

Case Report

Case Report: Diagnostic Dilemma between Atypical Meningioma and Glioblastoma-our Observation

Khan AH¹, Munna MH², Islam MS³, Rahman S⁴, Ahmed T⁵, Ripon JA⁶, Islam RU⁷, Islam A⁸, Mamun AA⁹, Dorji TW¹⁰, Biswas MA¹¹, Obaida ASMA¹², Rahman A¹³

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Contribution of Authors: Principal Investigator- Prof.

Manuscript preparation- Dr. Akhlaque Hossain Khan

Data collection- Dr. Mehedi Hasan Munna, Dr. Muhammad Saiful Islam, Dr. Sumaiya Rahman, Dr. Tofayel Ahmed, Dr. Jalal Ahmed Ripon, Dr. Rahatul Islam, Dr. Ariful Islam, Dr. Abdullah Al Mamun, Dr. Tandin Wangyel Dorji, Dr. Md. Anik Biswas, Dr. Abu Saleh Md. Abu Obaida

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Abstract:

Both meningioma and glioblastoma are very common intra-cranial neoplasm. Typically both have distinguishing clinical and radiological features which aid in diagnosis and thus pre-operative preparation, planning, counseling the patient etc. But dilemma may often arise in case of non-grade-I meningioma making confusion in favor of higher grade astrocytoma (glioblastoma). In that case total per-operative picture, surgical approach, post-operative managements, and disease prognosis will be altered. We faced such kind of confusion in a case so reported here.

Key Words: Meningioma, Glioblastoma

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Introduction:

Meningiomas, usually benign (WHO-I) are the most common primary intracranial slow-growing, extra-axial tumor, comprising 13-26 % of all intra cranial tumors (Robin A Buerki et al., 2018). Anaplastic meningiomas (WHO-III) comprise merely 1% of meningiomas exhibit

aggressive behavior and are malignant in nature. There has been recognition of a pathologic third type of meningioma known as "atypical," (WHO-II) with borderline histological and clinical features between benign and malignant meningiomas. On MRI, meningiomas typically appear as lobular masses with

1. Dr. Akhlaque Hossain Khan, Professor and Chairman, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
2. Dr. Mehedi Hasan Munna, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
3. Dr. Muhammad Saiful Islam, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
4. Dr. Sumaiya Rahman, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
5. Dr. Tofayel Ahmed, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
6. Dr. Jalal Ahmed Ripon, Resident, Department of Neurosurgery, BSMMU Shahbagh, Dhaka-1000, Bangladesh.
7. Dr. Rahatul Islam, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
8. Dr. Ariful Islam, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
9. Dr. Abdullah Al Mamun, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
10. Dr. Tandin Wangyel Dorji, Resident, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
11. Dr. Md. Anik Biswas, Resident. Department of Pathology, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
12. Dr. Abu Saleh Md. Abu Obaida, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.
13. Dr. Asifur Rahman, Associate Professor, Department of Neurosurgery, BSMMU, Shahbagh, Dhaka-1000, Bangladesh.

Address of Correspondence: Dr. Akhlaque Hossain Khan, Professor and Chairman, Department of Neurosurgery, Bangabandhu Sheikh Mujib Medical University, Dhaka-1000 Bangladesh. Mobile 01711471153, Email: akhlaque63neuro@yahoo.com

well-circumscribed margins and a broad-based dural attachment (Buetow et al., 1991), but occasionally may exhibit a more infiltrating pattern over the dura, which are seen as asymmetric thickened enhancing sheets on imaging (Whittle et al., 2004). The high-grade meningiomas are more likely to present with an unclear peri-tumoral rim and peri-tumoral edema (Nakano et al., 2002). After contrast administration, meningiomas usually demonstrate homogenous enhancement, although they may have areas of necrosis that do not enhance (Watts et al., 2014). A dural tail, which is enhancement of the dura infiltrating away from the lesion, may also be seen on MRI and could prove useful in distinguishing meningioma from other brain lesions (O'Leary et al., 2007). Despite exhibiting characteristic imaging features, there exist variations in appearance on imaging that prove to be diagnostically challenging and limit the value of routine MRI in predicting WHO grades (Watts et al., 2014).

