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Fundamental Neurosurgery

"Hitting all the right markers to save a life" Solitary fibrous tumors of the central nervous system: Case series and review of the literature

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Abstract

Background: Solitary fibrous tumors (SFTs) of the central nervous system are uncommon. Their biological features remain largely unknown; hence, the clinical management and prognosis is often challenging due to the lack of comprehensive data. For this reason, we present two cases of large SFTs to illustrate a comprehensive review.

Methods: This was a retrospective analysis of two patients: a 65-year-old male with a left parietooccipital lesion and a 70-year-old female with a right parietal convexity mass.

Results: Gross total resection was performed in the male patient with no recurrence 30 months after resection. The second patient received stereotactic radiosurgery for what was initially thought to be a parafalcine meningioma; however, continued growth 1 year later prompted an open resection, with pathology indicative of an SFT. The tumor recurred the following year requiring repeat resection. Unfortunately, due to the aggressive nature of the lesion, the patient eventually succumbed to tumor burden a year later.

Conclusion: Based on the literature review, the sometimes observed aggressive growth pattern, and also, the potential for malignant transformation, we recommend complete resection of SFTs with close sequential follow-up.

Key Words: Solitary fibrous tumor, menigeoma, immunohistochemistry, pathology, treatment

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INTRODUCTION

Solitary fibrous tumors (SFTs) are uncommon spindle cell tumors of mesenchymal origin, and were initially described as primary neoplasms of the mediastinum and visceral pleura.[36] The tumorigenesis remained debatable, and later, the tumor entity was carefully reexamined and further characterized as fascicles of spindle cells,

resembling CD34-positive interstitial dendritic cells intermingled with bands of collagen.[8,18,22,74] There have been more than 800 cases of pleural SFTs described in the literature with a peak incidence in the sixth and seventh decades of life found equally frequent among men and women[19] or with a slight preponderance among the female population.[72] However, they have also been reported under various other labels[14] in a large number of extrathoracic body sites, including head and neck, pericardium, peritoneum, liver, thyroid, mesentery, as well as the sinus and orbit[17,21,25,28,61,74,80,81] but only rarely in the central nervous system.

EPIDEMIOLOGY

SFTs of the central nervous system (CNS) were first classified as a distinct pathological entity in 1996[11] and are categorized as a rare mesenchymal, nonmeningothelial tumor of the CNS. To date, fewer than 100 SFTs have been reported in the cranial and spinal compartments[9,12,13,15,20,82] and the fact that this tumor is different from other benign and malignant spindle cell tumor entities remains largely unknown to most neurosurgeons.[30]

SFTs in the CNS are most often dura-based neoplasms and can occur in any location, with reports in the supratentorium,[66,67,71] parenchyma,[13] sella,[23] ventricle,[6,16,37,69,75] cerebellopontine angle,[5] orbit/paranasal sinuses,[78,80] tentorium,[65] posterior fossa meninges,[27,64] along cranial nerves V[48] and VI,[77] at the foramen of Monroi,[35] and in the infratemporal fossa.[62,68] There is a tendency to manifest in the posterior fossa and spine as well as along spinal nerve rootlets.[2,5,7,12,31,32,38,41,56,58,60,76]

It is of interest that most intracranial SFTs seem to be dura-based, whereas two-thirds of spinal SFTs lack a dural attachment.[42,47,54] They represent an entity that is clinically distinct from other mesenchymal extracranial soft tissue tumors,[3,11,73] often leading to an unusual clinicopathological presentation and outcome pattern.[1,23,34,55,75] Both benign and aggressive forms have been described, as well as a potential toward malignant transformation.[82] Intracranial SFTs occur across all ages, with one recent review of 60 cases reporting an age range of 11–73 years, the majority being meningeal with near equal gender distribution and the median age of occurrence of 47.6 years.[12]

