromboembolism.

Tables

Table 1. Clinical, imaging, and laboratory features that help discriminate pulmonary artery intimal sarcoma from pulmonary thromboembolism

Feature	PAIS	PTE	Present case
Clinical setting	No typical VTE risk factors; progressive dyspnea, cough, hemoptysis; often middle-aged adults	Immobilization, recent surgery, active cancer, thrombophilia	No VTE risk factors; cough, sputum, chest discomfort
D-dimer and coagulation	Frequently normal or only mildly elevated	Usually elevated	Normal
CT pulmonary angiography	Expansile, mass-like defect filling and distending the lumen; heterogeneous enhancement; vessel caliber increased	Eccentric, non-enhancing defect; vessel caliber normal or reduced; may resolve on anticoagulation	Expansile defects from the main pulmonary artery into both branches, with a 5.6 × 2.9 cm hilar mass
Wall eclipsing sign	Present (tumor eclipses the arterial wall)	Absent	Present
Response to anticoagulation	None	Expected improvement or resolution	No improvement; hemoptysis after rivaroxaban
18F-FDG PET/CT	Uptake within the intravascular lesion, exceeding that of thrombus	No or negligible uptake	SUVmax 5.6 within the filling defects; concordant parenchymal uptake
Histology and IHC	Spindle or polygonal cells; vimentin positive; lineage markers negative; MDM2 positive	Organizing thrombus without atypia or mitoses	Spindle and polygonal cells; EMA focal, SMA weak; lineage markers negative; MDM2 strongly positive
Molecular profile	MDM2 (12q13-15) amplification with or without CDK4 co-amplification; CDKN2A deletion	None	MDM2 amplification, CDKN2A deletion, TMB 1.95 mutations/Mb

PAIS, pulmonary artery intimal sarcoma; PTE, pulmonary thromboembolism; VTE, venous thromboembolism; IHC, immunohistochemistry; SUVmax, maximum standardized uptake value; TMB, tumor mutational burden.

Table 2. Reported cases of pulmonary artery intimal sarcoma with molecular characterization, treatment, and outcome

Study	Age/sex	Initial diagnosis	Molecular features	Treatment	Outcome
Liu et al. 2024(2)	34/F	PTE	MDM2 amplification, FRS2 amplification	Emergency surgery (incomplete resection)	Rapid relapse; death (OS 2 months)
Wu et al. 2019(21)	63/M	PTE	MDM2 positive, KIT amplification, PD-L1 negative, TMB-low	Pemetrexed, then anlotinib	Transient partial response; discontinued for toxicity; death (OS 8 months)
Moguillansky et al. 2019(26)	44/F	PTE	MDM2 positive	Doxorubicin/ifosfamide, then paclitaxel, imatinib, and NLG802	Progressive disease across lines; death (OS 12 months)
Takemoto et al. 2017(29)	52/F	PAS	PDGFRA positive, KIT negative	Doxorubicin, then salvage pneumonectomy	Brain and adrenal metastases after surgery; death (OS 12 months)
Atahan et al. 2024(24)	46/F	PTE	MDM2 amplification	Endarterectomy, adjuvant doxorubicin/ifosfamide, and radiotherapy (60 Gy)	No evidence of disease at 7 months
Liang et al. 2025(22)	38/M	PAS	MDM2 amplification, SATB2 positive	Ifosfamide/pirarubicin plus anlotinib, surgery, then radiotherapy	Sustained disease control (PFS 25 months)
Zhang et al. 2023(23)	52/M	PAS	CDK4 positive	Anlotinib, then sintilimab plus chemotherapy, then maintenance sintilimab	Partial response lasting 24 months
Present case	51/M	PTE	MDM2 amplification, CDKN2A deletion, PD-L1 negative, TMB 1.95 mutations/Mb	Sintilimab/docetaxel/cisplatin ×6, sintilimab/docetaxel ×2, radiotherapy (60 Gy), maintenance sintilimab	Stable disease, 27.9% regression; PFS 12.1 months

PAIS, pulmonary artery intimal sarcoma; PAS, pulmonary artery sarcoma; PTE, pulmonary thromboembolism; OS, overall survival; PFS, progression-free survival; TMB, tumor mutational burden; NLG802, an anti-PD-1 antibody.