Glioblastoma, the most common and most rapidly progressing intrinsic primary malignant tumor of the central nervous system, continues to portend a dismal prognosis, despite improvements in diagnostic and therapeutic strategies over the last 20 years (Gaurav Shukla et al., 2017). Glioblastomas are characterized by marked neo-vascularity, increased mitosis, greater degree of cellularity and nuclear pleomorphism, and microscopic evidence of necrosis. The most common imaging appearance of Glioblastoma is a large heterogeneous mass in the supra tentorial white matter that exerts a considerable mass effect. It typically contains central areas of necrosis, has thick irregular walls, and is surrounded by extensive, vasogenic edema, but the tumor may also have thin round walls, scant edema, or a cystic appearance with a mural nodule (John H. Rees et al., 1996).

Report of Case:

A 47 year old female presented with headache for 6 months, several episodes of vomiting and blurring of vision for 1 month. On neurological examination she was found to have right homonymous hemianopia and bilateral papilledema. However, she denied any history of convulsion, altered level of consciousness and all other cranial nerves, motor, sensory or cerebellar functions were intact.

On pre-operative MRI of brain with contrast showed a hypo-intense lesion in the left occipital region pushing the left parieto-occipital sulcus, effacement of left lateral

ventricle with midline shift to the right (Fig 1A) which was iso to mild hypo-intense with peri-lesional hypo-intensity on T2 (Fig 1B), with heterogenous contrast enhancement (Fig 1E), FLAIR showed huge perilesional edema (Fig 1C), patchy diffusion restriction was found on DWI (Fig 1D). MRS findings were increased choline peak, decreased NAA and the ratio between choline and creatine was 3.85 (Fig 1H)

There was a dilemma about the diagnosis. As the lesion did not have any clear cleavage giving it an intrinsic-like look along with the unusual pattern of contrast enhancement and huge perilesional oedema for a meningioma, we thought of some high-grade glioma. However, the crowding of the sulci and gyri, and the osposhto / thin CSF cleft around the lesion, and the contrast uptake pattern made us think of an extra-axial lesion like a high-grade meningioma.

On the basis of the clinic-radiological information we thought the lesion to be a meningioma, most likely a high-grade one. We kept Glioblastoma also in our DD.

The dura was opened in a C-shaped fashion keeping the base toward the superior sagittal sinus following a 4 cm X 4 cm craniotomy placing the patient in the left park-bench position to facilitate the brain retraction utilizing gravity. Right after the dural opening, a greyish-white, moderately vascular, firm, fibrous, non-suckable tumor was encountered that had no attachment to the dura. There was no well-delineated cleavage between the tumor and the brain parenchyma giving the peroperative impression of an intrinsic tumor. Gross total removal of the tumor was done in a piecemeal fashion. There were some necrotic parts as well as some thrombosed vessels in the tumor. The dura was closed in a watertight fashion and rest of the wound was closed in layers as usual. The postoperative period was uneventful.

The postoperative computerized tomography (CT) revealed no residual tumor or haematoma (Fig 3- A,B), and the patient was discharged on the 7th postoperative day.

Histopathology from one center revealed malignant glial tissue arranged in diffuse sheets and the cells were highly pleomorphic. Mitoses, micro-vascular proliferation, and necrosis were seen. All these features were compatible with Glioblastoma (NOS), WHO- grade 4.

