RESEARCH PAPER

The Role of MR Spectroscopy in a Massive Non-Enhancing Diffuse Astrocytoma Presenting with Transtentorial Herniation: A Case Report

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ABSTRACT

Diffuse infiltrative astrocytomas typically present as slow-growing, low-grade lesions that do not exhibit contrast enhancement. We present the case of a 25-year-old male who presented with clinical features of acutely raised intracranial pressure. Contrast-enhanced MRI (CEMRI) of the brain revealed a massive 6.7 x 10.2 x 6.7 cm lesion involving the left temporal and basifrontal lobes. Radiologically, the mass presented a severe diagnostic paradox: it exhibited aggressive mass effect, including a 10 mm contralateral midline shift and descending transtentorial herniation compressing the midbrain, yet demonstrated absolutely no post-contrast enhancement. Advanced neuroimaging, specifically MR Spectroscopy, was pivotal in resolving this dilemma. Spectroscopy revealed a marked elevation in the Choline peak (Cho/Cr ratio 2.9) and reduced N-acetylaspartate (NAA/Cr ratio 0.7), establishing a definitive metabolic signature of a cellular neoplasm over an ischemic or infectious process. This case underscores the indispensable utility of MR Spectroscopy in guiding the emergent radiological diagnosis of massive, non-enhancing brain lesions presenting with life-threatening herniation syndromes.

Keywords: Magnetic Resonance Spectroscopy, Diffuse Astrocytoma, Transtentorial Herniation, Neuro-oncology, Contrast-Enhanced MRI.

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INTRODUCTION

Diffuse infiltrative astrocytomas (WHO Grade II) are primary central nervous system neoplasms characterized by a slow growth rate and a relatively intact blood-brain barrier. On standard cross-sectional imaging, this intact barrier classically translates to a complete absence of post-contrast enhancement.(1) Due to their indolent nature, these tumors often grow to a substantial size before becoming clinically symptomatic.

However, initial presentation with acute, life-threatening mass effect and descending transtentorial herniation is a rare and critical neurosurgical emergency.(2)

In such emergent scenarios, massive non-enhancing space-occupying lesions present a significant radiological dilemma. The differential diagnosis for a non-enhancing mass causing severe edema and midline shift includes tumefactive demyelination, subacute extensive infarction, atypical encephalitis, or a low-grade infiltrative glioma.(3) Contrast-enhanced magnetic resonance imaging (CEMRI) combined with physiological imaging, such as Magnetic Resonance Spectroscopy (MRS), plays a definitive role in narrowing this differential.(4) We present a unique case wherein advanced MRI sequences and MRS successfully diagnosed a giant diffuse infiltrative neoplasm presenting with an impending herniation syndrome, despite a complete lack of contrast enhancement.

CASE PRESENTATION

A 25-year-old male presented to the emergency department with altered sensorium and clinical signs of severely raised intracranial pressure. To evaluate the cause of the acute neurological deficit, a contrast-enhanced MRI (CEMRI) of the brain was performed using standard neuro-oncology protocols, including T1-weighted (T1W), T2-weighted (T2W), Fluid-Attenuated Inversion Recovery (FLAIR), Diffusion-Weighted Imaging (DWI), Susceptibility-Weighted Imaging (SWI), and single-voxel MR Spectroscopy sequences.

Imaging revealed a large, mass-like area of altered signal intensity measuring approximately 6.7 cm x 10.2 cm x 6.7 cm (Craniocaudal x Anteroposterior x Transverse), extensively involving the entire left temporal lobe and the adjacent basifrontal lobe. The lesion appeared heterogeneously hypointense on T1W images and hyperintense on T2W images, with multiple tiny cystic areas noted peripherally. The mass showed partial signal suppression on FLAIR sequences and mild diffusion restriction on DWI. Notably, no blooming was observed on SWI, ruling out internal hemorrhage, and the lesion demonstrated absolutely no enhancement on the post-gadolinium contrast scans.

The mass involved the external capsule and extended along the ventricular margin of the posterior horn and trigone of the left lateral ventricle. Associated moderate perilesional edema extended into the frontoparietal and occipital lobes, as well as the left thalamus and lower basal ganglia. This resulted in a profound mass effect, evidenced by compression of the ipsilateral lateral ventricle, a marked contralateral midline shift of 10 mm, and compression of the third ventricle with consequent prominence of the contralateral lateral ventricle. Additional signs of severely raised intracranial pressure included a partially empty sella with a prominent perioptic CSF sheath, and critical descending transtentorial herniation compressing the midbrain.

Crucially, MR Spectroscopy of the lesion demonstrated a marked reduction in N-acetylaspartate (NAA) and a marked elevation in the Choline peak. The

Choline/Creatine (Cho/Cr) ratio was elevated to 2.9, while the NAA/Creatine (NAA/Cr) ratio was reduced to 0.7. Based on the infiltrative pattern, lack of enhancement, and the distinct neoplastic metabolic signature on spectroscopy, a radiological diagnosis of a diffuse infiltrative neoplasia (suggestive of a WHO Grade II diffuse astrocytoma) was established, allowing for immediate neurosurgical triage.

