10. Koeller, K.K., Rushing, E.J. From the archives of the AFIP: medulloblastoma: radiologic-pathologic correlation. Radiographics. 2003,23,6,1613–1637.
11. Riffaud, L., Morandi,, Massengo, S. et al. Primary extraneural medulloblastoma: a case report. Neurochirurgie. 2001,47,6,542–546.
12. Pollack, I.F. Multidisciplinary management of childhood medulloblastoma. Curr Treat Options Neurol. 2003,5,5,403–414.

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CORPUS CALLOSAL SPACE OCCUPYING LESION: AN INSTITUTIONAL EXPERIENCE AND REVIEW OF LITERATURE

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INTRODUCTION:

The corpus callosum is the main bridge connecting the two brain hemispheres. The fibers from the inferior frontal and lower parietal lobes cross through the genu, while those from the parietal lobe cross at the splenium. The remaining fibers cross through the body of the corpus callosum.[1] Disorders affecting the corpus callosum are diverse and can include congenital abnormalities, demyelination, infections, trauma, and tumors. Lesions in this area often cause bilateral hemispheric effects, creating a distinctive "butterfly" pattern on imaging.[2] Gliomas involving the corpus callosum (CC) represent a unique clinical subset of brain tumors due to their deep midline location, potential for bilateral spread, and significant neurocognitive consequences. These tumors, particularly high-grade gliomas (HGGs), often present with non-localizing symptoms such as personality changes, memory loss, or limb weakness. Corpus callosum invasion, especially in so-called 'butterfly gliomas,' has historically been associated with a poor prognosis and limited surgical options. Gliomas involving the corpus callosum are a heterogeneous group, ranging from low-grade oligodendrogliomas to high-grade astrocytomas. Advances in imaging and surgical techniques have shifted treatment paradigms toward safe maximal resection, improving both survival and cognitive outcomes. Histopathological and molecular classification (IDH1, ATRX, Ki-67) is crucial in guiding prognosis and adjuvant therapy. Currently, there is no universally accepted optimal treatment; the approach generally involves biopsy, followed by radiation and chemotherapy. Due to limited data, our focus in this study is to describe the clinical features, histological diagnosis, outcomes, and survival rates of lesions affecting both hemispheres.

CASE SERIES:

Case 1:

A 43-year-old female with no known comorbidities presented with a history of irritable behaviour and forgetfulness, holocranial headache 20 days prior, right side weakness 12 days prior followed by left side weakness 10 days prior, slurring of speech 10 days prior. On presentation, she was hemodynamically stable, conscious, oriented, and following verbal commands, with bilateral pupils normally reacting to light, moving all four limbs. KPS score was 80 on presentation. MRI brain with contrast (Figure 1A & 1B) suggestive of A large lobulated, bilobed Approx 5.8 x 3.8 x 3.2 cm solid lesion involving the splenium of the corpus callosum , extending to the posterior 1/3 of the corpus callosum (midline/paramedian). It shows Hypo to isointense on T1WI, Hyperintense on T2WI, Mild peripheral diffusion restriction on DWI, Multiple foci of blooming on SWI, on post contrast Irregular peripheral enhancement seen. The patient underwent left parieto temporo occipital craniotomy for excision of the lesion. Subtotal resection achieved. Post op CT Brain plain was done (Figure 1D). Histopathological examination (Figure 1C)demonstrated Tumor cells are moderately pleomorphic with central nuclei surrounded by clear cytoplasm. Chicken - wire type vasculature. Focal calcification and astrocytic differentiation. Micro calcification with fibrillary background. Brisk mitotic activity and microvascular proliferation is seen. Immunohistochemistry shows IDHR132H : Positive ATRX : Retained nuclear expression, p53: Occasional scattered positive cells, Ki 67: 35%, suggestive of Anaplastic oligodendroglioma, IDH 1 r132h mutant, CNS who grade 3. The patient was advised for adjuvant chemo and radiotherapy.

