

Advanced pulmonary carcinoid managed with chemoimmunotherapy and antiangiogenic agent: A case report and literature review

LIQIU SUN and MENG YING LIU

Department of Oncology, Tumor Hospital of Mudanjiang, Mudanjiang, Heilongjiang 157009, P.R. China

Received June 24, 2025; Accepted March 17, 2026

DOI: 10.3892/mco.2026.2962

Abstract. Pulmonary carcinoid (PC) tumors are rare, well-differentiated neuroendocrine neoplasms of the lung with indolent progression and limited therapeutic consensus in advanced stages. While surgery remains the mainstay for localized disease, optimal management strategies for progressive or unresectable cases remain debated. A case of a 56-year-old man diagnosed with a left upper lobe PC was presented. Initial treatment consisted of platinum-based chemotherapy followed by traditional Chinese medicine. Upon disease progression, he received thoracic radiotherapy, achieving transient stabilization. However, subsequent radiologic surveillance revealed enlargement of mediastinal lymph nodes, prompting the initiation of a triple-modality systemic regimen comprising lobaplatin, the PD-1 inhibitor sintilimab, and the antiangiogenic agent anlotinib. After two treatment cycles, follow-up imaging demonstrated marked lymph node regression with satisfactory tolerability. This case highlights the potential therapeutic value of integrating chemotherapy, immunotherapy and antiangiogenic therapy in advanced PCs, particularly when standard surgical approaches are unfeasible. Although randomized data remain scarce, experience suggests that multimodal systemic therapy may offer disease control in selected patients. Further prospective studies are warranted to refine treatment paradigms for this uncommon malignancy.

Introduction

Neuroendocrine tumors (NETs) are a heterogeneous class of neoplasms originating from peptidergic neurons and neuroendocrine cells. They are characterized by the capacity to produce biologically active amines or peptide hormones (1).

Correspondence to: Dr Liqiu Sun, Department of Oncology, Tumor Hospital of Mudanjiang, 333 Kangjia Street, Mudanjiang, Heilongjiang 157009, P.R. China
E-mail: liqius123@outlook.com

Key words: pulmonary carcinoid, neuroendocrine tumor, mediastinal lymph node metastasis, radiotherapy, lobaplatin, sintilimab, anlotinib, combined systemic therapy

These tumors can arise in various organs throughout the body, including the stomach, intestines and lungs. Pulmonary NETs account for ~30% of all NETs (2), whereas pulmonary carcinoid (PC) tumors comprise only about 1-5% of all lung NET cases (3). PCs are relatively well-differentiated neoplasms compared with other pulmonary NETs, yet currently there is no standardized therapeutic strategy for their management.

Surgery remains the preferred treatment for localized PC disease and offers the best long-term outcomes. However, the role of adjuvant chemotherapy is not well-defined, particularly in atypical carcinoids (ACs). For patients with inoperable or advanced stage PCs, treatment options are limited and evidence guiding systemic therapy is sparse. In the present study, a case of an advanced PC with progressive mediastinal lymph node metastases is presented, that responded to a combination of platinum-based chemotherapy, immune checkpoint blockade and antiangiogenic therapy. The clinical relevance of this case was discussed in light of current treatment strategies.

Case presentation

The patient was a 56-year-old male. Upon admission, the patient was fully conscious, with stable vital signs: Body temperature, 36.5°C; heart rate, 106 beats per min; respiratory rate, 18 breaths per min; and blood pressure, 116/79 mmHg. Physical examination revealed coarse breath sounds over the left lung field without adventitious sounds. No cyanosis or superficial lymphadenopathy was observed. Cardiac and abdominal exams were unremarkable. There was no peripheral edema. Performance status was ECOG 1 and the numerical rating scale score for pain was 0 (Table I). The time-structured flow of diagnosis and treatment was shown in Fig. 1.

Laboratory investigations revealed leukopenia with a white blood cell count of $3.36 \times 10^9/l$ and mild thrombocytopenia (platelets $104 \times 10^9/l$). Biochemical markers, including hepatic and renal function, coagulation profile and cardiac enzymes, were within normal limits. Thyroid function was unremarkable. Tumor marker screening, including carcinoembryonic antigen, carbohydrate antigen 125, neuron-specific enolase, progastrin-releasing peptide and squamous cell carcinoma antigen-yielded normal results. Imaging studies of the brain, abdomen and cervical lymph nodes revealed no abnormalities. Initial CT in April 2023 identified an occupying lesion in the left upper lobe (Fig. 2A).

