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Case Report

Intracranial Solitary Fibrous Tumour Masquerading as Recurrent Meningioma in Previously Irradiated Field: A Diagnostic Pitfall

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Abstract: Intracranial solitary fibrous tumours are rare mesenchymal neoplasms that may closely resemble meningiomas clinically and radiologically, leading to diagnostic difficulty, particularly in previously treated dural-based lesions. We report the case of a 64-year-old woman who had undergone gross total resection and adjuvant radiotherapy for a right parasagittal meningioma in 2016 and presented after a prolonged interval with involuntary micturition, stool evacuation, and left lower-limb weakness. Contrast-enhanced MRI demonstrated a heterogeneously enhancing right frontal parasagittal lesion, initially suggestive of recurrent meningioma. The patient underwent re-exploration and gross tumour resection. Histopathology revealed a spindle-cell neoplasm with staghorn-shaped vascular channels, and immunohistochemistry showed diffuse positivity for CD34, vimentin, and STAT6, with low Ki-67 proliferative activity and negative GFAP, confirming the diagnosis of solitary fibrous tumour. Postoperative imaging showed no residual disease, and the patient remains on regular follow-up. This case highlights the diagnostic pitfall of assuming that delayed lesions arising in a previously operated and irradiated meningioma field represent recurrence. The long interval may reflect an originally misclassified tumour, delayed manifestation of an indolent SFT, or a second primary lesion in the treated field. STAT6 immunohistochemistry is essential for accurate diagnosis, and contemporary re-evaluation should be considered in atypical or recurrent meningioma-like lesions. Long-term surveillance is warranted because of the known recurrence potential of intracranial solitary fibrous tumours.

Keywords: Intracranial, meningioma, Solitary fibrous tumor (SFT), STAT 6

INTRODUCTION

Primary intracranial solitary fibrous tumors (ISFTs) are uncommon mesenchymal tumors initially described by Carneiro et al. in 1996. ISFTs comprise only 0.09% of meningeal tumors. The prognosis, cytogenetics, histogenesis, and clinical course of ISFTs are still mostly up for debate and are a matter of concern for oncologists worldwide. SFT, believed to originate from the fibroblast, must be distinguished from certain tumors, such as fibrous meningioma, as well as from myxoid forms.[1] World Health Organization (WHO) 2021 classification has categorized this as a separate entity under the mesenchymal/non-meningothelial tumors.[2] The majority of these are dural-based and may arise in different areas, such as the falx cerebri, tentorium cerebelli, ventricles, cerebellopontine angle, and suprasellar region.[3] The present case is noteworthy because the lesion appeared after a prolonged interval in the same previously operated and irradiated territory of a tumour originally diagnosed as meningioma. Such a presentation creates an important diagnostic dilemma, as delayed dural-based lesions are often presumed to represent recurrent meningioma on imaging. However, the possibility of an originally misclassified SFT, delayed manifestation of an indolent tumour, or a second primary lesion in a previously treated field should also be considered.

CASE REPORT

A 64 year old lady, a followed-up patient of right anterior 1/3rd para sagittal meningioma previously operated (gross tumor resection) in 2016 and had received adjuvant radiotherapy (RT), presented to our institute with complaints of involuntary micturition and stool evacuation associated with weakness of left lower limb since 4 months. Contrast enhanced magnetic resonance imaging (CE-MRI) brain revealed a 3.4 x 8 x 4 cm enhancing lesion in the right frontal para-sagittal region, which was hyperintense on T1 and hetero- intense on T2 (Figure 1a and 1b), suggestive of a recurrent meningioma for which she underwent re-

exploration and gross tumor resection. Tumor appeared as a cellular tumor arranged in diffuse sheets suggestive of anaplastic meningioma or solitary fibrous tumor (SFT). Further, immunohistochemistry (IHC) markers applied on post-operative histopathology specimen were positive for CD34 (cluster of differentiation 34), Vimentin and STAT6 (Signal Transducer and Activator of Transcription 6), suggestive of solitary fibrous tumor with 1-2% Ki67 and negative for Glial fibrillary acidic protein (GFAP) (Figure 2). Post-op CE-MRI brain done was suggestive of no evidence of disease. Currently, the patient has an ECOG (Eastern Cooperative Oncology Group) 2 and is on regular follow-up.

