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# Focal cerebritis with vasculitis mimicking a high-grade glioma. A case report

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## ABSTRACT

Cerebritis is an inflammatory reaction in the brain which can be localized or diffuse and can be secondary to various etiologies like granulomatous demyelinating infections, vasculitis or neoplastic. Identifying the aetiology is very essential for the proper treatment. We report a case of 24 years male patient, who came with a history of headaches for 3 months. CT scan revealed intra-axial hypodense lesion suggesting high-grade glioma, which on histopathological examination revealed reactive gliosis with diffuse lymphocytic infiltrate and perivascular lymphocyte collection.

## INTRODUCTION

Differentiation between tumor like lesions and tumors of central nervous system is very essential for selecting the mode of treatment and prognosis. Both have similar features in ultrasound (US), computerized tomography (CT), and magnetic resonance imaging (MRI) studies. Misinterpretation leads to delay in the treatment of malignant tumors or over-treatment of tumor – like benign lesions. On imageology, tumor presents as focal density or signal alteration displacing or infiltrating adjacent structures surrounded by vasogenic edema and with or without matching contrast enhancement. But many tumor like lesions such as abscess, vascular malformations, resolving hematomas, tumefactive multiple sclerotic (MS) plaques also present with similar features. Development of functional MRI sequences such as Magnetic resonance spectroscopy, Diffusion tensor imaging (DTI), perfusion weighted imaging, PH weighted MRI and susceptibility weighted imaging (SWI) helps in differentiating tumor like lesions and tumors.[1] Histopathological examination of biopsied tissue can give confirmed diagnosis.

## Keywords

cerebritis,  
vasculitis,  
tumour like lesion



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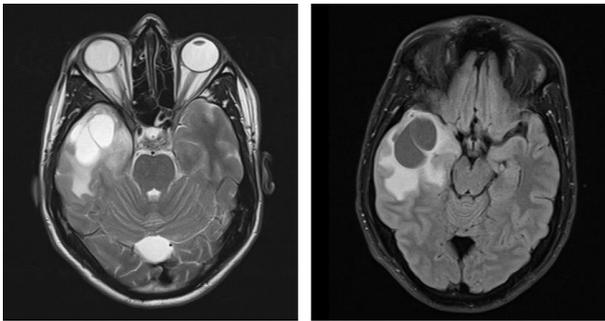
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### CASE REPORT

A 24-year-old male patient came to neurology OP with chief complaints of headache and paresthesia on right side since 3 months. In 2014, he had history of diplopia and headache. In 2018, patient had transient ischemic attack. Patient had no history of vomiting, seizures, fever, neck stiffness, photophobia, double vision or jaw deviation. He had no history of hypertension, diabetes, tuberculosis, chronic obstructive pulmonary disease, or chronic kidney disease.

On examination patient was conscious coherent, with no pallor, icterus, cyanosis, clubbing or pedal edema. Patient was afebrile with pulse rate 85/minute, blood pressure of 110/80mm Hg and respiratory rate of 20/minute.

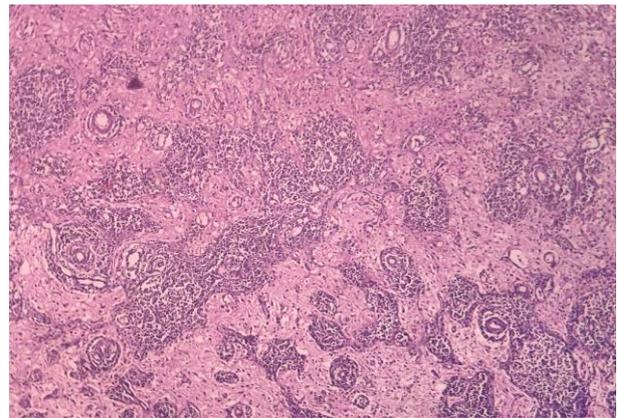
CNS examination revealed Glasgow coma score of 14/15. Higher mental functions and cranial nerves were normal. Motor examination revealed normal bulk, tone and power. Sensory system examination revealed intact cerebellar signs and no meningeal signs. No abnormality was detected in skull and spine.