Bronchoscopic biopsy performed on April 13, 2023 at Harbin Medical University Cancer Hospital, from the anterior segment of the left upper lobe demonstrated histological features consistent with PC. Immunohistochemical analysis confirmed the diagnosis of a well-differentiated NET. The tumor cells showed positive staining for neuroendocrine markers (Synaptophysin⁺, Chromogranin A⁺ and CD56⁺) and the Ki-67 proliferation index was low at 1%, consistent with a typical carcinoid (TC).

Following the initial diagnosis in April 2023, the patient underwent two cycles of platinum-based chemotherapy with carboplatin at Harbin Medical University Cancer Hospital. Despite radiologic evidence of shrinkage in both the pulmonary lesion and mediastinal lymphadenopathy, the patient chose to discontinue systemic therapy and instead pursue treatment with traditional Chinese medicine. By July 2023, a soft tissue density was noted within the left upper lobe bronchus, measuring ~0.8x1.6 cm, accompanied by mediastinal lymphadenopathy in station 4R measuring 3.3x2.0 cm. A follow-up scan in September 2023 revealed mild enlargement of both the intrabronchial mass (1.1x1.7 cm) and the 4R lymph node (2.5x3.2 cm). Upon subsequent evaluation at our institution, progressive enlargement of the bronchial lesion and mediastinal lymph nodes was observed. As a result, thoracic intensity-modulated radiotherapy (IMRT) was initiated and delivered between October 11 and November 23, 2023, with a total dose of 60 Gy administered in 30 fractions over 47 days. Post-radiotherapy CT imaging demonstrated a reduction in the size of both the primary tumor (from 1.1x1.7 to 1.2x0.7 cm) and the 4R lymph node (from 2.5x3.2 to 2.1x1.4 cm), indicating a partial response. After thoracic radiotherapy was completed in November 2023, the bronchial lesion had reduced to 1.2x0.7 cm, and the lymph node regressed to 2.1x1.4 cm (Fig. 2B). Detailed RECIST 1.1 measurements and percent changes are summarized in Table II.

However, on August 28, 2024, ~9 months after completing radiotherapy, follow-up chest CT revealed signs of disease progression, including re-enlargement of mediastinal lymph nodes (up to 3.7x2.5 cm) and soft tissue thickening at the bilateral upper lobe hilum (Fig. 2C). Given the radiographic findings and disease course, systemic therapy was initiated. Starting on August 30, 2024, the patient received a combination regimen consisting of lobaplatin (50 mg intravenously), sintilimab (200 mg intravenously) and anlotinib (12 mg orally once daily, administered on days 1-14 of a 21-day cycle). A second cycle was completed on September 24, 2024. Imaging assessment after one and two cycles demonstrated significant shrinkage of the mediastinal lymph nodes, with the 4R node reduced to 2.1x2.5 cm with primary bronchial lesion remained stable and no new metastatic lesions, qualifying stable disease (SD) according to RECIST 1.1 criteria (Fig. 2D and E). The most recent follow-up CT scan performed at a local hospital on November 11, 2025, revealed further mediastinal lymph nodes shrinkage with a maximum short-axis diameter of 1.2 cm and no evidence of new metastatic lesions (Fig. 2F). As of this writing, the patient remains alive and under regular follow-up. The patient's overall survival (OS) was at least 31 months from the initial diagnosis in April 2023 to the last follow-up in November 2025.

Table I. Clinical and laboratory findings.

Parameter category	Findings
Vital signs and physical examination	
Consciousness	Fully conscious
Body temperature	36.5°C
Heart rate	106 beats per min
Respiratory rate	18 breaths per min
Blood pressure	116/79 mmHg
Lung auscultation	Coarse breath sounds over left lung field; no adventitious sounds
Other findings	
	No cyanosis, superficial lymphadenopathy, peripheral edema, or cardiac/abdominal abnormalities
Performance status	ECOG 1
Pain score (numerical rating scale)	0
Laboratory investigations	
Complete blood count	
White blood cell count	3.36x10 ⁹ /l (Leukopenia)
Platelet count	104x10 ⁹ /l (Mild thrombocytopenia)
Biochemical and functional markers	
Hepatic/renal function, coagulation profile, cardiac enzymes	Within normal limits
Thyroid function	Unremarkable
Tumor Markers	
Carcinoembryonic antigen	Normal
Carbohydrate antigen 125	Normal
Neuron-specific enolase	Normal
Progastrin-releasing peptide	Normal
Squamous cell carcinoma antigen	Normal

