World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

PULMONARY PLEOMORPHIC CARCINOMA WITH KRAS G12C MUTATION AND HIGH PD-L1 EXPRESSION: A CASE REPORT AND LITERATURE REVIEW

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World Journal of Advanced Research and Reviews, 2026, 31(02), 647–654

Publication history: Received on 04 July 2026; revised on 10 August 2026; accepted on 12 August 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.31.2.2107

ABSTRACT

Pulmonary pleomorphic carcinoma (PPC) is a rare subtype of pulmonary sarcomatoid carcinoma (PSC) within the non-small cell lung cancer category, characterized by significant histological heterogeneity that complicates diagnosis. A 71-year-old male with a 55-pack-year smoking history and chronic obstructive pulmonary disease (COPD) presented with four months of progressive dyspnea, nonproductive cough, pleuritic chest pain, weight loss, and hemoptysis. Physical examination identified decreased breath sounds in the lower right lung field and hypoxemia.

Contrast-enhanced computed tomography (CT) revealed a large (7.5 cm) mass in the lower right lobe with associated hilar lymphadenopathy. PET-CT demonstrated intense FDG uptake (SUVmax 18.5) without evidence of distant metastasis. CT-guided core biopsy revealed a high-grade neoplasm composed of pleomorphic spindle cells and multinucleated giant cells with necrosis. Immunohistochemical (IHC) analysis confirmed PSC, positive for cytokeratin (AE1/AE3), vimentin, and p40, and negative for TTF-1 and Napsin A. Next-generation sequencing identified a KRAS G12C mutation, high tumor mutational burden, and 60% PD-L1 expression. The patient underwent right lower lobectomy with nodal dissection, achieving R0 resection of stage IIIB disease, followed by adjuvant carboplatin-paclitaxel chemotherapy and mediastinal radiotherapy.

At 12 months, surveillance CT identified bilateral pulmonary nodules confirmed as metastatic recurrence, prompting initiation of pembrolizumab. After six months, the disease progressed to involve the liver and bones, leading to death 22 months after diagnosis.

Keywords: Pulmonary pleomorphic carcinoma; Sarcomatoid carcinoma; Smoking history; Chronic obstructive pulmonary disease; Non-small cell lung carcinoma; Programmed Death-Ligand 1

1 INTRODUCTION

Pulmonary sarcomatoid carcinoma (PSC) is a rare and biologically distinctive group of poorly differentiated non-small cell lung carcinomas. According to the 2021 World Health Organization (WHO) classification of pulmonary tumors, the PSC family consists of five distinct histological subtypes. [1,2]

Pulmonary pleomorphic carcinoma (PPC) is the most frequently encountered pattern within this family, accounting for over 50% of all cases. It is diagnosed when a poorly differentiated adenocarcinoma, squamous cell carcinoma, or large cell carcinoma contains at least ten percent spindle cell and/or giant cell differentiation. [1,3,4]

Clinically, patients typically present in the seventh decade of life with a heavy smoking history and nonspecific respiratory complaints, including cough, dyspnea, chest pain, hemoptysis, and constitutional weight loss, often mimicking a pneumonic process or abscess on initial evaluation. [1] Because radiologic and cytologic features are frequently indistinguishable from conventional non-small cell lung cancer or, occasionally, from primary pulmonary sarcoma, definitive diagnosis rests on histopathology supplemented by a broad IHC panel. Ideally, diagnosis should be performed on a resection specimen rather than a limited biopsy. [3]

Management follows a multimodal paradigm combining surgery, chemotherapy, radiotherapy, and, increasingly, immune checkpoint inhibition, but outcomes remain considerably worse than for other non-small cell lung cancer histology owing to aggressive biology, early metastatic spread, and relative chemoresistance. [1] The case reported here illustrates these features and is discussed in the context of the current literature

2 CLINICAL PRESENTATION

2.1 Clinical Presentation

A 71-year-old male with a 55-pack-year smoking history and a background of COPD presented with a four-month history of progressive dyspnea on exertion, a persistent dry cough, and intermittent pleuritic right-sided chest pain. He reported an unintentional weight loss of approximately 15 pounds over the same period. Initial management by his primary care physician included a course of oral antibiotics for presumed community-acquired pneumonia and an inhaled bronchodilator, which provided no symptomatic relief. His symptoms had worsened in the preceding two weeks, with the onset of hemoptysis and profound fatigue, prompting him to seek urgent specialized medical attention. He denied any fevers or night sweats.

