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Tuberculosis-Associated Immune Reconstitution Inflammatory Syndrome (TB-IRIS): A Critical Strategy for Improved Outcomes

Kapil Bainade, V.Kotrashetti, Neelu Elon, Saylee Jagtap, Rhutu Shah.
Department of Pediatrics,
Dr. D.Y. Patil Medical College & Hospital, Navi Mumbai,
Maharashtra, India, 400706

***Corresponding Author:**
Dr. Rhutu Shah
Department of Pediatrics, Dr. D.Y. Patil Medical College & Hospital,
Navi Mumbai, Maharashtra, India, 400706

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Abstract

Tuberculosis-associated Immune Reconstitution Inflammatory Syndrome (TB-IRIS) is a clinical phenomenon observed in individuals undergoing or after completion of anti-tuberculosis therapy, characterized by a paradoxical worsening or new presentation of tuberculosis (TB) symptoms despite effective anti-TB treatment. TB-IRIS occurs as a result of a restored immune response to Mycobacterium tuberculosis antigens following immune recovery, typically within weeks of anti TB therapy initiation. Two forms are recognized: paradoxical TB-IRIS, which presents as a recurrence or worsening of previously diagnosed TB, and unmasking TB-IRIS, where previously undiagnosed TB manifests with an exaggerated inflammatory response. Risk factors include a low CD4 count at ART initiation, high mycobacterial burden, and a short interval between starting TB treatment and ART. Diagnosis is clinical, relying on exclusion of alternative causes such as TB treatment failure or drug resistance. Management involves continued ATT and TB therapy, with corticosteroids used in moderate to severe cases to reduce inflammation. Further research is warranted to show effective of corticosteroids in TB-IRIS.

Keywords: TB-IRIS, Tuberculosis, Neurotuberculosis, Immune Reconstitution Inflammatory Syndrome, Corticosteroids, Hydrocephalus

Introduction

TB-IRIS is a paradoxical deterioration or recurrence of preexisting tuberculous lesions, or a development of new lesions in patients on active antituberculosis treatment. It may occur during or even after completion of anti-TB therapy. Sometimes it is misdiagnosed as superimposed infection or failure of treatment. Hence a criteria has been formulated to identify TB-IRIS. [2]

1. initial improvement of TB-related symptoms and/or radiographic findings after adequate anti-TB treatment for a particular period of time.
2. paradoxical deterioration of TB-related symptoms and/or radiologic findings at the primary or at new site during or after anti-TB treatment.
3. absence of comorbidities that reduce the efficacy of anti-TB drugs (e.g., poor compliance, drug malabsorption, drugs side effects).
4. exclusion of other possible causes of clinical deterioration.

Pathophysiological Basis for Early Steroid Use:

During ART/ATT-induced immune restoration, a surge in pro-inflammatory cytokines (e.g., TNF-α, IFN-γ, IL-6) contributes to a hyperactive immune

response. Steroids exert early immunomodulatory effects by:

1. Suppressing inflammatory cytokine production
2. Reducing T-cell activation
3. Limiting tissue destruction

Early initiation targets the inflammatory cascade before it escalates into full-blown IRIS, potentially preventing irreversible organ damage.

Case Report:

A 5 year old male child diagnosed with Neurotuberculosis on ATT since 1 month came with complaints of right sided ptosis since 4 days associated with increased redness of right eye since 2 days, 2 episodes of projectile vomiting associated with headache since 1 day. He had a past history of fever and parietal region headache since 10 days. MRI Brain was s/o multiple small tuberculoma in pons and midbrain, leptomeningeal enhancement and mild odema of cerebellar parenchyma. CSF analysis showed no cells and MTB was not detected. He was started on anti-tubercular therapy without steroid coverage. There was clinical improvement of symptoms. However 1 month later, he presented with worsening of symptoms (projectile vomiting, headache, lethargy, rt sided ptosis). On examination irritation, lethargy, meningeal signs was present, all reflexes were diminished. Repeat MRI Brain was showed increase in size of Tuberculoma with basal exudates- increased prominence of bilateral lateral ventricle and third ventricle with flair hyper intense periventricular ooze s/o uncompensated obstructive hydrocephalus and V-P Shunt was placed for the same. He was started on Mannitol, Dexamethasone, AKT was continued. After which he showed clinical improvement, Inj Mannitol was stopped and Tab Acetazolamide was started, IV steroids were shifted to oral Prednisolone (2mkd) over 4 weeks and tapered over next 4 weeks along with AKT.

Fig 1: 5 year old ready for V-P shunting

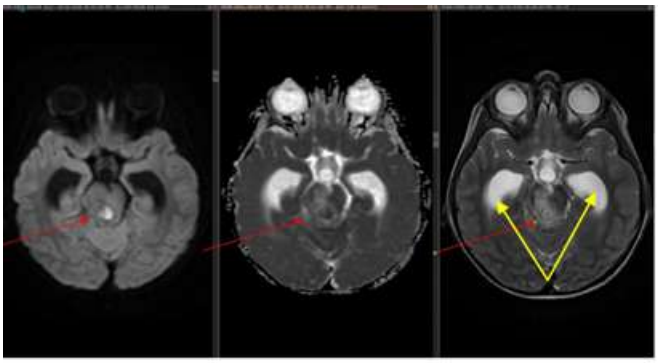

Fig 2:Mid-brain and right hemi Pons Tuberculoma (red arrow)

Temporal horns of bilateral lateral ventricles appears dilated s/o hydrocephalus (yellow arrow)

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Fig 3: Multiple Brainstem tuberculoma with basal exudates