Discussion

A multimodal systemic regimen was implemented to address the resistant disease of this patient. The patient was started on a triple combination of chemotherapy, immunotherapy and anti-angiogenic therapy. After two cycles of this combined therapy, our patient achieved a marked regression of mediastinal lymph node metastases, with the primary pulmonary lesion remaining stable. Equally notable, he tolerated the treatment well with minimal adverse effects. This favorable response suggests that integrating immunotherapy and antiangiogenic therapy with chemotherapy can be effective and feasible in an advanced PC case, even after multiple prior treatments. To the best of our knowledge, reports of such a chemo-immunotherapy plus antiangiogenic combination in PC are extremely limited,

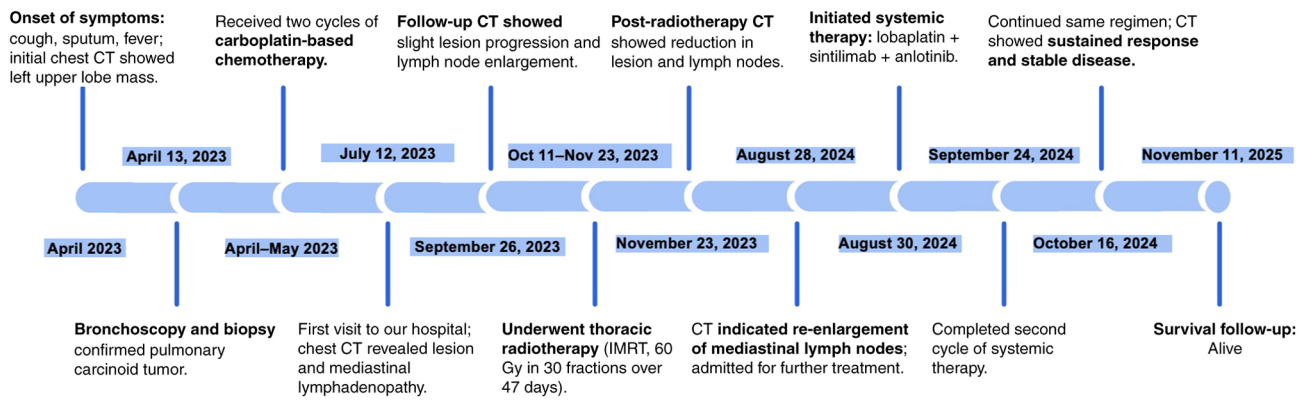


Figure 1. Time-structured flow of diagnosis and treatment.

