Delayed pulmonary metastasis and recurrence of intracranial malignant solitary fibrous tumor/hemangiopericytoma: Case report and literature review

QIHENG LI, CHUNMEI ZHANG and ZHENGJIN LI

Department of Pathology, The First Affiliated Hospital of Dali University, Dali, Yunnan 671000, P.R. China

Received March 29, 2022; Accepted May 24, 2022

DOI: 10.3892/ol.2022.13375

Abstract. Solitary fibrous tumors/hemangiopericytomas (SFTs/HPCs) are intracranial spindle cell tumors that originate from interstitial tissue. SFTs/HPCs that are primary malignant intracranial tumors are exceedingly uncommon. A case of intracranial malignant SFT/HPC that originated intracranially and spread to the pulmonary region is described herein. Furthermore, the specimens from two surgical resections obtained when the patient had undergone two prior procedures for intracranial 'meningiomas' were also reviewed. The results of the lung biopsy matched the morphologic appearance of the intracranial tumor. The patient died ~2 years after the chest pain started. In addition, the literature was reviewed. According to previous studies, STAT6 expression was positive in 100% of SFTs/HPCs and radiologic characteristics assisted in determining the tumor pathology and grade. Surgical management has been the mainstay treatment for SFTs. In cases of incomplete resection, adjuvant radiotherapy is effective and rigorous follow-up is required to monitor for recurrence.

Introduction

Solitary fibrous tumors (SFT) and hemangiopericytomas (HPC) are solid tumors that originate from mesenchymal tissue and typically occur in soft tissue (1). Although intracranial tumors are possible, they are uncommon, accounting for only 0.4 percent of all primary central nervous system (CNS) tumors (2). Repeated intracranial SFT/HPC recurs locally and has a high rate of metastasis even long after the initial treatment. At our department, a case with multiple well-enhanced masses in the left upper lobe was encountered. Initially, primary lung adenocarcinoma was suspected. The patient had previously undergone two separate surgeries to remove two intracranial meningiomas. The patient was eventually diagnosed with an extracranial metastasis of a primary intracranial SFT/HPC, as well as an intracranial recurrence and metastasis that had occurred 11 and 13 years after the first resection in 2004. Furthermore, the differential diagnoses of intracranial masses and how to distinguish them based on imaging characteristics and immunohistochemistry were discussed.

Case report

A 50-year-old male presented at The First Affiliated Hospital of Dali University (Dali, China) in September 2017 with paroxysmal left-side chest discomfort that had become increasingly severe over the preceding month. Routine biochemical and hematological test results were within normal limits, but CT revealed a well-defined oval lung mass in the left upper lobe near the hilum measuring 41x35 mm in size (Fig. 1A). Pre-operative CT indicated heterogeneous enhancement of a round mass of up to 31x21 mm in the left thalamus, and a significant cavity was observed on post-operative review after resection of the brain tumor (Fig. 1B and C). MRI revealed well-defined masses in the left thalamus, corpus callosum body, left frontal lobe and right temporal lobe measuring 31x21 mm in size. The tumor had a low signal on T1- and T2-weighted images and the tumor in the bilateral cerebellum was isointense on T1- and T2-weighted images; furthermore, the tumor had uneven enhancement. The patient had previously undergone multiple intracranial tumor resections (Table I). The first was an excision of a right cerebellar tentorium tumor 17 years previously, which had been identified as a fibrous meningioma. The second procedure was the excision of a tumor that was diagnosed as meningioma in the left cerebellopontine angle, World Health Organization (WHO) grade I. According to the WHO 2016 revised guidelines, meningiomas are classified as follows: Grade I, benign meningiomas with <4+ mitoses per 10 consecutive high-power fields (HPF; objective magnification, x40); Grade II, atypical with a mitotic rate of 4-19 per 10 HPF or brain invasion (if neither feature is present, at least three of the following five histologic criteria must be evident to arrive at a Grade II diagnosis: Intratumoral micronecrosis not caused by presurgical thrombosis therapy; patternless sheets of tumor cells; prominent nucleoli; high cellularity; and tumor cells with scant cytoplasm relative to nuclear size); Grade III, anaplastic (malignant) with >20+ mitoses per 10 consecutive

Correspondence to: Professor Chunmei Zhang, Department of Pathology, The First Affiliated Hospital of Dali University, 32 Carlsberg Avenue, Dali, Yunnan 671000, P.R. China
E-mail: zhangchunmeiemail@163.com

Key words: primary brain tumors, solitary fibrous tumors, meningiomas, hemangiopericytoma

HPF (2). Given that the tumor in the upper lobe of the left lung was the largest, it was first assumed to be the primary lesion but this was later disproven, as it was a pulmonary metastasis from a primary intracranial tumor. The upper lobe of the left lung tumor was subjected to a CT-guided biopsy. Based on these findings, a definitive diagnosis of malignant SFT/HPC pulmonary metastases was made.