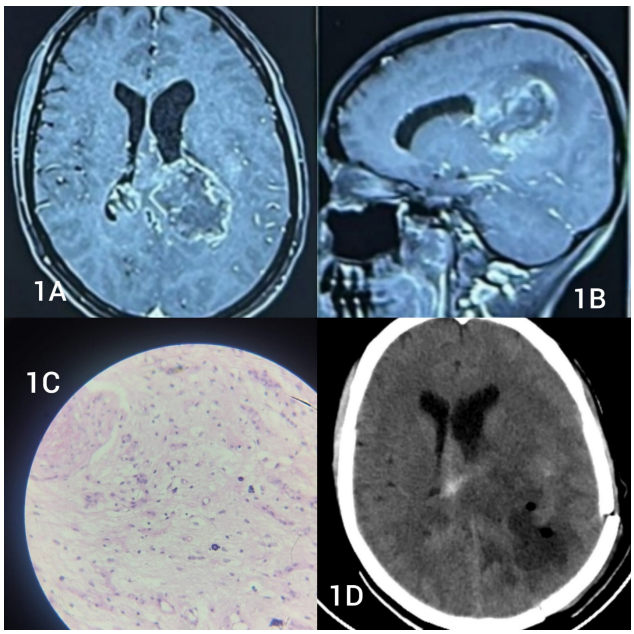


Figure 1: (A) Axial and (B) sagittal T1-weighted post-contrast MRI showing a large bilobed lesion involving the splenium of the corpus callosum with irregular peripheral enhancement and "butterfly" pattern (Case 1). (C) Histopathology (H&E, 400×) showing oligodendroglial cells with perinuclear halos and chicken-wire vasculature. (D) Postoperative non-contrast CT brain showing subtotal excision.

Case 2:

A 72-year-old female with known comorbidities of hypertension for 12 years and parkinsonism for 5 years on regular medications, presented with left side upper limb lower limb weakness 5 months prior. On presentation, she was hemodynamically stable, conscious, oriented, and following verbal commands, with bilateral pupils normally reacting to light, left upper limb power MRC grade 2 Lower limb power MRC grade 3. KPS score was 60 on presentation. MRI brain with contrast (Figure 2A & 2B) revealed a fairly defined, heterogeneous intra-axial lesion is seen in the right sided body of corpus callosum with involvement of cingulate gyrus , measuring approximately 58 x 40 x 44 mm (AP x TR x CC) . Hypointense on T1WI, Heterogeneously hyperintense on T2WI, Diffusion restriction on DWI, with peripheral blooming on SWI, Post contrast Heterogeneous peripheral enhancement with central non-enhancing necrosis seen. On MR Spectroscopy : Reduced NAA, elevated choline uptake seen. The patient underwent right parasagittal fronto parietal craniotomy for excision of the lesion. Near total resection was achieved. Post operative CT brain plain was done (Figure 2D). Histopathological examination (Figure 2C) revealed tumor cells with nuclear atypia, giant cells, bizarre cells and atypical mitotic figures. There are presence of foci of coagulative necrosis and microvascular proliferation, Immunohistochemistry shows IDH1 : Mutant Positive (R132H IHC) ATRX : Nuclear expression lost (Consistent with mutant) Ki67 : ~30% CD 68 : Positive P53 : Tumor cells are positive, suggestive of Astrocytoma, IDH mutant, CNS who grade 4. The patient was advised for adjuvant chemo and radiotherapy.

