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Tectal Plate Cryptococcoma Mimicking Glioma: Diagnostic Pitfalls and the Value of MR Spectroscopy

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Abstract: A man in his early fifties with chronic hepatitis B presented with two days of altered mental status. CT showed a tectal plate lesion causing hydrocephalus, initially thought to be a glioma. MRI findings were atypical for glioma, suggesting an infectious process. CSF analysis revealed high cryptococcal antigen titers, and MR spectroscopy demonstrated lipid and lactate peaks, supporting an infectious etiology. A diagnosis of tectal plate cryptococcoma complicating cryptococcal meningitis was made. Antifungal therapy led to clinical improvement, and follow-up imaging showed slight decreased size of the tectal lesion. This case of tectal plate cryptococcoma highlights the diagnostic challenge of identifying a fungal infection in an uncommon location and the value of advanced imaging contributing to the diagnosis, avoiding unnecessary interventions.

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I. INTRODUCTION

Cryptococcosis is a fungal infection predominantly affecting immunocompromised individuals [1]. While cryptococcal meningitis is well-described, mass-forming fungal granulomas known as cryptococcomas are rarer, particularly in the brainstem [2,3]. Causative organisms include Cryptococcus gattii and Cryptococcus neoformans[4]. Cryptococcomas most commonly occur in the cerebral cortex and ventricles, with occasional reports in the pons [3,5]. This case report describes a tectal plate cryptococcoma in a patient with chronic hepatitis B, initially mimicking a tectal plate glioma, emphasizing the need for a broad differential diagnosis when evaluating midbrain lesions.

II. CASE PRESENTATION

A man in his 50s who initially presented to the emergency department with a two-day history of confusion. He was newly diagnosed with chronic hepatitis B and latent TB during hospitalization, both untreated, and had emigrated from China more than 20 years ago. There was no history of trauma, prior history of similar symptoms, recent travel or known sick exposures. On initial examination, the patient was alert and oriented to self and year, following commands with prodding by a family member. Vital conditions were stable and blood parameters were within normal limits. Central nervous system examination demonstrated no significant findings.

- *Investigations:*

A non-contrast computed tomography (CT) scan of the head was performed to investigate the cause of altered mental status. It revealed a heterogeneously hyperdense 1.5 cm mass in the midbrain, compressing the cerebral aqueduct and causing dilation of the lateral and 3rd ventricles, consistent with obstructive hydrocephalus. A Brain MRI with contrast was recommended to assess it further (Figure 1).