Figure Legends

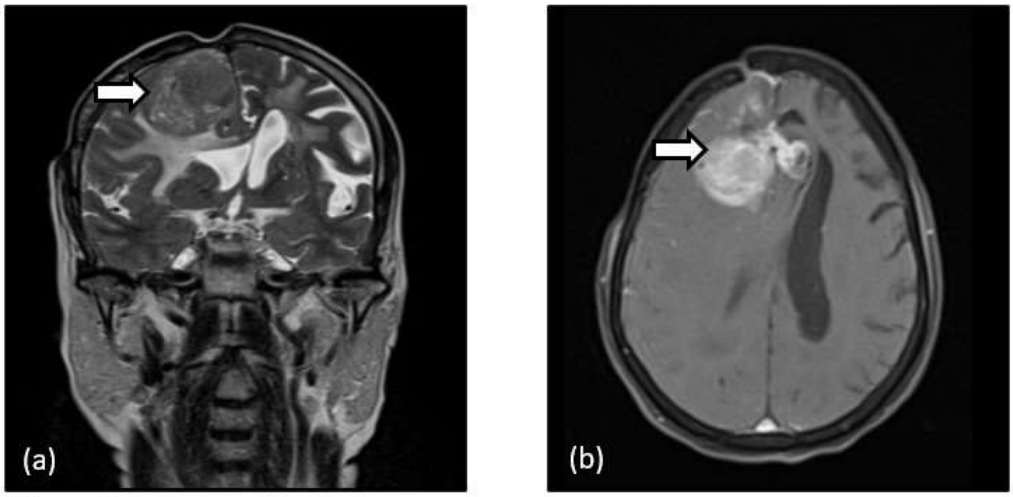

Figure 1

Figure 1: (a) T2w coronal section, (b) T1w axial section: An irregularly shaped heterogeneously enhancing T2 hetero-intense with hypointense core and T1 hyperintense lesion seen in the right frontal para-saggital region, measured – 3.4 x 8 x 4 cm, causing a mass effect in the form of effacement of adjacent sulci and ipsilateral lateral ventricle

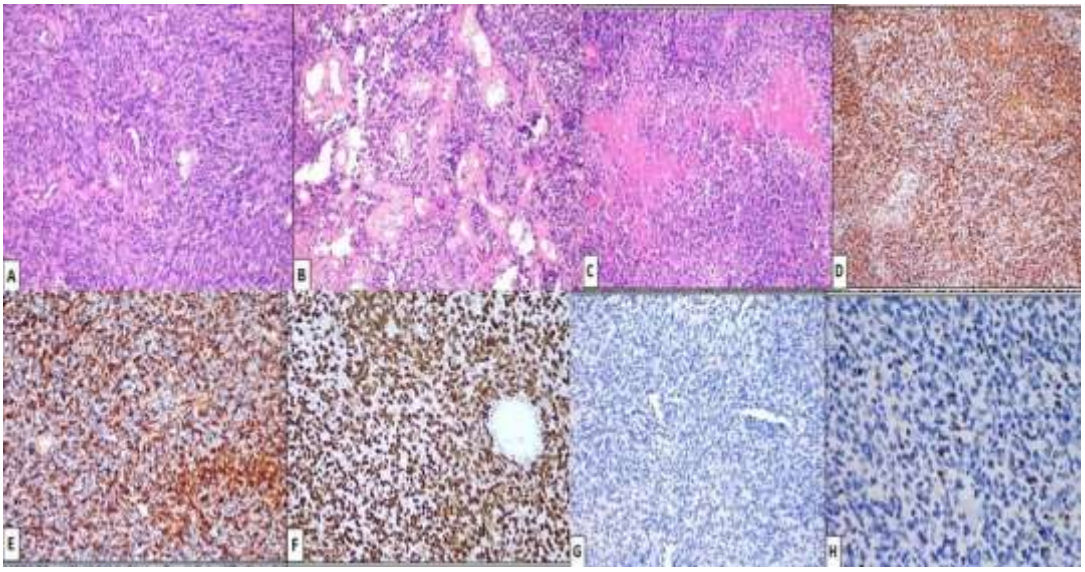

Figure 2 : A- Hematoxylin and eosin (H& E) stained sections show a tumor composed of spindled to ovoid monomorphic cells. (H&E; x100); B- Admixed area shows hyalinized, dilated, thin-walled, branching (staghorn-shaped) blood vessels. (H&E; x200); C- Cellular areas have densely packed round to oval cells with little or no intervening stroma and less apparent vasculature, mixed with pale zones or foci of necrosis. (H&E; x100); D- Tumor cells diffusely immunopositive for CD34. (CD34; x40); E- Tumor cells diffusely immunopositive for Vimentin. (Vimentin x100); F- Tumor cells diffusely immunopositive for Signal transducer and activator of transcription 6. (STAT6; x100); G- Tumor cells are negative for Glial fibrillary acidic protein. (GFAP; x100); H- Tumor cells show 1-2% Ki67 activity. (Ki67; x100)

