

Suprasellar Anaplastic Meningioma Masquerading As Craniopharyngioma: A Case Report

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Abstract

Anaplastic meningiomas are uncommon. We report the clinical, radiological and pathological features of an anaplastic meningioma in a young male Egyptian patient presenting as a suprasellar solid/cystic enhancing mass resembling a craniopharyngioma.

Keywords: Meningioma; Anaplastic; Suprasellar; Craniopharyngioma

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INTRODUCTION

Meningiomas are common intracranial neoplasms arising from arachnoidal cells. They occur most commonly in middle-aged female patients. Examples in young patients are often associated with hereditary tumor syndromes (particularly NF2) and affect either sex equally [1]. Unlike benign meningiomas, anaplastic meningiomas are rare and tend to show a male predominance [1].

Anaplasia is defined by morphologic features of frank malignancy exceeding those present in atypical meningioma. Those features include obvious malignant cytomorphology (resembling carcinoma, melanoma or sarcoma), or a high mitotic index with more than 20 mitoses per 10 high-power fields [1]. Anaplastic meningiomas exhibit an aggressive clinical course including rapid growth, local recurrence, metastasis and poor prognosis. They are often fatal with a median survival of 1.5 years [2].

We present the clinicoradiological, histopathological and immunohistochemical features of a case of anaplastic meningioma in a young male patient, which presented radiologically as a suprasellar mass mimicking craniopharyngioma.

CASE REPORT

21 years old male patient presented with diminution of vision in both eyes. MRI showed a sizable suprasellar expanding lesion (4.6x2.3x4 cm). The lesion showed complex solid and smaller cystic component. The solid component expressed isointense signals in T1 and T2 weighted images with focal low signal denoting focal calcification. The solid component showed moderate heterogeneous enhancement with a thin marginal enhancement of the cystic component. The lesion exerted mass effects on the surrounding structures. Radiologically, the lesion raised the possibility of a craniopharyngioma. (Figure 1a) Intra-operative, the lesion was retro-chiasmatic hypothalamic in location and attached to the optic nerve.

Histopathological examination revealed a cellular tumor with a discrete interface with the adjacent brain parenchyma and heterogeneous growth patterns. Epithelioid appearance (Figure 1b) was imparted by the disposition of tumor cells in cohesive sheets of pleomorphic cells. Pseudopapillary pattern and arrangement of tumor cells in interlacing cords emphasized this epithelioid appearance. Other foci showed arrangement of tumor cells in cords embedded in a hyaline eosinophilic material creating a

mesenchymal appearance (Figure 1c). Individual tumor cells had pleomorphic vesicular to hyperchromatic nuclei with occasional prominent nucleoli and eosinophilic to vacuolated to clear cytoplasm. Mitotic figures (2-6/10 HPF) and areas of necrosis were seen.

Reticulin histochemical stain showed that reticulin fibers surrounded clusters of tumor cells without extensive pericellular deposition (Figure 1d). The provisional diagnosis was poorly differentiated malignant tumor.