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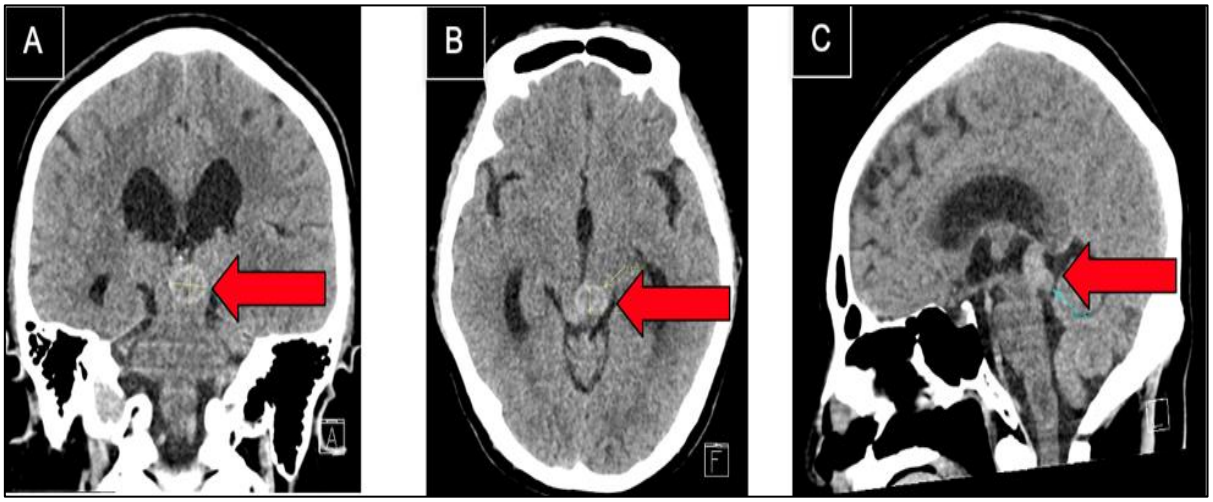
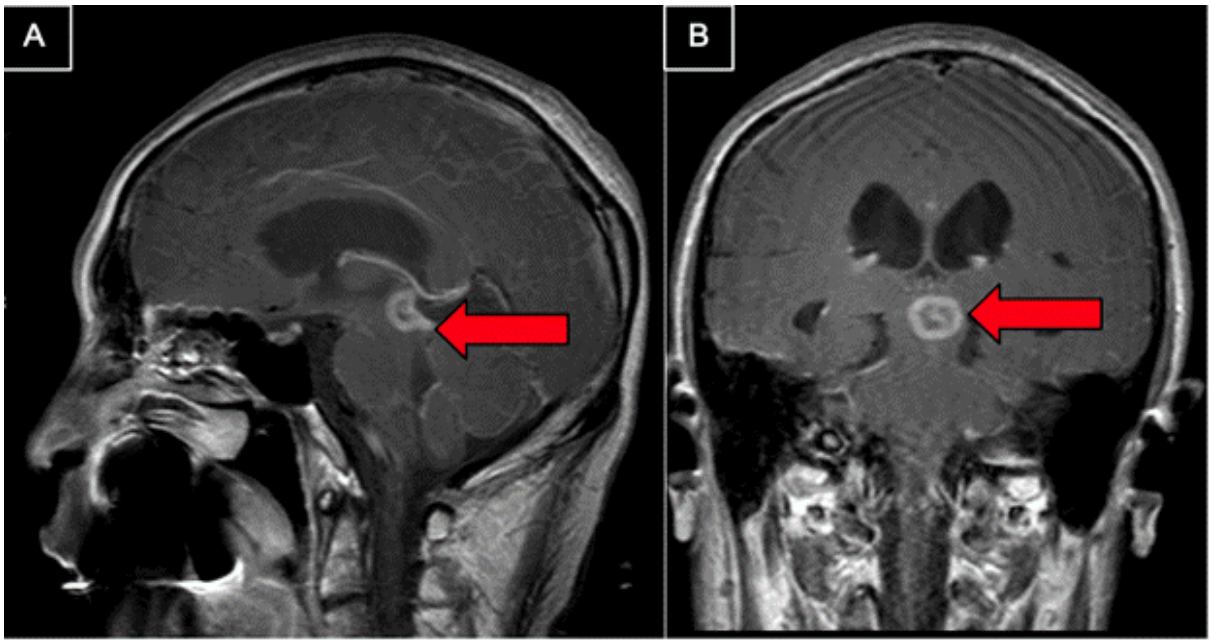


Fig 1 Non-Contrast CT Brain A. Coronal View with 1.5 cm Well-Circumscribed Ring-Enhancing Lesion. B and C. Axial and Sagittal Views Demonstrate a Midbrain Lesion Obstructing the Cerebral Aqueduct with Enlargement of the Lateral and Third Ventricles.

Magnetic resonance imaging (MRI) revealed a 1.5 cm well-circumscribed lesion in the left aspect of the midbrain, characterized by a T2 hyperintense central cavity with a hypointense peripheral ring and rim enhancement in the post-contrast T1 images. The lesion demonstrated peripheral restricted diffusion and appeared hypointense on T1-weighted imaging. A small nodular enhancing component was also noted along the right lateral margin. Associated findings included obstructive hydrocephalus of the supratentorial ventricular system and minimal periventricular white matter hyperintensity consistent with transependymal CSF flow, indicating acute hydrocephalus.

The initial interpretation of the MRI (Figure 2 A-F) was initially considered as a tectal plate low grade glioma. This was due to its location , mass effect and secondary hydrocephalus and perilesional edema .

But other differentials were also considered following this a multidisciplinary case discussion was conducted . Due to lack of diffusion restriction , pyogenic abscess was ruled out and other atypical. Due to increased hydrocephalus and decreased mental status overnight , a right frontal approach ventriculostomy catheter for ICP monitoring to see if this changes his mental status. Further the CSF was sent for profiling at the same time to rule out other etiologies (Table 1).