RADIOGRAPHIC APPEARANCE

Based on standard CT and MR imaging (MRI) sequences, SFTs appear as heterogenous, hyperattenuated masses compared to adjacent brain parenchyma on noncontrast CT studies resembling meningiomas or hemangiopericytomas, with the possible erosion of the overlying skull but usually with a sharp demarcation toward the surrounding parenchyma and vivid contrast enhancement.[48] Resemblance to meningiomas and hemangiopericytomas also holds true intraoperatively.[4,39] SFTs are isointense on T1-weighted and mixed to low signal intensity on T2-weighted images with marked heterogenous enhancement.[16,40,79] They demonstrate restricted diffusion with an elevated peak of myoinositol on PET imaging.[16]

PATHOLOGY

Histological features of SFTs include monomorphic spindle cells arranged in a patternless architecture or arranged in straight, curving, or undulating fascicles; prominent collagenous bands; branching vascular channels with thin walls; lack of other architectural features such as well-formed lobules, whorls, or psammoma bodies, which help to distinguish SFTs from meningiomas.[11,44,57]

CLINICAL OUTCOMES

Although outcome data are very limited, the extent of resection seems to be the most important prognostic factor.[45,73] Invasion or delayed seeding of the CSF space can occur even in the setting of seemingly benign SFTs.

To raise the awareness of challenges posed to the neurosurgeon in managing this distinct tumor entity, we present two representative cases of large SFTs and discuss their imaging and histological findings, and also, review the literature in regard to treatment and prognosis.

RESULTS

Case 1–65-year-old male with a left parietooccipital lesion

This patient presented to our clinic in October 2009 with visual disturbances and word-finding difficulties for several months. Physical exam revealed a right homonymous hemianopsia.

Preoperative imaging

MRI showed a multilobulated, heterogeneous contrast-enhancing lesion in the left parietooccipital lobe measuring 4 cm in diameter [Figure 1a] abutting the superior aspect of the tentorium [Figure 1b and c]. The mass appeared to be predominantly within the occipital horn of the left lateral ventricle [Figure 1d]; however, we felt that there was also the possibility that the lesion was dura based and simply compressing the ventricle. Given the imaging characteristics, a provisional diagnosis of a meningioma was made.

Management

The patient was counseled extensively and given the size, localization, and symptomatic nature of the lesion, surgery was recommended. An image-guided, left-

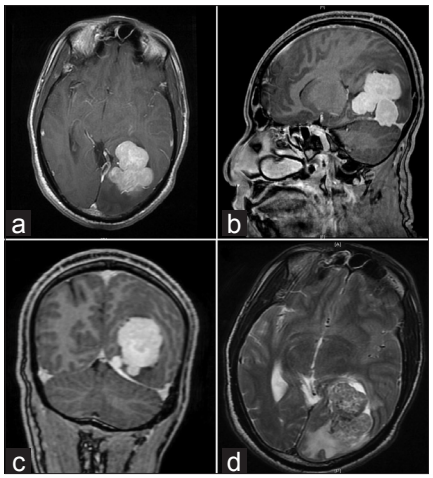

Figure 1: Preoperative MRI of a solitary fibrous tumor involving the occipital horn of the left lateral ventricle also infiltrating the superior aspect of the tentorium cerebelli. (a) Axial, (b) sagittal, and (c) coronal T1-weighted imaging shows a multifocal tumor with uniform gadolinium enhancement. (d) The tumor is seen to invade the left occipital horn on T2-weighted imaging

sided parietooccipital craniotomy was performed with intraoperative histopathology suggestive of an aggressive malignant neoplasm in keeping with a malignant meningioma, ependymoma, or sarcoma. A gross total resection (GTR) was achieved with the removal of the intraventricular portions of the lesion along with its attachments to the choroid plexus, as well as complete resection of the inferior aspect abutting the tentorium.