DISCUSSION

Here we presented a case of an intracranial parafalcine SFT masquerading as an anaplastic meningioma. The distribution of SFT within the central nervous system is comparable to that of meningiomas. 70% are supratentorial, 15% are in the posterior fossa, and 15% are spinal.[1] Consistent with our case, the majority of intracranial SFTs are seen in females and occur in the fifth decade. SFTs are slow-growing, and patients may experience a variety of nonspecific symptoms related to tumor location or raised intracranial pressure (ICP). Clinical symptoms like sensory disturbance, gait imbalance, hemiplegic paralysis, and epileptic seizures appear only when the lesions grow large enough to impinge on important functional areas.[4] MRI imaging features that may suggest a diagnosis of SFT remain non specific. As per Clarençon et al., when an extra-axial, multilobulated, and heterogeneous tumor has hypointense T2 regions that strongly increase upon gadolinium injection without surrounding meningeal enhancement, ISFT may be recommended as the diagnosis. Multimodal MR with Diffusion, perfusion, and spectroscopy could provide additional clues that aid in ISFT recognition which could not be done in our case.[5] Radiological features like vascular flow voids, absence of dural tail, higher ADC (apparent diffusion coefficient), and less perilesional oedema may help differentiate SFT from meningiomas.[6] The yin-yang sign, a black-and-white patchy pattern on post contrast T2-weighted scans with alternating regions of hyperintensity and hypointensity brought on by changes in cellularity and staghorn vascularity, is a radiological finding usually seen in an ISFT.[7] However, we could not find a yin-yang sign in our case. As per the 2021 CNS WHO classification, SFT may be classified into grades 1, 2, or 3. Grade 1 tumors are characterized by a highly collagenous spindle-cell lesion with little cellularity. Grade 2 tumour is less collagenous and more cellular, with plump cells, staghorn vasculature, and mitosis <5 per 10 high-power fields (HPF). Finally, grade 3 is indicated by necrosis and/or ≥5 mitoses/10 HPF.[2] Lower progression-free survival (PFS) and local recurrence-free survival (LRFS) were linked to CNS WHO grade III.[8] A combination of positive CD34 and Bcl-2 is highly indicative of SFT. Also NAB2-STAT6 fusion genes are unique for SFTs, and they can be detected using IHC for STAT6. Intense and diffuse nuclear staining of STAT-6 is seen in more than 90% of the cases. [9]

Because of the dearth of cases, literature on the standard management of SFTs remains limited. Reducing the percentage of local and distant recurrences must be the goal of treatment since this tumor has an extremely high recurrence rate. Surgery remains the universal mainstay of SFT treatment. GTR patients had a considerably reduced 5-year recurrence risk than STR patients (20.8% vs. 72.7%; p = 0.006), according to Kim et al. The effect of GTR on overall survival (OS), however, is still debatable.[10] Adjuvant radiation therapy was substantially linked to higher PFS and LRFS, according to Lottin et al., but it did not have any effect on OS.[8] Prospective trials are required to assess novel treatment options following surgery, such as a revised chemotherapy regimen or advanced contemporary methods like proton therapy. In the present case prolonged gap between the initial treatment and the current presentation is noteworthy. One possible explanation is that the original tumour may have been an SFT misdiagnosed as meningioma before routine use of STAT6 immunohistochemistry. Alternatively, the present lesion may represent a second primary SFT or a delayed manifestation in a previously operated and irradiated field. Because retrospective review of the original tumour was not possible, the exact relationship between the initial meningioma and the present STAT6-positive SFT remains uncertain.

CONCLUSION

The case highlights the diagnostic difficulty of distinguishing recurrent meningioma from CNS SFT in a previously operated and irradiated dural-based lesion, and emphasizes the value of STAT6 immunohistochemistry and re-evaluation of presumed recurrent meningioma.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest There are no conflicts of interesta

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