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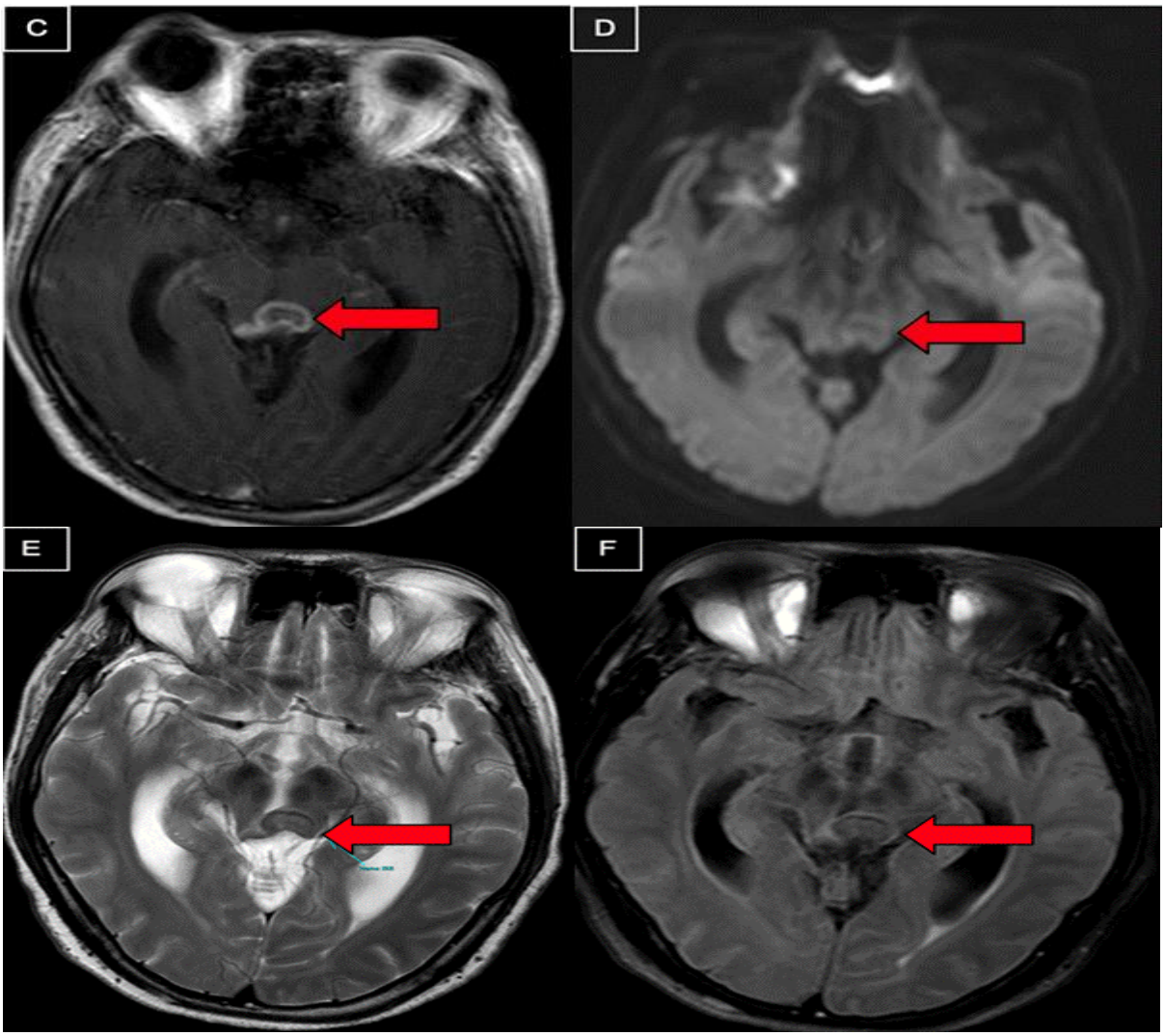

Fig 2 MRI Brain with gadolinium contrast A.Sagittal B.Coronal T1 view with 1.5 cm lesion with peripheral enhancement and hypointense centre hyperdensity. C.Axial T1 view with a ring component and nodular component from the right posterior region. D.Diffusion weighted image with restricted diffusion in the periphery with no enhancement of the central region. E.Axial T2 view with hypointense lesion with peripheral hypointense rim. F.Axial FLAIR view with transependymal edema in the posterior horn of the left lateral ventricle.