Histopathology

Final histology revealed a solid tumor which composed of a dense population of spindle cells with thick fascicles of collagen and scattered infrequent vasculature [Figure 2a]. The trichrome stain confirmed the dense collagenous background [Figure 2b] with dense CD34 and BCL-2 but patchy Desmin staining [Figure 2c and d, respectively]. The tumor was negative for EMA, S-100, and HMB-45, thus ruling out a meningioma [Figure 2e and f]. The MIB-1 index came back as 3%–5% of tumor cells [Figure 2g]. The microvasculature was positive for CD-31 but not in the tumor cells proper (results not shown). Factor XIIIa was negative. The staining pattern was consistent with that of a solitary fibrous tumor. This was corroborated on electron microscopy showing tumor cells abundant with rough endoplasmic reticulum [Figure 2h, arrow] surrounded by dense parallel collagen fibrils [Figure 2h, asterisk].

Clinical outcome

The patient tolerated the surgery well and had no new focal deficits. As we were confident that we had achieved a GTR and the patient remained seizure free with no new symptoms, he chose not to pursue adjuvant radiotherapy and opted for expectant management. Repeat imaging at 30 months demonstrated no evidence of disease recurrence [Figure 3].

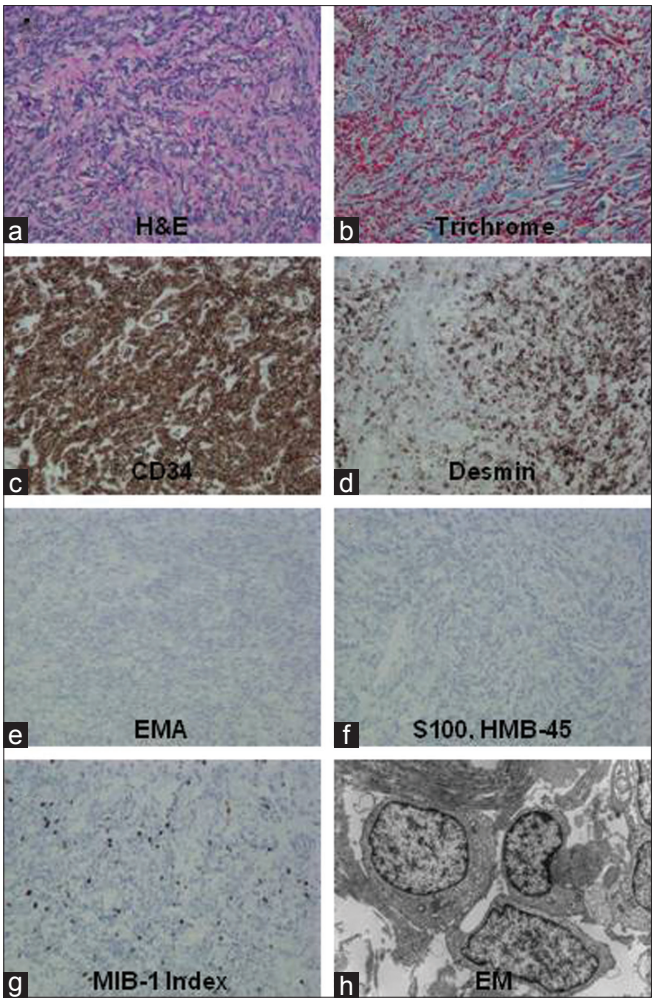

Figure 2: Histology of a solitary fibrous tumor. (a) H and E staining demonstrates a dense fibrous background with spindle-like cells. (b) Trichrome stain brings out the collagenous background. (c) Microvasculature is CD34 positive but in the tumor cells, (d) desmin shows patchy positivity. Cells are negative for (e) EMA and (f) S-100 and HMB-45 with (g) a moderate MIB-1 index of 3%–5%. (h) Electron microscopy shows abundance of rough endoplasmic reticulum (arrow) surrounded by dense collagen fibrils (*). No basement membrane is visible

Case 2 – 70-year-old female with a right parietal convexity lesion

This patient presented initially in 2004 with a several-year history of headaches, intermittent double vision, and occasional numbness and weakness in her left leg leading to multiple falls. Her exam was significant for horizontal diplopia, and bilateral decreased sensation to pinprick and vibration in a glove-stocking distribution.

