Solitary fibrous tumor of the central nervous system invading and penetrating the skull: A case report

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Received September 28, 2022; Accepted December 20, 2022

DOI: 10.3892/ol.2023.13667

Abstract. Solitary fibrous tumor (SFT) of the central nervous system is a rare spindle cell tumor of mesenchymal origin. The present study reports the case of a 44-year-old male patient with SFT. Magnetic resonance imaging demonstrated that the majority of the intracranial tumors exhibited uneven low signals on T1-weighted imaging (T1WI) and low mixed signals on T2WI, and there was an enhancement on enhanced scanning. Furthermore, the distal part of the left occipital lobe exhibited hypersignals on T1WI and T2WI, and this was significantly enhanced following enhanced scanning. The lower part of the scalp exhibited low signals on T1WI and high signals on T2WI, and there was no notable enhancement following enhanced scanning. Magnetic resonance spectroscopy demonstrated an elevated choline/creatine peak in the solid part of the tumor. Under the microscope, the tumor exhibited characteristic 'staghorn-shaped' blood vessels. As SFT is difficult to differentially diagnose via imaging, immunohistochemical analysis of CD34, vimentin and signal transducer and activator of transcription 6 was performed for the definitive diagnosis of SFT. Of note, surgical resection was the preferred treatment for SFT; however, due to the rarity of the tumor, subsequent adjuvant therapy and prognosis require further investigation.

Introduction

Solitary fibrous tumor (SFT) is a rare spindle cell tumor of mesenchymal origin, initially reported by Klempere and Rabin (1) in 1931. SFT is commonly found in the mediastinum and visceral pleura; however, it also occurs in the pleura external sites, such as the head and neck, pericardium, peritoneum, liver, thyroid, mesentery, and sinuses and orbits (2). Due to the lack of a true connective tissue component in the central nervous system (CNS), primary SFT of the CNS is rare, accounting for ~1% of all primary CNS tumors (3,4). Most CNS SFTs occur in the cranial cavity and just over one-fifth were intraspinal (5-7). Primary spinal SFT may occur at any age (5). The mean age of onset was 40.9 years for males and 35.0 years for females (5). There was no significant difference in morbidity between males and females (5). Primary spinal SFT usually occurs in the thoracic spinal cord, followed by the cervical and lumbar spinal cord, and the sacral spinal cord is rarely affected (5). Intracranial SFT occurs most commonly in adults aged 20-70 years, with similar incidence rates in males and females (8). When CNS SFTs occur intracranially, they are frequently extra-axially located (9,10). Hemangiopericytomas (HPCs) are also rare mesenchymal tumors that exhibit similar clinical, radiological and histological features to SFTs (11). The NGFI-A-binding protein (NAB2) and signal transducer and activator of transcription 6 (STAT6) gene fusion was identified as a driver mutation of SFT (12,13). Previous pathological findings demonstrated that SFT and HPCs contain identical genetic abnormalities and these prompted the World Health Organization (WHO) to classify the two tumor types as a new combined entity in 2016 (14). This classification described three grades of SFT/HPC, namely grade I, II and III. Of note, the distinction between the two types was no longer clinically significant due to the pronounced clinical and histopathological overlap. In the 2021 WHO classification of CNS tumors, the term 'hemangiopericytoma' was removed and replaced with SFT (15).

A solid lesion located in the CNS distinct from fibrous meningioma, termed primary SFT, was initially reported by Carneiro *et al* (16) in 1996. Intracranially, SFT may occur at the cerebellopontine angle, spinal dura, parasagittal region, meninges and the intraventricular region (17). The present article reports on a 44-year-old male patient with SFT. The SFT originated from the superior sagittal sinus and not only penetrated through the skull, but also invaded the bilateral occipital lobes distally. This is the first case of SFT completely penetrating the skull, to the best of our knowledge. The imaging data, histopathological features and treatment of SFT were briefly reviewed and the imaging features of this case were discussed.