In the present case, the patient had already undergone two surgeries to remove intracranial meningiomas. The specimens that had been removed in 2004 were also evaluated in 2015, since it was suspected that the excised tumors were indeed SFT/HPC. A pathological examination on biopsy specimens from 2017 was also performed. The morphological appearance of the larger intracranial mass was confirmed to be consistent with the biopsy. The tumor histopathology revealed an abundance of typical 'staghorn' vascularization in the tissue (Fig. 2A) and tumor cells grew around blood vessels. A large number of spindle-shaped tumor cells arranged in bundles and alternating between sparse and dense distribution was observed between the vessels (Fig. 2B) and mitotic bodies were visible (≥5 mitoses per 10 high-power fields) (Fig. 2C). Histopathological and immunohistochemical findings of the tumor from 6 years previously revealed spindle cells positive for STAT6 (Fig. 3A), vimentin (Fig. 3B), CD34 (Fig. 3C) and Bcl-2 (Fig. 3D), and negativity for smooth muscle actin (Fig. 4A), S-100 (Fig. 4B), epithelial membrane antibody (EMA) (Fig. 4C) and progesterone receptor (PR) (Fig. 4D), findings that were compatible with malignant SFT/HPC, WHO III. Therefore, all intrapulmonary and intracranial lesions were considered recurrence and metastasis from a primary intracranial SFT/HPC. The patient was discharged after 15 days of radiotherapy. One year and 9 months after the onset of chest pain, the patient's systemic condition worsened and CT indicated thoracolumbar metastasis. The patient died two months after the detection of multiple metastases throughout the body.

Discussion

SFT/HPC tumors are perivascular cell cancers. Although such tumors may arise intracranially, they are infrequent, accounting for <1% of all primary CNS tumors, with the majority occurring in the fifth decade of life and with no apparent sex differences (3). Solitary fibrous tumors and hemangiopericytomas of the CNS exhibit overlapping pathology and immunohistochemical characteristics, such as occurrence in the neuraxis, inversions at 12q13 and overexpression of the NGFI-A-binding protein 2 (NAB2)-STAT6 gene fusion; according to recent research, quantitative PCR revealed high expression levels of the 5'-end of NAB2 and the 3'-end of STAT6, which, on deep sequencing of enriched DNA corresponded to NAB2/STAT6 fusions (2,4). Their diagnoses inevitably overlap as a result of this representation (5,6).

In the report for 2016, the WHO classification of CNS tumors has created the combined term SFT/HPC (7,8). Thus, as HPC and SFT have similar imaging features, their combined diagnosis may decrease the incidence of presurgical misdiagnosis. Until the tumor reaches a particular size or invades brain regions that produces measurable effects or has functional implications, there are no identifiable clinical symptoms.

Although radiologic characteristics may assist in predicting and grading tumor pathology, pathological examination remains the gold standard for diagnosis and pathological grading. On T2-weighted MRI (T2WI), the majority of cases with WHO grade I exhibit intermediate-low signal intensity (3). A minority of the cases have 2 different signal intensity areas on T2WI. T2 hyperintense regions indicate fibrotic components with distinct difference enhancement, whereas T2 iso- or hyperintense areas represent hypercellular components with mild heterogeneous enhancement. The so-called black-and-white or yin-yang signals are associated with intracranial SFTs/HPCs when these two components are combined (9-11). For WHO grades II and III, the tumors generally have intermediate-high signal intensity on T2WI. The existence of a tortuous flow-empty vascular shadow inside or on the surface of these two grades of tumors is critical for distinguishing WHO grade I from WHO grade II and III. Preoperatively, SFTS/HPCs are frequently misdiagnosed as fibrous meningiomas or nerve sheath tumors, which are difficult to differentiate. SFTs/HPCs are more prone than meningiomas to develop necrosis, cystic degeneration and areas of signal void, whereas meningiomas usually present with dural caudal symptoms.

Owing to the diversity of histological patterns exhibited by SFT, they frequently pose a diagnostic challenge and integration of clinical, histomorphological, immunohistochemical and molecular features is necessary for establishing a correct diagnosis (12). However, the final pathological and immunohistochemical findings remain the gold standard for diagnosing intracranial SFTS/HPCs, which are classified into three categories (I-III) based on a set of characteristics (13). Grade I SFT/HPC has more collagen and a relatively low cell density with spindle-like cells. Grade II has more cells arranged in no specific direction and less collagen, and staghorn-like vascular branches were observed. There were at least 5 mitotic figures per 10 high-power microscopes at Grade III (2). In SFT, high mitosis, necrosis and atypia are all crucial indicators of malignant and aggressive behavior (14). Since intracranial SFT/HPC is similar to meningioma in terms of clinical presentation and pathological diagnosis, it is critical to distinguish between them. The histopathological feature of SFT is the coexistence of sparse and dense regions, which are separated by fibrous stroma and have hemangiopericytoma branching vessels (15). Tumors with the SFT phenotype had a patternless architecture or a short fascicular pattern, with alternating hypocellular and hypercellular areas and thick collagen bands on histopathology (2). Tumors with an HPC phenotype have a high level of cellularity across the entire area. Meningioma cells may be observed to be arranged in nest-like clusters under the microscope, with abundant cytoplasm and poorly defined cells (syncytium-like). Pseudo-inclusions are common in the nucleus and the cells have weakly defined cell borders (syncytial cell-like) (16). Within the meningioma, sand granule formation is also seen. In SFT/HPCs, STAT6 is always positive, CD34 is positive to varying degrees, and most of them also express Bcl-2 and CD99. STAT6 immunohistochemistry is both a highly specific and sensitive surrogate for NAB2-STAT6 gene fusions, and the specificity and sensitivity of nuclear STAT6 for SFT/HPCs were 100 and 96.6%, respectively (17-19). Detection of STAT6 nuclear expression, which is

Table I. Timeline of recurrence, metastasis and treatment of the present case of malignant SFT/HPC.