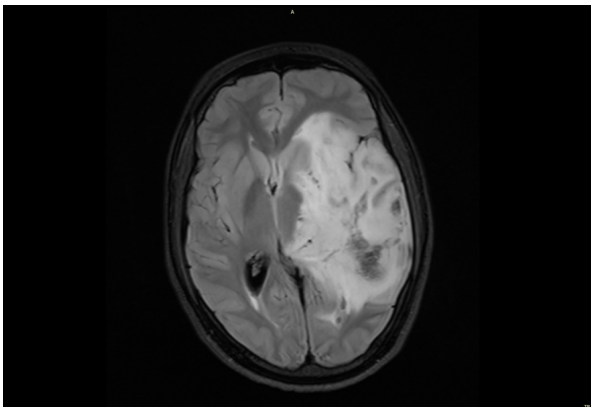

Figure 1: Axial T2-weighted MRI demonstrating a massive hyperintense lesion in the left temporal lobe causing severe mass effect and a 10 mm contralateral midline shift.

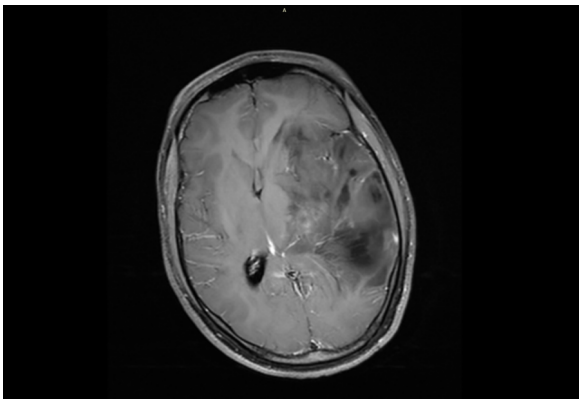

Figure 2: Axial Post-Contrast T1-weighted MRI revealing a complete absence of enhancement within the lesion.

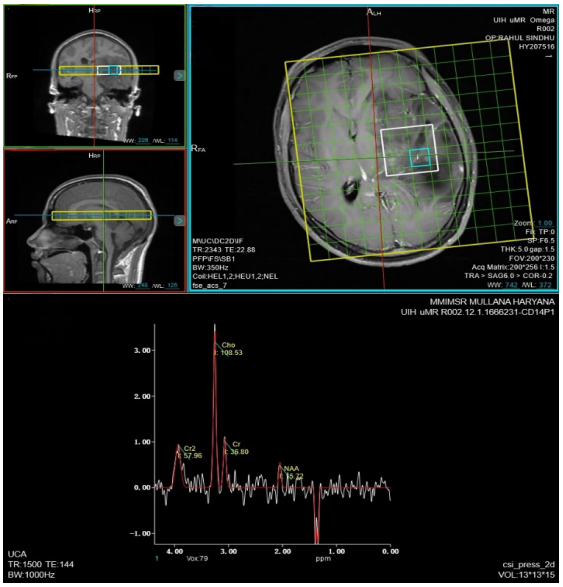

Figure 3: MR Spectroscopy of the left temporal lesion demonstrating a markedly elevated Choline peak and reduced NAA peak, consistent with a neoplastic process.

DISCUSSION

In neuro-oncologic imaging, contrast enhancement serves as a reliable surrogate marker for angiogenesis and blood-brain barrier breakdown, typically signifying a high-grade malignancy (WHO Grade III or IV) or an active inflammatory process. The presentation of a massive 10.2 cm lesion causing profound oedema and transtentorial herniation without any post-contrast enhancement presents a highly discordant radiological picture.[1,5]

In the absence of immediate tissue histopathology, MR Spectroscopy (MRS) is the definitive non-invasive tool to differentiate a non-enhancing low-grade glioma from aggressive non-neoplastic processes. MRS interrogates the metabolic microenvironment of the lesion. Choline is a marker of cellular membrane turnover and proliferation, while NAA is a marker of healthy neuronal integrity.[6] The striking Choline elevation (Cho/Cr ratio of 2.9) observed in our patient reflects high neoplastic cellularity, while the depleted NAA (NAA/Cr ratio of 0.7) indicates the destruction and replacement of normal neurons—a metabolic signature that is virtually pathognomonic for a diffuse infiltrative glial neoplasm.[6,7]

This case highlights a critical teaching point for radiologists and emergency physicians: while low-grade astrocytoma's lack the contrast enhancement associated with aggressive glioblastomas, their silent, extensive infiltrative growth can eventually breach critical intracranial compliance.[8] This culminates in an acute neurosurgical emergency, such as the descending trans-tentorial herniation and midbrain compression seen in this patient. Advanced MRI sequences, specifically the utilization of MRS, are therefore essential not only for establishing the initial diagnosis of non-enhancing lesions but for expediting life-saving decompressive interventions.

CONCLUSION

Massive, non-enhancing brain lesions presenting with acute herniation syndromes represent a significant diagnostic challenge. This case illustrates that a complete absence of contrast enhancement does not rule out a life-threatening neoplasm. By utilizing MR Spectroscopy, radiologists can identify the classic metabolic markers of elevated Choline and reduced NAA to confidently diagnose a diffuse infiltrative astrocytoma. This non-invasive metabolic profiling is crucial in the emergency setting to differentiate low-grade gliomas from tumefactive demyelination or ischemia, thereby guiding immediate and appropriate neurosurgical management.

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