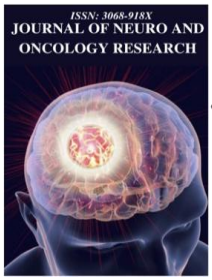

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Case Report

Autoimmune-Associated Lymphocytic Central Nervous System (CNS) Vasculitis Presenting as a Tumefactive Cerebral Infarct Mimicking Multicentric Glioma

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Abstract

Purpose: Central nervous system vasculitis may rarely present as a tumor-like lesion with enhancement, edema, mass effect and multifocality, closely mimicking glioma. We report an uncommon case of autoimmune-associated lymphocytic CNS vasculitis presenting as a tumefactive cerebral infarct radiologically mimicking multicentric glioma.

Methodology: Clinical history, neurological examination, preoperative imaging, operative findings, histopathology, immunohistochemistry, postoperative course and blood-based autoimmune serology were reviewed and summarized.

Key Findings: A 50-year-old hypertensive woman presented with increased forgetfulness, slurring of speech, expressive speech difficulty and mild headache. MRI demonstrated an irregular, ill-defined, heterogeneously enhancing left fronto-temporo-parietal/perisylvian lesion with significant perilesional edema, mass effect, approximately 5 mm midline shift and an additional right cerebellar lesion, raising suspicion of multicentric glioma. She underwent exoscopic neuronavigation-guided maximum safe resection under intraoperative neurophysiological monitoring. Histopathology revealed cerebral infarction, while follow-up pathology/IHC assessment supported lymphocytic vasculitis. Blood-based autoimmune serology showed ANA, anti-dsDNA, anti-RNP/RNP-C, p-ANCA and ANCA-lactoferrin positivity. Corticosteroid therapy was initiated by the rheumatology team. Postoperatively, speech improved and no new neurological deficit was observed.

Conclusion: Autoimmune-associated lymphocytic CNS vasculitis may present as a tumefactive cerebral infarct mimicking multicentric glioma. Tissue diagnosis is essential when clinical and radiological findings are discordant.

Keywords: CNS Vasculitis; Lymphocytic Vasculitis; Tumefactive Infarct; Cerebral Infarct; Multicentric Glioma Mimic; Autoimmune Vasculitis; Anti-RNP; p-ANCA

Abbreviations

ANA: Antinuclear Antibody; ANCA: Antineutrophil Cytoplasmic Antibody; CNS: Central Nervous System; CUSA: Cavitron Ultrasonic Surgical Aspirator; GRE: Gradient Recalled Echo; HPE: Histopathological Examination; IHC: Immunohistochemistry; MCA: Middle Cerebral Artery; MRI: Magnetic Resonance Imaging; P-ANCA: Perinuclear Antineutrophil Cytoplasmic Antibody; RNP: Ribonucleoprotein; SWI: Susceptibility-Weighted Imaging.

Introduction

Central nervous system vasculitis is an inflammatory disorder of cerebral blood vessels that may result in ischemic infarction,

hemorrhage, seizures, cognitive decline, focal neurological deficits or encephalopathy. It may occur as a primary disorder confined to the CNS or as secondary CNS involvement associated with systemic autoimmune, inflammatory, infectious or vasculitic diseases. Tumor-like presentation of CNS vasculitis is rare but clinically important because it can mimic high-grade glioma, lymphoma, metastasis or tumefactive demyelinating disease on neuroimaging.

Previous reports have documented primary angitis of the CNS and lymphocytic vasculitis presenting as mass-like brain lesions mimicking neoplasm. Panchal et al. reported biopsy-proven lymphocytic vasculitis mimicking aggressive multifocal cerebral neoplasm on MRI and MR spectroscopy [1]. Jin, et al., described primary angiitis of the CNS mimicking glioblastoma [2]. Suthiphosuwan et al. reported a biopsy-confirmed series of tumefactive primary CNS vasculitis, supporting tumefactive CNS vasculitis as a rare but recognized neuroinflammatory tumor mimic [3].

We report a rare case of autoimmune-serology-positive lymphocytic CNS vasculitis presenting as a tumefactive left perisylvian cerebral infarct, radiologically mimicking multicentric glioma. The case is clinically relevant because preoperative imaging suggested a neoplastic lesion, whereas histopathology established cerebral infarction, follow-up pathology/IHC assessment supported lymphocytic vasculitis and blood-based autoimmune serology suggested a secondary autoimmune vasculitic mechanism.

Case Description and Methodology

Patient Presentation

A 50-year-old hypertensive woman presented with recurrent forgetfulness, speech difficulty with slurring and mild headache for 2 months.

She developed forgetfulness which increased gradually along with slurred speech. MRI brain at previous hospital showed a left fronto-temporo-parietal lesion, interpreted as a subacute infarct with hemorrhagic transformation. She was managed conservatively with cerebral decongestants after which she improved to some extent.