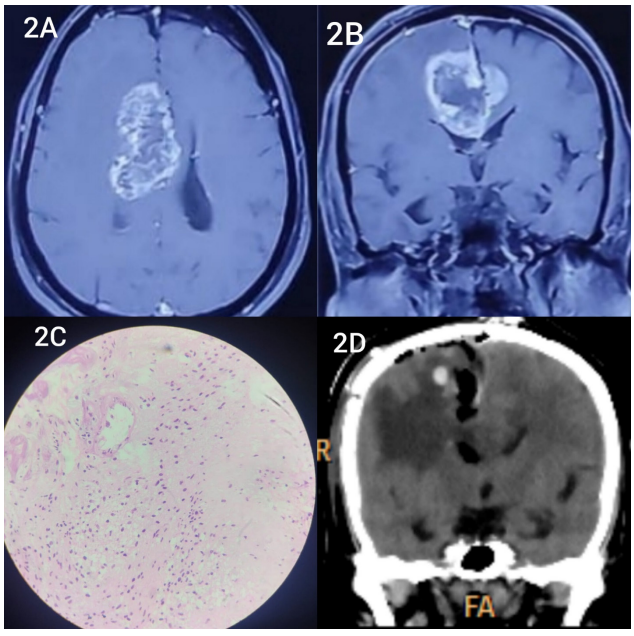


Figure 2: (A) Axial and (B) coronal T1-weighted post-contrast MRI showing a heterogeneous mass in the right body of the corpus callosum with peripheral enhancement and central necrosis (Case 2). (C) Histopathology (H&E, 400×) demonstrating nuclear atypia, microvascular proliferation, and necrosis. (D) Postoperative CT brain plain showing near-total resection.

Case 3:

A 48-year-old male with nil comorbidities presented with a 10-day history of frontal and occipital headache on and off associated with left side upper and lower limb weakness and forgetfulness. On presentation, he was hemodynamically stable, conscious, oriented, and following verbal commands, with bilateral pupils normally reacting to light, left side upper limb MRC grade 4 lower limb MRC Grade 4. KPS score was 80 on presentation. MRI brain with contrast (Figure 3A,3B) revealed inhomogeneously enhancing heterogenous lesion is seen along posterior right lateral ventricle extending in parafalcine parietal region protruding in body of lateral ventricle size measures 75 x 45 x 35 mm. mass effect is seen in form of compression over body of lateral ventricle with buckling of posterior corpus callosum with contra-lateral midline shift of 13 mm towards left side. The patient underwent right parieto temporal craniotomy for excision of lesion. Subtotal resection was achieved. Post op CT Brain plain was done (Figure 3D). Histopathological examination (Figure 3C) showed tumor cells with nuclear atypia, bizarre cells and atypical mitotic figures. There is presence of foci of coagulative necrosis and microvascular proliferation, Immunohistochemistry shows p53 -Patchy positive, Ki 67 - ~10%, IDH1 - Positive, ATRX -Loss of nuclear expression, GFAP -Positive, OLIG2 -Positive, suggestive of Astrocytoma, IDH mutant, CNS who grade 4. The patient was advised for adjuvant chemo and radiotherapy.

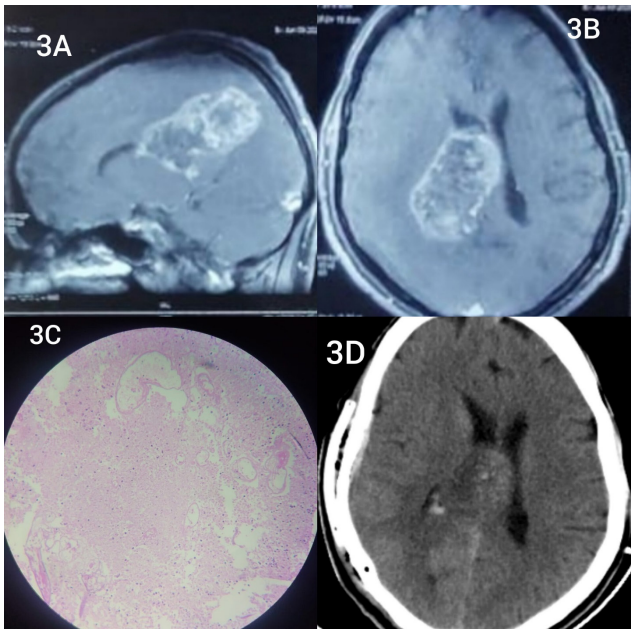


Figure 3: (A) Sagittal and (B) axial T1-weighted post-contrast MRI showing a large right parafalcine parietal mass protruding into the body of lateral ventricle and corpus callosum (Case 3). (C) Histopathology (H&E, 400×) showing hypercellular tumor with necrosis and mitotic figures. (D) Postoperative CT brain plain showing subtotal excision

Case 4 :