Initial imaging

MRI of the brain showed a uniformly enhancing parafalcine lesion with a predominantly right-sided component with no associated peritumoral edema [Figure 4a and b], suggestive of a meningioma.

Initial management

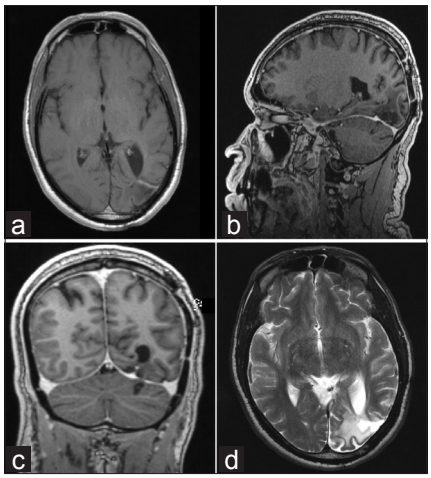

Figure 3: Postoperative imaging at 2 years following a gross total resection shows absence of disease recurrence. (a) Axial, (b) sagittal, and (c) coronal T1-weighted postcontrast scans as along with (d) axial T2-weighted imaging show no tumor recurrence

Given the assumption that this was a slow-growing meningioma of the falx with partial occlusion of the sinus compounded with risks of perioperative morbidity and impairment, the patient opted for stereotactic CyberKnife radiosurgery in November 2005, receiving five fractions of 600 cGy each for a total of 3000 cGy to the 84% idodose line. The patient was initially maintained on dexamethasone but was weaned off in February 2006 due to weakness from steroid myopathy. By April 2006, she was still having persistent bilateral leg weakness, left worse than right. Repeat MRI of the brain showed further enlargement of the mass with peritumoral edema despite radiotherapy [Figure 4c and d]. Given the lack of response to radiation and worsening of the neurologic function, the patient consented to surgical resection, receiving a bifrontal craniotomy with a wide excision of the dura, leaving only parts that had infiltrated the superior sagittal sinus and convexity veins [Figure 4e and f].

Histopathology

Staining of the specimen showed a solid tumor with prominent nucleoli, sheeting, and nuclear pleomorphism. Immunoperoxidase stains showed no reactivity to AE1, AE3, CAM5.2, or CD34, thus excluding metastasis, meningioma, and SFT. EMA staining was only focally and very weakly positive. The initial diagnosis was an atypical meningioma; however, dispersed among the tumor were features consistent with a spindle cell neoplasm such as SFT, interrupted by areas resembling transitional meningioma-type histology. Because the tumor had previously been treated with radiation, it was felt that it could no longer be graded by formal WHO criteria.

Clinical course

The patient returned in October 2007 with a sudden onset of right-sided weakness as well as increasing frequency of