Eight days before the admission, she had increased forgetfulness with expressive speech difficulty. She could comprehend speech but had difficulty forming words. Mild non-progressive headache was present for two days before admission which was relieved with medication. There was no documented history of vomiting, giddiness, limb weakness, loss of consciousness, complete aphasia or seizures.

Clinical Examination on Admission

On admission, the patient was conscious and obeying commands. Pupils were bilaterally equal and reactive to light. Extraocular movements were full; however, left eye squint with esotropia was noted. Speech examination revealed expressive dysphasia, suggestive of dominant perisylvian language center involvement, while comprehension was preserved. Motor examination showed a power of 5/5 in all four limbs. The overall admission status was consistent with a conscious, cooperative patient with predominant language dysfunction and forgetfulness, without limb weakness.

Diagnostic Assessment

MRI brain with contrast demonstrated an irregular, ill-defined, heterogeneous lesion involving the left fronto-temporo-parietal opercular and insular cortical-subcortical regions, measuring approximately 4.7 × 5.7 × 4.2 cm.

The lesion was heterogeneously hyperintense on T2/FLAIR images and predominantly hypointense on T1-weighted images. Areas of gyriform T1 hyperintensity were noted. Blooming was seen on gradient echo images, suggestive of hemorrhagic or calcific components. Mild heterogeneous post-contrast enhancement was present. The lesion was associated with significant perilesional edema and mass effect, causing partial effacement of the left lateral ventricle and approximately 5 mm midline shift toward the right.

A second smaller cortical-based nodular lesion was seen in the inferomedial right cerebellar hemisphere, showing similar imaging characteristics with patchy enhancement. Based on these findings, the preoperative radiological impression favored multifocal cortical neoplastic lesions, with multicentric glioma considered as a major diagnostic differential (Fig. 1).

