Successful treatment with tepotinib followed by pembrolizumab in pulmonary pleomorphic carcinoma harbouring a *MET* exon 14 skipping mutation: A case report

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Abstract. Pulmonary pleomorphic carcinoma (PPC) is a subtype of sarcomatoid carcinoma that accounts for 0.1-0.3% of all lung cancer cases. PPC is typically resistant to chemotherapy and radiotherapy, and no standard treatment protocols are currently established. Moreover, to the best of our knowledge, the effectiveness of the MET inhibitor tepotinib in the treatment of PPC has not yet been reported. Herein, the present study describes the case of a 75-year-old woman with PPC harbouring a *MET* exon 14 skipping mutation who was successfully treated with tepotinib as first-line systemic therapy, followed by pembrolizumab as subsequent immune checkpoint inhibitor therapy. Although the tumour relapsed 4 months postoperatively, treatment with tepotinib resulted in a favourable response and subsequent pembrolizumab therapy achieved a durable response. This case suggests that patients with sarcomatoid carcinoma, which is generally associated with a poor prognosis, may experience improved outcomes with the use of molecular targeted therapies and immune checkpoint inhibitors.

Introduction

Pulmonary sarcomatoid carcinoma is a rare tumour that accounts for approximately 0.5% of all lung tumours (1). Pulmonary pleomorphic carcinoma (PPC), the most common subtype of pulmonary sarcomatoid carcinoma, is a poorly differentiated non-small cell lung cancer (NSCLC) that accounts for 0.1-0.3% of all lung tumours (2). Pleomorphic carcinoma is diagnosed when tumours consist exclusively of spindle and/or giant cells or when spindle and/or giant cells account for ≥10% of the total tumour (3). Therefore, to confirm the pathological diagnosis of PPC, the entire tumour needs to be evaluated via surgical resection, and a biopsy specimen is insufficient for a definitive diagnosis. This kind of tumour is highly malignant, rapidly progressing, and refractory to various therapies (4). The outcome of surgical resection is poor, with 57.1% of pN0 cases reported to have vascular invasion (5), and distant metastases is commonly detected early after surgery. The median survival time for patients with PPC is 10-12 months, with a 5-year survival rate of approximately 10%, and the response to chemotherapy is poorer than that of NSCLC (6). However, recent advances in molecular targeted therapy and immune checkpoint inhibitors (ICIs) have led to significant progress in therapeutic strategies.

MET exon 14 skipping mutations are found in approximately 3% of NSCLC cases (7). Unlike other driver gene mutations/translocations, including those in *EGFR*, *ALK*, and *ROS1*, the *MET* exon 14 skipping mutation has also been detected in older patients and smokers (7). It is characterised by the fact that it is not only found in pulmonary adenocarcinomas but also in approximately 5% of pulmonary adenosquamous carcinomas and 10-32% of pulmonary sarcomatoid carcinomas (6,8,9). Although tepotinib and capmatinib have been approved by the Food and Drug Administration (FDA) for the treatment of unresectable advanced or recurrent NSCLC with *MET* exon 14 skipping mutations (7,10), to our knowledge, no previous study has reported on the use of MET inhibitors in patients with pulmonary sarcomatoid carcinoma. Herein, we report the case of a patient with PPC with a *MET* exon 14 skipping mutation that was successfully treated with tepotinib as the primary drug and with pembrolizumab as the subsequent ICI therapy.

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Case report

A 75-year-old woman with no history of smoking underwent left upper lobectomy, pulmonary angioplasty, and lymph node dissection (ND2a-2) for an approximately 4-cm mass in the left upper lobe with pulmonary artery invasion (Fig. 1A and F) in May 2022 at Niigata University Medical and Dental Hospital (Niigata, Japan). The patient had comorbidities such as hypertension, chronic kidney disease, and atrial fibrillation.