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Key words: solitary fibrous tumor, central nervous system, diagnosis

Case report

A 44-year-old male patient presented at the neurology outpatient clinic of Xiaolan People's Hospital of Zhongshan (Zhongshan, China) in August 2020 due to dizziness and blurred vision for one month. Neurological examination of the patient appeared normal; however, a visual field defect was observed below the central visual field of both eyes. Bone window computed tomography angiography (Ingenuity CT; Philips Medical Systems, Inc.; slice thickness, 0.8 mm; center, 450; width, 1,600) of the head demonstrated that the mass had invaded and penetrated the skull (Fig. 1A).

Head magnetic resonance imaging (MRI) findings demonstrated a mass shadow near the occipital cerebral falx, with an irregular shape and unclear boundaries. The mass was ~64x44x64 mm in size and stretched across both sides of the cerebral falx. The mass filled the posterior portion of the superior sagittal sinus to form a filling defect, invading and penetrating the occipital bone, and the boundary between the mass and bilateral occipital lobe brain tissue was unclear. Of note, the majority of the intracranial tumors exhibited uneven low signals on T1-weighted imaging (T1WI) and low mixed signals on T2WI, with notable enhancements following enhanced scanning. The distal part of the left occipital lobe demonstrated hypersignals on T1WI and T2WI, with significant enhancements following enhanced scanning. In addition, the lower part of the scalp exhibited low signals on T1WI and high signals on T2WI, and there were not notably enhanced following enhanced scanning (Fig. 1B-D).

Results of the magnetic resonance spectroscopy (MRS) demonstrated a multi voxel collection with no notable N-acetylaspartic acid peak in the mass. The choline/creatine peak in the solid area was increased; however, no notable abnormalities were found in the spectral lines of the tissues surrounding the mass (Fig. 1E).

A craniotomy was performed for tumor resection and follow-up MRI demonstrated complete tumor resection (Fig. 1F). During the operation, the tumor was gray and red in color with an abundant blood supply (Fig. S1A). Foci indicative of previous bleeding far from the origin occurred in the tumors in both the occipital lobe and the scalp. In addition, the tumor texture was uneven, with both soft and tough sections, with an incomplete capsule and lobulated invasive growth. The tumor broke through the brain tissue of the occipital lobe and the demarcation between the tumor and the brain tissue of the occipital lobe was unclear. The occipital pia mater was markedly edematous. The adjacent occipital skull demonstrated osteolytic bone destruction, the tumor penetrated the occipital bone to form a local mass under the scalp and the local scalp thickened reactively.

Pathological examination revealed that the tumor was composed of alternately distributed cell-rich areas and cell-sparse areas. The tumor cells in the cell-rich area were short-spindle or oval, with little cytoplasm and uniform nuclear chromatin. There was no notable atypia in the two areas. The frequency of mitotic figures was 1/10 high-power fields (HPF) and the tumor cells were arranged in sheets and striae. These were hemangiopericytoma-like, with abundant blood vessels in the tumor. Thus, these were labeled 'staghorn-shaped blood vessels' (Fig. 2A). Tumor cells presented with diffuse strong immunoreactivity to CD34, vimentin and STAT6 (Fig. 2B-D). The Ki-67 labeling index was ~10% with no signs of necrosis (Fig. 2E). The tumor cells presented as weakly positive for epithelial membrane antigen (EMA) and negativity for progesterone receptor (PR) (Fig. S1B and C). Grade I SFT cells are fusiform, with lower cell density and higher collagen content. Grade II SFT has more cells, less collagen, no specific cell arrangement and typical staghorn-shaped blood vessels. There are more than 4 mitotic figures per 10 HPF of grade III SFT (14). Intracranial SFT is mainly differentiated from meningioma because they are similar in clinical presentation and pathological diagnosis. The histopathological features of SFT are sparse and dense areas separated by fibrous stroma, with hemangiopericytoma branching vessels (18). The phenotype of SFT is characterized by a patternless architecture or a short fascicular pattern (14). SFT is histopathologically characterized by alternating low-cell and high-cell areas and thick collagen bands (14). Microscopically, the meningioma cells are nested, with abundant cytoplasm and unclear cells (syncytioid) (19). Pseudo-inclusions are common in meningioma nuclei, where cells have weakly defined cell boundaries (syncyti-like) (19). In SFT, STAT6 is positive in almost all patients, while CD34 is positive to varying degrees (20-22). However, all forms of meningiomas characteristically expressed EMA and PR; CD34 reactivity was patchy and weak; STAT6 was not expressed (21,22). Histopathological examination confirmed WHO grade II SFT.