Year/month	Diagnosis	Location	Treatment
2004	Fibrous meningiomas	Right cerebellar tentorium	Gross total resection
2015	Meningiomas (WHO I)	Left cerebellopontine angle	Gross total resection
2017.2	Possibility of meningioma recurrence	Upper lobe of left lung near the hilum	Follow-up
2017.9	Central lung malignant tumor	Upper lobe of left lung near the hilum	Radiation
2017.10	Lung spindle cell tumor	Upper lobe of left lung near the hilum	Radiation
2017.12	SFT/HPC	Lung and bilateral cerebral hemisphere	Radiation
2018.1	SFT/HPC	Lung and bilateral cerebral hemisphere	Radiation
2018.4	SFT/HPC	Thoracic spine and other parts of the body	Radiation

SFT/HPC, solitary fibrous tumors/hemangiopericytomas; WHO, World Health Organization.

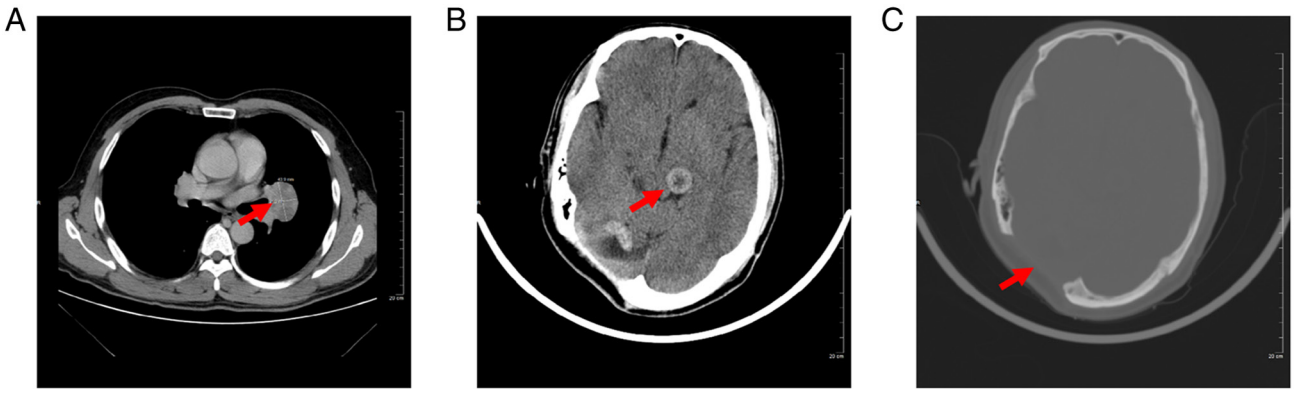

Figure 1. CT images prior to the puncture biopsy in 2017. (A) CT indicating a heterogeneously enhanced lung mass measuring up to 44x31 mm in the left upper lobe near the hilum. The arrow indicates the lung mass. (B) Preoperative CT revealed a heterogeneously enhanced round mass measuring up to 31x21 mm in the left thalamus. The arrow indicates the left thalamic mass. (C) CT (bone window) indicated a cavity after resecting the brain tumor and no local recurrence (scale bar, 20 cm). The arrow indicates the cavity after resecting the brain tumor.

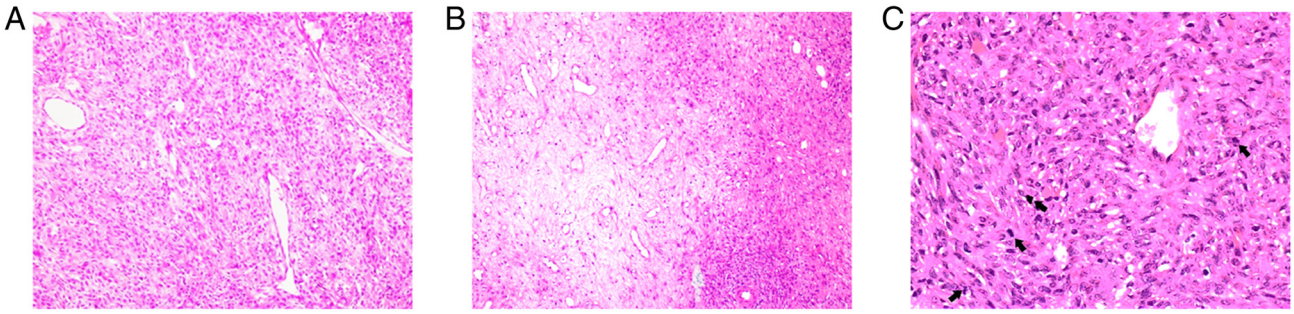

Figure 2. Hematoxylin and eosin staining of different regions of the same solitary fibrous tumor/hemangiopericytoma specimen. (A) An abundance of blood vessels was observed in tumor tissue, with a typical 'staghorn' vascularization and a large number of fusiform tumor cells arranged in bundles between blood vessels (magnification, x200). (B) HE staining indicated that sparse areas and dense areas coexisted (magnification, x100). (C) Mitotic bodies were visible (black arrow; ≥5 mitoses per 10 high-power fields) (magnification, x400).

a molecular hallmark of SFTs, is recommended to confirm the diagnosis of SFT/HPC as per the 2021 WHO guidelines (20). SFT/HPCs are positive for CD34 and STAT6 on immunohistochemistry and negative for EMA and PR. However, for all forms of meningiomas, the opposite is true (21). When the histology results make it difficult to distinguish between the two, immunohistochemical examination of markers such as STAT6, CD34, EMA and PR may be a valuable tool.