Pathological examination of the resected specimen showed the presence of PPC consisting of spindle cell carcinoma (50%), giant cell carcinoma (30%), and adenocarcinoma components (20%) (Fig. 2A-C). Immunohistochemical analysis showed that the spindle cells, giant cells, and adenocarcinoma components (Fig. 2D-F) were weakly positive, weakly positive, and positive, respectively, for thyroid transcription factor-1 (TTF-1) while they were positive, positive, and negative, respectively, for vimentin (Fig. 2G-I). For haematoxylin and eosin staining, surgically resected specimens were fixed in 10% neutral buffered formalin at room temperature for 48 h. Representative tissue samples were selected, embedded in paraffin and sectioned at a thickness of 4 μm. The sections were then deparaffinized in xylene, rehydrated through a graded series of ethanol, and stained with haematoxylin for 5 min and eosin for 2 min at room temperature. Immunohistochemical staining was performed on the 4-μm sections prepared from formalin-fixed, paraffin-embedded tissue. Briefly, antigen retrieval was carried out using citrate buffer (pH 6.0) and endogenous peroxidase blocking was performed with 3% hydrogen peroxide (cat. no. 081-04215; FUJIFILM Wako Pure Chemical Corporation) for 5 min at room temperature. The primary antibodies used were TTF-1 (1:200; for 30 min at room temperature; clone SPT24; cat. no. NCL-L-TTF-1; Leica Biosystems) and vimentin (1:1; for 30 min at room temperature; clone V9; cat. no. 718511; Nichirei Biosciences, Inc.). Histofine® Simple Stain™ MAX PO (MULTI) (1:1; for 30 min at room temperature; cat. no. 424151; Nichirei Biosciences, Inc.) was used as a secondary antibody. Immunoreactivity was visualized using 3,3'-diaminobenzidine, and nuclei were counterstained with haematoxylin. All slides were examined under a light microscope.

According to the 8th TNM classification, the pathological stage was T2aN0M0 stage IB.

Three months postoperatively, the patient experienced left shoulder pain, and visited the hospital, with an Eastern Cooperative Oncology Group performance status of 2 owing to the pain and state of recovery postoperatively. Computed tomography (CT) showed a 26-mm nodule on the left diaphragm, which was considered to indicate pleural dissemination, bilateral adrenal metastases (right: 11 mm, left: 21 mm), and multiple bone metastases, including to the fourth cervical vertebra and left ribs (Fig. 1B and G). The CEA level was 11.9 ng/ml (normal range, ≤5.0 ng/ml), SLX level was 110 U/ml (normal range, ≤38 U/ml), and CYFRA level was 12.5 ng/ml (normal range, ≤3.5 ng/ml). Palliative radiotherapy was administered for bone metastases, with 30 Gy in 10 fractions to the fourth cervical vertebra and 20 Gy in 5 fractions to the left eighth rib.

Analysis of the resected tumour using the Oncomine™ Dx Target Test Multi CDx System with next-generation sequencing (NGS) revealed a *MET* exon 14 skipping mutation, with a PD-L1 expression level of >75% by PD-L1 IHC 22C3 pharm Dx Dako. As both analyses were performed as outsourced tests, the raw data were not available. In September 2022, four months after surgery, the patient was hospitalized and received tepotinib (500 mg/day) as primary drug therapy after the completion of radiotherapy. On day 10 of tepotinib treatment, the dose was reduced to 250 mg/day owing to a grade 2 creatinine increase, according to the Common Terminology Criteria for Adverse Events, version 5.0. On day 17, grade 2 liver injury occurred and tepotinib was discontinued. One week after withdrawal, both hepatic and renal functions recovered and tepotinib was restarted at 125 mg/day. Subsequently, the patient experienced no apparent adverse events.

Two months after initiating tepotinib, CT imaging showed a reduction in pleural dissemination on the left diaphragm (5 mm) and in bilateral adrenal metastases (right: 5 mm, left: 12 mm) (Fig. 1C and H), and the response was assessed as a partial response (PR).

However, 5 months after the initiation of tepotinib, a CT scan showed that although the reduction in the extent of the bilateral adrenal metastases was maintained, the pleural dissemination had progressed and the amount of left pleural effusion had increased (pleural dissemination: 93 mm, left adrenal metastasis: 13 mm, Fig. 1D and I), leading to the diagnosis of progressive disease (PD).

