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CASE REPORT

# Intracranial Solitary Fibrous Tumor Masquerading as Meningioma

**Preeti Joseph John, Pushpinder Kaur, Ankit Mittal, Ombir Saini**

**Abstract**

Intracranial solitary fibrous tumour (SFT) is a rare primary neoplasms of the meninges. It is often misdiagnosed on imaging as meningioma. Herein, we present two cases of intracranial SFT, which were diagnosed clinically as meningioma based on imaging findings. Microscopically, the tumors showed hypercellular and hypocellular areas with characteristic "patternless-pattern" and staghorn like vessels. Immunohistochemistry for STAT 6 showed diffuse nuclear positivity and EMA was negative, favours the diagnosis of SFT. The present study discusses the radiological, histomorphological, and immunohistochemical features of SFT, with emphasis on potential diagnostic pitfalls that may lead to erroneous diagnosis.

**Key Words**

Intracranial, Solitary Fibrous Tumour, Meningioma.

## Introduction

Solitary fibrous tumor (SFT) is a spindle cell neoplasm of mesenchymal origin that are typically found in pleura.[1] Primary SFT involving the central nervous system is rare and has been attributed to the paucity of true connective tissue elements.[2] Intracranial SFTs arise from the dura and comprise only 0.09% of all dural-based tumors.[1,3] SFTs lack pathognomonic imaging characteristics and it is challenging to differentiate from other dural-based lesions, such as meningiomas. The definitive diagnosis of SFT therefore, it mainly depends on the post operative histomorphology and immunohistochemical staining.[3,4] The treatment for the tumours mainly depends upon complete surgical resection. However, due to the potential for recurrence and extracranial metastasis, rigorous long term follow up is recommended.

## Case Report

Case 1- A 27-year-old female presented with history of headache, decreased vision, and two episodes of seizures for the past few months. CE-MRI brain demonstrated a well-defined lobulated dural based lesion (T1/T2 heterointense predominantly isointense) measures 5.9 x 5.8 x 6.4 cm along the left parieto-occipital convexity with perifocal edema and evidence of dural tail, cortical buckling and CSF cleft. On post contrast images avid enhancement seen. The lesion was having central non-enhancing area shows subtle diffusion restriction on DWI/ADC images and blooming on GRE images suggesting haemorrhage/calcification. Effacement of ipsilateral cortical sulci and minimal midline shift towards the right seen. Findings were suggestive of left parieto-occipital convexity meningioma with mass effect *(Fig.1a)*.

On gross examination tumour was in multiple pieces with soft to firm in texture measuring 10 x 8 x 3 cm *(Fig. 1b)*.

Histopathological examination revealed a neoplasm with variable cellularity composed of alternately hypocellular and hypercellular areas. The cellular areas show oval to short spindle cells lying in a patternless pattern in a collagenous stroma. The cells had uniform nuclei with vesicular chromatin. Areas of staghorn-like, branching vasculature were appreciated. Mitotic count

**Departments of Pathology , Adesh Medical College and Hospital, NH-44, Vill. Mohri Teh. Shahabad (M) near Ambala Cantt. Haryana, India.**

**Correspondence to**: Dr Preeti Joseph, Associate Professor, Department of Pathology, AMCH, Shahabad, Haryana, India.

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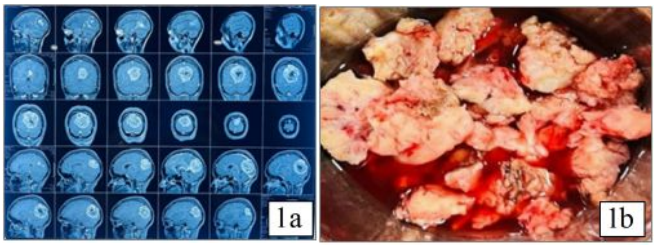


**Figure 1a : CE-MRI brain demonstrated a well-defined lobulated dural based lesion measures 5.9x 5.8x 6.4 cm.**
**Figure 1b : Gross image, multiple partly circumscribed firm white to reddish brown mass.**

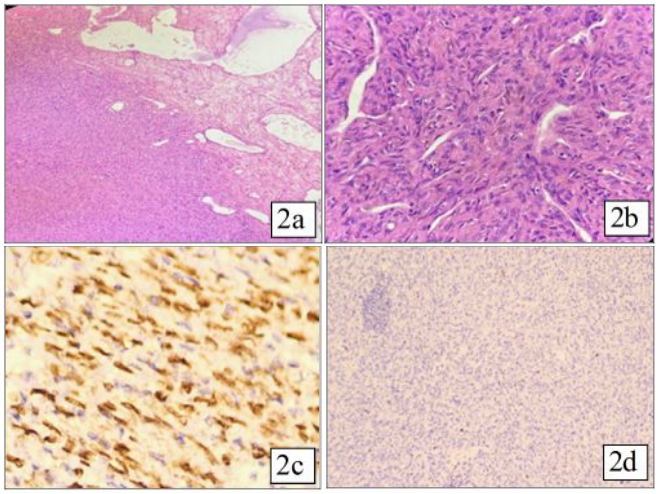


**Figure 2a : Cellular tumour with thin-walled branching vessels (H&E 4x).**
**Figure 2b : Cellular area with oval to spindled fusiform nuclei and staghorn vessels (H&E 10x).**
**Figure 2c : STAT-6 nuclear positivity in neoplastic cells (IHC).**
**Figure 2d : EMA negative tumour cells (IHC).**

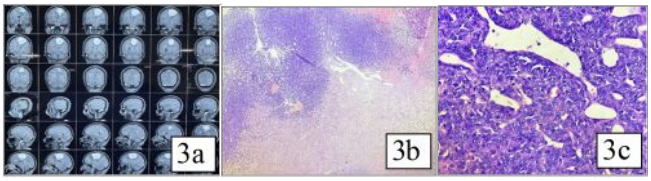


**Figure 3a : NCCT brain showed well defined extra-axial mass lesion measuring 4.5 x 6.2x4.8 cm in left frontoparietal parafalcine location.**
**Figure 3b : SFT with cellular and hypocellular areas (H&E 4x).**
**Figure 3c : Cellular area with oval uniform nuclei and thin-walled vessels (H&E 10x).**

was 0-1/10 HPF. The morphological features were suggestive of solitary fibrous tumor WHO grade I *(Fig. 2a & 2b)*.