Figures

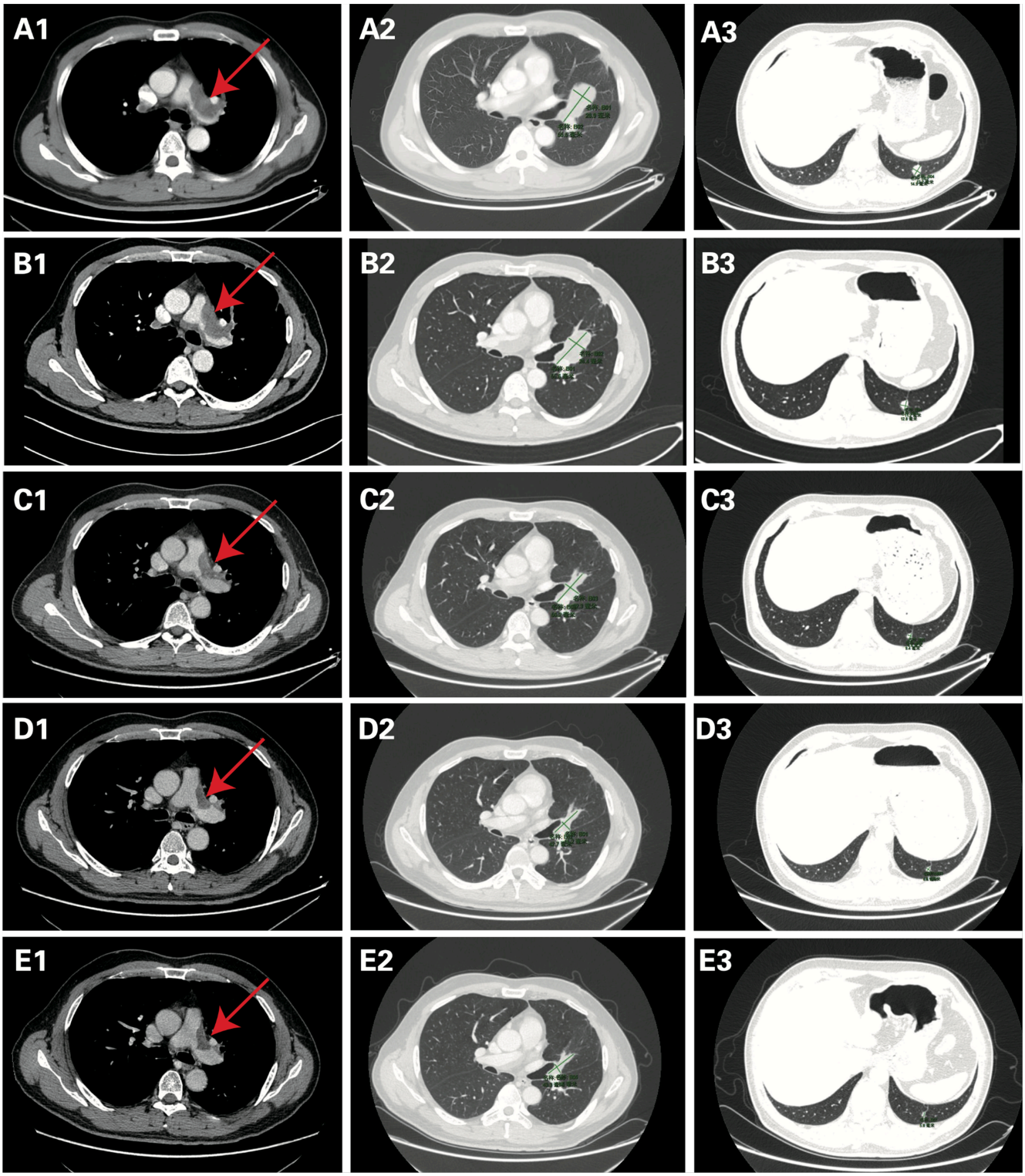


Figure 1

Serial contrast-enhanced chest computed tomography during immunochemotherapy. The figure is arranged as a matrix in which rows (A-E) represent sequential time points and columns (1-3) represent the three target and non-target lesions: column 1 (A1-E1) shows the filling defects in the main pulmonary artery extending

into both branches (arrowheads), column 2 (A2-E2) the left hilar/upper lobe mass, and column 3 (A3-E3) the left lower lobe nodule. (A) Baseline on admission to our institution (April 21, 2025): extensive filling defects, a 5.6 × 2.9 cm left upper lobe mass, and a 1.5 × 1.2 cm lower lobe nodule. (B) Initial diagnostic computed tomography at first presentation (March 8, 2025), which showed filling defects interpreted as pulmonary embolism and led to anticoagulation. (C) After two cycles (July 24, 2025): the mass decreases to 5.0 × 2.2 cm and the nodule to 0.9 × 0.6 cm. (D) After four cycles (September 18, 2025): the mass decreases to 4.3 × 1.5 cm and the nodule to 0.5 × 0.6 cm. (E) After six cycles (December 18, 2025): no further interval change; the sum of target-lesion diameters has fallen from 68 mm to 49 mm, corresponding to a best response of stable disease by RECIST 1.1.