Consequently, the patient underwent pleural drainage and pleurodesis using talc. At the beginning of the secondary treatment, the patient was found to have inflammation in the left lower lobe, which was possibly caused by pleurodesis. Owing to concerns regarding the progression of pulmonary inflammation, ICIs were avoided. Therefore, nab-paclitaxel was administered as the second-line therapy. After the first course of nab-paclitaxel, the size of the pleural dissemination increased, accompanied by an increase in chest pain, leading to the assessment of PD. At that time, the ground-glass opacity of the left lower lobe had disappeared; therefore, pembrolizumab was started as third-line therapy. Thereafter, the left pleural dissemination shrunk and the chest pain disappeared; the response was assessed as PR (pleural dissemination: 25 mm, left adrenal metastasis: 13 mm, Fig. 1E and J). The patient had no apparent adverse events. Nine months after the initiation of pembrolizumab, the patient required palliative radiotherapy for enlargement of a left iliac metastasis. No significant disease progression was observed at other sites, and pembrolizumab treatment was successfully continued for a total of 19 months. Subsequently, the patient developed infection and heart failure. Owing to a decline in performance status, anticancer therapy was discontinued, and the patient died of primary disease 36 months after surgery at the receiving hospital.

Discussion

In the present case, PPC was pathologically diagnosed using a resected specimen and the patient experienced recurrence soon after surgery. NGS revealed a *MET* exon 14 skipping mutation, and PD-L1 was highly expressed. The optimal first-line therapy for *MET* exon 14 skipping-positive NSCLC remains