The protocols of the imaging examinations, histopathological staining and immunohistochemistry (IHC) are provided in the supplemental data.

Three-dimensional emphasis radiotherapy was performed 18 days after surgery [tumor absorbed dose: Planning target volume (PTV)1, 60.2 Gray/28 fractions; PTV2, 54.6 Gray/28 fractions]. At the follow-up 3 months after the surgery, the patient reported that the headache and dizziness symptoms had gradually disappeared after the surgery. The patient's visual field was examined using a Humphrey II 740 Visual Field (Carl Zeiss Meditec), indicating that the patient's visual field was significantly improved compared with that prior to surgery. During the two-year follow-up, the patient experienced no recurrence of SFT.

Discussion

The majority of intracranial SFTs are dural masses originating predominantly from thick collagen bands, which are produced by fibroblasts, most frequently occurring in the parasagittal sinus and spinal canal (3,23-25). In the present study, the patient experienced SFT originating in the superior sagittal sinus, consistent with previous reports (3,23-25).

Symptoms of SFT vary and patients may present with several non-specific symptoms associated with elevated intracranial pressure or tumor location. These include headache, nausea, vomiting, dizziness, gait disturbance, hemiplegia, hearing loss and memory disturbance (26).

Sugiyama *et al* (27) reported on an 86-year-old male with SFT, which was located in the right parietal lobe and invaded the parietal bone, who presented with sustained progressive motor weakness in the left lower extremity for 1 month. Another study reported on a 30-year-old male with SFT, which was