2.2 Physical Examination and Imaging Findings

Physical examination revealed an ill-appearing male in mild respiratory distress, with a performance status of ECOG 1. Vital signs were stable except for a resting oxygen saturation of 91% on room air. Chest auscultation demonstrated decreased breath sounds and dullness to percussion over the right lower lung field, with no audible wheezes. There was no palpable lymphadenopathy or digital clubbing. Significant personal history included hypertension and hyperlipidemia; family history was unremarkable for malignancy. Laboratory studies were notable for a normocytic anemia (Hb 11.2 g/dL) and a mildly elevated platelet count.

Contrast-enhanced computed tomography (CT) of the chest revealed a large, irregular, heterogeneous mass measuring 7.5 cm with central low attenuation in the right lower lobe, associated with ipsilateral hilar lymphadenopathy. A whole-body PET-CT showed intense FDG-avid uptake within the primary tumor (SUVmax 18.5) and the hilar node, with no evidence of distant metastases. The clinical and radiological differential diagnosis included primary bronchogenic carcinoma (specifically non-small cell lung cancer), a benign necrotizing infection or abscess, and less commonly, pulmonary sarcoma or lymphoma.

2.3 Multidisciplinary Tumor Board Discussion

The case was presented at the multidisciplinary thoracic tumor board, where the discussion centered on the diagnostic uncertainty given the imaging characteristics and the patient's symptoms. The central debate involved the optimal approach to establishing a definitive diagnosis and the subsequent treatment strategy. The team weighed the risks and

benefits of a CT-guided transthoracic needle biopsy versus a more definitive surgical approach, such as a video-assisted thoracoscopic surgery (VATS) wedge resection.

Given the patient's borderline performance status and the location of the mass with associated hilar lymphadenopathy, the consensus favored a CT-guided core needle biopsy for histological and molecular characterization. The recommended next step was to perform the biopsy and, if malignancy was confirmed, to proceed with a comprehensive staging workup, including a brain MRI, to rule out occult intracranial disease.

2.4 Special Studies, Pathology, and Diagnosis

Histopathological examination of the core biopsy revealed a high-grade malignant neoplasm with substantial necrosis (25%). The tumor comprised mixed cell populations, including pleomorphic spindle cells arranged in fascicles (approximately 50%) and large, bizarre, multinucleated giant cells with abundant eosinophilic cytoplasm (approximately 35%). The remaining cells were highly malignant but lacked a distinct architectural pattern. Overall, the biopsy histomorphology was consistent with NSCLC showing pleomorphic and spindle cell features, pending evaluation of the entire tumor.

IHC was pivotal in confirming the diagnosis, with the tumor cells showing strong positivity for cytokeratin (AE1/AE3), vimentin, and p40, and negativity for TTF-1, Napsin A, Desmin, Myogenin, S100, synaptophysin, and CD45. This profile supported sarcomatoid carcinoma with epithelial and mesenchymal differentiation. Molecular testing by next-generation sequencing revealed a KRAS G12C mutation and a high tumor mutational burden (TMB) of 18 mutations per megabase, with no actionable EGFR, ALK, or ROS1 alterations. Programmed Death-Ligand 1 (PD-L1) expression was 60% in the tumor cells.

2.5 Treatment, Outcome, and Follow-up

After staging confirmed localized disease (cT4N1M0, Stage IIIA), the patient underwent a right lower lobectomy with en bloc resection of the involved hilar lymph nodes, achieving a complete (R0) resection. Examination of the entire tumor mass confirmed the diagnosis of Sarcomatoid carcinoma of the lung, pleomorphic subtype. Given the large tumor size, high-grade features, and nodal involvement, adjuvant therapy was recommended. The patient received four cycles of carboplatin and paclitaxel, followed by adjuvant radiotherapy to the mediastinum.