Most SFTs grow slowly, but low-grade SFTs/HPCs may progress to higher-grade tumors (22). Of note, only a small number of cases of malignant progression from lower-grade SFT/HPC tumors have been reported in the literature, owing to the lack of a comprehensive review of tumor recurrence. As in the present case, a WHO grade II right cerebellar curtain mass had developed into a WHO grade III left cerebellar horn mass. Surgical resection is the treatment of choice for SFT/HPC and

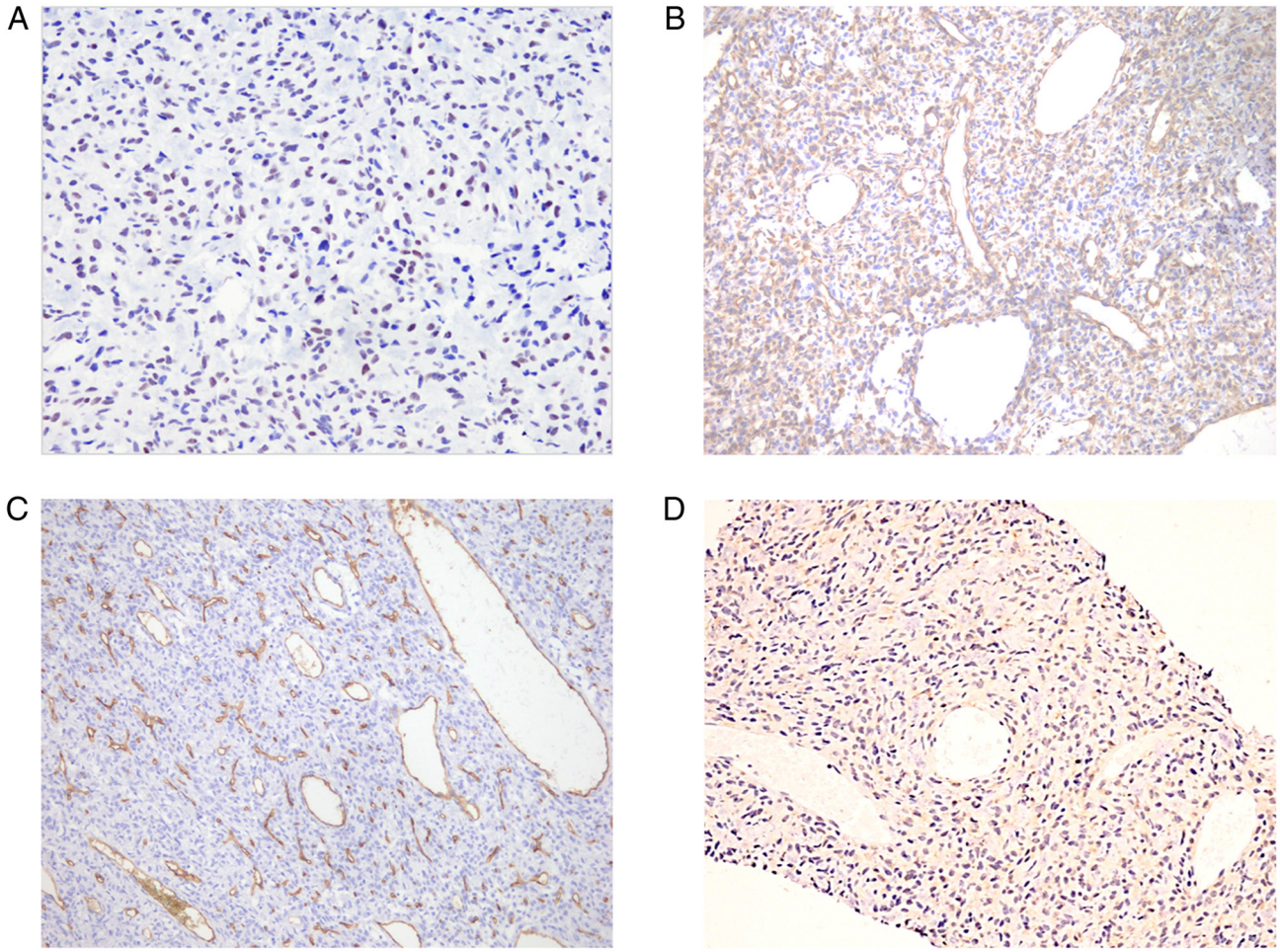

Figure 3. Immunostaining of lung puncture biopsy specimen in 2017. Immunohistochemistry indicated (A) STAT6 positivity of the tumor cell nuclei, (B) Vimentin positivity in the tumor cells' cytoplasm, (C) CD34 positivity of tumor cell membrane and cytoplasm, (D) Bcl-2 positivity of tumor cell cytoplasm and membrane (magnification, x200 for all, tumor cells with brownish-yellow staining are immunohistochemically positive).

gross total resection is the most important factor in tumor management (23). However, this is technically difficult, as most higher-grade tumors invade important surrounding anatomical structures, such as arteries, venous sinuses and nerves, and because the tumor has a rich blood supply, intraoperative bleeding frequently occurs. Preoperative embolization of the tumor's blood supply artery has been documented in the literature to reduce intraoperative bleeding and surgical difficulty (24). Complete excision of the mass is superior to incomplete excision and sub-totally removed SFTs/HPCs may recur or continue to grow. Subtotal resection carries a recurrence risk of up to 54%, compared to the 14% recurrence rate of total resection. As a result, the extent of resection is the most important predictor of SFTS/HPC recurrence. RT positively affects patient outcomes; in particular, patients undergoing gross total tumor resection + radiotherapy treatment exhibited the best survival advantage. Recurrence and metastasis of SFT/HPC are common and cases may progress to advanced SFT (25). Patients with advanced SFT exhibit a certain response to traditional chemotherapy drugs, but there are not many options and alternative treatments for unresectable tumors are urgently required.

