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Case Report

Left Insular Low-Grade Glioma: A Rare Comprehensive Case Report

Sapthami Ramya V.G*, Deva Dharshini, Charanya A.G, Shailaja.K

C L Baid Metha College of Pharmacy, Affiliated to "The Tamil Nadu Dr. MGR Medical University", Chennai, Tamil Nadu, INDIA.

ABSTRACT

Low-Grade Gliomas (LGGs) are a specific type of brain tumor that are characterized by slow-growing, less aggressive cancerous cells that originate from glial cells, which support and protect neurons in the brain and spinal cord. The insula is a small region of the cerebral cortex located deep within the lateral sulcus, and tumors in this area are particularly challenging to treat due to the critical functions it supports, such as language, sensory processing, and autonomic control. Left Insular Low-Grade Gliomas (LILGGs) are a rare and particularly complex form of LGG. Although these tumors grow slowly, they have the potential to become malignant over time. They arise from neuroepithelial tissue and are derived from glial cells. Despite their rarity, LILGGs are well-documented in medical literature. This case report discusses a 50-year-old female patient diagnosed with a left insular low-grade glioma. The patient presented with symptoms that warranted further investigation, leading to the diagnosis of this uncommon tumor. The detailed analysis of her case adds to the existing body of knowledge, providing insights into the presentation, diagnosis, and potential treatment approaches for this rare and challenging condition.

Keywords: Case Report, Left Insular Low-Grade Glioma, Brain Tumour, Rare Cerebral Cancer.

Correspondence:
Ms. Sapthami Ramya V.G
C L Baid Metha College of Pharmacy, Affiliated to "The Tamil Nadu Dr. MGR Medical University", Chennai, Tamil Nadu, INDIA.
Email: sapthami35@gmail.com

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INTRODUCTION

Gliomas are a diverse group of tumors originating from glial cells in the Central Nervous System (CNS). Glial cells, which include astrocytes, oligodendrocytes, and ependymal cells, provide support and protection for neurons. Gliomas are the most common type of primary brain tumor, with an incidence of approximately six cases per 100,000 people annually in the United States. They are notable for their diffuse and infiltrative nature, making them challenging to treat as they often extend into surrounding brain tissue.[1]

Gliomas can vary significantly in their malignancy and prognosis. The World Health Organization (WHO) classifies gliomas into four grades based on histopathological and molecular features:

Grade I: Pilocytic astrocytoma, which are the least malignant and often affect children.

Grade II: Low-grade gliomas, which grow more slowly and can include diffuse astrocytoma and oligodendrogliomas.

Grade III: Anaplastic gliomas, which are more aggressive and rapidly growing.

Grade IV: Glioblastomas, which are the most malignant and aggressive form of gliomas.[1]

The classification of gliomas has evolved over time, with recent advancements emphasizing molecular and genetic markers. These include mutations in the Isocitrate Dehydrogenase 1 (IDH1) and Isocitrate Dehydrogenase 2 (IDH2) genes, 1p/19q co-deletion status, and the presence of certain genetic alterations like O (6)-Methylguanine-DNA-Methyltransferase (MGMT) promoter methylation. These markers have significant prognostic and therapeutic implications, allowing for more tailored and effective treatment strategies.[2]

Gliomas present with a variety of symptoms depending on their location within the CNS. Common symptoms include headaches, seizures, cognitive or personality changes, and focal neurological deficits. Imaging studies, particularly MRI, play a crucial role in the diagnosis and assessment of gliomas.[3]

Treatment for gliomas typically involves a combination of surgery, radiation therapy, and chemotherapy. The approach depends on the tumor's grade, location, and genetic characteristics. Despite advances in treatment, gliomas, especially high-grade ones, remain challenging to cure, and research continues to focus on improving outcomes and developing new therapies.[4]

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CASE PRESENTATION

A 50-year-old female presented with complaints of sudden onset of giddiness on and off since 1 month associated with impaired consciousness, seizure disorder and has been on anti-epileptic drugs T. Clinidipine-5 mg BD, T. Levipil-500 mg BD, T. Olanzapine-5 mg HS. The patient is a known case of Hypertension for 5 years and is on regular medication. From the clinical history, it was found that she had undergone Laparoscopic Oophorectomy done 5 years back. The patient was admitted for further evaluation and management.

On examination, the patient was found to be conscious and oriented to time place, and person, afebrile, vitals showed GCS-15/15, PERTL 2 mm, EOM full, VAFC@ 3Ft, power 5/5 at all limbs, no cranial nerve defects and vision was grossly normal.

All the hematological investigations were within normal limits. She underwent a surgical procedure Awake craniotomy-Left Fronto temporal navigation guided and subtotal excision of the lesion under intraoperative language mapping of the left side speech area, motor and sensory areas. Insula reached beyond the Middle Cerebral Artery (MCA) and its branches. Insular colectomy and lesion entered and debulked. Tumor tissue was sent for frozen section biopsy which reported Low low-grade glioma. (Table 1). The MRI Status post left Frontotemporal Craniotomy is shown in Figure 1.