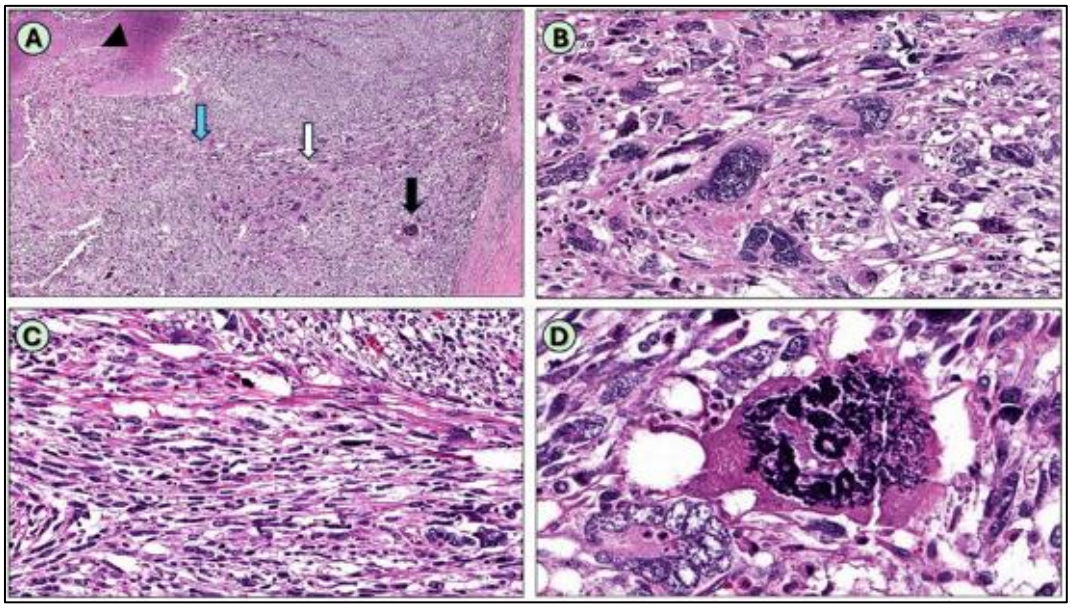

Figure 1: Microscopic features of sarcomatoid carcinoma of the lung, pleomorphic variant

- 1A**:** Low-power view showing three different components, spindle cells (blue arrow), giant cells (white arrow), and pleomorphic cells (black arrow). Black arrowhead indicates area of necrosis (H&E X20)
- 1B**:** High-power view showing malignant giant cells (H&E X40)
- 1C**:** High-power view showing malignant spindle cells (H&E X40)
- 1D: High-power view showing bizarre pleomorphic cells with abnormal mitoses (H&E X60)

Follow-up surveillance at 12 months with a CT scan revealed multiple new, small bilateral pulmonary nodules, consistent with metastatic recurrence. A repeat biopsy of a new nodule confirmed metastatic sarcomatoid carcinoma with an identical molecular profile. Second-line therapy with the immunotherapy agent pembrolizumab was initiated, given the high PD-L1 expression. The patient initially had a mixed response, but after 6 months of therapy (18 months from initial diagnosis), progressive disease was documented with new liver and bone metastases. The patient's performance status declined significantly, and he was transitioned to best supportive care, subsequently expiring due to complications of progressive disease 22 months after his initial diagnosis.

3 DISCUSSION

3.1 Background

Sarcomatoid carcinoma of the lung has been recognized as a distinct clinicopathologic entity within the non-small cell lung cancer spectrum since the unification of its diagnostic criteria after 1999, when tumors displaying pleomorphic, sarcomatoid, or sarcomatous elements were formally consolidated within the carcinoma category rather than classified as true sarcomas. [5,6] According to the World Health Organization (WHO) 2021 classification of pulmonary tumors, the pulmonary sarcomatoid carcinoma (PSC) family consists of five distinct histological subtypes: Pleomorphic, spindle cell, giant cell, carcinosarcoma, and blastoma, each with its own definition. [2,3,5,6]