In the present case, the patient had undergone partial surgical resection of a meningioma diagnosed in 2004 and 2015, respectively. SFT/HPC lung metastases at our institution were not surgically excised in the present case and the patient received radiotherapy treatment in 2017. The patient died two months later in 2018 after presenting with multiple metastases in the thoracic spine and throughout the body. It is necessary to be aware that recurrence and metastasis may occur even after a lengthy period of resection treatment, up to 10 years (26-28). The clinical course of patients with SFT/HPC is unpredictable, as local recurrence occurs in 25-85% of cases and whole-body metastases occur in 15-36% (29). SFT is a malignant condition that exhibits different clinical behaviors ranging from low to highly aggressive SFT. Malignant progression may be just one of several mechanisms provoking recurrence and metastasis (30). High-grade SFT/HPCs are more likely to recur and have an unfavorable overall survival rate. Higher histological grade and subtotal resection were associated with recurrence, while higher histological grade and recurrence were associated with metastasis formation. Recurrence was also revealed to be a risk factor for the establishment of metastases. The most prevalent locations of distant metastasis are the bone, liver, lung and abdominal cavity (31). Intraspinal spread of metastases from an intracranial HPC, particularly thoracic metastasis, is rare (32). Intracranial SFT/HPC is a tumor with moderate to low malignancy and a long survival

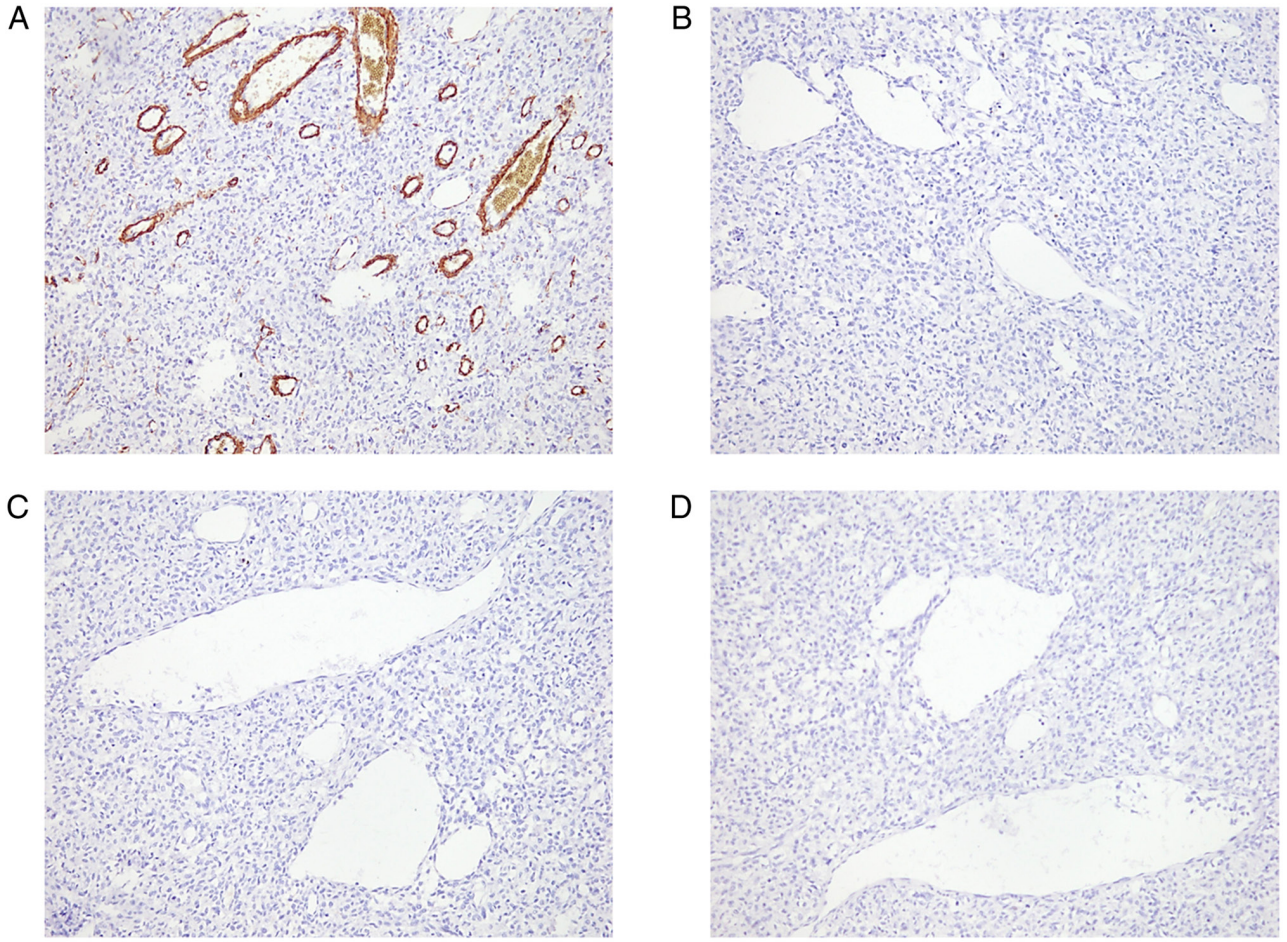

Figure 4. Immunostaining of lung puncture biopsy specimen in 2017. Immunohistochemistry indicated (A) SMA negative for tumor cells and positive for vascular smooth muscle, (B) S-100 negative for tumor cells, (C) EMA negative for tumor cells, (D) PR negative for tumor cells (magnification, x200 for all, brownish yellow indicates positive staining). SMA, smooth muscle actin; EMA, epithelial membrane antibody; PR, progesterone receptor.

period, and even if the tumor recurs or metastasizes distantly, as long as it is discovered early and treated immediately, it is possible to achieve a better outcome. Thus, therapy for intracranial SFT/HPC should be based on surgical resection with long-term vigilant monitoring (33). Follow-up of patients with SFT/HPC would probably reveal new recurrences and histological progression. The latest risk stratification model by Demicco *et al* (34) is based on assessment of patient age, mitoses/mm^2, tumor size and percentage of tumor necrosis to predict metastatic recurrences. It stratifies SFTs into low, intermediate and high-risk categories and is more accurate in predicting the prognosis. Therefore, it is appropriate to monitor disease progression based on risk prediction stratification model categories in combination with follow-up.