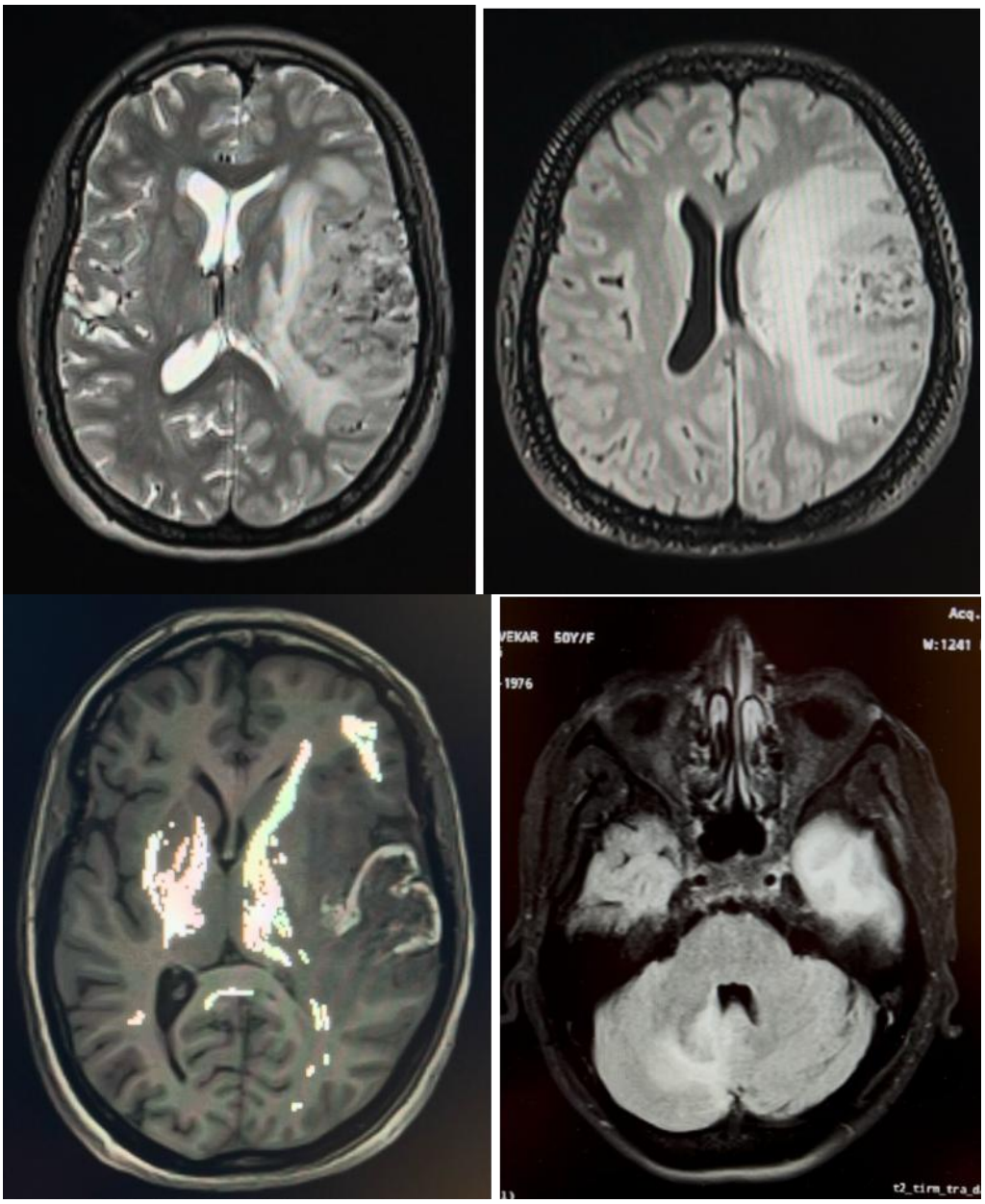

Figure 1: Preoperative MRI brain. Preoperative MRI brain showing an irregular, ill-defined, heterogeneously enhancing lesion involving the left fronto-temporo-parietal/perisylvian region with significant perifocal edema, mass effect, partial effacement of the left lateral ventricle and midline shift. An additional smaller lesion is seen in the right cerebellar hemisphere (last image), raising suspicion of multicentric glioma.

The lesion was radiologically concerning for glioma because of its irregular infiltrative appearance, cortical-subcortical involvement, heterogeneous enhancement, edema, mass effect, midline shift and multifocality. However, the prior imaging impression of subacute infarct with hemorrhagic transformation created diagnostic uncertainty. In view of recurrent neurological symptoms, interval lesion evolution, mass effect and suspicion of neoplasm, operative intervention was planned for maximum safe resection and definitive tissue diagnosis.

Therapeutic Intervention

The patient underwent exoscopic neuronavigation-guided maximum safe resection of the left perisylvian lesion under intraoperative neurophysiological monitoring. Under general anesthesia, a left temporo-parietal C-shaped skin incision was made, followed by left temporo-parietal craniotomy. The dura was opened with its base inferiorly. The lesion was seen surfacing at the cortical surface. Intraoperatively, it appeared yellowish-red, soft, highly vascular and was amenable to ultrasonic aspiration.

The lesion involved the eloquent perisylvian language region. Using the Cavitron Ultrasonic Surgical Aspirator (CUSA), maximum safe resection was performed through two small

corticectomies, one temporal and one parietal. Tissue involving eloquent cortex and tissue adherent to branches of the middle cerebral artery within the Sylvian fissure was deliberately left behind to minimize postoperative neurological morbidity. The extent of excision was confirmed using neuronavigation.

Cerebrospinal fluid egress was noted intraoperatively, after which the brain became lax. Meticulous hemostasis was achieved. Motor evoked potentials remained unchanged from baseline throughout the procedure. The dura was closed watertight, the bone flap was replaced and the wound was closed in layers. The operative procedure was uneventful and maximum safe resection was achieved (Fig. 2).