Table 1 CSF Report with Infectious Disease Workup

CSF	**Levels**
Cryptococcal Antigen titer	1:256
Color	Pink
Appearance	Slightly cloudy
RBC,CSF	9000.0 Cell/uL
TNC,CSF	20 Cells/uL
Neutrophils% CSF	83%
Lymphocytes% CSF	11%
Mono/Macrophage% CSF	4%
Protein,CSF	51 mg/dL
Glucose, CSF	67 mg/dL

During the interdisciplinary case discussion, these findings were considered more consistent with infectious etiology, a broad lab workup was made to find infectious etiology differentials including atypical infections such as TB, toxoplasmosis, neurocysticercosis, cryptococcal infection or inflammatory conditions. CSF cryptococcal antigen was positive (titer 1:256) and other labs for TB, Toxoplasmosis came back negative. Laboratory studies showed Hepatitis B surface antigen was positive and the surface antibody was non-reactive but the core antibody was reactive indicating a chronic infection . The HBV levels were 3,198 IU/ml and Hepatitis B DNA viral load was 10,905copies/ml. The HIV test was negative.

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The MRI finding had no diffusion restriction to suggest a pyogenic abscess. Additionally along with the meningitis findings in the CSF and the positive Antigen titres , cryptococcoma was considered the probable diagnosis.

MR spectroscopy was done additionally and revealed elevated lactate, absent choline peak, and reduced NAA levels which favour an infection over a malignancy (Figure 3). There is no significant increased relative cerebral blood volume in this lesion.

In the absence of a choline peak a neoplastic process would be less likely. There were no gelatinous pseudocysts but occasionally cryptococcal infection presents with a local mass. Presence of hyperintensity within the sulci on FLAIR images also favours an infectious etiology.

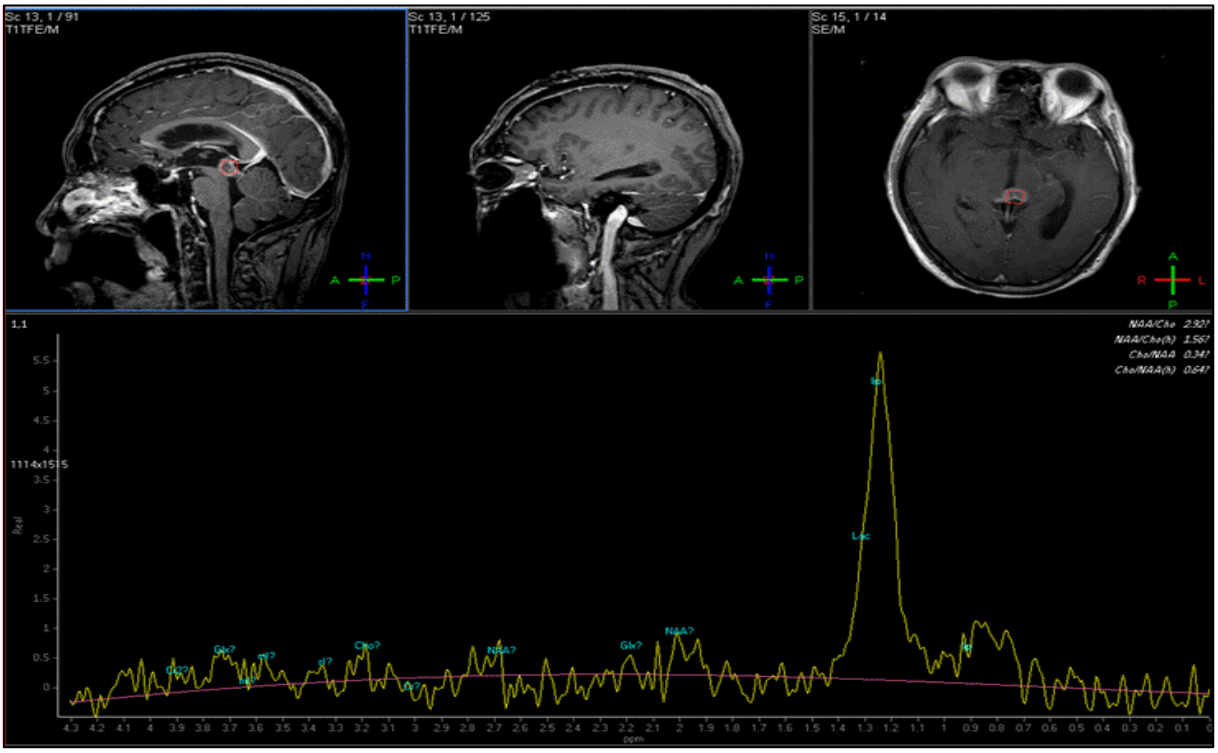

Fig 3 MR Spectroscopy Shows Elevated Lactate Peaks as Compared to Choline.