A 46-year-old male with hypertension for 4 years on irregular medications, presented with a complaint of holocranial headache 15 days prior, giddiness and left upper and lower limb weakness for 4 days. He was intubated in view of low GCS in an outside hospital. On presentation he had no eye opening, intubated on VCV mode, abnormal flexor response to pain, pupils bilateral normal sluggish reacting to light with left upper limb MRC grade 3 and lower limb MRC grade 3. KPS score was 20 on presentation. MRI brain with contrast (Figure 4A, 4B) revealed well defined T2/FLAIR hyperintense and TIW iso to hypointense peripherally enhancing mass lesion of size 40x46x30 mm (APXTRAXCC) is seen in the body of corpus callosum and cingulated gyrus. The patient underwent right parasagittal Fronto Parietal craniectomy for excision of lesion. Subtotal resection was achieved. Post op CT Brain Plain was done (Figure 4D). Histopathological examination (Figure 4C) shows tumor cells are moderately pleomorphic with central nuclei surrounded by clear cytoplasm with peri-nuclear halo. Chicken - wire type vasculature. Focal calcification and astro-

cytic differentiation. Micro calcification with fibrillary background. Immunohistochemistry shows IDH1 (R132H), OLIG2, ATRX: Retained Nuclear expression, p53: Occasional scattered weakly positive cells, Ki 67: ~ 4%, , suggestive of Oligodendroglioma, IDH 1 r132h mutant, CNS who grade 2. Unfortunately, the patient succumbed to sepsis on Post op day 12.

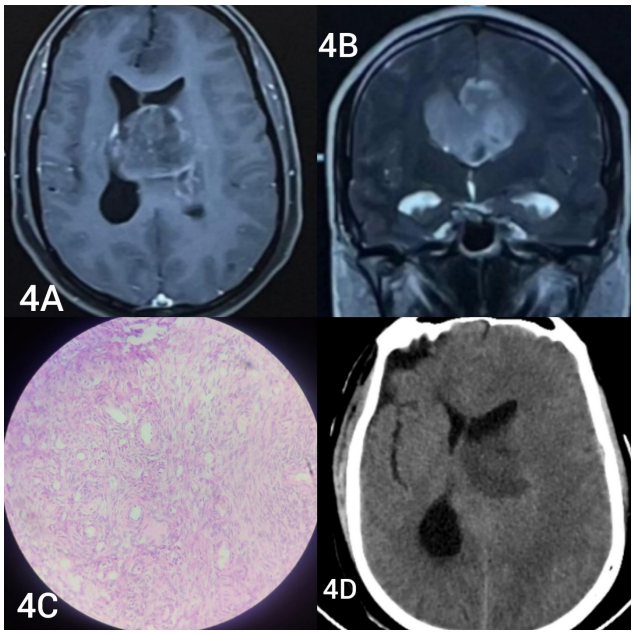


Figure 4: (A) Axial and (B) coronal T1-weighted post-contrast MRI showing a well-defined peripherally enhancing mass in the body of the corpus callosum and cingulate gyrus (Case 4). (C) Histopathology (H&E, 400×) showing classic oligodendroglial morphology with perinuclear halos and chicken-wire vasculature. (D) Postoperative CT brain plain showing subtotal excision.

DISCUSSION

Tumors involving the corpus callosum represent a complex neurosurgical and neuro-oncological challenge due to their deep midline location, tendency for bilateral spread, and potential for neurocognitive deficits.

The clinical signs of these tumors commonly include progressive headaches and motor weakness, which are often caused by large tumors that may extend into the motor pathways. The corpus callosum contains intricate fiber connections linking the two hemispheres of the brain. Using diffusion tensor imaging, Rimkus et al. examined the neuronal damage in the corpus callosum of patients with relapsing-remitting multiple sclerosis, finding significant destruction, particularly in the posterior midbody and splenium. Additionally, research on traumatic brain injury has shown that acceleration forces affecting the corpus callosum, especially at the genu, are linked to poorer outcomes. [3,4,5] Conversely, tumors that invade the splenium of the corpus callosum are also associated with a worse prognosis, as this area is involved in various pathways related to memory and cognitive functions. Pathological changes in the splenium thus contribute to a more negative prognosis.[6] Radiologically, these tumors demonstrated classical features of callosal involvement—T2/FLAIR hyperintensity, irregular peripheral enhancement, blooming on SWI, and midline crossing in high-grade cases. In our series Splenial and posterior body involvement, seen in Case 1, has been associated with poorer outcomes as reported by Tunthanathip et al. (2017). [7] other 3 cases (case 2,3,4) originate from the body of corpus callosum. Importantly, two patients (Cases 2 & 3) demonstrated midline-crossing lesions, consistent with "butterfly glioma" patterns, which are known for aggressive behaviour and limited resectability.