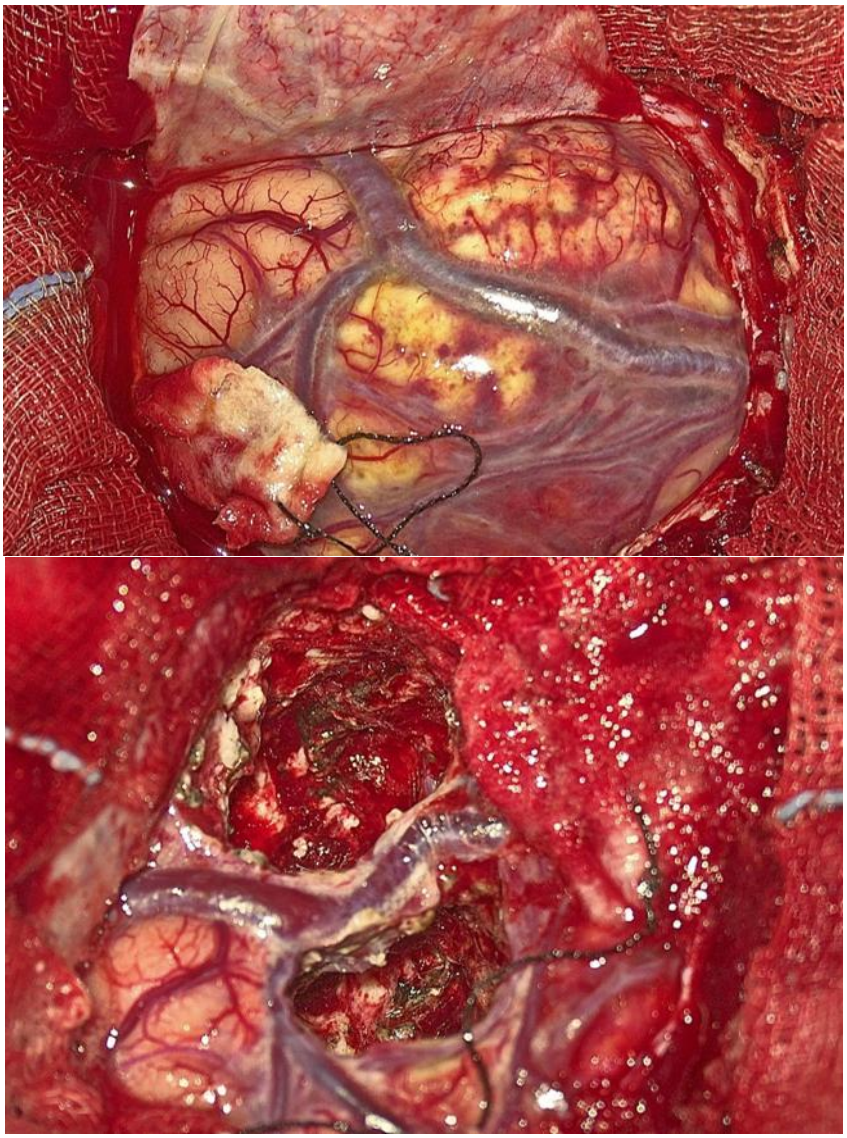

Figure 2: Intra-operative findings: The lesion was seen surfacing at the cortical surface. Intraoperatively, it appeared yellowish-red, soft, highly vascular and was amenable to ultrasonic aspiration.

Histopathology, Immunohistochemistry and Blood-Based Autoimmune Serology

Gross examination showed multiple submitted tissue bits, together measuring approximately 3.1 × 2.5 × 1.5 cm.

Microscopy revealed cerebral tissue with large areas of ischemic-type necrosis and foci of extravasated red blood cells. The necrotic areas were surrounded by capillaries and a mixed inflammatory cell infiltrate rich in foamy histiocytes. The surrounding brain parenchyma showed astrogliosis with a few gemistocytic astrocytes. A mixed inflammatory cell sprinkling was present, along with moderate to severe perivascular lymphoid infiltrate.

The initial histopathological impression was consistent with cerebral infarct. On follow-up pathology/IHC assessment, the diagnosis was refined to cerebral infarct with lymphocytic vasculitis, with the report noting perivascular lymphoid infiltrate with vasculitis and some histiocytic/epithelioid cell collections. This finding was discordant with the preoperative radiological impression of glioma or multicentric glioma and supported an inflammatory vascular mechanism rather than neoplasm (Fig. 3).