Pulmonary Pleomorphic Carcinoma (PPC) is defined as a poorly differentiated conventional lung cancer, such as adenocarcinoma, squamous cell carcinoma, or large cell carcinoma, that contains a significant sarcoma-like component. The tumor must consist of at least 10% spindle cells and/or giant cells, or be a carcinoma composed entirely of a mix of these two cell types. This is the most common subtype in the family. [4]

Pulmonary Spindle Cell Carcinoma (SpCC) is a rare variant of sarcomatoid carcinoma characterized by a uniform, elongated appearance. The tumor must consist of at least 10% spindle cells and/or giant cells, or be a carcinoma composed entirely of a mix of these two cell types. [7]

Pulmonary Giant Cell Carcinoma (PGCC) is defined as a highly aggressive variant comprised of massively oversized, abnormally shaped cancer cells. The tumor must consist almost entirely of highly malignant giant cells, either multinucleated or giant cells, and must lack any traditional spindle cell or differentiated lung cancer structures. [8]

Pulmonary Blastoma (PB) is a rare tumor that resembles the developing embryonic lung. An incredibly rare tumor that mimics the appearance of a developing embryonic lung. [6]

Pulmonary sarcomatoid carcinoma is exceedingly uncommon, accounting for roughly 0.1 to 0.4 percent of malignant lung tumors and approximately 1 percent of all non-small cell lung cancers [1], a rarity corroborated by large national registries, in which the entity represented well under 1 percent of the malignant lung tumor burden. [9] Population-based series place the mean age at diagnosis in the mid-to-late sixties with a consistent male predominance, a right-sided and upper-lobe predilection, and a strong association with substantial cigarette smoking exposure, mirroring the demographic and exposure profile of squamous-lineage high-grade lung cancers more broadly. [5,10] Broader epidemiologic surveillance of sarcomatoid carcinoma across all primary sites has documented a marked rise in age-adjusted incidence in recent decades, with the respiratory tract among the sites showing the steepest increase. However, this trend must be interpreted with caution given the heterogeneity of the underlying registries. [11,12]

3.2 Pathogenesis, Pathophysiology

Pulmonary pleomorphic carcinoma (PPC), the most prevalent subtype of pulmonary sarcomatoid carcinoma, is believed to originate from a single monoclonal bronchogenic epithelial precursor of conventional non-small cell lung cancer, rather than representing a collision of two independently arising neoplasms. The spindle and giant cell components arise through epithelial–mesenchymal transition (EMT) rather than from true mesenchymal cells. [1,13,14] EMT is mediated by TGF-β and MAPK signaling pathways with loss of E-cadherin and upregulation of TWIST, SNAIL, and ZEB1, and is triggered by chronic tobacco exposure, hypoxia, and acidosis. [15]

Genomic profiling consistently identifies TP53 as the most frequently altered gene, together with recurrent KRAS mutations; in this patient, the latter is KRAS G12C. MET exon 14 skipping occurs in a substantial minority of cases, while EGFR, ALK, and ROS1 alterations are comparatively rare. EMT imparts increased motility, invasiveness, and intrinsic chemoresistance, promoting early vascular and lymphatic dissemination.

Proliferation that exceeds neovascularization leads to the central necrosis observed on imaging. Bronchial compression and ventilation–perfusion mismatch account for dyspnea and hypoxemia; vessel erosion by necrotic tumor causes hemoptysis; and pleural involvement results in pleuritic pain.

Histopathologically, co-expression of cytokeratin (AE1/AE3) and vimentin represents the morphologic hallmark of EMT, while p40 positivity with TTF-1 negativity excludes true sarcoma. Elevated tumor mutational burden (18 mut/Mb) and PD-L1 expression (60%) plausibly underlie both chemoresistance and potential susceptibility to checkpoint inhibitors. [16,17,18,19]