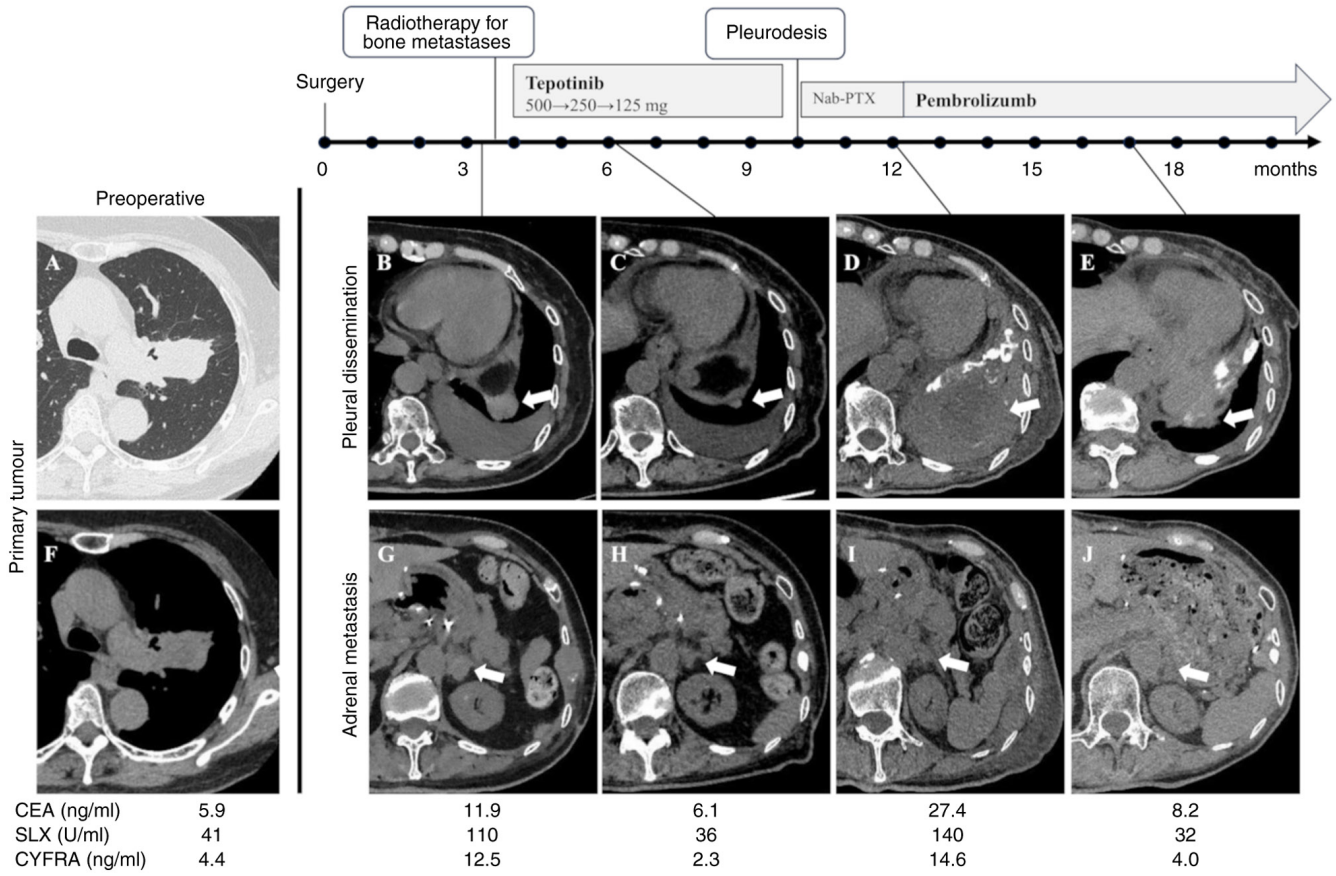

Figure 1. Computed tomography images over the clinical course. (A) Primary tumour of the left upper lobe shown on lung window. (B) Pleural dissemination of the left diaphragm at the time of postoperative recurrence, 26 mm in diameter. (C) Pleural dissemination of the left diaphragm decreased to 5 mm in diameter 2 months after tepotinib initiation. (D) Pleural dissemination of the left diaphragm had grown to 93 mm in diameter prior to the initiation of pembrolizumab. (E) Pleural dissemination of the left diaphragm decreased to 25 mm in diameter 5 months after initiation of pembrolizumab. (F) Primary tumour of the left upper lobe shown on mediastinal window. (G) Left adrenal metastasis at the time of postoperative recurrence, 21 mm in diameter. (H) Left adrenal metastasis decreased to 12 mm in diameter 2 months after initiation of tepotinib. (I) Size of left adrenal metastasis remained largely unchanged, measuring 13 mm before initiation of pembrolizumab. (J) Size of left adrenal metastasis remained unchanged, measuring 13 mm 5 months after initiation of pembrolizumab. Nab-PTX, nanoparticle albumin-bound paclitaxel; CEA, carcinoembryonic antigen; SLX, Sialyl Lewis X antigen; CYFRA, cytokeratin 19 fragment.

undetermined, with differing recommendations across guidelines regarding whether to use a MET inhibitor or standard systemic therapy including ICI (11-13). In this case, given the patient's symptomatic condition and poor performance, a MET inhibitor was prioritized considering the expectation of a favourable response characteristic of molecularly targeted drugs. Tepotinib as a primary therapy achieved a good short-term response. Subsequently, pembrolizumab treatment resulted in a favourable long-term response.

Although tepotinib and capmatinib have been approved by the FDA and are standard therapies for NSCLC with *MET* exon 14 skipping mutations, little is known about their efficacy in sarcomatoid carcinoma with *MET* exon 14 skipping mutations. Previously, in a phase II VISION study on the use of tepotinib for treating *MET* exon 14 skipping mutation-positive unresectable advanced NSCLC, the overall response rate (ORR) was 46%, with a progression-free survival (mPFS) of 8.5 months and overall survival (mOS) of 17.1 months. However, the majority of the cases were adenocarcinomas, and only 1/99 cases (approximately 1%) was that of sarcoma-like NSCLC (14). Another phase II study on the use of capmatinib (GEOMETRY mono-1 study) showed an ORR of 68% and mPFS of 12.4 months in untreated patients and an ORR of 41% and mPFS of 5.4 months in previously treated patients (15). However, it was not specified whether patients with sarcomatoid carcinomas were included in this study. In contrast, a phase II study of savolitinib monotherapy in locally advanced or metastatic *MET* exon 14 skipping-positive NSCLC in China reported an ORR of 40% and mPFS of 5.5 months in 25 patients with pulmonary sarcomatoid carcinoma, suggesting a potential benefit in patients with pulmonary sarcomatoid carcinoma (16). Nonetheless, there are insufficient data on the effect of MET inhibitors on sarcomatoid carcinomas with *MET* exon 14 skipping mutations. To the best of our knowledge, this is the first report of a PPC harbouring a *MET* exon 14 skipping mutation that was successfully treated with tepotinib.