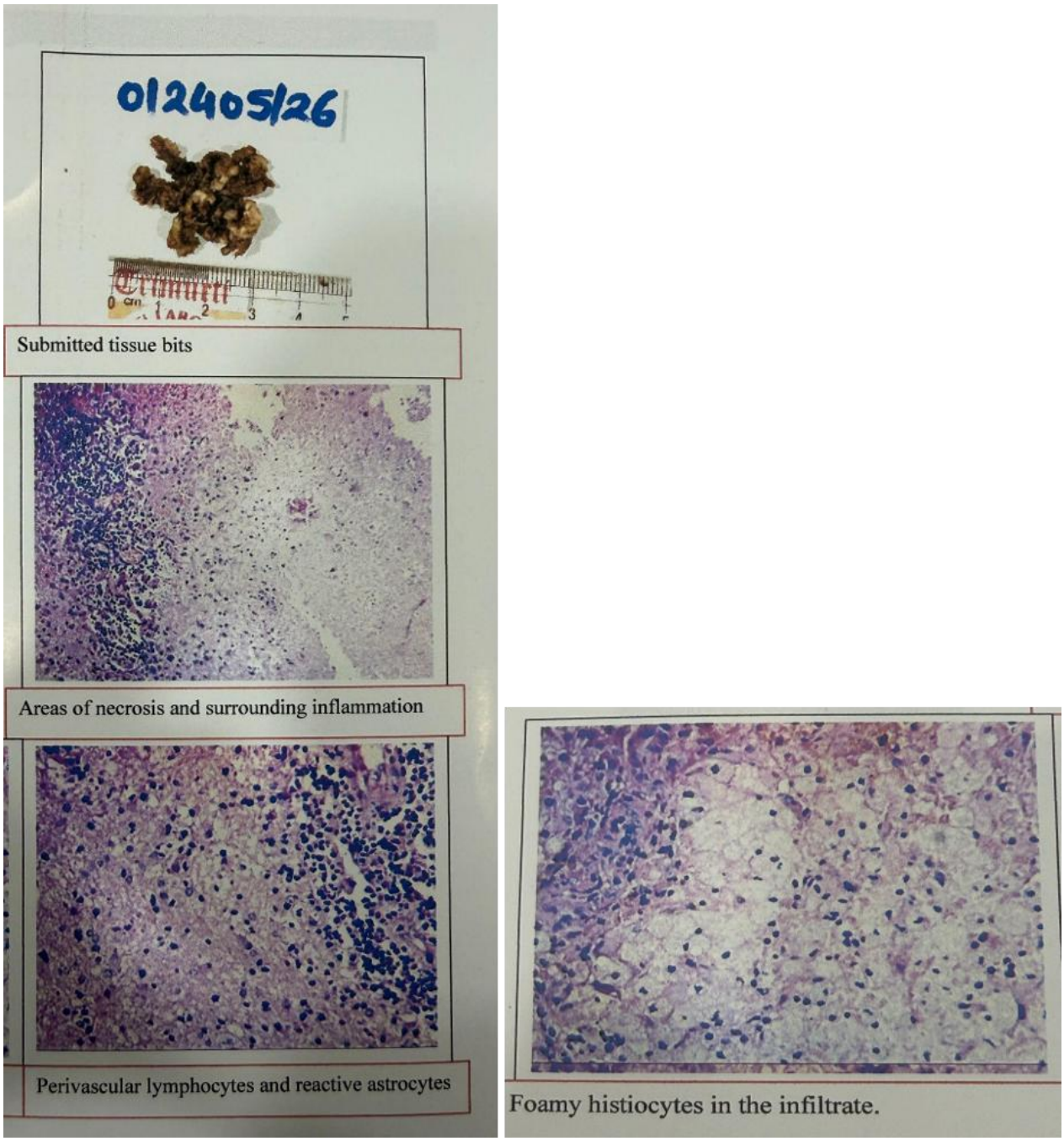

Figure 3: Microscopy Findings: Areas of necrosis are of ischemic type and are surrounded by some capillaries and mixed inflammatory cell infiltrate. The infiltrate is rich in foamy histiocytes.The surrounding brain tissues show astrocytosis with a few gemistocytic astrocytes. There is mixed inflammatory cell sprinkling.

In view of the histopathology-radiology discordance and the finding of lymphocytic vasculitis, rheumatology consultation was obtained to evaluate for an underlying systemic autoimmune or inflammatory etiology.

Blood-based autoimmune serology included (Table 1):

Test/Marker	Result Reported
ANA by IFA	Positive, reported as ++
ANA Pattern	Homogeneous and cytoplasmic reticular/AMA pattern, reported as ICAP AC-1 and AC-21
ANCA ELISA Profile	Positive antibody to lactoferrin, value 2.44
Anti-RNP	Positive on blood test
RNP-C	Positive on blood test
p-ANCA	Positive on blood test
Anti-dsDNA	Positive on blood test

Table 1: Blood-based autoimmune serology.

ANA positivity and anti-dsDNA positivity may support lupus-spectrum disease in the appropriate clinical setting. Anti-RNP/RNP-C positivity may be seen in mixed connective tissue disease, overlap connective tissue disease or lupus-spectrum autoimmunity, whereas ANCA-related/lactoferrin antibody positivity may support an associated autoimmune or inflammatory vascular process. However, this should not be equated with classic MPO/PR3 ANCA-associated vasculitis unless MPO/PR3 positivity and systemic features are documented. Antibody positivity alone is not sufficient to assign a final systemic autoimmune diagnosis without full clinical correlation.

In view of the tissue diagnosis of lymphocytic vasculitis and positive autoimmune profile, an autoimmune-associated secondary CNS vasculitic process was considered. Corticosteroid therapy was initiated by the rheumatology team.

Postoperative Course and Outcome

Postoperatively, the patient remained hemodynamically stable. There were no new neurological deficits. Her speech improved significantly compared with the preoperative state and she was able to speak in short phrases and sentences with effort. Postoperative CT brain performed on postoperative day 1 showed left fronto-temporal craniotomy changes with a postoperative cavity in the left temporal lobe, near-complete excision of the space-occupying lesion, interval improvement in midline shift and residual perilesional edema.

Neutrophil-to-Lymphocyte Ratio (NLR)

Before surgery and corticosteroids: WBC 6,400 cells/mm3, neutrophils 60%, lymphocytes 38% (ANC 3,840; ALC 2,432 cells/mm3), giving an NLR of 1.58. After surgery/treatment: WBC 9,000 cells/mm3, neutrophils 60%, lymphocytes 36% (ANC 5,400; ALC 3,240 cells/mm3), giving an NLR of 1.67. The small change was interpreted cautiously because the second sample followed surgery and corticosteroid exposure (Fig. 4).