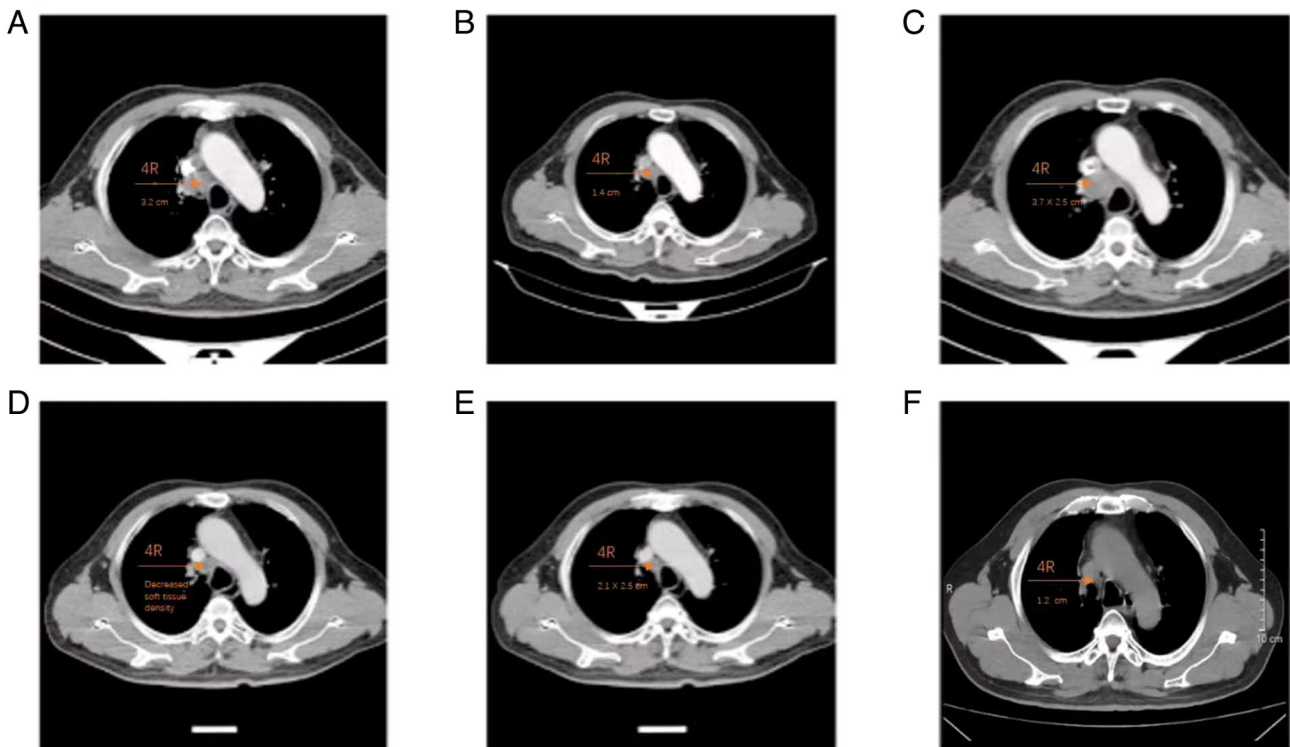


Figure 2. Axial CT images at representative treatment stages in a patient with pulmonary carcinoid. (A) Initial diagnosis (April 2023): Contrast-enhanced chest CT showing a left upper lobe endobronchial lesion (not visible at this level) and an enlarged mediastinal lymph node at station 4R (not annotated), with a maximum diameter of ~3.2 cm. (B) Post-radiotherapy (November 2023): Significant reduction in size of the 4R lymph node, with the maximum diameter decreasing to 1.4 cm. (C) Before systemic therapy (August 2024): Re-enlargement of the 4R lymph node to 3.7x2.5 cm, indicating disease progression. (D) After one cycle of systemic therapy (September 2024): Decreased soft tissue density in the 4R lymph node region. (E) After two cycles of therapy: Continued regression of the lesion, meeting the criteria for stable disease. (F) Surveillance (November, 2025): No newly emerging lesions and further lesion size shrinkage, indicating partial response. CT, computed tomography.

making the present case a valuable addition to the increasing clinical experience. It illustrates that, in selected patients, an individualized regimen addressing multiple tumor pathways may yield disease control where conventional modalities alone have fallen short.

NETs arise from the malignant transformation of neuroendocrine cells, with the gastrointestinal tract and lungs being the most commonly affected organs (4). Pulmonary NETs originate from bronchial Kulchitsky cells and may manifest either as isolated or clustered lesions. These cells are capable of producing various neuroendocrine mediators such as serotonin

and adrenocorticotropic hormone. In hypoxic conditions, they may also contribute to epithelial proliferation and tissue repair. Aberrant proliferation of these cells can ultimately lead to tumorigenesis.

PCs are histologically classified into TC, atypical carcinoid (AC), large cell neuroendocrine carcinoma (LCNEC) and small cell lung carcinoma (SCLC), with progressively increasing malignancy and correspondingly decreasing prognosis. TC and AC fall under the category of well-differentiated NETs, with AC exhibiting a higher mitotic rate and more aggressive behavior, highlighting a key epidemiological distinction. Unlike

Table II. Summary of treatment responses according to RECIST 1.1 criteria.