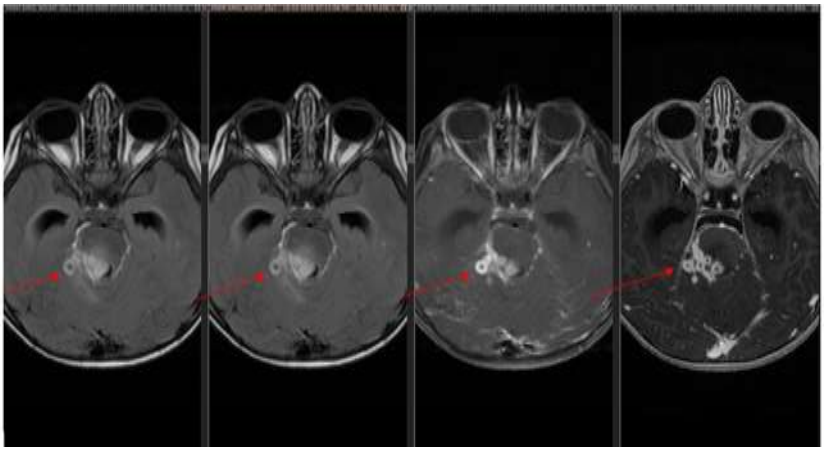
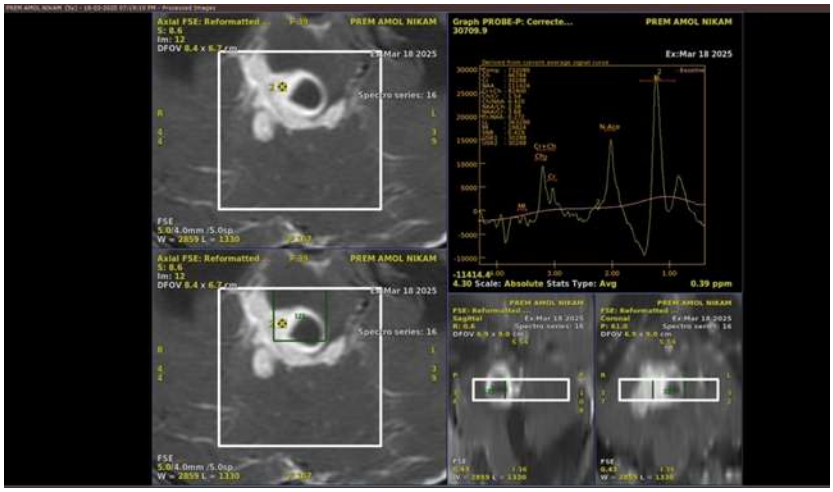


Fig 4: MR spectroscopy with elevated choline:creatinine ratio



Discussion:

Corticosteroids exert broad anti-inflammatory effects, including suppression of cytokine release, inhibition of T-cell activation, and stabilization of capillary membranes. These mechanisms help reduce the immune-mediated tissue damage observed in TB-IRIS. Early initiation of corticosteroids aims to intercept the inflammatory cascade before it causes irreversible tissue damage, particularly in cases involving the central nervous system (CNS), respiratory tract, or pericardium. The diagnosis of IRIS was determined in this case due to the increasing size of tuberculoma during ATT, even though the initial symptoms of tuberculosis (headache and vomiting) had improved and there was good adherence to ATT. Studies report higher rates; for instance, a 54.2% incidence was reported in patients with culture-confirmed pulmonary TB in India, and a 47% incidence of paradoxical TB-IRIS patients with TB meningitis. Paradoxical neurologic TB-IRIS is a possibly life threatening condition and symptoms tend to manifest later than in forms not involving the central nervous system. Neurologic TB-IRIS generally presents with new or worsening meningitis and/or features of raised intracranial pressure, due to enlarging cerebral tuberculomas or intracranial abscesses; mortality is high and ranges from 12% to 25%[4]. It may also present with spondylitis, epidural abscesses, and radiculomyelopathy. Landmark study by Meintjes et al. (2010) provided strong evidence for the use of corticosteroids in TB-IRIS with 30% reduction in symptoms, lowers the need for hospitalization and improves quality of life[3].

In present report, the patient exhibited with increase in size of the previous tuberculomas which eventually lead to periventricular ooz and acquired communicating hydrocephalus. MRI with gadolinium contrast is considered the most reliable method for

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diagnosing central nervous system (CNS) tuberculosis and the presence of obstruction was present on MRI, however presence of MTB was not detected on CBNAAT sputum and GENEXPERT CSF analysis. GeneXpert's sensitivity in diagnosing meningitis tuberculosis ranges from 61 to 85 %, and it is generally accepted that negative CSF results do not rule out the diagnosis of tuberculosis itself. Furthermore, CSF culture only has a modest role in diagnosing meningitis tuberculosis, with sensitivity around 50–70 %.

As previously indicated, neuroinflammation is a pivotal element in CNS TB, and it becomes even more intricate in the context of CNS TB-IRIS.The major approach for managing TB-IRIS is high-dose corticosteroid therapy, which has been supported by controlled clinical trials.

Conclusion

TB-IRIS is an important clinical entity that may result in significant morbidity and mortality, particularly in patients with central nervous system tuberculosis. Early recognition, exclusion of alternative diagnoses, and prompt initiation of corticosteroid therapy while continuing anti-tubercular treatment are essential for favourable outcomes. Neuroimaging remains critical for diagnosis and monitoring. Further prospective studies are needed to establish evidence-based recommendations regarding optimal timing, dosage, and duration of corticosteroid therapy in TB-IRIS.

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