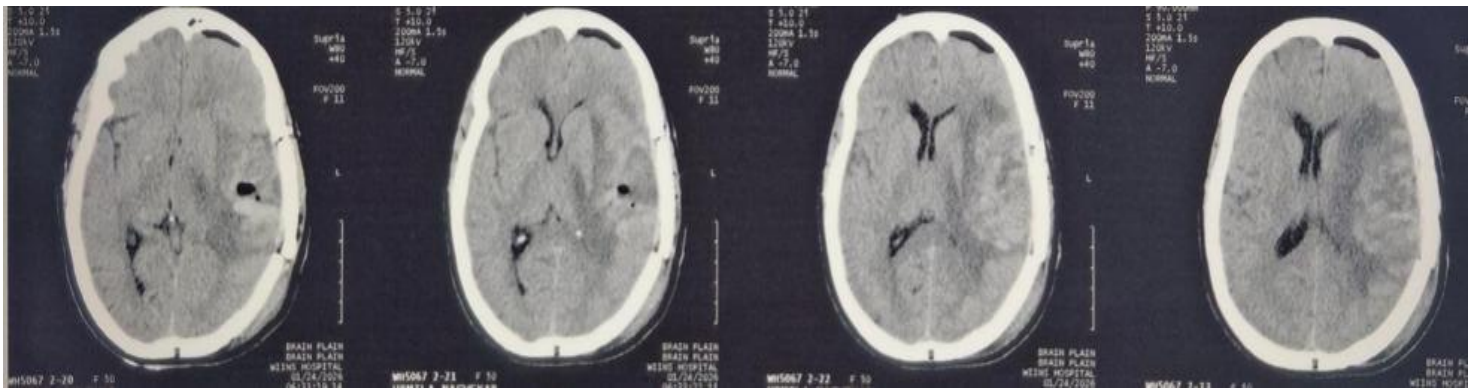

Figure 4: Post-op CT Brain.

Postoperative MRI performed on postoperative day 10 showed left temporal craniotomy changes with a postoperative defect in the left temporal lobe and near-complete excision of the previously seen left fronto-temporo-parietal lesion. Persistent perifocal edema was present in the left temporal and frontoparietal regions, with mass effect causing effacement of adjacent sulcal spaces, the left Sylvian fissure and the left lateral ventricle. The right cerebellar lesion showed altered signal with T2/FLAIR hyperintensity and GRE blooming, suggestive of resolving hemorrhagic infarct (Fig. 5).

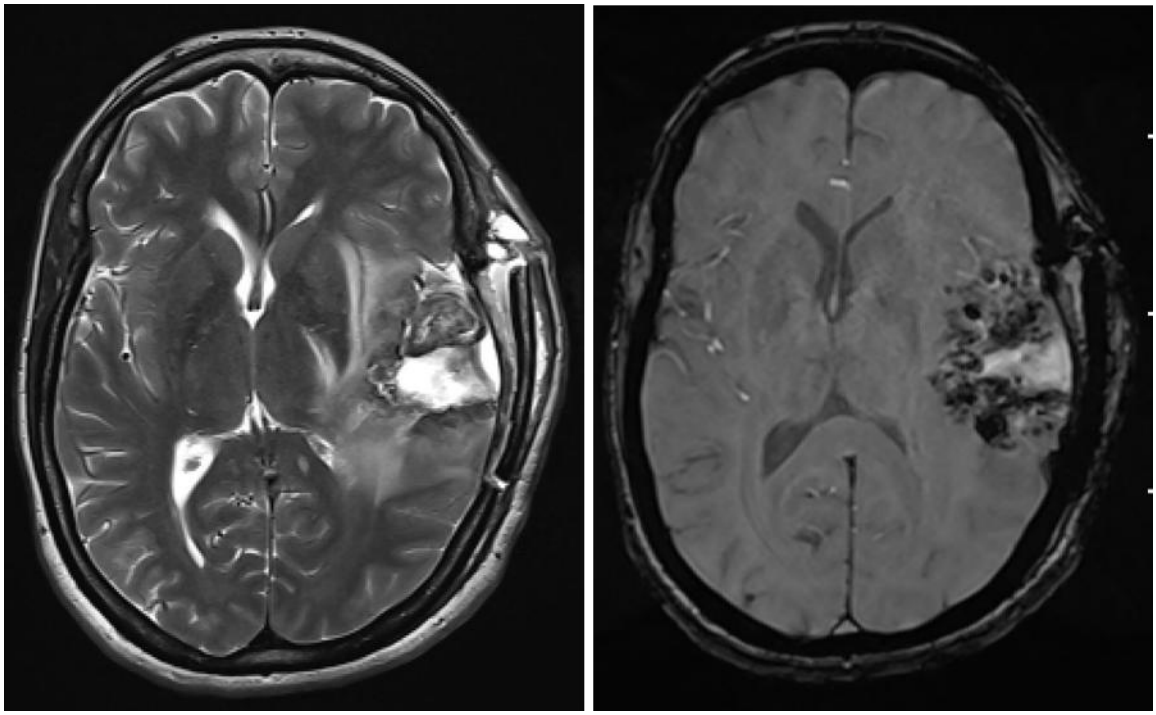

Figure 5: Postoperative MRI brain. Postoperative MRI brain showing near-complete excision of the left fronto-temporo-parietal/perisylvian lesion with residual perifocal edema and mass effect. The right cerebellar lesion showed features suggestive of resolving hemorrhagic infarct.

Postoperatively, diffuse perifocal edema was managed with cerebral decongestant therapy. The patient remained neurologically stable, with no deterioration after discontinuation of decongestants. By discharge, her speech had improved substantially, comprehension was preserved and motor power was 5/5 in all four limbs. The surgical wound was healthy and she was discharged in a stable neurological condition with advice for follow-up (Table 2).