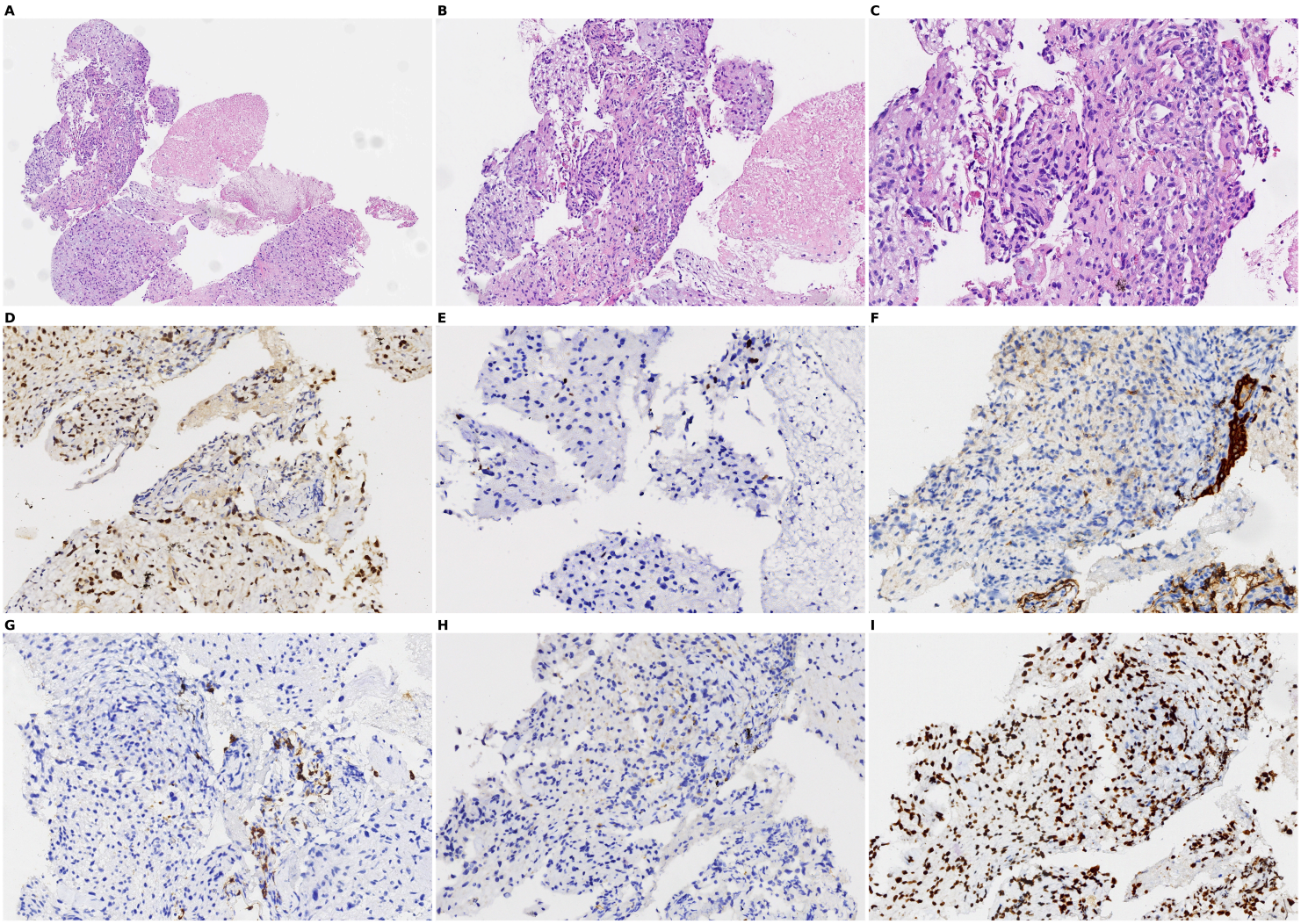


Figure 2

Histopathological and immunohistochemical features of the percutaneous lung biopsy specimen. (A-C) Hematoxylin and eosin staining at low (×100), intermediate (×200), and high (×400) magnification, respectively, showing a malignant spindle and polygonal cell neoplasm with a fascicular growth pattern, pronounced nuclear atypia, and mitotic activity. (D) Strong, diffuse nuclear positivity for MDM2 (×400). (E) Ki-67 proliferation labeling index of approximately 10% (×400). (F) Focal and equivocal positivity for epithelial membrane antigen (×400). (G,H) Complete negativity for lineage-specific markers CK-pan (G) and desmin (H)

(×400), aiding exclusion of carcinoma and muscle-derived sarcoma. (I) Retained nuclear expression of the SWI/SNF complex protein BRG1/SMARCA4 (×400).