Intracranial SFT/HPCs are uncommon mesenchymal neoplasms. SFT/HPCs may recur and metastasize even long after initial treatment. In our group, a case of recurrence and pulmonary metastasis 11 years after treatment with intracranial primary SFT/HPC resection was encountered. It should also be noted that SFT/HPC was previously thought to be a subtype of meningioma (35). As the clinical features and imaging presentation of SFT/HPC are similar to those of common meningiomas, they are frequently difficult to recognize. Thus, clinicians should depend on tissue biopsy and immunohistochemistry to make a definitive diagnosis. Preoperative imaging helps to clarify the diagnosis and determine the tumor grade. STAT6 immunohistochemistry is also a valuable and sensitive diagnostic method. Adjuvant radiation therapy is effective for malignant tumors that cannot be completely resected (36). In recent years, the molecular genetics of soft tissue tumors have been developing rapidly and the new generation of molecular tests, represented by second-generation sequencing, may not only provide an accurate clinical diagnosis but also assist the search for therapeutic targets in clinical research, formulate treatment strategies, assist in determining prognosis and provide relevant testing information for individualized and precise treatment of patients with soft tissue tumor (37,38).

Molecular target therapy is a promising approach for unresectable or metastatic SFT and an improved knowledge of the molecular biology of the neoplasm may support such therapy in the near future. It was reported that certain growth factors and kinases are overexpressed in SFTs, including platelet-derived growth factor (PDGF) α, PDGFβ, PDGF receptor (PDGFR)-α, PDGFR-β, insulin-like growth factor (IGF) 1 receptor, epidermal growth factor receptor, vascular endothelial growth factor (VEGF), IGFII, cellular-mesenchymal epithelial transition, c-kit, c-erbB2, phosphatase and tensin homolog deleted on chromosome 10, phosphorylated (p)AKT, pS6, phosphorylated 4E-binding protein, ERBB2, FGFR1 and JAK2 (39,40). Overexpression of these markers leads to activation of the

Akt/mTOR pathway and appears to be associated with tumor necrosis, targeted therapies toward the IGF signaling pathway and the Akt/mTOR pathway is considered a candidate therapeutic target, whereas it was not possible to directly establish an association with the actual clinical outcome (40). The 2021 National Comprehensive Cancer Network guidelines recommend the use of four targeted agents, bevacizumab, sunitinib, pazopanib and sorafenib, for the treatment of SFT/HPC, and all have activity against VEGF receptor (VEGFR)-1, -2 and -3, whose broad spectrum of targets may achieve in potential antitumor as well as antiangiogenic effects in tumors (41-43). Combination therapy with temozolomide and bevacizumab appears to provide a clinical benefit (44). Pazopanib is an anti-angiogenesis-based, small molecule, multi-targeting agent that interferes with angiogenesis inhibitors required for intractable tumor survival and growth, and has activity against VEGFR-1, -2 and -3, as well as PDGFR and KIT (45). Sorafenib inhibits the tyrosine kinases VEGFR-1, -2 and -3, PDGFR, RET/PTC as well as the Raf/Mek/Erk pathway (46). It is suggested that the detection of molecular targets (such as VEGFR-1, -2 and -3, BRAF, RET and PTC) is performed in patients with SFT, which will help to screen the potential beneficiaries of targeted therapy and extend the survival of the patient.

Acknowledgements

Not applicable.

Funding

The present study was supported by the Yunnan Provincial Education Department Scientific Research Fund Project (grant no. 2022Y837).

Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Authors' contributions

QL was responsible for collecting clinical, imaging and pathological data of the patient and drafting the manuscript. and CZ analyzed the data and revised the manuscript. ZL participated in making the pathological diagnosis. QL, CZ and YL confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of The First Affiliated Hospital to Dali University (approval no. 20200612).

Patient consent for publication

Written consent for publication of the case report and any accompanying images, without any potentially identifying information, was provided by the patient's family.

Competing interests

The authors declare that they have no competing interests.