In recent years, research on the biological characteristics of pulmonary sarcomatoid carcinoma has progressed, and it has become clear that therapeutic intervention based on genetic mutations is as important as that in other NSCLCs. Microdissection analysis of the epithelial and sarcomatoid components of PPCs has revealed that both components harbour similar genomic alterations, including activating driver mutations (17). Therefore, even in heterogeneous cancers such as pleomorphic carcinoma, a certain effect of molecular

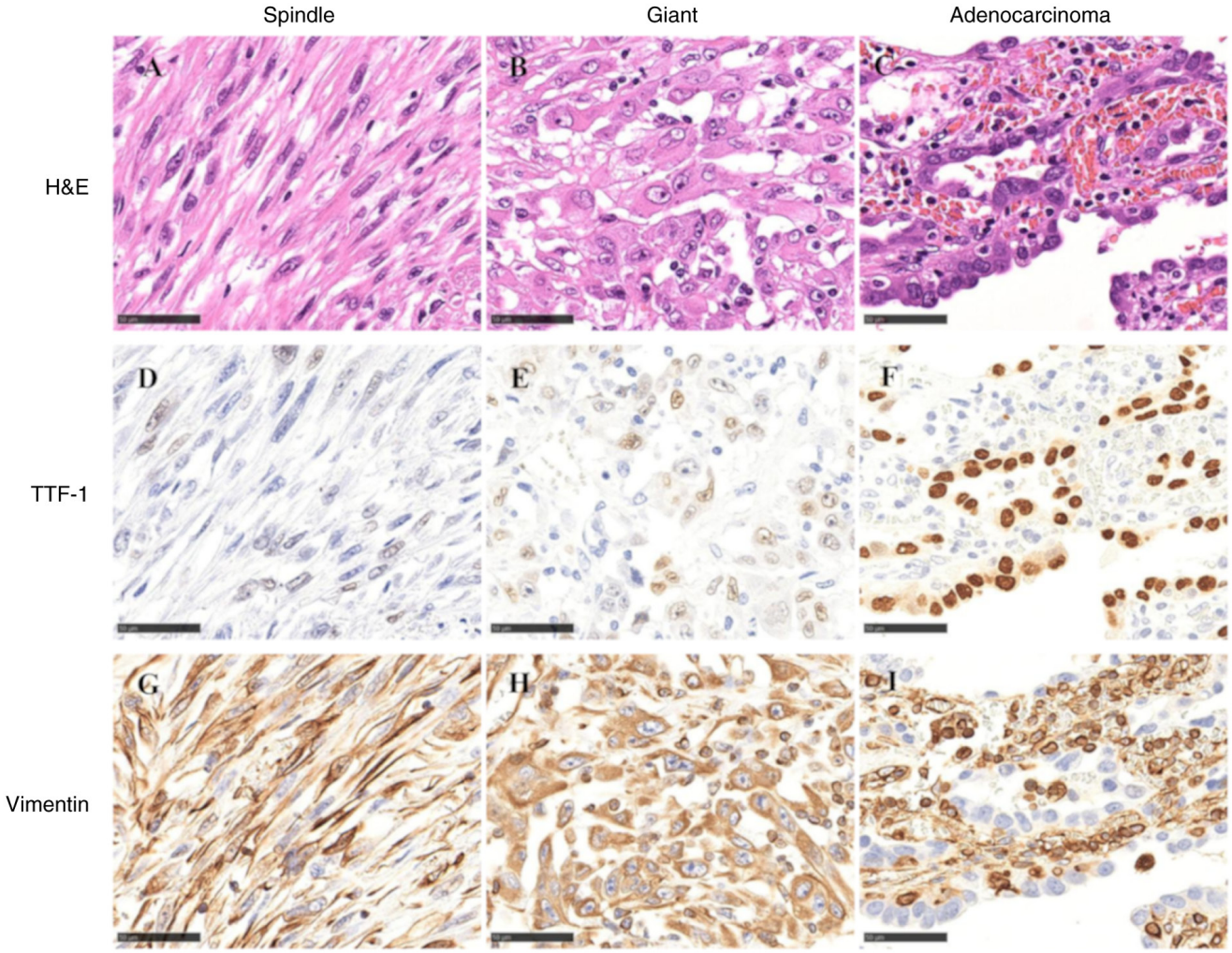

Figure 2. Histological findings of the surgical specimens. H&E staining of (A) spindle cells component, (B) giant cells component and (C) adenocarcinoma component. Immunohistochemical staining for TTF-1 was (D) weakly positive in the spindle cells component, (E) weakly positive in the giant cells component and (F) positive in the adenocarcinoma component. Immunohistochemical staining for Vimentin was (G) positive in spindle cells component, (H) positive in giant cells component and (I) negative in adenocarcinoma component. Black bar, 50 μm. H&E, haematoxylin and eosin; TTF-1, thyroid transcription factor-1

targeted therapies can be expected when driver mutations are detected.