➢ *Differential Diagnosis:*

The differentials can be divided into neoplastic lesions, vascular and infectious disease lesions based on radiology appearance.

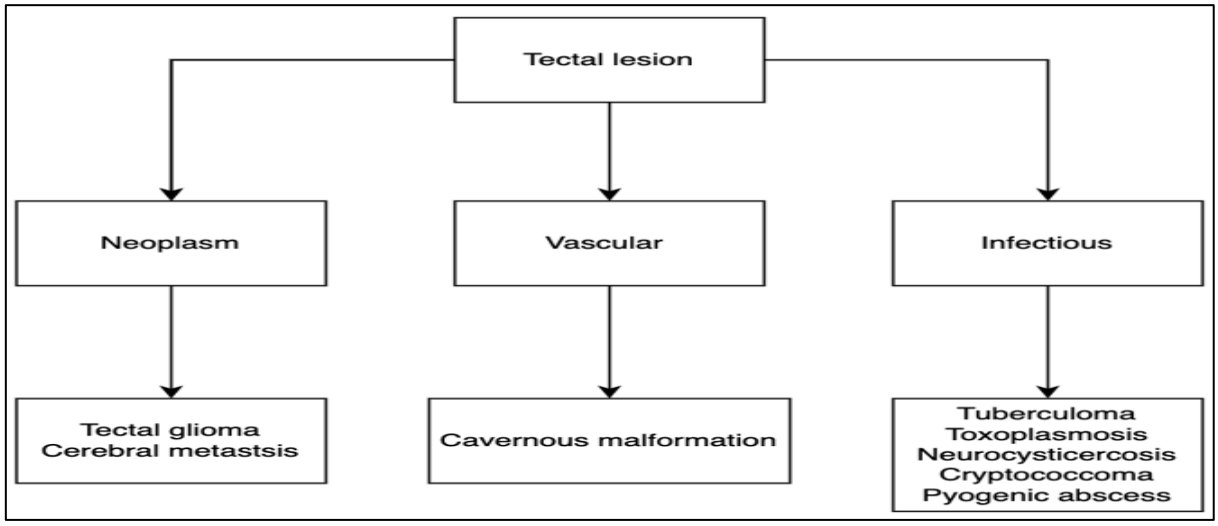

Fig 4 Flowchart of Radiographic Differentials of a Tectal Plate Lesion.

Initially, the patient's tectal lesion on CT was thought to be a tectal plate glioma due to its location , expansion and secondary hydrocephalus and age of the patient. On MRI with contrast a rim enhancing lesion was seen , and tectal gliomas do not enhance with contrast. Thus other differentials including atypical infections and vascular lesions were considered .

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An LP was pursued due to inconclusive findings and MR spectroscopy results were interpreted. Her markedly elevated cryptococcal antigen level in the CSF were concerning for cryptococcoma and cryptococcal meningitis. Additionally MR spectroscopy showed elevated levels of lactate and not choline which helped confirm the infectious etiology as opposed to a neoplastic etiology.

➢ *Treatment:*

The patient was started on induction antifungal therapy of cryptococcal meningitis and cryptococcoma with intravenous amphotericin and oral flucytosine for six weeks. Following the induction phase, he was transitioned to oral fluconazole 400 mg daily for consolidation therapy.

He also received treatment for latent tuberculosis with isoniazid and pyridoxine, which he tolerated well. Physical therapy and occupational therapy evaluations recommended discharge to home.

➢ *Outcome and Followup:*

The patient was discharged home approximately two months after his initial presentation. Clinically, he showed gradual improvement. During subsequent outpatient follow-up visits with infectious disease (ID) clinic, he reported feeling well, denying fevers, chills, headaches, confusion, dizziness, visual changes, or pulmonary symptoms. His sister noted improvement in his memory and weight gain. He continues to tolerate fluconazole, isoniazid, and pyridoxine well as part of his post-induction therapy. A repeat brain MRI scheduled in 3 months showed post -treatment reduction of the rim thickness and a decrease on the nodular component. Long-term follow-up with ID is planned.