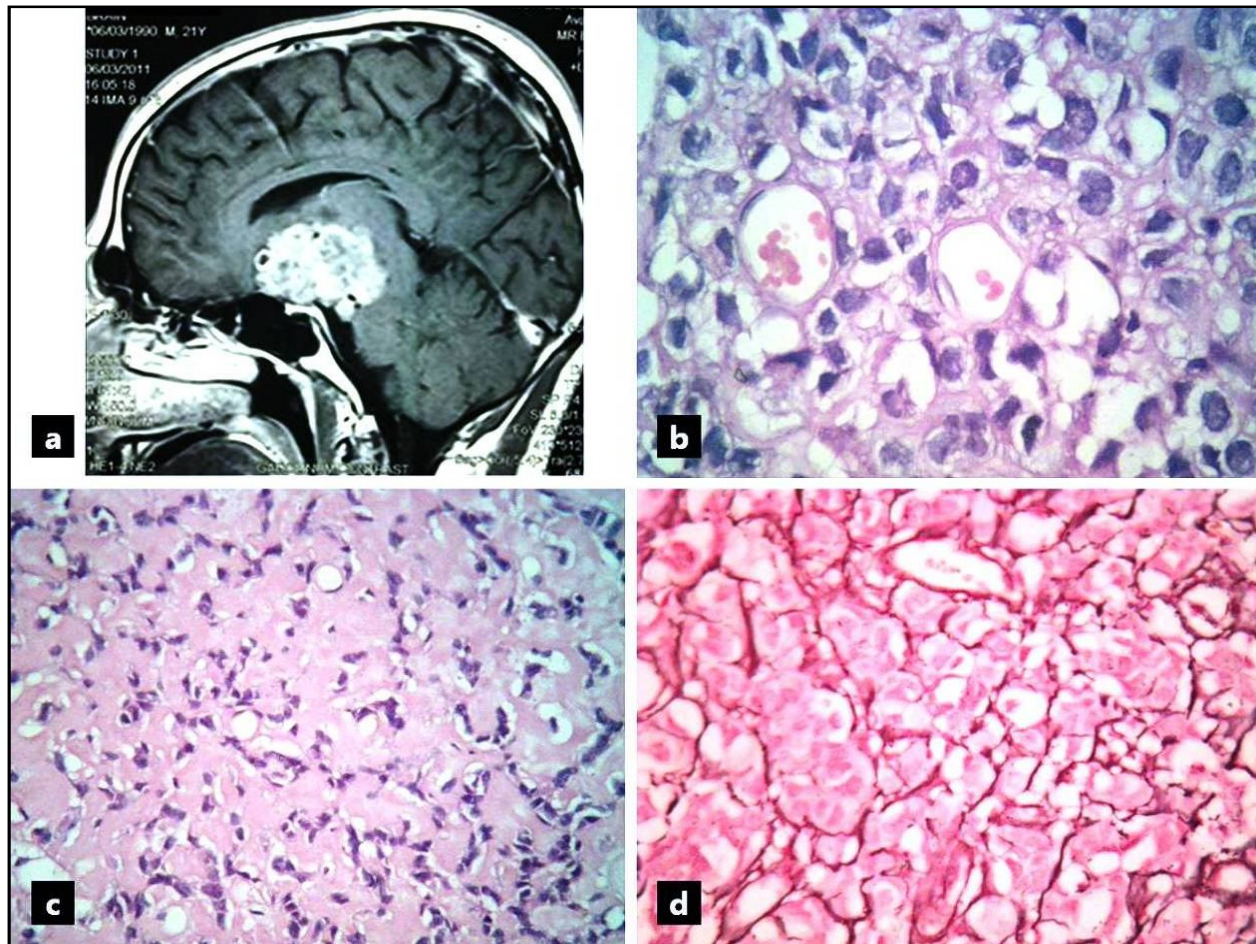


Figure 1: [a] MRI T1 weighted image with contrast showing the suprasellar S.O.L. with enhancement and cystic component. [b]: Poorly differentiated pleomorphic tumor cells with epithelioid features (H&E, x400). [c]: Cords of tumor cells in a hyaline background imparting a mesenchymal appearance (H&E, x200). [d]: Reticulin fibers surrounding groups of tumor cells (Reticulin, x200).

By immunohistochemistry, tumor cells were positive for epithelial membrane antigen (EMA), vimentin, and integrase interactor 1 (INI 1); focally positive for S 100 protein and progesterone receptor (PgR); and negative for glial fibrillary acidic protein (GFAP), synaptophysin, placental alkaline phosphatase (PLAP), cytokeratin, CD30, CD31 and smooth muscle actin (SMA). Ki 67 proliferative index was about 7% (Figure 2).

Based on histological and immunohistochemical results (EMA-positive, vimentin-positive, PgR-focally positive) the diagnosis of anaplastic meningioma, WHO grade III was considered. The case was referred to Mayo clinic and the diagnosis was confirmed.

The patient received post-operative radiotherapy. Six months later, follow up radiology showed evidence of a huge tumor recurrence. Intra-operative, the tumor had a small soft component and a large calcified component compressing the brain stem and hypothalamus.

The recurrent tumor was histologically composed of paucicellular tissue showing few spindled to plump cells, with hyperchromatic to vesicular nuclei and occasional distinct nucleoli, embedded in a hyalinized eosinophilic background. Focal metaplastic bone formation was noted.