Immunohistochemistry for STAT-6 showed diffuse nuclear positivity and EMA was negative (*Fig. 2c & 2d)*.

Case 2- A 45 years old male presented with headache and right sided weakness for 1 month. CT/MRI brain showed well defined extra-axial mass lesion measuring 4.5 x 6.2 x 4.8 cm in left frontoparietal parafalcine location. It appeared heterogenously hyperintense on T2 W/FLAIR images with significant white matter edema and mass effect. The findings were suggestive of meningioma (*Fig. 3a*).

Histopathology revealed a neoplasm composed of predominantly cellular areas with few hypocellular areas. The cellular areas show cells have oval to spindle fusiform nuclei arranged in sheets with scanty intervening collagen in the background. Many ectatic thin walled staghorn like vascular channels were noted within the tumour. Mitosis was 1-2/10 hpf. The morphological features were of SFT WHO grade 1 (*Fig. 3b & 3c*).

## Discussion

Primary SFT is rare and accounts for ~0.09% of all meningeal tumors.[1,5] Most of SFTs are dural based often supratentorial followed by frontal convexity, skull base, parasagittal, falcine location, cerebellopontine angle, ventricles.[1,2,3,6]

Most SFT instances occur in females between the fifth and sixth decades of life, with 18% of patients being younger than 40 years old.[1,4,6] Common clinical presentation include headache, gait imbalance, sensory disturbance, epileptic seizure and hemiplegic paralysis.[4,7]

It is challenging to differentiate SFT on imaging due to its radiological characteristics, which might mimic those of other, more prevalent CNS tumors. SFTs are frequently misdiagnosed as fibrous meningioma, schwannomas, neurofibroma, hemangiopericytoma and fibrosarcoma.[2] MRI is the preferred imaging modality for diagnosis. The most common presentation is heterogenous pattern on T2- weighted imaging with hypointense areas and iso- or hyperintense areas. This may be explained by existence of hypocellular and hypercellular components within the tumor. Combining two separate components gives rise to the 'yin yang' sign associated with CNS SFTs. An avid contrast enhancement at the lesion's periphery (dural tail) is typically seen.[1,3,8]

No specific features on CT or MRI can be used to distinguish SFT from meningioma.[3,8,9] Radiological diagnosis of meningioma was rendered to the current cases before it was proved on histopathology. Pretreatment differentiation is essential as the behavior

and treatment of these tumors are different.[8]

The definitive diagnosis of SFT is based on histomorphology and immunohistochemical staining, with imaging serving as a supplemental diagnostic tool. The characteristic histology features of SFT includes hypocellular and hypercellular areas. The hypocellular phenotype display short spindle and oval-round cells arranged in so called "patternless pattern" with alternating thick bands of hyalinized collagen and thin walled branching hemangiopericytoma-like (staghorn) vessels. The hypercellular area is characterized by oval-round cells arranged in haphazard pattern with minimal intervening stroma.[5,6,8] Nested syncytial pattern, with abundant cytoplasm with indistinct borders and pseudoinclusions characteristic of meningioma are not observed. The WHO grading system assigns a grade 1 to tumours with mitotic count of <5/10 HPF whereas tumours are classified as grade 2 with mitotic count of e"5 /10 HPF without necrosis, and tumours with mitosis e"5/10HPF with necrosis are classified as grade 3.[6]

SFT are diffusely positive for STAT6 and CD34. STAT6 IHC has a very high specificity and sensitivity surrogate for NAB2-STAT6 fusion and is considered a definitive marker SFT.[2,3,6,7] Fibrous meningiomas must be distinguished from SFT. Meningiomas are typically reactive for EMA and negative for nuclear STAT6 expression.[6,8]

More than half of SFT cases express positivity for CD99 and bcl-2.[1] Dura-based Ewing sarcoma/primitive neuroectodermal express CD99 on IHC and may resembles hypercellular phenotype of SFT but lacks nuclear STAT6 staining and is characterized by EWSR1 gene rearrangement.[6,8] Monophasic synovial sarcoma is another differential diagnosis of SFT based on CD99 and Bcl2 expression. This diagnosis is supported by positive staining with cytokeratin, EMA, TLE1, and absence of nuclear STAT6.[6,8]

Rarely, malignant peripheral nerve sheath tumours may resemble the hypercellular phenotype of SFT. However, MPNST shows localized positivity for S100 and SOX10 and is negative for CD34 and STAT6.[6,8]

The initial course of treatment is complete surgical excision of the mass. Conventional radiotherapy has been described as adjuvant treatment for residual SFTs.[1]

SFT is characterized by high rates of local recurrence and extracranial metastasis and high fatality rates even decades after resection.[2,5]

To conclude, SFT is often misdiagnosed on imaging because of its similarities to other more prevalent brain tumours. Accurate diagnosis of SFT mainly relies on histomorphology and immunohistochemical staining. Following early surgical treatment, SFT has an excellent neurological outcome. However, due to potential of recurrence, long term follow up is recommended.

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