In our patient's case, the PFS with tepotinib was relatively shorter than that reported in studies on NSCLC with *MET* exon 14 skipping. Acquired resistance to the selective tepotinib in *MET* exon 14 skipping NSCLC involves both on-target and off-target mechanisms. On-target resistance is predominantly caused by secondary mutations in the MET kinase domain, especially at residues D1228 and Y1230, which impair drug binding and restore MET signalling despite continued tepotinib exposure. Off-target or bypass resistance mechanisms include alterations in KRAS, EGFR/HER family genes, and other downstream effectors, which reactivate proliferative signalling independent of MET inhibition (18,19). In this case, the presence of pleomorphic carcinoma, a histologic subtype known for its marked heterogeneity, may have contributed to the tumour's susceptibility to phenotypic change. In addition, the reduction of the systemic therapy dose to one-quarter of the standard level due to renal and hepatic dysfunction likely influenced the short PFS.

Pulmonary sarcomatoid carcinoma is known to be an immune-hot tumour with a high tumour mutation burden, abundant $CD8^+$ T cell infiltration, and high expression of PD-L1 (20). For treating these tumours, ICIs such as anti-PD-1/PD-L1 and anti-CTLA-4 antibodies are expected to be effective. In fact, a previous analysis of 124 patients with advanced or metastatic pulmonary sarcomatoid carcinoma treated with ICI revealed an ORR of 59%, mPFS of 10.5 months, and mOS of 32.8 months, and 81 patients (65%) were strongly positive for PD-L1 (21). Additionally, in the analysis of 37 patients with pulmonary sarcomatoid carcinoma who were treated with ICI monotherapy as secondary treatment, an ORR of 40.5%, mPFS of 4.9 months, and mOS of 12.7 months were obtained regardless of PD-L1 expression (22). In another single-arm phase II study conducted at a Korean institution to evaluate the efficacy of durvalumab and tremelimumab in 18 patients with recurrent or metastatic pulmonary sarcomatoid carcinoma, the ORR, mPFS, and mOS were 26.7%, 5.9 months, and 15.4 months, respectively (23). In our case, PPC showed high PD-L1 expression, for which pembrolizumab led to a durable response. Thus, ICIs are gradually improving clinical outcomes of patients with sarcomatoid carcinomas such as PPC. Therefore, ICIs should be considered as key drugs for the treatment of sarcomatoid carcinoma.

This case report has several limitations. First, the patient's advanced age, poor performance status and the need for pleurodesis and palliative care during the course of treatment required careful consideration of treatment selection at each stage. In addition, dose reduction of tepotinib was necessary, which likely limited its efficacy. Given these constraints, determining the optimal first-line treatment for this case, characterized by *MET* exon 14 skipping mutation, high PD-L1 expression, and sarcomatoid carcinoma rather than conventional NSCLC, remains challenging. Another limitation is that a tumour re-biopsy could not be performed at the time the patient developed resistance to tepotinib because of the patient's poor performance status and the need for an urgent change in treatment, which ultimately precluded elucidation of the underlying resistance mechanism.

In conclusion, the present study describes the case of patient with PPC with a *MET* exon 14 skipping mutation that was treated with tepotinib and subsequently pembrolizumab therapy that led to a durable response. Therefore, sarcomatoid carcinoma, which is generally considered to have a poor prognosis, may have a better prognosis when treated with ICI and MET inhibitors. Consequently, it is critical to consider driver mutations in PPC and other pulmonary sarcomatoid carcinomas and to seize the opportunity to treat patients with these carcinomas using molecular targeted medications or ICIs.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

ST contributed to conceptualization and writing the original draft. YS conceptualized the study, wrote the original draft, and reviewed and edited the manuscript. YA, JA, RY, TW, HW, KK, RS, NY, MA, MS, TT, KN and SK contributed to data collection. SW, HU and TK contributed to analysis and interpretation of the data, supervision and editing. YS, SW, and TK confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Written informed consent was obtained from the patient, including consent to participate.

Patient consent for publication

Written informed consent was obtained from the patient, including consent for publication of the findings.

Competing interests

The authors declare that they have no competing interests.

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