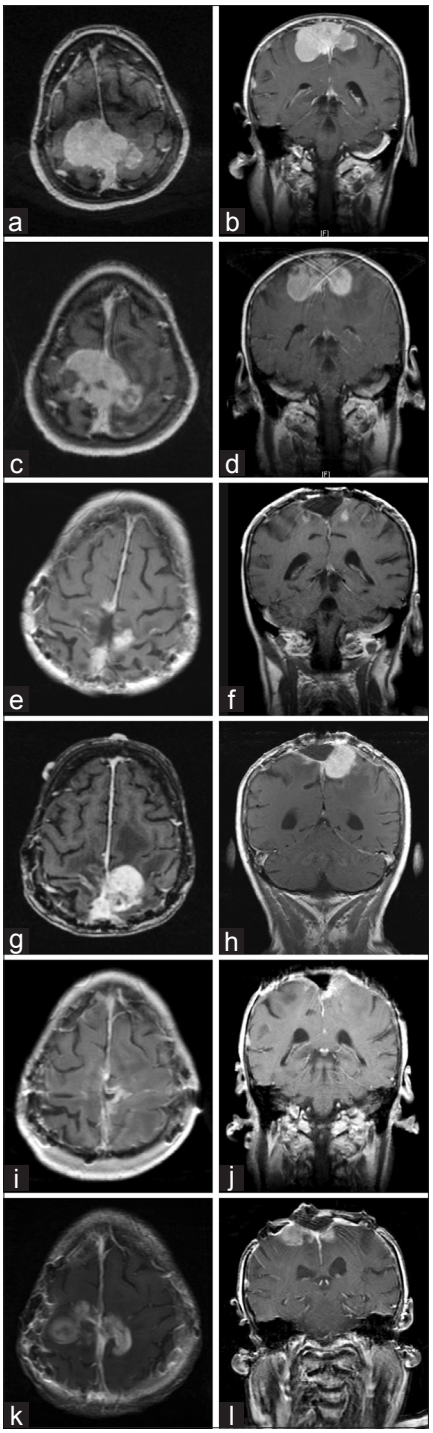

Figure 4: Parafalcine convexity SFT in a 70-year-old female with malignant transformation following a course of radiotherapy. All images are T1-weighted postcontrast scans. A large, enhancing parafalcine dura-based lesion without surrounding edema seen on (a) axial and (b) coronal slices, initially thought to be a slow-growing meningioma, treated with a total radiotherapy dose of 3000 cGy. (c and d) Tumor enlargement despite radiotherapy. Postoperative imaging shows subtotal resection with residual tumor left attached to the superior sagittal sinus (e and f). Tumor recurrence 1 year following initial resection predominantly on the left side of the falx (g and h). Repeat resection shows adequate decompression with some left-sided residual tumor (i and j). Malignant transformation with recurrence on both sides of the falx 1 year following repeat resection (k and l)

seizures. A repeat MRI revealed an enlarging recurrent tumor on the left side posterior to the resection bed and abutting the falx, causing a significant mass effect as well as edema [Figure 4g and h]. The patient consented for a second resection and underwent a bifrontal redo craniotomy with image guidance for resection. She tolerated the procedure well with no complications and had a good surgical result [Figure 4i and j].

Histopathology

Tissue examination of this recurrent tumor revealed dense bands of thick collagen interspersed with fascicles of spindle cells. Immunohistochemistry revealed positive staining with CD34 in regions of increased cellularity. EMA and cytokeratin cocktail showed high-background staining in brain tissue. GFAP, S100, and synaptophysin highlighted areas of entrapped and infiltrated brain parenchyma. MIB-1 revealed many proliferating cells, but LCA and CD68 revealed extensive infiltration of the tumor with lymphocytes and macrophages which likely accounted for the majority of proliferating cells. Actin highlighted smooth muscles in vessels, but was largely negative in the tumor. Trichrome stain revealed extensive collagen deposition. Reticulin stain revealed a fine network of reticulin outlining all neoplastic cells. These findings were diagnostic for a solitary fibrous tumor. Additional findings of cellular atypia in the neoplastic spindle cell population with features of brain invasion suggested malignant sarcomatous transformation of the tumor.

Postoperative course

In follow-up, the patient was seen to have improved strength in her right leg and decreased frequency of her seizures. She was able to stand and be mobilized again and was transferred to rehabilitation. Postoperative imaging demonstrated an adequate repeat resection [Figure 4i and j]. Unfortunately, she presented in April 2008 again with progressive weakness and lethargy. MRI at this time showed bilateral parafalcine masses at the site of surgical resection, consistent with a recurring tumor [Figure 4k and l]. The patient and her family were resistant to the idea of having repeat surgery or further radio- or chemotherapy and the decision was made to provide palliative care for the patient, who expired a few months later in a nursing facility.