Date	Target lesion	Size (cm) (longest diameter/ long axis)	Sum of longest diameters (SLD, cm)	Change from baseline (%)	Best overall response
Baseline-Apr 2023	Left upper lobe bronchus 4R Lymph node	2.5 3.2	5.7	N/A	N/A
Jul 2023 (after 2 cycles of platinum-based chemotherapy with carboplatin)	Left upper lobe bronchus 4R lymph node	1.6 2.0	3.6	-36.8%	Partial response
Sep 2023	Left upper lobe bronchus 4R lymph node	1.7 2.5	4.2	-26.3%	SD
Nov 2023 (after IMRT)	Left upper lobe bronchus 4R lymph node	1.2 1.4	2.6	-54.4%	Partial response
Aug 2024	Left upper lobe bronchus 4R lymph node	2.1 2.5	4.6	-19.3%	Disease progression ^a
Oct 2024	Left upper lobe bronchus 4R lymph node	1.7 2.1	3.8	-17.4%	SD ^b
Nov 2025	Left upper lobe bronchus	1.5	2.7	-41.3%	Partial response

^aCompared with the nadir sum of 2.6 cm in November 2023 after IMRT, a 76.9% increase was observed, meeting the RECIST 1.1 criteria for disease progression (requiring both a $\geq 20\%$ increase and an absolute increase of ≥ 5 mm from the nadir). ^bEvaluated against a new baseline (4.6 cm) established after progression. A 17.4% reduction from this baseline qualifies as SD per RECIST 1.1. IMRT, intensity-modulated radiotherapy. SD, stable disease.

most pulmonary malignancies, PCs are not strongly linked to tobacco exposure (5). Rather, their development is considered to involve intrinsic dysregulation of neuroendocrine cell proliferation or genetic predisposition. Most patients present between the ages of 40 and 50 years, often with non-specific respiratory complaints such as cough, hemoptysis, or dyspnea. A minority may present with paraneoplastic symptoms such as carcinoid syndrome. Imaging with CT or MRI is essential for identifying lesion size and distribution, but definitive diagnosis relies on histopathological confirmation. This observation is consistent with the clinical profile of our patient, who had no history of smoking, no familial predisposition, and presented with non-specific respiratory symptoms, features broadly in line with the typical presentation described in prior studies.

Surgical resection remains the cornerstone of curative treatment for patients with resectable PCs. According to the National Comprehensive Cancer Network guidelines, adjuvant chemotherapy consisting of platinum-based agents and etoposide is recommended for patients with stage II-III AC, whereas such treatment is not advised for TC (6). Similarly, the European NET Society guidelines recommend adjuvant chemotherapy only for AC cases in which lymph node metastasis is present (2). For unresectable or progressive cases, the benefit of chemotherapy has been documented (7), although standardized regimens remain undefined. In addition to surgery and chemotherapy, radiotherapy may also confer benefit. A retrospective analysis of 59 patients demonstrated excellent outcomes, particularly among those who achieved complete remission post-treatment, with 5- and 10-year survival rates of 80% and a disease-free survival (DFS) of

98% (3). All but four patients underwent surgical resection, which included 54 lung-sparing resections and 1 pneumonectomy. Among the 55 surgically treated patients, five received concurrent chemoradiotherapy: Four with AC and one with TC. An additional three patients with AC were treated with adjuvant radiotherapy, palliative radiotherapy, or palliative chemotherapy alone. The Kaplan-Meier analysis showed that both 5-year and 10-year OS rates were 80%. Among the 88% of patients who achieved complete remission, the DFS rate was 98%. These results reinforce that surgical resection is the primary and often sufficient treatment for most cases of TC, offering high rates of OS and DFS. For patients with AC with adverse pathological features, adjuvant chemotherapy or radiotherapy may be considered. It is generally accepted that for AC cases with positive surgical margins or those achieving R0 resection but demonstrating nodal metastases on pathology, postoperative radiotherapy may be beneficial, although its role in PC remains unclear (5). Thus, this case aligns with current literature in terms of surgical and radiotherapeutic approaches, while providing additional insights into systemic treatment in the post-progression setting. In contrast to existing studies that emphasize the predominance of surgery or traditional cytotoxic therapy, this patient underwent chemotherapy and later radiotherapy as early interventions, which diverges from guideline-recommended practice. This deviation was driven by institutional availability and patient choice, but it also provides a useful reference for the spectrum of real-world treatment sequences.