Time Point	Clinical Event
Approximately 2 months before admission	Sudden onset of forgetfulness and slurring of speech. MRI performed elsewhere suggested a left fronto-temporo-parietal lesion, initially interpreted as a subacute infarct with hemorrhagic transformation.
After initial treatment	Partial clinical improvement with persistent word-finding difficulty.
8 days before admission	Recurrence of forgetfulness and expressive speech difficulty.
2 days before admission	Mild, non-progressive headache relieved with medication.
Preoperative MRI	MRI demonstrated a left fronto-temporo-parietal/perisylvian lesion with surrounding edema, contrast enhancement and mass effect, along with an additional right cerebellar lesion. Multicentric glioma was considered in the differential diagnosis.
Hospital admission	Admitted for further evaluation and surgical management.

Surgery	Underwent exoscopic neuronavigation-guided maximum safe resection under neurophysiological monitoring.
Postoperative day 1	CT scan showed expected postoperative changes, residual edema and interval improvement in the midline shift.
Postoperative day 10	MRI demonstrated near-complete excision of the lesion and a resolving right cerebellar hemorrhagic infarct.
Postoperative period	Histopathological examination showed cerebral infarction; subsequent pathology and immunohistochemistry assessment supported a diagnosis of lymphocytic vasculitis.
Rheumatology evaluation	Blood investigations revealed positive ANA, anti-dsDNA, anti-RNP/RNP-C, p-ANCA and ANCA-lactoferrin antibodies. Corticosteroid therapy was initiated.
Discharge	Discharged in stable condition with improved speech and no residual motor deficit.

Table 2: Timeline details with the events.

Discussion

This case demonstrates a rare and clinically important diagnostic dilemma: autoimmune-associated lymphocytic CNS vasculitis presenting as a tumefactive cerebral infarct and mimicking multicentric glioma. The patient had recurrent cognitive and language symptoms with headache. MRI showed an irregular enhancing left perisylvian/fronto-temporo-parietal lesion with edema, mass effect, midline shift and an additional cerebellar lesion. These features reasonably suggested a multifocal neoplastic process before surgery (Table 3).

Literature Comparison		
Author/Year	**Reported Diagnosis**	**Tumor Mimic**
Panchal, et al., 2005 [1]	Lymphocytic vasculitis	Aggressive multifocal cerebral neoplasm
Jin, et al., 2019 [2]	Primary angiitis of the CNS	Glioblastoma
Suthiphosuwan, et al., 2020 [3]	Tumefactive primary CNS vasculitis	Mass-like brain lesions
Zedde, et al., 2024 [4]	Tumor-like lesions in primary angiitis of the CNS	Brain tumors, including glioma-like lesions
Present case	Autoimmune-associated lymphocytic CNS vasculitis with tumefactive cerebral infarction	Multicentric glioma

Table 3: Literature comparison.

Tumor-like CNS vasculitis is uncommon but well documented. Panchal et al. described lymphocytic vasculitis mimicking aggressive multifocal cerebral neoplasm, closely resembling the present case in terms of multifocal tumor-like imaging and lymphocytic vasculitic pathology [1]. Jin, et al., reported primary angiitis of the CNS initially misdiagnosed as glioblastoma in a 35-year-old woman with expressive dysphasia, again highlighting the close overlap between vasculitic lesions and malignant glioma on imaging [2]. Suthiphosuwan, et al., reviewed 10 biopsy-confirmed cases of tumefactive primary CNS vasculitis, further supporting that vasculitis can present as a mass-like inflammatory brain lesion [3]. The present case differs from many reported primary CNS vasculitis mimics because of its autoimmune serological profile. The patient had blood-based ANA positivity, anti-dsDNA positivity, anti-RNP/RNP-C positivity and ANCA-related/lactoferrin antibody positivity. These findings support an autoimmune-associated secondary CNS vasculitic process rather than isolated primary angiitis of the CNS. This distinction is important because primary CNS vasculitis is generally considered a diagnosis of exclusion after systemic autoimmune, infectious and inflammatory causes have been ruled out. In this patient, systemic autoimmune serology was positive and corticosteroids were initiated by the rheumatology team. The present case is distinctive because it combines several uncommon features: a tumefactive cerebral infarct, immunohistochemical support for lymphocytic vasculitis, a multifocal radiological appearance mimicking multicentric glioma and positive blood-based autoimmune serology including ANA, anti-dsDNA, anti-RNP, RNP-C and ANCA-related serology. While tumor-like CNS vasculitis has been reported previously, autoimmune-serology-positive lymphocytic CNS vasculitis presenting as a glioma-mimicking tumefactive infarct remains uncommon and represents an important neurosurgical diagnostic pitfall.