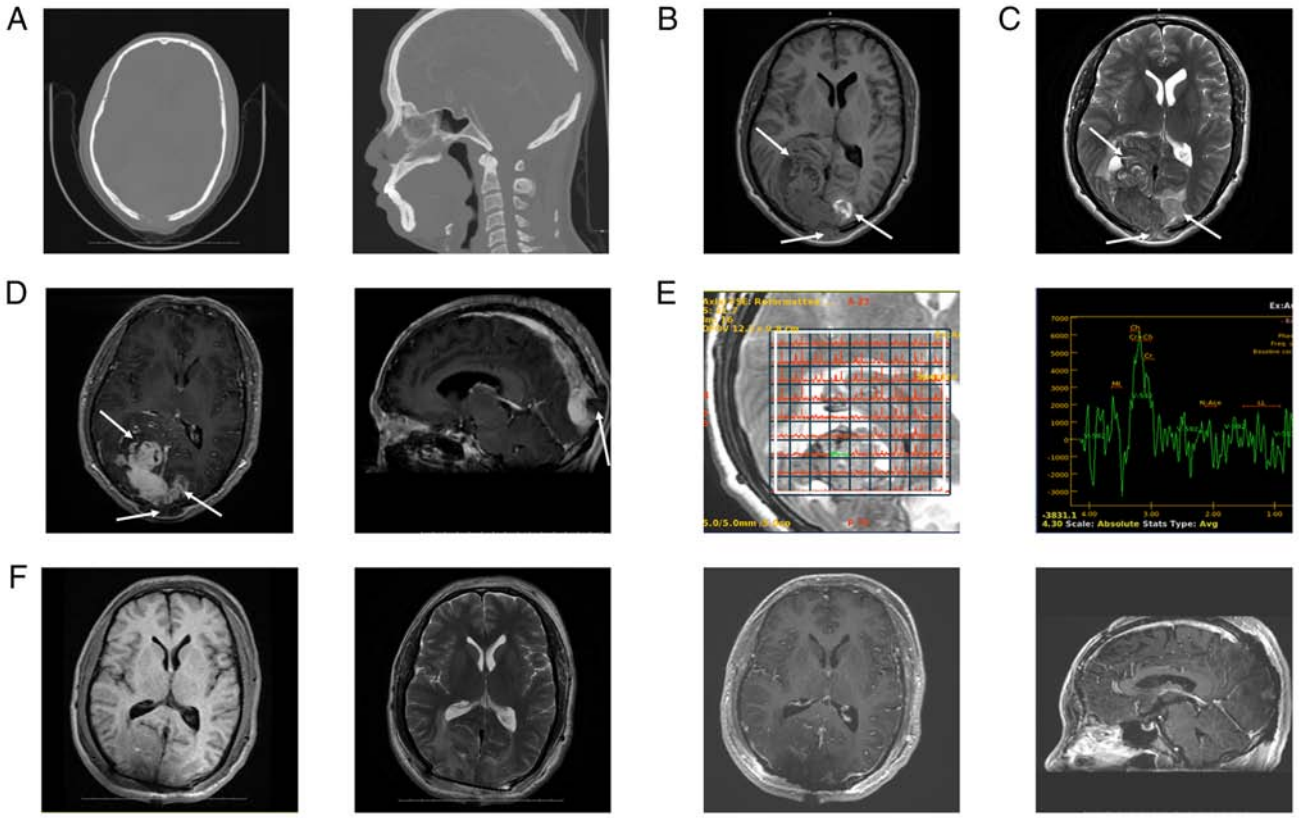

Figure 1. Preoperative and postoperative imaging profiles of the tumor. (A) The axial (left panel) and sagittal (right panel) images of the bone window of the head computed tomography angiogram demonstrated that the mass invaded and penetrated the skull. MRI on admission demonstrated a mass shadow near the occipital cerebral falx, with an irregular shape and unclear boundaries. The mass was ~64x44x64 mm in size. (B) The majority of the intracranial and subcutaneous tumors demonstrated uneven low signals on T1WI, while the tumors at the far end of the left occipital lobe demonstrated high signals on T1WI. (C) The majority of the intracranial tumors demonstrated low mixed signals on T2WI, while the tumors at the far end of the left occipital lobe and under the scalp demonstrated high signals on T2WI. (D) The axial (left panel) demonstrated that the tumor crossed both sides of the cerebral falx. There were unclear boundaries with the bilateral occipital lobe brain tissue. The majority of intracranial tumors and the tumors at the far end of the left occipital lobe were significantly enhanced following enhanced scanning, while the tumors under the scalp were not significantly enhanced following enhanced scanning. The sagittal (right panel) images of the contrast-enhanced MRI demonstrated that tumors filled the posterior section of the superior sagittal sinus to form a filling defect, invading and penetrating the occipital bone. (E) The voxel (left panel) and corresponding magnetic resonance spectroscopy map (right panel) demonstrated no notable N-acetylaspartic acid peaks in the mass collected by multivoxel, elevated choline/creatine peaks in the solid area, and no notable abnormalities in the spectral lines of tissues around the mass. (F) Postoperative MRI demonstrated that the tumor was completely removed. The bilateral occipital lobes surrounding the surgical area demonstrated T1WI low signal (left panel) and T2WI high signal (second left panel), and the axial (second right panel) and sagittal (right panel) images of enhanced scanning demonstrated obvious enhancement along the edges of the surgical area. White arrows indicate the tumor. MRI, magnetic resonance imaging; T1W1, T1-weighted imaging; T2W1, T2-weighted imaging.

located near the right temporal lobe and led to the thickening of the temporal bone of its neighbor; the patient developed left facial nerve paralysis and dysarthria, and decreased muscle strength of the left upper and lower limbs (28). SFT in the present case reported was located in the parietal occipital area and invaded and penetrated the skull and the patient presented with headache, dizziness and blurred vision. Headache and dizziness are mainly caused by increased intracranial pressure. Blurred vision is caused by a tumor pressing on the visual center. The patient of the present study had no symptoms of limb weakness, facial nerve paralysis or dysarthria.