The selection of this triple regimen, integrating a PD-1 inhibitor (sintilimab), an antiangiogenic agent (anlotinib)

and platinum chemotherapy (lobaplatin), was grounded in a synergistic biological rationale. Antiangiogenic therapy is postulated to remodel the tumor microenvironment through vascular normalization, reducing immunosuppression and enhancing T-cell infiltration, thereby potentially sensitizing tumors to PD-1 blockade (8). Meanwhile, platinum chemotherapy contributes not only direct cytotoxicity but also immunogenic cell death, which exposes tumor antigens and primes antitumor immunity, creating a more favorable context for immune checkpoint inhibition (9). Together, these three modalities act synergistically by targeting cellular proliferation through chemotherapy, inhibiting angiogenesis via antiangiogenic agents, and counteracting immune evasion with immunotherapy. This multitarget strategy is designed to yield additive or synergistic effects, particularly in overcoming acquired resistance after prior therapies. This rationale is further supported by emerging clinical and molecular evidence specific to PCs. Studies exploring immune checkpoint pathways in PCs remain limited but suggest a potentially actionable role for immunotherapy. In a previous study, PD-1 and PD-L1 expression were evaluated in tissue samples from 20 patients with PC, revealing positivity rates of 40 and 45%, respectively (10). These findings indicate that PD-1/PD-L1 overexpression may be involved in the pathogenesis of PCs, providing a theoretical basis for the clinical application of immune checkpoint blockade. In parallel, a phase III prospective randomized controlled trial compared octreotide combined with bevacizumab vs. octreotide plus interferon- α 2b in patients with advanced carcinoid tumors. The median progression-free survival (mPFS) was 15.4 months in the bevacizumab group, compared with 10.6 months in the interferon arm, supporting the therapeutic role of anti-VEGF agents in this setting (11). In the present case, the patient underwent multiple sequential treatments throughout the course of disease evolution. Initial management involved two cycles of carboplatin-based chemotherapy, followed by traditional Chinese medicine. Upon radiologic progression, thoracic radiotherapy was administered and achieved transient disease control. However, ~9 months after completion of radiotherapy, mediastinal lymph node enlargement suggested renewed disease activity, prompting reevaluation. This therapeutic decision was informed not only by the patient's specific clinical trajectory but also by emerging evidence supporting similar combinations across neuroendocrine neoplasms. In SCLC, adding anti-PD-L1 agents (atezolizumab/durvalumab) to first-line platinum-etoposide chemotherapy improves survival, establishing a precedent for chemo-immunotherapy synergy in aggressive neuroendocrine carcinomas (12,13). In well-differentiated gastro-entero-pancreatic NETs, a basket study of atezolizumab plus bevacizumab achieved encouraging disease control, highlighting the potential synergy between immunotherapy and antiangiogenics (14). A phase II study of nivolumab and temozolomide reported notable activity in the lung primary subgroup. Among 11 patients with lung NETs, the objective response rate (ORR) was 64% (7 of 11 patients), which was significantly higher compared with patients with NETs of other primary origins ($P=0.02$) (15). Similarly, results for pembrolizumab-based therapy in LCNEC have also been reported. In treatment-naïve patients, first-line pembrolizumab plus chemotherapy yielded an ORR of 70% (7/10) and a

disease control rate of 90% (9/10), with a mPFS of 5.5 months and a median OS of 13.0 months. Among pretreated patients, mPFS was 3.8 months and mOS was not reached (16). This compelling data suggests that pulmonary NETs may represent a particularly immuno-responsive subset within the broader NET spectrum. Considering the clinical trajectory and prior treatments, a triple systemic regimen consisting of lobaplatin, sintilimab and anlotinib was initiated. This approach incorporated a platinum agent for cytotoxic backbone, a PD-1 inhibitor for immunomodulation, and a VEGFR-targeted agent for antiangiogenic activity.

After two cycles of this combination therapy, repeat imaging demonstrated a marked reduction in mediastinal lymph node size, with the pulmonary lesion remaining radiographically stable. The patient tolerated the regimen without notable toxicity, suggesting that such a combination may offer a safe and effective option in patients with advanced PCs. While the present findings are consistent with limited existing literature supporting immune and targeted interventions in carcinoid tumors, this case provides an example of integrating these therapies in a rational, stepwise fashion following local treatment failure. Recent case reports (15,17-22) and clinical studies in PC are summarized in Table SI. Collectively, these data suggest that long-term survival in these cases appears to be founded on three cornerstones: Sustained surveillance for late recurrence, multidisciplinary team collaboration and treatment strategies personalized to both tumor biology and patient factors.

A limitation of the present case report is that the original IHC images were unavailable, as the initial pathological diagnosis was established at an external institution and the images were not retained in a retrievable digital format. No repeat biopsy was clinically indicated. Nonetheless, the diagnosis was clearly documented and consistent with the clinical course and treatment response.