The tumour was found not vascular. MCA and its branches were identified and preserved. Small feeding arteries to tumour coagulated and cut. During the tumour resection, the patient's speech was checked repeatedly. At one stage during the tumour removal on the frontal side, the brain started swelling and hematoma was evacuated from the frontal lobe inferiorly. Brain swelling worsened and patient was becoming drowsy, had difficulty breathing and maintain airway. At thi stage tumour removal was stopped. The patient was aroused assessed and was conscious but not speaking and right-side limb weakness. Patient was shifted for imaging.

Post op CT showed Status post left frontal-temporal craniotomy. Intraparenchymal haemorrhage with surrounding edema seen in left frontal region measuring-38*27 mm. Post operative changes seen in adjacent frontal and temporal region with pneumocephalus and haemorrhagic changes. Gyrus thickening with FLAIR hyperintensity noted in left inferolateral Basi frontal and median temporal regions. Subtle mass effect with compression of ipsilateral lateral ventricle seen. On post-op day, the patient GCS E4V4M6, PERTL1.5mm, right sided weakness, vitals stable. Patient was started on 3% NaCl at 30 mL per hour. Continuous critical care monitoring was given and continued on Antibiotics, Steroids and AEDs and Inj. Nimodipine. Patient was weaned off from 3% NaCl and Nimodipine and further started on Cilnidipine.

The neurological status was explained and counselled the patient regarding redo surgery. After three days Left Fronto temporal parietal Re-do craniotomy and evacuation of Left frontal ICH and Gross total excision of left Basi frontal and left mesial temporal Low-grade glioma done. Post op, patient was started on Inj. Magnex forte, Plasmolyte 100 mL/hr and 3% NS at 30 mL/hr, Analgesics, Steroids and AEDs were continued. Broca Aphasia was found and advised to follow up for speech therapy. On the post-op examination, the patient had same neurological exam along with stable vitals and Right hemiparesis and was started on T. Quietiapine-25 mg stat dose and antibiotics were stopped along with steroids. Patient improved symptomatically and was discharged with T. Levipil-500 mg, T. Clinidipine-5mg, T. Qutipine-25 mg, T. Dolo-650 mg, T. A-Z, Syrup.Cremaffin-15 mL.

DISCUSSION

In our patient, the diagnosis of Left insular low-grade glioma was based on the awake craniotomy (Left frontotemporal navigation guided and subtotal excision of lesion under intra operative language mapping done on 30/08/2022). The common risk factors of low-grade glioma includes genetic factors, age, exposure to radiation, environmental, Immune system factors and may be due to life style modification.[5]

On 30/09/22 Awake craniotomy procedure, patient during intra OP mapping of left side speech area motor and sensory area was done. Sylvain fissue split. Insula reached beyond MCA and its

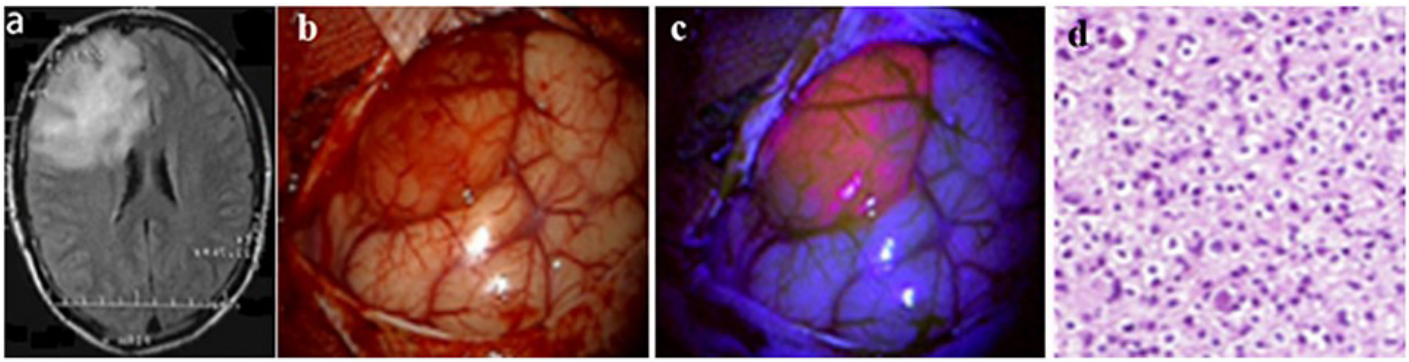

Figure 1: MRI: Status post left frontotemporal craniotomy.

Table 1: Pathology Lab Tests Results.