3.3 Comparative Analysis of Our Case with Existing Literature

The present case shares substantial overlaps with several recently published reports describing molecularly annotated pulmonary sarcomatoid or pleomorphic carcinoma. Huang and colleagues described a heavily pretreated patient with a KRAS G12C mutation and a concurrent ALK rearrangement whose disease, refractory to targeted therapy and chemotherapy, achieved a durable response to a PD-1 inhibitor combined with an anti-angiogenic agent, underscoring the potential of checkpoint blockade in KRAS-mutant sarcomatoid disease even though the driver mutation itself differed structurally from the present case, which lacked a concurrent fusion event. [20]

Hayashi and colleagues reported a series of three driver-negative, high-PD-L1 pleomorphic carcinoma patients treated with checkpoint inhibitors in whom outcomes were markedly discordant, ranging from no response and early death to durable partial remission, directly illustrating that high PD-L1 expression, as observed in the present patient, does not reliably forecast benefit, an observation that needs further studies. [21]

Cimpeanu and colleagues described a 69-year-old, 60-pack-year smoker with PD-L1 expression exceeding 50 percent who achieved a dramatic and durable response to first-line pembrolizumab, a demographic and biomarker profile strikingly similar to the index case. However, that patient received immunotherapy up front rather than after relapse. [22]

Wen Y and colleagues reported a patient with comparably high PD-L1 expression and tumor mutational burden treated with combined anti-angiogenic and checkpoint inhibitor therapy who ultimately developed hyperprogressive disease, a cautionary parallel to the eventual treatment failure observed in our case. [23]

An additional report documented durable disease control with pembrolizumab after failure of another checkpoint inhibitor in stage III sarcomatoid carcinoma, further reinforcing the emerging yet inconsistent role of immunotherapy in this type of tumor. [24]

Collectively, these reports situate the present case within a recognizable but heterogeneous pattern of biomarker-high disease with unpredictable immunotherapeutic response.

4 WHAT HAVE WE LEARNED FROM THIS CASE?

This case reinforces several practical lessons for clinicians encountering suspected PSC. First, the initial clinical and radiologic picture, an FDG-avid, centrally necrotic mass with hilar adenopathy in a heavily smoking older adult, is frequently indistinguishable from conventional bronchogenic carcinoma or a necrotizing infectious process. A trial of antibiotics without tissue diagnosis should not delay biopsy when red-flag features such as hemoptysis, rapid symptom progression, and weight loss are present.

Second, the diagnosis and its subtyping often cannot be fully established on limited core biopsy material alone, since sampling may under-represent the spindle or giant cell component, and definitive classification frequently awaits examination of the resected specimen, as occurred here.

Third, comprehensive molecular and immune biomarker testing, including PD-L1 and tumor mutational burden, should be pursued routinely given the comparatively high prevalence of actionable or immunotherapy-relevant findings in this histology, even though, as this case and several comparators demonstrate, high PD-L1 expression and tumor mutational burden are favorable but not fully reliable predictors of durable immunotherapeutic response [16,18].

Finally, close surveillance imaging following resection is warranted given the substantial risk of recurrence even after margin-negative surgery, and clinicians should maintain a low threshold for rebiopsy of recurrent disease to confirm molecular concordance before selecting subsequent therapy, as was appropriately done in this patient. This case neither overturns nor is inconsistent with existing knowledge. However, it adds a well-annotated data point supporting the incorporation of comprehensive upfront molecular profiling into the standard diagnostic pathway for this rare and aggressive tumor.

Abbreviations

- Pulmonary pleomorphic carcinoma (PPC)
- Sarcomatoid carcinoma (PSC);
- Chronic obstructive pulmonary disease (COPD)
- Non-small cell lung carcinoma (NSCLC)
- Immunohistochemical (IHC)
- Programmed Death-Ligand 1 (PD-L1)

5 CONCLUSION

This case contributes a clinically detailed and molecularly annotated example of pulmonary pleomorphic carcinoma arising in a heavily smoking older man, capturing the full arc from diagnostic ambiguity through surgical cure, biomarker-guided salvage immunotherapy, and eventual disease progression. It adds to the still-limited body of published cases correlating KRAS G12C mutation status with high PD-L1 expression, tumor mutational burden, and tumor trajectory in this histology. The tumor showed an initial mixed response to pembrolizumab, followed by progression, echoing the discordant outcomes reported elsewhere in literature.