In summary, the present case report contributes to the evolving therapeutic landscape for advanced PC, where established options (for example, somatostatin analogs, everolimus and chemotherapy) frequently provide limited benefit, and other strategies such as peptide receptor radionuclide therapy await robust validation in this setting. The present case report demonstrates the potential clinical benefit of a tailored multimodal approach in advanced PC, incorporating chemotherapy, immunotherapy and antiangiogenic therapy in sequence, adding to the increasing evidence base for innovative multimodal approaches in this uncommon malignancy. For practitioners, an important implication is that when standard options, notably surgery or established medical therapies, are unfeasible or no longer effective, exploring novel combinations in the context of clinical evidence can be considered on a case-by-case basis. Our experience aligns with the limited literature suggesting that immune checkpoint inhibitors and anti-VEGF agents may have activity in well-differentiated NETs, and it provides a practical example of how these therapies can be combined safely. However, given the rarity of PC tumors, high-level evidence to guide such interventions is lacking. This case underscores the need for further research and collaborative studies to define optimal management strategies. Prospective trials evaluating the efficacy of immunotherapy and targeted agents (such as VEGF inhibitors or mTOR pathway inhibitors) in PCs would help clarify

their roles and inform future guidelines. Until more data are available, management of advanced PC will continue to rely on clinical judgment and extrapolation from related NET experience. The present case adds to the increasing evidence base and highlights that innovative, multimodal treatment approaches may offer hope for improved outcomes in this uncommon malignancy.

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.

Authors' contributions

LS and ML conceptualized the study and wrote the original manuscript. LS collected clinical data and conducted literature search. ML analyzed the data and reviewed the manuscript. All authors read and approved the final version of the manuscript and confirm the authenticity of all the raw data.

Ethics approval and consent to participate

The ethics approval was waived by the Ethics Committee of Tumor Hospital of Mudanjiang. The study was conducted in accordance with the Declaration of Helsinki (1975). The patient has provided written informed consent.

Patient consent for publication

Patient consent was obtained for publication of images and associated data.

Competing interests

The authors declare that they have no competing interests.