The tumor-like appearance can be explained by the underlying vasculitic pathophysiology. Inflammatory injury to cerebral vessel walls may lead to vessel narrowing, thrombosis, ischemia, hemorrhagic transformation, edema and contrast enhancement. When this occurs in a focal cortical-subcortical distribution, especially in eloquent regions such as the perisylvian cortex, the resulting lesion may resemble glioma both clinically and radiologically. Multifocality, as seen in this case with an additional cerebellar lesion, can further strengthen the erroneous impression of multicentric glioma. From a neurosurgical perspective, the case highlights the role of surgery in resolving diagnostic uncertainty. The lesion involved eloquent perisylvian language cortex and was adherent to branches of the middle cerebral artery within the Sylvian fissure. Therefore, aggressive total excision would have risked language center or vascular injury. Exoscopic neuronavigation-guided maximum safe resection with intraoperative neurophysiological monitoring allowed diagnostic tissue acquisition and lesion debulking while minimizing neurological morbidity. The stable intraoperative motor evoked potentials, absence of new postoperative deficits and improvement in speech support the safety of this approach.

This case also emphasizes the importance of multidisciplinary management. The diagnosis required correlation between neurosurgical findings, neuroimaging, histopathology, immunohistochemistry and rheumatological serology. Once lymphocytic vasculitis was demonstrated and autoimmune markers were positive, steroid therapy was appropriately started. Long-term follow-up is essential because disease recurrence, additional infarcts or systemic autoimmune manifestations may emerge over time. Hemorrhagic manifestations also merit emphasis in CNS vasculitis. Although ischemic infarction is more typical, inflammatory destruction of the vessel wall may cause rupture, intracerebral hemorrhage or hemorrhagic transformation of an ischemic lesion. Dawoud et al. described primary angiitis of the CNS presenting with a large cortical intracerebral hemorrhage and emphasized that vasculitis should be considered in otherwise unexplained cortical hemorrhage [7]. In the present patient, the initial scan was interpreted as a subacute infarct with hemorrhagic transformation; subsequent GRE blooming, histological extravasation of red blood cells and the resolving hemorrhagic right cerebellar infarct support hemorrhagic involvement within the vasculitic-infarct spectrum. Recognition of this possibility is important because hemorrhage may increase mass effect, complicate radiological interpretation and influence decisions regarding antithrombotic treatment, biopsy or surgical decompression. The Neutrophil-to-Lymphocyte Ratio (NLR), calculated from a routine differential leukocyte count, is an inexpensive and readily available marker of systemic inflammatory balance. Its potential value in vasculitic disorders is supported indirectly by a meta-analysis of 17 studies comprising 6,334 patients with Kawasaki disease, in which NLR was significantly higher among patients who developed coronary artery abnormalities and coronary aneurysms [8]. In the present case, the pretreatment NLR was 1.58 and the post-treatment NLR was 1.67. The relatively low pretreatment value despite tissue-supported CNS vasculitis illustrates that NLR is a nonspecific adjunct and that a low value does not exclude a predominantly localized CNS vasculitic process. The small post-treatment increase should not be interpreted as disease progression because surgery, physiological stress and corticosteroid exposure can alter circulating neutrophil and lymphocyte counts. Furthermore, the cited evidence is from Kawasaki disease rather than CNS vasculitis; NLR cannot establish the diagnosis or replace imaging, cerebrospinal fluid evaluation, angiography or tissue examination.

The main limitation of this case report is that the exact systemic autoimmune diagnosis has not yet been defined in the available data. Additional information such as CRP, complement levels, MPO/PR3 status, antiphospholipid antibody profile, CSF analysis, CTA/DSA findings and long-term follow-up would further strengthen the etiological classification. Interpretation of the small NLR change is also limited by the single-patient design and by postoperative physiological stress and corticosteroid exposure; NLR should not be used as a disease-specific diagnostic or treatment-response marker. Nevertheless, the combination of tumefactive infarct, histopathologically proven cerebral infarction, immunohistochemically supported lymphocytic vasculitis and positive autoimmune serology makes this case a valuable addition to the literature on glioma-mimicking vasculitic brain lesions.

Conclusion

Autoimmune-associated lymphocytic CNS vasculitis may rarely present as a tumefactive cerebral infarct mimicking multicentric glioma. In the present case, preoperative imaging suggested a multifocal neoplastic lesion, but histopathology revealed cerebral infarction and follow-up pathology/IHC assessment supported lymphocytic vasculitis. Positive blood-based ANA, anti-dsDNA, anti-RNP, RNP-C and ANCA-related/lactoferrin antibody serology supported an autoimmune-mediated secondary CNS vasculitic process. This case emphasizes the need to consider inflammatory vasculitic infarction in atypical tumor-like brain lesions and highlights the importance of tissue diagnosis in resolving clinicoradiological discordance.

Conflict of Interest
The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement
All relevant anonymized clinical data are included in the manuscript. Additional data may be available from the corresponding author on reasonable request, subject to institutional policy and patient consent..

Ethical Statement
This is a single-patient retrospective case report. Institutional ethics committee approval obtained.

Informed Consent Statement
Informed consent was obtained from all participants included in the study.

Authors' Contributions
All authors contributed equally to this paper.

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