**Figure 1.** Pre and post IV contrast MRI Brain showing well defined peripherally enhancing lesion with perilesional edema in the right temporal region which is hyperintense on T2W image, suppressed on FLAIR, hypointense on T1W images with evidence of enhancing pachymeninges adjacent to the lesion.

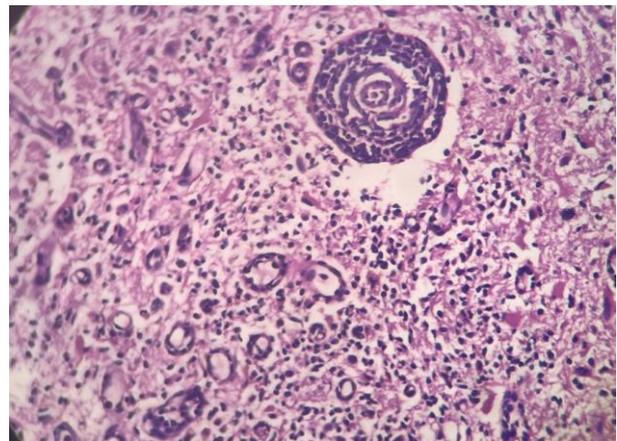
CT scan revealed intra axial hypodense lesion measuring 3.5X2.9cm in the right temporal lobe with adjacent perilesional edema in the surrounding white matter of the right temporal and parietal lobes. MRI scan with contrast study revealed ill-defined mixed signal intensity lesion with solid and cystic components. Solid component was T2/FLAIR (Fluid Attenuated Inversion Recovery) hypointense (Figure 1), and isointense on T1 in the right anterior and medial temporal lobe which was showing no evidence of restricted diffusion on DWI with diffuse

contrast enhancement. Cystic component was T2/FLAIR hyperintense and hypointense on T1 with no evidence of restricted diffusion on DWI (Diffusion Weighted magnetic resonance Imaging) with evidence of peripheral contrast enhancement. Medially the lesion was invading the cavernous sinus. Irregular T2 hyperintensity was not suppressed on FLAIR showing no evidence of contrast enhancement in perilesional region involving right temporal lobe, insular cortex, right external capsule and posterior limb of internal capsule suggesting edema. Lesion was causing mass affect in the form of midline shift of 5mm towards left side. Imageology suggested the clinical diagnosis of high-grade glioma.



**Figure 2.** Glial tissue showing diffuse lymphoplasmacytic infiltrate and perivascular lymphocytic cuffing (H&E, X40).

All the hematological investigations were within normal limits except for Erythrocyte sedimentation rate which was 82mm/1st hour.



**Figure 3.** Glial tissue with vessels showing lymphocytic infiltration in the vessel wall (H&E, X100).

Patient underwent right fronto temporo parietal minicraniotomy and right temporal pole excision. Excised tissue was sent for histopathological examination. We received multiple grey, brown soft tissue bits altogether measuring 2X1.5X1cm. Sections studied shows glial tissue with diffuse lymphoplasmacytic infiltrate. Adjacent foci showed reactive gliosis with gemistocytes. Perivascular lymphocytic cuffing was noted (Figure 2). Some of the vessels showed lymphocytic infiltrate into the vessel wall (Figure 3). Few vessels showed fibrinoid necrosis of the wall. Dense lymphocyte predominant lymphoplasmacytic infiltrate was seen in the meninges.

Immunohistochemistry showed LCA, CD3 and CD20 positivity in the dense inflammatory infiltrate indicating the polyclonal nature of lymphocytes. GFAP and S-100 showed positivity in glial tissue. CD 68 was positive in scattered histiocytes. CD138 was positive in plasma cells. Ki67 was seen positive in some inflammatory cells. Special stains for acid fast tubercle bacilli and fungal elements were negative. Due to the above histopathological and immunohistochemical features diagnosis of cerebritis, probably secondary to vasculitis was considered.