The differential diagnosis of SFT via imaging is difficult due to its variable signal intensity on MRI scans (29). Differentiation from meningioma, schwannoma, neurofibroma, metastases and lymphoma was required (30-33).

Computed tomography and MRI are important imaging techniques for the diagnosis of SFT. The medical imaging of intracranial SFT reveals numerous characteristics and previous imaging revealed that intracranial SFT is more likely to occur at the base of the skull (34), sagittal sinus (35), falx cerebri and peritentorium cerebelli (36), or near the venous sinus (37). In addition, intracranial SFT is characterized by extracranial tumors, which are lobular or irregular, and some may appear oval- or dumbbell-shaped (38-40). Previous CT scans demonstrated high or equal density. The majority of boundaries were clear; however, a small number of the boundaries with the brain tissue were not clear (27,28,40,41). Cystic degeneration and necrosis in the tumor were common, but there was no calcification (27,28,40,41). The density of the tumor following necrosis and cystic degeneration was uneven and destruction of the skull adjacent to the tumor may occur (27,28,40,41). In general, SFT appears as isointense to slightly high on T1WI and isointense on T2WI, compared with gray matter. T1WI demonstrated isointense to slightly high signals (27,28,36) and isointense mixed signals (36) in the case of cystic degeneration and necrosis. In addition, T2WI demonstrated slightly high or isointense mixed signals (27,36), and isointense mixed signals in the case of

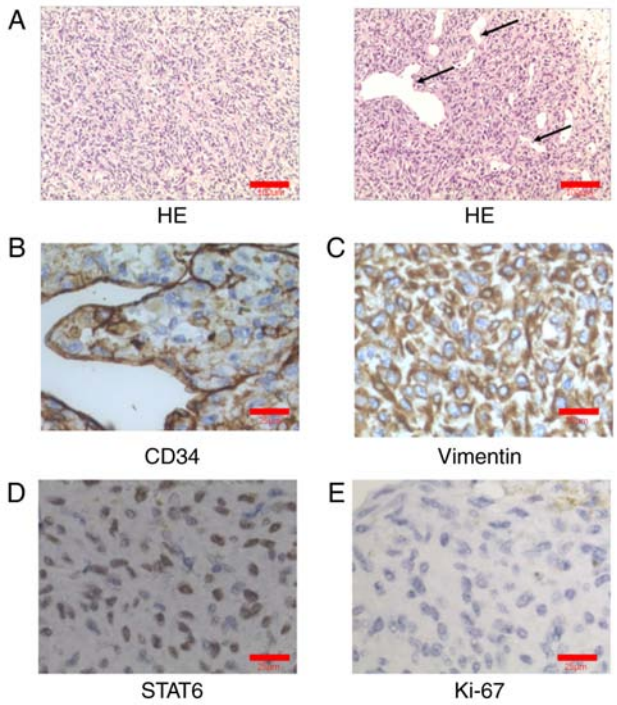

Figure 2. Histopathological and immunohistochemical features of the tumor. (A) The tumor was composed of alternately distributed cell-rich areas and cell-sparse areas (left panel). The tumor cells in the cell-rich area were short spindle- or oval-shaped, with little cytoplasm and uniform nuclear chromatin (left panel). There was no notable atypia in the two areas (left panel). The frequency of mitotic figures was 1/10 high-power fields and the tumor cells were arranged in sheets and striae (left panel). These were hemangiopericytoma-like with abundant blood vessels in the tumor (right panel). Thus, these were labeled 'staghorn-shaped blood vessels', indicated using black arrows (magnification, x100; scale bar, 100 μm; H&E staining). Immunohistochemical examination demonstrated strong expression of (B) CD34, (C) vimentin and (D) STAT6. (E) The Ki-67 labeling index was ~10% (magnification, x400; scale bar, 25 μm). A brown color in the cells indicated positive staining for CD34, vimentin, STAT6 and Ki-67. STAT6, signal transducer and activator of transcription 6.

cystic degeneration and necrosis (36). Following enhanced MRI scanning, the tumor appeared significantly strengthened, and those with cystic degeneration demonstrated heterogeneous enhancement (36,38,40,42-44). Peritumoral edema is often mild (41,44,45).