The present case series includes two high-grade astrocytomas (WHO grade IV) and two oligodendrogliomas (WHO grade II and III), all with corpus callosum involvement. Notably, all tumors were IDH1 mutant, indicating secondary glioma origin and potentially more favourable prognosis compared to IDH-wildtype counterparts.

Previous studies have identified several clinical factors that negatively impact the prognosis of low-grade gliomas. These include being older than 40 years at the time of diagnosis, having neurological deficits (such as seizures) before surgery, a tumor diameter greater than 6 cm, a Karnofsky Performance Status (KPS) score of less than 70, tumors crossing the midline, and an astrocytoma histology subtype.[8,9,10] For high-grade gliomas, negative factors include being over 50 years old, a KPS score under 70, the glioblastoma subtype, tumor resection exceeding 98%, and postoperative treatment with radiation and temozolomide.[11,12,13,14]. In our study, two cases (case 1 & case 3) have a KPS score of 80, one case (case 2) has a KPS score of 60 and one case (case 4) has a KPS score of 20. Multivariable analysis has shown that the glioblastoma histology subtype is a significant unfavourable predictor of survival. In gliomas, both the histologic subtype and grade play a critical role in determining prognosis.[15] The median overall survival for low-grade glioma patients ranges from 100 months to 10.5 years [8,9], 16 months for anaplastic astrocytoma, and between 6 to 14 months for glioblastoma.[16,17].

From the literature, studies by Cui et al. (2021) [18] and Forster et al. (2020) [19] support the role of multimodal surgical techniques (neuronavigation, intraoperative MRI, neuromonitoring) in safely achieving higher extent of resection (EOR), which correlates with better survival and functional outcomes. However, due to limited resources and lower socioeconomic status of patients we could not use all resources. In our cases, all patients underwent surgical excision tailored to tumor location, followed by adjuvant chemoradiotherapy. Molecular profiling further guided therapeutic strategies, particularly in distinguishing astrocytic from oligodendroglial lineages using ATRX and p53 markers. These findings reinforce the emerging view that even tumors involving the corpus callosum, once considered inoperable, can benefit from aggressive resection and individualized treatment. Integration of imaging, surgical planning, and molecular diagnostics is essential for optimizing outcomes in these patients.

CONCLUSION:

Butterfly Gliomas involving the corpus callosum are a heterogeneous group of tumors with distinct radiological and molecular characteristics. While traditionally associated with poor prognosis, advances in surgical and diagnostic techniques have enabled more aggressive and individualized management. Age, KPS score and neurological condition at presentation, IDH mutation status, histological subtype, and extent of resection remain critical determinants of outcome. Early diagnosis, integrated molecular profiling, and tailored multimodal therapy are essential to optimize survival and functional outcomes in these challenging cases.