## DISCUSSION

Cerebritis is an inflammatory reaction in the brain which can be due to systemic or local etiologies and can mimic neoplasm. [2] Imaging features of various tumor like lesions like resolving hematoma, tumefactive MS plaque, vascular malformation and abscess are similar and are difficult to differentiate. [1] Solitary lesion in various demyelinating diseases including acute disseminated encephalomyelitis, progressive multifocal leukoencephalopathy and multiple sclerosis may mimic tumor. Histopathological examination is gold standard for diagnosis.

Idiopathic focal cerebritis could be secondary to infectious disease, vasculitis, granulomatous or demyelinating diseases. Tumor like mass lesions are reported in patients having systemic vasculitis such as SLE (systemic Lupus Erythematosus), and primary angiitis of CNS. Huang *et al* reported a large temporal mass lesion mimicking tumor in 14-year-old boy with systemic lupus erythematosus.[3] Molloy *et al* described a solitary tumor-like mass lesion in a subset of primary angiitis of CNS presenting with

edema and contrast enhancement. [4] Mohamad Ezzeldin *et al* described idiopathic focal cerebritis, presenting as tumor like lesion in right posterior parietal lobe in 35-year-old male. In this case ESR, C reactive protein (CRP) and Anti-nuclear antibodies (ANA) were normal and had no involvement of arteries. In our case, ESR was raised, and vessels showed lymphocytic infiltrate indicating possible cause may be vasculitis leading to cerebritis.

Differential diagnosis for this condition on imageology is focal cortical dysplasia, ganglioglioma, oligodendroglioma, Dysembryoplastic neuroepithelial tumor (DNET). [5] Focal cortical dysplasia type 1 occurs in adults with changes in the temporal lobe. Type 2 occurs in children and presents in frontal lobes with severe clinical symptoms. MRI may show focal cortical thinning or thickening with increased signal on T2 and gray and subcortical matter tapering towards ventricles on FLAIR weighted images. [6] Gangliogliomas contain cystic, solid and calcified components which gives inhomogeneous appearance to tumor. [7] Oligodendrogliomas commonly arise in frontal lobe, has well defined margins and with frequent calcifications. On MRI, T1 weighted images of tumor are hypointense and T2 weighted images of tumor are hyperintense. Surrounding vasogenic edema is uncommon [5]. DNET shows hypointensity on T1 weighted images and hyperintensity on T2 weighted images, without or with minimal mass effect and with surrounding vasogenic edema. 30% of cases show contrast enhancement. [7]

However histopathological examination is gold standard for diagnosis. Microscopically focal cortical dysplasia are two types. Type I shows cortical architectural abnormality (Type Ia) with hypertrophic neurons and immature neurons (Type Ib). Type II shows dysmorphic neurons with dislayering abnormalities (Type IIa) and with balloon cells (Type IIb). [6] Gangliogliomas are well differentiated benign neuroepithelial tumors. Histologically these tumors show combination of glial and neuronal elements exhibiting marked heterogeneity. [8] Oligodendroglioma is a low grade gliocytoma (WHO grade II) composed of monotonous cells having uniform round nuclei with perinuclear clearing (fried egg appearance) and thin branching vasculature (chicken wire vasculature). [9] Dysembryoplastic neuroepithelial tumor is characterized by presence of

abundant mucinous background with small round oligodendroglial like cells without dysplasia.[10]

Cerebritis secondary to vasculitis can be confused with lymphoma as in both the conditions diffuse lymphocytic infiltrate can be present. [11] Immunohistochemistry helps in differentiating polyclonality of lymphocytes which rules out the possibility of lymphoma.

### CONCLUSION

Focal cerebritis can present as neoplasm on imageology. Focal cerebritis can be idiopathic or can be secondary to infectious disease, vasculitis, granulomatous or demyelinating diseases. Correct diagnosis and appropriate treatment are essential for prognosis. Proper awareness in neurologist and radiologist is essential regarding the tumor like lesions of brain. Histopathology is the gold standard for diagnosis.

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