The imaging findings of SFT were similar to those of meningioma and MRS may be used to distinguish SFT from meningioma. The relative ratios of choline and myo-inositol are increased in SFT compared with meningioma (40,46). Chen *et al* (47) also reported that the normalized apparent diffusion coefficient ratios and intratumoral susceptibility signal intensity are useful for differentiating SFT/HPC from meningioma. In the present case reported, SFT occurred near the cerebral falx. STF was irregular, exhibited unclear boundaries with the occipital lobe brain tissue, displayed notable enhancements in the intracranial section following enhanced scanning and exhibited an elevated choline peak in the MRS analysis, which was consistent with previous reports (40,46). SFT in the present case reported was located in the distal part of the left occipital lobe, demonstrated high signals on T1WI and T2WI, and was significantly enhanced following enhanced scanning. These results were also consistent with those previously reported (27,28,36,38,40,42-44).

The majority of the intracranial tumors in the patient in the present report demonstrated uneven low signal intensity on T1WI and low mixed signal intensity on T2WI, which differed from the results obtained from previous reports. Due to the intraoperative situation, it was hypothesized that the tumor demonstrated low signal intensity on T1WI and T2WI due to intra-tumor hemorrhage. SFT may cause skull destruction when adjacent to the skull, which is manifested as hyperostosis, bone erosion or bone destruction (27,28,40,41,48). To the best of our knowledge, this is the first case of SFT that completely penetrated the skull. The SFT signal penetrating the skull under the scalp demonstrated a low signal on T1WI and a high signal on T2WI, and no notable enhancements were observed following enhanced scanning. There was no notable enhancement of SFT under the scalp following enhanced scanning, which was also inconsistent with the results obtained from previous reports (36,38,40,42-44). Thus, the tumor was considered heterogeneous. As tumor cells were dense, the interstitial components were relatively sparse with few vascular components. Of note, the sub-scalp tumor was not enhanced in the conventional enhancement time window. Thus, SFT under the scalp may require delayed enhancement for accurate development. In addition, for SFT located inside and outside the skull, and under the scalp, dynamic enhancement of multiple time windows is required in MRI to fully display the scope and nature of the tumor, and to avoid miscalculation.

The diagnosis of SFT mainly relies on pathological examination. Histological staining demonstrates that the tumor tissue is rich in spindle-shaped or polygonal cells. In typical cases, a large number of 'staghorn-shaped' blood vessels and collagen fibers may be observed. The tumor cells are arranged in concentric circles around the blood vessels, and these may form dense or sparse areas (49). IHC staining demonstrated that CD34, vimentin and STAT6 are positive in SFT tissues, and the Ki-67 proliferation index is frequently indicative of patient prognosis (50). Various studies recommended that high Ki-67 (>5%) should be included as an adverse prognostic parameter in assessing the prognosis of SFT of the CNS (7,51). At present, CD34 is considered the most consistent marker in SFT and positive staining is reported in 95-100% of patients; however, its absence does not rule out this tumor (52,53). STAT6 is positive in almost all patients with intracranial SFT (22,54). STAT6 may be associated with the fusion of the NAB2-STAT6 gene caused by 12q chromosome rearrangement (25). Thus, detection of STAT6 or the NAB2-STAT6 fusion gene is recommended for the diagnosis of intracranial SFT (54-56). NAB2 and STAT6 are neighbour genes localized on the long arm of chromosome 12 and transcribed in opposite directions (57). In SFT, an intrachromosomal inversion places the genes in the same orientation, which results in an in-frame fusion transcribed from the NAB2 promoter, leading to STAT6 nuclear expression that may be detected by IHC (14,57). The expression of STAT6 in intracranial SFT tissue was detected using IHC staining, and the NAB2-STAT6 fusion gene was accurately detected with both high specificity and sensitivity (54-56). STAT6 IHC 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 (20). In the present study,