REFERENCES

1. Bourekas, E.C., Varakis, K., Bruns, D., Christoforidis, G.A., Baujan, M., Slone, H.W., et al. Lesions of the corpus callosum: MR imaging and differential considerations in adults and children. AJR Am J Roentgenol., 200,179,1,251–257.
2. Georgy, B.A., Hesselink, J.R., Jernigan, T.L. MR imaging of the corpus callosum. AJR Am J Roentgenol. 1993,160,5,949–955.
3. Mendelsohn, D.B., Levin, H.S., Harward, H., Bruce, D. Corpus callosum lesions after closed head injury in children: MRI, clinical features and outcome. Neuroradiology. 1992,34,5,384–388.
4. Cicuendez, M., Castaño-León, A., Ramos, A., Hilario, A., Gómez, P.A., Lagares, A. Prognostic value of corpus callosum injuries in severe head trauma. Acta Neurochir (Wien). 2017,159,1,25–32.
5. Matsukawa, H., Shinoda, M., Fujii, M., Takahashi, O., Yamamot,o D., Murakata, A., et al. Genu of corpus callosum in diffuse axonal injury induces a worse 1-year outcome in patients with traumatic brain injury. Acta Neurochir (Wien). 2011,153,9,1687–1693.
6. Sammler, D., Kotz, S.A., Eckstein, K., Ott, D.V., Friederic,i A.D. Prosody meets syntax: The role of the corpus callosum. Brain. 2010,133,Pt 9,2643–2655.
7. Tunthanathip, T., Ratanalert, S., Sae-heng, S., Oearsakul, T. Butterfly tumor of the corpus callosum: Clinical characteristics, diagnosis, and survival analysis. J Neurosci Rural Pract. 2017,8,Suppl 1,S57–S65.
8. Leighton, C., Fisher, B., Bauman, G., Depiero, S., Stitt, L., MacDonald, D., et al. Supratentorial low-grade glioma in adults: An analysis of prognostic factors and timing of radiation. J Clin Oncol. 1997,15,4,1294–1301.
9. Lote, K., Egeland, T., Hager, B., Stenwig, B., Skullerud, K., Berg-Johnsen, J, et al. Survival, prognostic factors, and therapeutic efficacy in low-grade glioma: A retrospective study in 379 patients. J Clin Oncol. 1997,15,10,3129–3140.
10. Pignatti, F., van den Bent, M., Curran, D., Debruyne, C., Sylvester, R., Therasse, P., et al. Prognostic factors for survival in adult patients with cerebral low-grade glioma. J Clin Oncol. 2002,20,8,2076–2084.
11. Lacroix, M., Abi-Said, D., Fourney, D.R., Gokaslan, Z.L., Shi, W., DeMonte, F., et al. A multivariate analysis of 416 patients with glioblastoma multiforme: Prognosis, extent of resection, and survival. J Neurosurg. 2001,95,2,190–198.
12. Sarica, F.B., Cekinmez, M., Tufan, K., Sen, O., Onal, H.C., Mertsoylu, H., et al. Five-year follow-up results for patients diagnosed with anaplastic astrocytoma and effectiveness of concomitant therapy with temozolomide for recurrent anaplastic astrocytoma. Asian J Neurosurg. 2012,7,4,181–189.
13. Stupp, R., Hegi, ME., Mason, W.P., van den Bent, M.J., Taphoorn, M.J., Janzer, R.C., et al. Effects of radiotherapy with concomitant and adjuvant temozolomide versus radiotherapy alone on survival in glioblastoma in a randomised phase III study: 5-year analysis of the EORTC-NCIC trial. Lancet Oncol. 2009,10,5,459–466.
14. Wen, P.Y., Kesari, S. Malignant glioma in adults. N Engl J Med. 2008,359,5,492–507.
15. Iwamoto, F.M., Reiner, A.S., Nayak, L., Panageas, K.S., Elkin, E.B., Abrey, L.E. Prognosis and patterns of care in elderly patients with glioma. Cancer. 2009,115,23,5534–5540.
16. Filippini, G., Falcone, C., Boiardi, A., Broggi, G., Bruzzone, M.G., Caldiroli, D., et al. Prognostic factors for survival in 676 consecutive patients with newly diagnosed primary glioblastoma. Neuro Oncol. 2008,10,1,79–87.
17. Ma, X., Lv, Y., Liu, J., Wang, D., Huang, Q., et al. Survival analysis of 205 patients with glioblastoma multiforme: Clinical characteristics, treatment and prognosis in China. J Clin Neurosci. 2009,16,12,1595–1598.
18. Cui, M., Chen, H., Sun, G., Liu, J., Zhang, M., Lin, H., et al. Combined use of multimodal techniques for the resection of glioblastoma involving corpus callosum. Acta Neurochir (Wien). 2022,164,3,689–702. doi:10.1007/s00701-021-05008-6
19. Forster, M.T., Behrens, M., Lortz,.I, et al. Benefits of glioma resection in the corpus callosum. Sci Rep. 202,10,16630. doi:10.1038/s41598-020-73928-x

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