By documenting the full diagnostic workup, multidisciplinary decision-making, and longitudinal molecular concordance between primary and recurrent disease, this report offers the medical community a concrete reference point for counseling patients and selecting therapy in molecularly high-risk, biomarker-high PSC, while reaffirming that further prospective study is needed to identify which biomarker-high patients will truly benefit from checkpoint blockade.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Acknowledgments

Special thanks to Andressa Balbi, MD, for their assistance in reviewing the final manuscript. Additionally, we appreciate the assistance of Grammarly's language editor and the Manus 1.6 program, which provided valuable writing support by identifying and correcting errors in grammar, spelling, punctuation, and writing style, ultimately enhancing the manuscript.

Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

This work did not involve any studies on human or animal subjects performed by the authors. It was a retrospective review of archival pathology material collected during routine clinical care, and all data were fully de-identified before analysis.

Statement of informed consent

The patient expired, and all attempts to reach the family members were unsuccessful. Therefore, the paper has been sufficiently anonymized to maintain patient confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

REFERENCES

[1] Li X, Wu D, Liu H, Chen J. Pulmonary sarcomatoid carcinoma: progress, treatment and expectations. Therapeutic Advances in Medical Oncology. 2020 Aug; 12:1758835920950207.

[2] Nicholson AG, Tsao MS, Beasley MB, Borczuk AC, Brambilla E, Cooper WA, Dacic S, Jain D, Kerr KM, Lantuejoul S, Noguchi M. The 2021 WHO classification of lung tumors: impact of advances since 2015. Journal of Thoracic Oncology. 2022 Mar 1;17(3):362-87

[3] Tsao MS, Nicholson AG, Maleszewski JJ, Marx A, Travis WD. Reprint of "Introduction to 2021 WHO classification of thoracic tumors". Journal of Thoracic Oncology. 2022 Mar 1;17(3):337-40.

[4] Boland JM, Larsen BT. Sarcomas and sarcomatoid neoplasms of the lungs and pleural surfaces. In Practical Pulmonary Pathology: A Diagnostic Approach 2022 Jan 1 (pp. 497-558). Elsevier

[5] Chen Z, Liu J, Min L. Clinicopathological characteristics, survival outcomes and prognostic factors in pleomorphic carcinoma: a SEER population-based study. BMC Pulmonary Medicine. 2022 Mar 31;22(1):116.

[6] Baldovini C, Rossi G, Ciarrocchi A. Approaches to tumor classification in pulmonary sarcomatoid carcinoma. Lung Cancer: Targets and Therapy. 2019 Dec 5:131-49

[7] Samanta RP, Agarwal S, Agrawal R, Saha K, Sengupta S. Pulmonary spindle cell carcinoma: as rare as a hen's teeth. Cureus. 2024 Feb 15;16(2): e54266

[8] Moran CA. Primary pulmonary carcinomas with spindle and/or giant cell features: a review with emphasis on classification and pitfalls in diagnosis. Diagnostics. 2023 Jul 26;13(15):2477

[9] Xie Y, Lin Z, Shi H, Sun X, Gu L. The prognosis of pulmonary sarcomatoid carcinoma: development and validation of a nomogram based on SEER. Technology in Cancer Research & Treatment. 2022 Jun; 21:15330338221109647

[10] Steuer CE, Behera M, Liu Y, Fu C, Gillespie TW, Saba NF, Shin DM, Pillai RN, Pakkala S, Owonikoko TK, Khuri FR. Pulmonary sarcomatoid carcinoma: an analysis of the national cancer database. Clinical Lung Cancer. 2017 May 1;18(3):286-92.

[11] Xu Z, Wang L, Tu L, Liu Y, Xie X, Tang X, Luo F. Epidemiology of and prognostic factors for patients with sarcomatoid carcinoma: a large population-based study. American Journal of Cancer Research. 2020 Nov 1;10(11):3801.

[12] Chen M, Yang Q, Xu Z, Luo B, Li F, Yu Y, Sun J. Survival analysis and prediction model for pulmonary sarcomatoid carcinoma based on SEER database. Frontiers in Oncology. 2021 May 31; 11:630885.