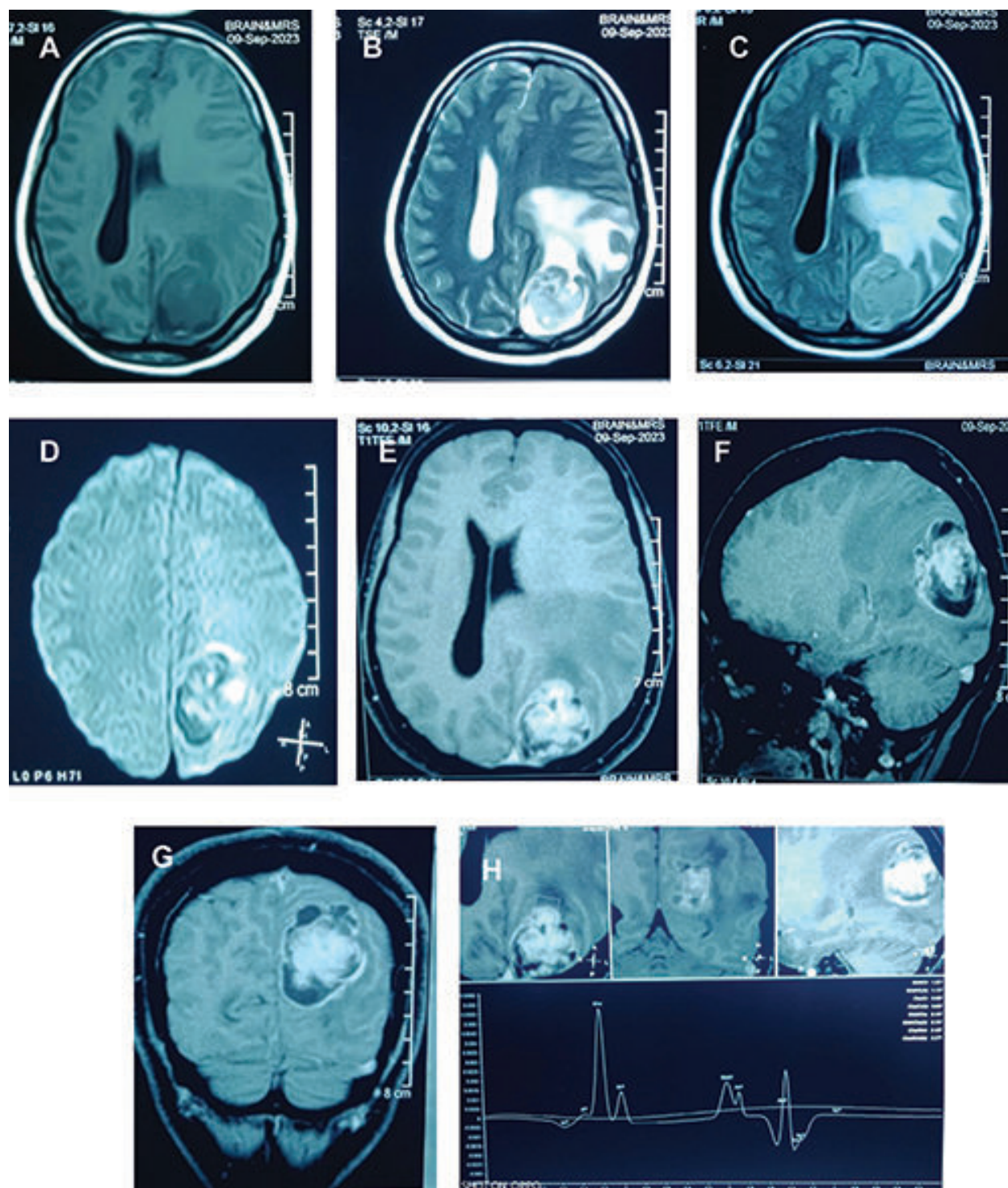


Figure 1: Preoperative (A) MRI axial T1 image showing hypo-intense irregular lesion occupying left occipital lobe adjacent to pole with perilesional hypo-intensity, effacement of left lateral ventricle, shifting of midline to right, (B) axial T2 iso to hypo-intensity with peri-lesional hyper-intensity, (C) which is also hyper-intense on FLAIR suggesting peri-lesional edema (D) patchy restriction of diffusion, (E) Heterogenous contrast enhancement with intralesional hypo-intensity(non-enhancing) may be necrosis on axial contrast, (F) sagittal contrast suggesting heterogenous contrast enhancing may be extra axial lesion surrounded by cystic fluid or CSF cleft (G) coronal contrast, (H) MRS showing high Choline peak, low NAA, Choline: Creatine- 3.85