References

1. Stout AP and Murray MR: Hemangiopericytoma: A vascular tumor featuring zimmermann's pericytes. Ann Surg 116: 26-33, 1942.
2. Louis DN, Perry A, Reifenberger G, von Deimling A, Figarella-Branger D, Cavenee WK, Ohgaki H, Wiestler OD, Kleihues P and Ellison DW: The 2016 world health organization classification of tumors of the central nervous system: A summary. Acta Neuropathol 131: 803-820, 2016.
3. Wang ZY, Qiu K, Ma YH, Wang XT, Bao JJ, Zhang ZF and Liu XZ: Intracranial solitary fibrous tumors: A report of two cases and a review of the literature. Oncol Lett 11: 1057-1060, 2016.
4. Yamashita D, Suehiro S, Kohno S, Ohue S, Nakamura Y, Kouno D, Ohtsuka Y, Nishikawa M, Matsumoto S, Bernstock JD, *et al*: Intracranial anaplastic solitary fibrous tumor/hemangiopericytoma: Immunohistochemical markers for definitive diagnosis. Neurosurg Rev 44: 1591-1600, 2021.
5. Fritchie K, Jensch K, Moskalev EA, Caron A, Jenkins S, Link M, Brown PD, Rodriguez FJ, Guajardo A, Brat D, *et al*: The impact of histopathology and NAB2-STAT6 fusion subtype in classification and grading of meningeal solitary fibrous tumor/hemangiopericytoma. Acta Neuropathol 137: 307-319, 2019.
6. Georgiesh T, Namløs HM, Sharma N, Lorenz S, Myklebost O, Bjerkehagen B, Meza-Zepeda LA and Boye K: Clinical and molecular implications of NAB2-STAT6 fusion variants in solitary fibrous tumour. Pathology 53: 713-719, 2021.
7. Kim BS, Kim Y, Kong DS, Nam DH, Lee JII, Suh YL and Seol HJ: Clinical outcomes of intracranial solitary fibrous tumor and hemangiopericytoma: Analysis according to the 2016 WHO classification of central nervous system tumors. J Neurosurg 129: 1384-1396, 2018.
8. Shin DW, Kim JH, Chong S, Song SW, Kim YH, Cho YH, Hong SH and Nam SJ: Intracranial solitary fibrous tumor/hemangiopericytoma: Tumor reclassification and assessment of treatment outcome via the 2016 WHO classification. J Neuroncol 154: 171-178, 2021.
9. Frazier AA: The yin and yang of solitary fibrous tumor. Radiographics 34: 294, 2014.
10. Yang H, Zhang Y, Zheng T, Li C, Tang G and Chen G: A solitary fibrous tumor/hemangiopericytoma of the fourth ventricle: Case report and literature review. J Int Med Res 47: 6349-6355, 2019.
11. Clarençon F, Bonneville F, Rousseau A, Galanaud D, Kujas M, Naggara O, Cornu P and Chiras J: Intracranial solitary fibrous tumor: Imaging findings. Eur J Radiol 80: 387-394, 2011.
12. Ronchi A, Cozzolino I, Marino FZ, Accardo M, Montella M, Panarese I, Roccuzzo G, Toni G, Franco R and De Chiara A: Extrapleural solitary fibrous tumor: A distinct entity from pleural solitary fibrous tumor. An update on clinical, molecular and diagnostic features. Ann Diagn Pathol 34: 142-150, 2018.
13. Ma L, Wang L, Fang X, Zhao CH and Sun L: Diagnosis and treatment of solitary fibrous tumor/hemangiopericytoma of central nervous system. Retrospective report of 17 patients and literature review. Neuro Endocrinol Lett 39: 88-94, 2018.
14. Kallen ME and Hornick JL: The 2020 WHO classification: What's new in soft tissue tumor pathology? Am J Surg Pathol 45: e1-e23, 2021.
15. Huang SC and Huang HY: Solitary fibrous tumor: An evolving and unifying entity with unsettled issues. Histol Histopathol 34: 313-334, 2019.
16. Buerki RA, Horbinski CM, Kruser T, Horowitz PM, James CD and Lukas RV: An overview of meningiomas. Future Oncol 14: 2161-2177, 2018.
17. Doyle LA, Vivero M, Fletcher CD, Mertens F and Hornick JL: Nuclear expression of STAT6 distinguishes solitary fibrous tumor from histologic mimics. Mod Pathol 27: 390-395, 2014.
18. Schweizer L, Koelsche C, Sahm F, Piro RM, Capper D, Reuss DE, Pusch S, Habel A, Meyer J, Göck T, *et al*: Meningeal hemangiopericytoma and solitary fibrous tumors carry the NAB2-STAT6 fusion and can be diagnosed by nuclear expression of STAT6 protein. Acta Neuropathol 125: 651-658, 2013.
19. Yoshida A, Tsuta K, Ohno M, Yoshida M, Narita Y, Kawai A, Asamura H and Kushima R: STAT6 immunohistochemistry is helpful in the diagnosis of solitary fibrous tumors. Am J Surg Pathol 38: 552-559, 2014.