[13] Liu X, Wang F, Xu C, Chen X, Hou X, Li Q, Li P, Xie Z, Liu Y, Chang L, Guan Y. Genomic origin and intratumor heterogeneity revealed by sequencing on carcinomatous and sarcomatous components of pulmonary sarcomatoid carcinoma. Oncogene. 2021 Jan 28;40(4):821-32.

[14] Kwon HJ, Lee S, Han YB, Lee J, Kwon S, Kim H, Chung JH. Genomic landscape of pulmonary sarcomatoid carcinoma. Cancer Research and Treatment: Official Journal of Korean Cancer Association. 2024 Apr 1;56(2):442-54.

[15] Wang Q, Guo H, Wang Y, He T, Diao M, Wu Y, Xiong A, Zhou F, Li W, Cheng L, Zhao C. The pathogenesis and therapeutic management of rare pulmonary sarcomatoid carcinoma: a narrative review. Translational Lung Cancer Research. 2025 Nov 27;14(11): 5137..

[16] Zhou F, Huang Y, Cai W, Li J, Su C, Ren S, Wu C, Zhou C. The genomic and immunologic profiles of pure pulmonary sarcomatoid carcinoma in Chinese patients. Lung Cancer. 2021 Mar 1; 153:66-72.

[17] Liu XW, Chen XR, Rong YM, Lyu N, Xu CW, Wang F, Sun WY, Fang SG, Yuan JP, Wang HJ, Wang WX. MET exon 14 skipping mutation, amplification and overexpression in pulmonary sarcomatoid carcinoma: A multi-center study. Translational Oncology. 2020 Dec 1;13(12):100868.

[18] Wang F, Cali Daylan AE, Deng L, Yang J, Sharma J, Su C, Li S, Zang X, Halmos B, Borczuk A, Cheng H. Heterogeneous expression of PD-L1, B7x, B7-H3, and HHLA2 in pulmonary sarcomatoid carcinoma and the related regulatory signaling pathways. Cancers. 2023 Jun 27;15(13):3372.

[19] Stephan-Falkenau S, Streubel A, Mairinger T, Blum TG, Kollmeier J, Mairinger FD, Bauer T, Pfannschmidt J, Hollmann M, Wessolly M. Integrated clinical, molecular and immunological characterization of pulmonary sarcomatoid carcinomas reveals an immune escape mechanism that may influence therapeutic strategies. International Journal of Molecular Sciences. 2023 Jun 23;24(13):10558.

[20] Huang N, Qu T, Zhang C, Li J. Case report: Successful treatment of advanced pulmonary sarcomatoid carcinoma with BUBIB-ALK rearrangement and KRAS G12C mutation by sintilimab combined with anlotinib. Frontiers in Oncology. 2024 Feb 13; 14:1269148.

[21] Hayashi K, Tokui K, Inomata M, Azechi K, Mizushima I, Takata N, Taka C, Okazawa S, Kambara K, Imanishi S, Miwa T. Case series of pleomorphic carcinoma of the lung treated with immune checkpoint inhibitors. in vivo. 2021;35(3):1687-92.

[22] Cimpeanu E, Ahmed J, Zafar W, DeMarinis A, Bardarov SS, Salman S, Bloomfield D. Pembrolizumab-emerging treatment of pulmonary sarcomatoid carcinoma: a case report. World Journal of Clinical Cases. 2020 Jan 6;8(1):97.

[23] Wen Y, Dong Y, Yi L, Yang G, Xiao M, Li Q, Zhao C, Ye D, Yao Y. Anlotinib combined with pembrolizumab as first-line treatment for advanced pulmonary sarcomatoid carcinoma: a case report and literature review. Frontiers in Oncology. 2023 Oct 18; 13:1241475.

[24] Nishino K, Kunimasa K, Kimura M, Inoue T, Tamiya M, Kuhara H, Kumagai T. Favorable response to pembrolizumab after durvalumab failure in stage III sarcomatoid carcinoma of the lung: a case report. BMC Pharmacology and Toxicology. 2020 Apr 3;21(1):26.