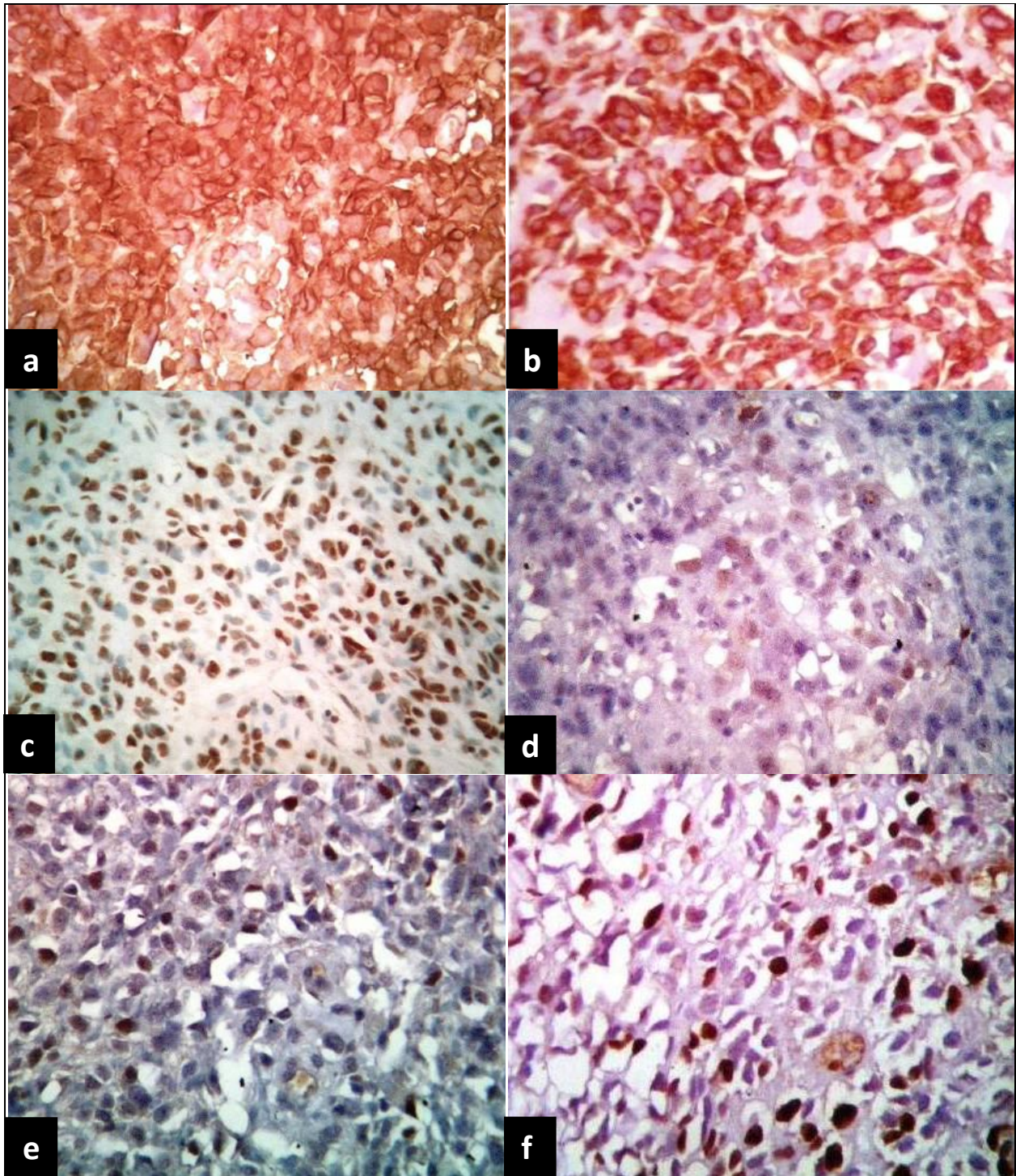


Figure 2: Immunohistochemically, tumor cells were positive for EMA [a], vimentin [b], and INI 1 [c]; and focally positive for S-100 [d] and PgR [e]. Ki67 immunostaining showed moderate proliferative activity [f] (Original magnification, x 200).

DISCUSSION

Anaplastic meningiomas exhibit an aggressive clinical course [1, 2]. Herein, we present a case of anaplastic meningioma presenting radiologically as a suprasellar mass resembling craniopharyngioma in a young male patient. The patient did not show any evidence of hereditary syndromes. Histologically, the tumor was poorly differentiated with epithelioid features. In the presented case, an extensive panel of immunostains was used to cover many differential diagnostic entities. The tumor was only reliably positive for EMA and vimentin.

It is well known that meningiomas are distinctive for its dual mesenchymal and epithelial differentiation, which is reflected on the immunohistochemical level by positivity for vimentin and EMA [1]. The age of the patient and site of the tumor favored a primary neoplasm. Nevertheless, negative reaction to cytokeratin excluded any doubts regarding the possibility of metastatic carcinoma. Although the tumor was positive for EMA, positivity for vimentin further supported the primary nature of the tumor. Despite that vimentin can be expressed by some carcinomas, such as renal cell carcinomas, vimentin can be a useful marker distinguishing malignant meningioma from metastatic carcinoma [3].

In the present case, the dual EMA and vimentin positivity raised the possibility of atypical teratoid/rhabdoid tumor (AT/RT); however this was excluded by the positive nuclear staining of INI1 in tumor cells. Negative reaction to SMA further supported the exclusion. Germ cell tumors were excluded based on negative reaction to PLAP. For further confirmation, cytokeratin and CD30 were employed to exclude embryonal carcinoma and both markers were negative in our case. Negative reaction to GFAP and synaptophysin excluded the possibility of glial or neuronal differentiation.

Vascular tumors, although rare, can arise intracranially [1]. Negative reaction to CD31 excluded this

possibility. Focal positivity for PgR further substantiated the possibility of meningioma and the final diagnosis was anaplastic meningioma, WHO grade III.

The use of reticulin histochemical stain was helpful. Reticulin fibers surrounded groups of cells rather than individual cells, a pattern expected in meningiomas.

Presentation as a suprasellar solid/cystic lesion raised the possibility of craniopharyngioma, however, this was not the case both intra-operatively and histologically.

In conclusion, we report a case of anaplastic meningioma radiologically resembling craniopharyngioma and histologically showing poorly differentiated phenotype. We conclude that anaplastic meningiomas can present as poorly differentiated neoplasms that require an extensive panel of immunostains to reach a final diagnosis.

CONFLICTS OF INTEREST

Authors declare that there are no conflicts of interest.

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