References

1. Araujo-Castro M, Pascual-Corrales E, Molina-Cerrillo J, Moreno Mata N and Alonso-Gordoa T: Bronchial carcinoids: From molecular background to treatment approach. *Cancers (Basel)* 14: 520, 2022.
2. Caplin ME, Baudin E, Ferolla P, Filosso P, Garcia-Yuste M, Lim E, Oberg K, Pelosi G, Perren A, Rossi RE, *et al*: Pulmonary neuroendocrine (carcinoid) tumors: European Neuroendocrine Tumor Society expert consensus and recommendations for best practice for typical and atypical pulmonary carcinoids. *Ann Oncol* 26: 1604-1620, 2015.
3. Herde RF, Kokeny KE, Reddy CB, Akerley WL, Hu N, Boltax JP and Hitchcock YJ: Primary pulmonary carcinoid tumor: A Long-term single institution experience. *Am J Clin Oncol* 41: 24-29, 2018.
4. Kasajima A and Klöppel G: Diagnostic issues in neuroendocrine neoplasms of the lung. *Pathologie (Heidelberg)* 45: 51-55, 2024.
5. Mackley HB and Videtic GM: Primary carcinoid tumors of the lung: A role for radiotherapy. *Oncology (Williston Park)* 20: 1537-1545, 1549, 2006.
6. Shah MH, Goldner WS, Benson AB, Bergsland E, Blazzkowsky LS, Brock P, Chan J, Das S, Dickson PV, Fanta P, *et al*: Neuroendocrine and adrenal tumors, version 2.2021, NCCN clinical practice guidelines in oncology. *J Natl Compr Canc Netw* 19: 839-868, 2021.
7. Marquez-Medina D and Papat S: Systemic therapy for pulmonary carcinoids. *Lung Cancer* 90: 139-147, 2015.
8. Zhou Y, Liu Z, Yu A, Zhao G and Chen B: Immune checkpoint inhibitor combined with antiangiogenic agent synergistically improving the treatment efficacy for solid tumors. *Immunotargets Ther* 13: 813-829, 2024.
9. Chang X, Bian M, Liu L, Yang J, Yang Z, Wang Z, Lu Y and Liu W: Induction of immunogenic cell death by novel platinum-based anticancer agents. *Pharmacol Res* 187: 106556, 2023.
10. Li M, Xu S, Fan H, Zhang H, Li Y, Li Y, Liu M, Liu H and Chen J: Expression and clinical significance of PD-1 and PD-L1 in pulmonary carcinoids. *Chin J Lung Cancer* 19: 847-853, 2016 (In Chinese).
11. Yao JC, Guthrie KA, Moran C, Strosberg JR, Kulke MH, Chan JA, LoConte N, McWilliams RR, Wolin EM, Mattar B, *et al*: Phase III prospective randomized comparison trial of depot octreotide plus interferon Alfa-2b versus depot octreotide plus bevacizumab in patients with advanced carcinoid tumors: SWOG S0518. *J Clin Oncol* 35: 1695-1703, 2017.
12. Horn L, Mansfield AS, Szczesna A, Havel L, Krzakowski M, Hochmair MJ, Huemer F, Losonczy G, Johnson ML, Nishio M, *et al*: First-line atezolizumab plus chemotherapy in Extensive-stage small-cell lung cancer. *N Engl J Med* 379: 2220-2229, 2018.
13. Goldman JW, Dvorkin M, Chen Y, Reinmuth N, Hotta K, Trukhin D, Statsenko G, Hochmair MJ, Özgüroğlu M, Ji JH, *et al*: Durvalumab, with or without tremelimumab, plus platinum-etoposide versus platinum-etoposide alone in first-line treatment of extensive-stage small-cell lung cancer (CASPIAN): Updated results from a randomised, controlled, open-label, phase 3 trial. *Lancet Oncol* 22: 51-65, 2021.
14. Halperin DM, Liu S, Dasari A, Fogelman D, Bhosale P, Mahvash A, Estrella JS, Rubin L, Morani AC, Knafel M, *et al*: Assessment of clinical response following atezolizumab and bevacizumab treatment in patients with neuroendocrine tumors: A nonrandomized clinical trial. *JAMA Oncol* 8: 904-909, 2022.
15. Owen DH, Benner B, Wei L, Sukrithan V, Goyal A, Zhou Y, Pilcher C, Suffren SA, Christenson G, Curtis N, *et al*: A Phase II clinical trial of nivolumab and temozolomide for neuroendocrine neoplasms. *Clin Cancer Res* 29: 731-741, 2023.
16. Song L, Zhou F, Xu T, Zeng L, Xia Q, Wang Z, Deng L, Li Y, Qin H, Yan H, *et al*: Clinical activity of pembrolizumab with or without chemotherapy in advanced pulmonary large-cell and large-cell neuroendocrine carcinomas: A multicenter retrospective cohort study. *BMC Cancer* 23: 443-452, 2023.
17. Trpezanovski J, Karpelowsky J, Hesketh E and London K: Pediatric theranostics in a 13-Year-Old female with bronchial carcinoid. *World J Nucl Med* 24: 270-277, 2025.
18. Cooper AJ, Rekhman N, Baine MK, Thomas MC, Lynch AC and Gentzler RD: First report of response to tarlatamab in a patient with DLL3-Positive pulmonary carcinoid: Case report. *JTO Clin Res Rep* 5: 100750, 2024.
19. Hu W, Zhao J, Wang G, Wang Q, Deng M, Shen J, Hofman P, Urbanska EM, Santoni-Rugiu E, Christopoulos P, *et al*: A rare case report of a primary lung cancer comprising adenocarcinoma and atypical carcinoid tumor, with the carcinoid component harboring EML4-ALK rearrangement. *Transl Lung Cancer Res* 13: 1150-1162, 2024.
20. Çağan P, Safaei S, Kimiaei A, Yapıcıer Ö and Kutlu CA: Contralateral metachronous pulmonary carcinoid tumor 7 years after lobectomy. *Indian J Thorac Cardiovasc Surg* 41: 591-595, 2025.
21. Aldrete K and Shahla L: Severe ectopic adrenocorticotrophic hormone syndrome due to pulmonary carcinoid tumor: A case report and literature review. *AACE Clin Case Rep* 10: 232-235, 2024.
22. Bostan H, Duger H, Akhanli P, Calapkulu M, Turkmenoglu TT, Erdol AK, Duru SA, Sencar ME, Kizilgul M, Ucan B, *et al*: Cushing's syndrome due to adrenocorticotrophic hormone-secreting metastatic neuroendocrine tumor of unknown primary origin: A case report and literature review. *Hormones (Athens)* 21: 147-154, 2022.