Clinical Notes
H/O of sudden onset giddiness since 1 month. Seizure disorder. MRI-Insular space occupying lesion, minimal contrast enhancing.
Specimen
H22-2615) Insular temporal space occupying lesion, Frozen section H22-2628) Tumour tissue in formalin.
Macroscopy
H22-2615) Insular temporal space occupying lesion (Frozen section): Received multiple grey, white to grey, brown soft tissue fragments altogether measuring 0.4*0.3 cm. All embedded in one block. Received multiple grey, white to grey, brown soft tissue fragments altogether measuring 1.2*0.5 cm. All embedded in one block. H22-2628) Tumour tissue: Received multiple grey, white to grey, brown soft tissue fragments altogether measuring 2.5*2*0.5 cm. All embedded in one block.
Microscopy: H22-2615&H22-2628 Insular temporal space occupying lesion, Excision biopsy (Frozen & H&E section): Sections studied showed glial tumour with fibrillary background. There is minimal increase in cellularity of astrocytes with mild nuclear pleomorphism. Few scattered large ganglion-like cells with abundant eosinophilic cytoplasm and large nuclei made out. No evidence of atypia, increase cellularity, mitosis, microvascular proliferation or tumour necrosis. No evidence of foamy histiocyte collections/inflammatory cells for pseudo-tumour. No high-grade features made out.
Immunohistochemistry GFA: Stronlgy positive in tumour cells. Chromogranin: Strong cytoplasmic positivity in ganglion cells. CD-34: Positive in ganglion cells. ATRX: Patchy positivity in tumour cells. Synaptophysin: Weak cytoplasmic positivity in ganglion cells. IDH-1 (R132H): Negative in tumour cells. p53: Negative in glial & ganglion cells. CD-68: Negative in glial & ganglion cells. BRAF (V600E mutation): Negative in glial and ganglion cells. Ki-67(Mitotic proliferative index): 1.2%.
Diagnosis Low Grade Glioma, Consistent with Ganglioglioma (Who Grade I).

branches. Tumor tissue was sent for frozen section biopsy and reported Low Grade Glioma.

The insular tumor was resected using CUSA and suction up to the circular sulcus. The tumor was found to be not vascular. During the removal stage on the frontal side the brain started to swell, and hematoma was evacuated from the frontal lobe inferiorly. The patient's condition worsened, and surgery was stopped.

MRI Brain revealed about status post left frontal temporal craniotomy, Intra parenchymal hemorrhage with surrounding edema seen in frontal region measuring 38*27 mm. Post operative changes seen in adjacent frontal and temporal region with pneumocephalus and hemorrhagic changes. Gyral thickening with FLAIR hyperintensity noted in left inferolateral Basi frontal and median temporal regions. Subtle mass effect with compression of ipsilateral lateral ventricle seen.

Complications of Glioma include seizure, headache, nausea, vomiting, personality changes and problem in speaking.[6]

Brain tumors are ranked I-IV on the World Health Organization (WHO) scale, where I and II are considered low grade and III and IV are considered high grade. A tumor's potential growth rate and size determine its grade. Benign tumors known as low-grade gliomas can result from environmental or genetic alterations. Low-grade gliomas might show differently according on the size and location of the tumor.[7] According to WHO our case represent Grade I Left Insular Low-Grade Glioma.

CONCLUSION

In conclusion, a low-grade glioma in the left insular region presents unique challenges due to its location and potential impact on critical brain functions, such as language, sensory processing, and autonomic control. These tumors, while generally less aggressive than high-grade gliomas, can still lead to significant complications, including neurological deficits, seizures, cognitive and behavioral changes, and treatment-related side effects.

Advancements in molecular and genetic profiling have improved gliomas' classification, prognostication, and treatment, allowing for more personalized therapeutic approaches. Despite these advancements, managing a low-grade glioma in the left insular region requires careful consideration of the risks and benefits of various treatment modalities, including surgery, radiation, and chemotherapy.

A multidisciplinary approach involving neurology, neurosurgery, oncology, and supportive care is essential to address the complex needs of patients, aiming to optimize outcomes and maintain quality of life. Ongoing research and clinical trials continue to seek better treatment options and improve the prognosis for patients with low-grade gliomas in this sensitive and functionally important brain region.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

LGGs: Low-grade gliomas; **LILGGs:** Left insular low-grade gliomas; **CNS:** Central nervous system; **WHO:** World Health Organization; **IDH1:** Isocitrate dehydrogenase 1; **IDH2:** Isocitrate dehydrogenase 2; **MGMT:** O (6)-Methylguanine-**DNA:** methyltransferase; **MRI:** Magnetic Resonance Imaging; **GCS:** Glasgow Coma Scale; **MCA:** Middle Cerebral Artery; **GFA:** Glial Fibrillary Acidic; **ATR-X:** Alpha thalassemia and mental retardation X: linked; **BRAF:** B-Raf Proto-Oncogene, Serine/ Threonine Kinase; **CUSA:** Cavitation ultrasonic surgical aspiration; **FLAIR:** Fluid-attenuated inversion recovery.

SUMMARY

Low-grade gliomas in the left insular region present unique challenges due to their impact on brain functions. Although advances in molecular and genetic profiling have been made, managing these tumors necessitates careful consideration of treatment modalities. A multidisciplinary approach that involves neurology, neurosurgery, oncology, and supportive care is crucial to optimize outcomes and maintain the quality of life.

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