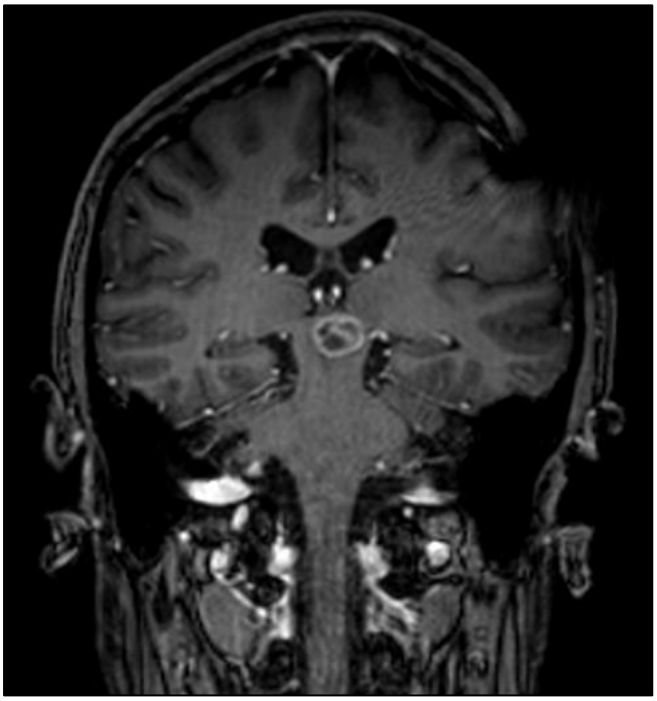

Fig 5 MRI Coronal T1W Post Contrast View Post Treatment after 3 Months Shows a Decrease in Thickness of the Rim and Mild Decrease in Size and Absence of Nodularity Seen before.

III. DISCUSSION

To our knowledge, this is the first case of tectal plate cryptococcoma reported in an adult with chronic Hepatitis B infection. Cryptococcoma remains a rare condition in adults, seen among immunocompromised and immunocompetent individuals [4,5]. Cryptococcosis most likely spreads to the CNS by means of hematogenous dissemination from a pulmonary foci [7,9]. Most commonly, it is caused more often by C.gattii than C. neoformans. Radiographic findings have been described previously as multiple lesions with surrounding vasogenic edema. It is commonly seen in males and in those from the age group of 50[4,5,9].

Most common sites include the basal ganglia, thalamus, cerebellum, and the cortex through its spread through the PVS[9,14]. A PubMed search was conducted for reported cases of adults with tectal plate lesions as a primary lesion, and no results were found.

On CT images, these lesions may have a high or low attenuation. On MRI, each image gives us a clue to the diagnosis. A ring-like or solid lesion is seen, which is typically T1 hypointense, with contrast enhancement, T2 hyperintense, hypointense on DWI, and ADC hyperintensity [6,8,9,15]. Cryptococcomas are commonly misdiagnosed as neoplasms, abscesses (pyogenic or tubercular), neurocysticercosis, or demyelinating lesions [5,6]. It is especially challenging to differentiate a solitary Cryptococcoma from a neoplasm[6]. Cases that are misdiagnosed as neoplasms underwent brain biopsy or resection [10,11]. Tectal plate gliomas also cause obstructive hydrocephalus, and most cases are diagnosed purely on the basis of radiologic appearance, but can often be an indication for invasive biopsy to confirm the diagnosis [12,13]. The spectroscopic data indicated a decline in N-acetyl aspartate concentrations, along with a rise in both choline and lactate levels [15]. Management of this condition has been well established once diagnosed and has good recovery rates. Initial management consists of CSF diversion and relieving clinical findings. Treatment of cryptococcoma includes antifungal induction therapy for 6 weeks with Amphotericin B or Fluconazole and flucytosine, followed by consolidation and maintenance therapy with fluconazole for [5,7,9,16-18]. Amphotericin has strong fungicidal activity but it has a poor penetration into the CSF thus intrathecal administration is used [1]. Corticosteroids should not be used even as adjuvant therapy [2].

This case underscores that *Cryptococcus* infection should be considered in the differential diagnosis of brainstem lesions.

➢ *Learning Points or take Home Messages:*

- For tectal plate lesions that have ring enhancing effects , infectious etiology such as cryptococcoma should be considered as a differential.
- Interdisciplinary management is required for tectal plate lesion including CSF workup,infectious disease workup and Imaging Role of spectroscopy : absent choline peak

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rule out neoplastic etiology and elevated lactate peak supported an infectious etiology.

- Pyogenic abscess are ruled out onMRI on the basis of absence of restricted diffusion in the center.
- Tectal plate gliomas are more common among pediatric age groups and cause deformation and widening of tectal plate lesions with decreased response to fungal treatment.

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