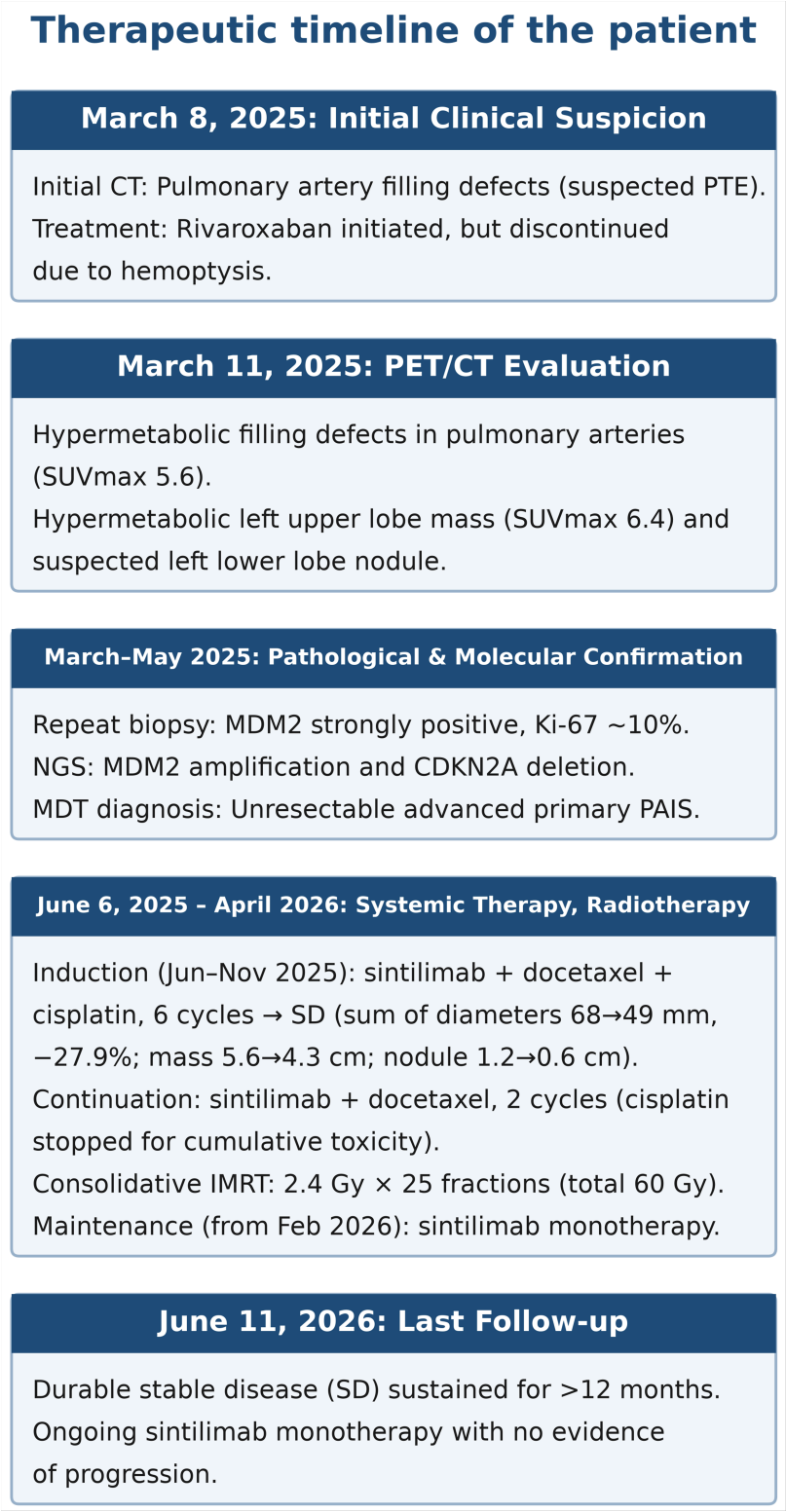


Figure 3

Clinical timeline from initial presentation through the latest follow-up. The timeline summarizes the diagnostic trajectory, multimodal treatment (six cycles of sintilimab/docetaxel/cisplatin followed by two

cycles of sintilimab/docetaxel, consolidative intensity-modulated radiotherapy 60 Gy in 25 fractions, and maintenance sintilimab monotherapy), and outcome (stable disease, progression-free at 12.1 months).

Supplementary Files

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- Additionalfile1CAREchecklist.docx