STAT6 expression was detected by IHC instead of detecting the NAB2-STAT6 fusion gene. Different NAB2-STAT6 fusion variants may be related to clinical pathology and prognosis (12,58-60). Therefore, the lack of NAB2-STAT6 fusion gene detection was a possible limitation of the present report. The SFT tissue of the patient described in the present study was positive for CD34, vimentin and STAT6, which was consistent with the results of previous reports (50,52-54). The patient experienced no tumor recurrence following surgery.

SFT is characterized by high rates of local and extracranial metastases (61). Results of previous studies demonstrated that in patients with SFT for a prolonged period, there is a risk of recurrence, even after 10 years of the initial resection (62-64). Therefore, patients with SFT require active treatment and long-term follow-up. As the tumor described in the present study is rare, treatment and prognosis require further investigation.

Yu *et al* (65) retrospectively studied patients treated for intracranial SFT between January 2009 and June 2019. Their results demonstrated reduced WHO grading, and patients who underwent gross total resection and adjuvant therapy, such as Gamma Knife surgery, exhibited prolonged progression-free survival (PFS) (65). Of note, the aforementioned previous study was retrospective in nature, with a small sample size and selection bias, leading to biased results. Results of a multi-center study demonstrated that postoperative radiotherapy, including 2-dimensional conventional radiotherapy, 3-dimensional conformal radiotherapy and intensity-modulated radiotherapy, may significantly improve the PFS of patients with SFT, irrespective of the surgical extent and grade (61). Of note, the present study did not investigate the effects of different radiotherapy techniques on SFT. At present, there are no standardized treatment guidelines for intracranial malignant SFT. Surgical resection and postoperative radiotherapy are not effective in the treatment of intracranial malignant SFT. Anlotinib, a newly multitargeted tyrosine kinase inhibitor with anti-neoplastic and anti-angiogenic activities, inhibits tumor angiogenesis and proliferation (66). Anti-angiogenesis may be a potential option for the treatment of SFT (67-69). Surgery, radiotherapy and anlotinib alone are effective in the treatment of malignant intracranial SFT (13). However, the present article reports one case and further research and larger randomized controlled trials are required to verify its findings. Pazopanib, a multi-target receptor tyrosine kinase inhibitor with potent anti-angiogenic properties, is approved for the treatment of advanced renal cell carcinoma and certain subtypes of advanced soft tissue sarcoma (70). Of note, pazopanib is effective in treating patients with metastatic or unresectable SFT (69,71). The present study demonstrated that surgical resection is the optimal choice for the treatment of SFT, and postoperative radiotherapy may significantly improve PFS in patients. Molecular targeted therapy, such as tyrosine kinase inhibitors anlotinib and pazopanib, is a promising approach for malignant, unresectable or metastatic SFT.

In conclusion, SFT is a rare tumor type. Due to the rarity and similarity to other more common brain tumors, SFTs exhibit a high rate of misdiagnosis following imaging. Of note, histopathological testing is critical for differentiating SFT from other CNS disorders. In addition, complete tumor resection is the preferred treatment option for SFT. The indications for adjuvant therapy following surgery remain to be elucidated. Due to the potential for recurrence, rigorous long-term follow-up, including periodic imaging surveillance, is recommended.

Acknowledgements

Not applicable.

Funding

No funding was received.

Availability of data and materials

The datasets used and/or analyzed during the present study are available from the corresponding author on reasonable request.

Authors' contributions

QL and XZ designed the study and drafted the manuscript. JZ collected and analyzed the clinical data. XZ critically revised the manuscript. All authors have read and approved the final manuscript. QL and XZ confirm the authenticity of all the raw data.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the patient for publication of the present manuscript, including the medical data and any accompanying images.

Competing interests

The authors declare that they have no competing interests.

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