DISCUSSION

SFTs bear clinical and radiographical resemblance to both fibrous meningiomas and hemangiopericytomas. They therefore require diligent work-up to reveal all histological and immunohistochemical features to distinguish between these rare but distinct CNS tumors and possible differentials.[9,11,24,29,33,73,81] Immunohistochemically, SFTs are in most cases diffusely positive for CD34,[57,74,80] while meningiomas are typically reactive for EMA. However, CD34 is not specific for SFTs, as weak and usually patchy staining may be visualized in meningiomas, neurofibromas, and hemangiopericytomas.[17,42,70] Positivity for CD99 and bcl-2 is found in more than half of all cases of SFTs[26,44] and they also stain strongly with the intermediate filament vimentin, but are usually negative for the neural crest markers S-100, GFAP, EMA, cytokeratin, or vascular antigens.[43] Chromosomal imbalances have been investigated via comparative genomic hybridization, and multiple sites of allelic losses and gains were identified, but no single pathognomonic underlying feature has been found thus far. Electron microscopy has not yielded a unique distinctive feature, but some typical aspects include a well-developed rough endoplasmatic reticulum, occasional primitive junctions, and a lack of desmosomes as well as basal lamina. For these reasons, immunocytochemistry is essential in making the primary diagnosis, when a complex differential diagnosis is entertained.

Treatment and outcomes

Although an infrequently encountered tumor entity, it is important to raise awareness of SFTs in neurosurgeons and neuro-oncologists alike as a distinct entity in the differential diagnosis of CNS tumors. In principle, SFTs should be carefully considered in suspicious cases when entertaining other benign differential diagnoses, including hemangiopericytomas, fibrous meningiomas, schwannomas, neurofibromas, and less favorable entities such as fibrosarcomas.[10,12,39,52,53]

The prognosis of SFTs remains yet to be fully elucidated since follow-up data of the few reported cases are limited, however, it is believed that these tumors generally pursue a slow, indolent, and nonaggressive course. As is illustrated in our first case, surgery offers the best first-line treatment and achieves excellent local control.[45] Recurrence (as in our second case), malignant transformation,[49] or cerebrospinal fluid dissemination has also been described, though seemingly not as frequently.[12,44,46,51,54,59]

The only 2 available larger series of 18 cases each of solitary fibrous tumors in the central nervous system[45,73] were carefully analyzed and in the first study, the reported 5-year survival rate was 100%, with only 3 of the 18 tumors recurring during the follow-up period[73] – as could be observed in our second case illustration. This differs slightly from the second series in which the median follow-up was only 45 months.[45] Here, 15 of 18 patients were alive at the time of the report, but a significantly higher portion (50%) had suffered a recurrence that required further treatment in 9 patients. Only one of the SFTs in the first above-mentioned review revealed anaplastic histological features and the significance of this is still unclear.[73] A GTR has been possible for most CNS SFTs reported in the literature and it appears that surgery alone is the appropriate initial management for

most SFTs.[12,45] In these cases, meticulous and complete resection, rather than histological grading, is believed to be the most important prognostic factor and may preclude downstream malignant behavior.[42]

Conventional radiotherapy as well as stereotactic radiosurgery (SRS) has been described in occasional reports as adjuvant treatment for residual SFTs.[50] However, the role of adjuvant postoperative radiotherapy in improving long-term prognosis remains unclear and the number of patients who underwent complementary chemotherapy treatment is too small to evaluate any possible benefit.[45] A recent study was published recently reporting two cases that yielded reasonable treatment outcomes using Gamma Knife radiosurgery (GKRS) in patients who had recurrence of their intracranial SFTs following subtotal resection.[63] In the first case, the patient was referred for GKRS following a subtotal resection for recurrence with stable shrinking of the tumor at 20 months after treatment. The second case presented with multiple tumors that recurred following seven surgeries in the posterior fossa. Initial treatment with GKRS demonstrated effective local tumor control at 13 months; however, an out-of-field recurrence prompted repeat treatment at 15 months. This novel case series suggests that GKRS is a feasible adjunct for treating SFT; however, we still recommend close and indefinite follow-up for all patients.

CONCLUSION

SFTs of the CNS are rare entities that are challenging to manage. Higher powered prospective studies are needed to delineate the best management options, to further characterize the benefits of additional radio/chemotherapies, and also define the necessary duration of follow-up imaging in this patient population. Since the potential for malignant transformation exists, we recommend diligent long-term follow-up including regular imaging surveillance.

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