20. Louis DN, Perry A, Wesseling P, Brat DJ, Cree IA, Figarella-Branger D, Hawkins C, Ng HK, Pfister SM, Reifenberger G, *et al*: The 2021 WHO classification of tumors of the central nervous system: A summary. Neuro Oncol 23: 1231-1251, 2021.
21. Barresi V, Caffo M and Tuccari G: Classification of human meningiomas: Lights, shadows, and future perspectives. J Neurosci Res 94: 1604-1612, 2016.
22. Apra C, Mokhtari K, Cornu P, Peyre M and Kalamarides M: Intracranial solitary fibrous tumors/hemangiopericytomas: First report of malignant progression. J Neurosurg 128: 1719-1724, 2018.
23. Ramakrishna R, Rostomily R, Sekhar L, Rockhill J and Ferreira M: Hemangiopericytoma: Radical resection remains the cornerstone of therapy. J Clin Neurosci 21: 612-615, 2014.
24. Patel SR, Vachhani P and Moeslein F: Embolic brain infarcts: A rare fatal complication of preoperative embolization of a massive solitary fibrous tumor of the pleura. Cardiovasc Intervent Radiol 40: 306-309, 2017.
25. Sonabend AM, Zacharia BE, Goldstein H, Bruce SS, Hershman D, Neugut AI and Bruce JN: The role for adjuvant radiotherapy in the treatment of hemangiopericytoma: A surveillance, epidemiology, and end results analysis. J Neurosurg 120: 300-308, 2014.
26. Hashida S, Yokota H, Oyama Y, Kawakami M, Murakami S and Kawakami H: Bulky cardiac metastasis of intracranial solitary fibrous tumor/hemangiopericytoma: Delayed metastasis after cranial tumor resection. Radiol Case Rep 14: 1175-1180, 2019.
27. Nakada S, Minato H, Takegami T, Kurose N, Ikeda H, Kobayashi M, Sasagawa Y, Akai T, Kato T, Yamamoto N and Nojima T: NAB2-STAT6 fusion gene analysis in two cases of meningeal solitary fibrous tumor/hemangiopericytoma with late distant metastases. Brain Tumor Pathol 32: 268-274, 2015.
28. Wu Z, Yang H, Weng D and Ding Y: Rapid recurrence and bilateral lungs, multiple bone metastasis of malignant solitary fibrous tumor of the right occipital lobe: Report of a case and review. Diagn Pathol 10: 91, 2015.
29. Damodaran O, Robbins P, Knuckey N, Bynevelt M, Wong G and Lee G: Primary intracranial haemangiopericytoma: Comparison of survival outcomes and metastatic potential in WHO grade II and III variants. J Clin Neurosci 21: 1310-1314, 2014.
30. Giordan E, Marton E, Wennberg AM, Guerriero A and Canova G: A review of solitary fibrous tumor/hemangiopericytoma tumor and a comparison of risk factors for recurrence, metastases and death among patients with spinal and intracranial tumors. Neurosurg Rev 44: 1299-1312, 2021.
31. Patel AR, Flores BC, Ban VS, Hatanpaa KJ, Mickey BE and Barnett SL: Intracranial hemangiopericytomas: Recurrence, metastasis, and radiotherapy. J Neurol Surg B Skull Base 78: 324-330, 2017.
32. Ali HSM, Endo T, Endo H, Murakami K and Tominaga T: Intraspinal dissemination of intracranial hemangiopericytoma: Case report and literature review. Surg Neurol Int 7 (Suppl 40): S1016-S1020, 2016.
33. Demicco EG, Park MS, Araujo DM, Fox PS, Bassett RL, Pollock RE, Lazar AJ and Wang WL: Solitary fibrous tumor: A clinicopathological study of 110 cases and proposed risk assessment model. Mod Pathol 25: 1298-1306, 2012.
34. Demicco EG, Wagner MJ, Maki RG, Gupta V, Iofin I, Lazar AJ and Wang WL: Risk assessment in solitary fibrous tumors: Validation and refinement of a risk stratification model. Mod Pathol 30: 1433-1442, 2017.
35. Schiariti M, Goetz P, El-Maghraby H, Tailor J and Kitchen N: Hemangiopericytoma: Long-term outcome revisited. Clinical article. J Neurosurg 114: 747-755, 2011.
36. Chen LF, Yang Y, Yu XG, Gui QP, Xu BN and Zhou DB: Multimodal treatment and management strategies for intracranial hemangiopericytoma. J Clin Neurosci 22: 718-725, 2015.
37. Anderson WJ and Doyle LA: Updates from the 2020 world health organization classification of soft tissue and bone tumours. Histopathology 78: 644-657, 2021.
38. von Mehren M, Kane JM, Bui MM, Choy E, Connelly M, Dry S, Ganjoo KN, George S, Gonzalez RJ, Heslin MJ, *et al*: NCCN guidelines insights: Soft tissue sarcoma, version 1.2021. J Natl Compr Canc Netw 18: 1604-1612, 2020.
39. Hajdu M, Singer S, Maki RG, Schwartz GK, Keohan ML and Antonescu CR: IGF2 over-expression in solitary fibrous tumours is independent of anatomical location and is related to loss of imprinting. J Pathol 221: 300-307, 2010.
40. Yamada Y, Kohashi K, Fushimi F, Takahashi Y, Setsu N, Endo M, Yamamoto H, Tokunaga S, Iwamoto Y and Oda Y: Activation of the Akt-mTOR pathway and receptor tyrosine kinase in patients with solitary fibrous tumors. Cancer 120: 864-876, 2014.
41. Stacchiotti S, Negri T, Libertini M, Palassini E, Marrari A, Troia BD, Gronchi A, Tos APD, Morosi C, Messina A, *et al*: Sunitinib malate in solitary fibrous tumor (SFT). Ann Oncol 23: 3171-3179, 2012.
42. Machado I, Nieto-Morales G, Cruz J, Navarro S, Giner F, Ferrandez A, López-Soto MV, Lavernia J and Llombart-Bosch A: Controversial issues in soft tissue solitary fibrous tumors: A pathological and molecular review. Pathol Int 70: 129-139, 2020.
43. Sawada N, Ishiwata T, Naito Z, Maeda S, Sugisaki Y and Asano G: Immunohistochemical localization of endothelial cell markers in solitary fibrous tumor. Pathol Int 52: 769-776, 2002.
44. Biermann JS, Chow W, Reed DR, Lucas D, Adkins DR, Agulnik M, Benjamin RS, Brigman B, Budd GT, Curry WT, *et al*: NCCN guidelines insights: Bone cancer, version 2.2017. J Natl Compr Canc Netw 15: 155-167, 2017.
45. Maruzzo M, Martin-Liberal J, Messiou C, Miah A, Thway K, Alvarado R, Judson I and Benson C: Pazopanib as first line treatment for solitary fibrous tumours: The royal marsden hospital experience. Clin Sarcoma Res 5: 5, 2015.
46. Park MS, Ravi V and Araujo DM: Inhibiting the VEGF-VEGFR pathway in angiosarcoma, epithelioid hemangioendothelioma, and hemangiopericytoma/solitary fibrous tumor. Curr Opin Oncol 22: 351-355, 2010.