Figure 2: (A) Park bench position (B) Incision (C) (D) per-operative pictures

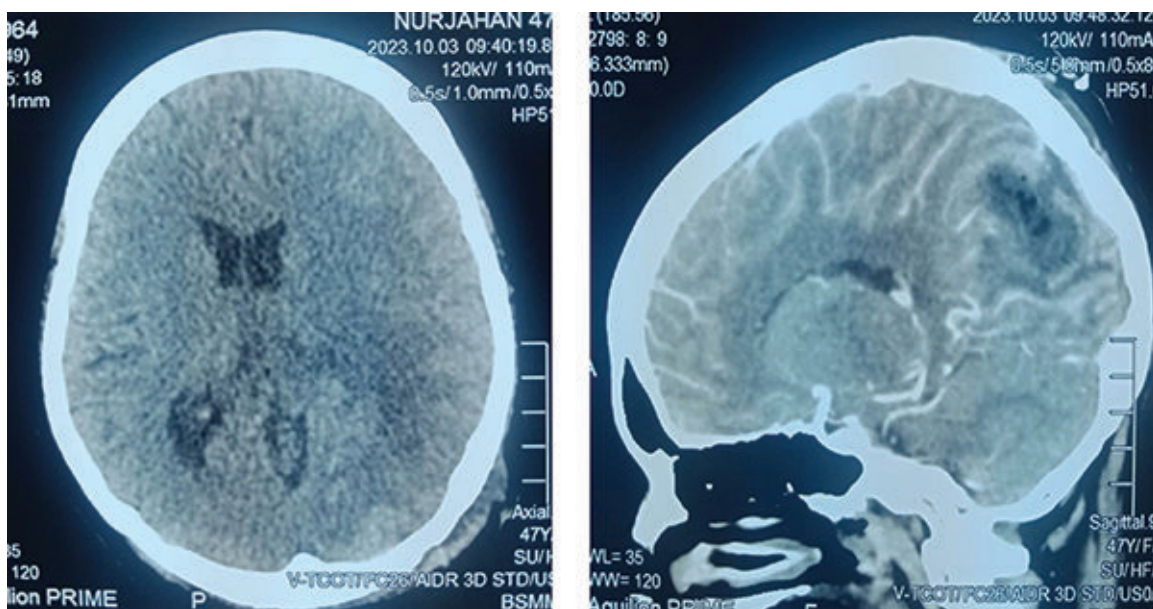


Figure 3: Post-operative CT brain (A) axial non-contrast, (B) sagittal contrast, showing no residual tumor and no tumor bed hematoma.

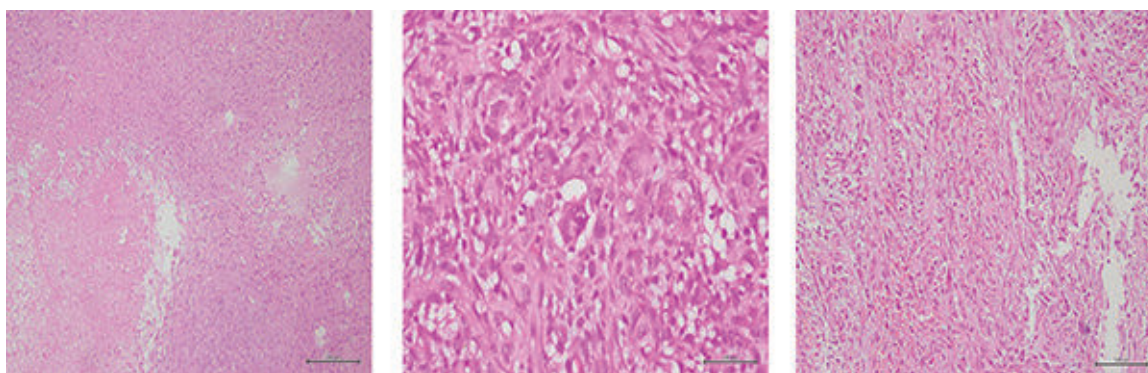


Figure 4: Histopathological pictures of resected specimen from left occipital SOL (A) (B) (C)

Conclusion:

Even such most common intracranial tumors like meningiomas and glioblastoma with distinctive clinical and radiological features may create confusion in

diagnosis which often can mislead in management plan. This kind of mishaps might be avoided by careful evaluation of each individual cases and tactful pre-operative patient counseling.

Declarations:

Conception, Diagnosis and Design:

Prof. Dr. Akhlaque Hossain Khan, Dr. Mehedi Hasan Munna

Radiological Diagnosis and Final approval of manuscript:

Prof. Dr. Akhlaque Hossain Khan, Dr. Mehedi Hasan Munna, Assoc. Professor Dr. Asifur Rahman

Manuscript Preparation, Technical Revision:

Prof. Dr. Akhlaque Hossain Khan, Dr. Mehedi Hasan Munna, Dr. Sumaiya Rahman, Dr. Tofayel Ahmed, Dr. Jalal Ahmed Ripon, Dr. Rahatul Islam,

Manuscript editing and revision:

Prof. Dr. Akhlaque Hossain Khan, Dr. Mehedi Hasan Munna, Dr. Ariful Islam, Dr. Abdullah Al Mamun, Dr. Tandin Wangyel Dorji, Dr. Md. Anik Biswas,

Literature Review:

Prof. Dr. Akhlaque Hossain Khan, Dr. Mehedi Hasan Munna, Dr. Muhammad Saiful Islam, Dr. Abu Saleh Md. Abu Obaida

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