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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/15938
DOI URL: http://dx.doi.org/10.21474/IJAR01/15938

RESEARCH ARTICLE

CASE REPORT: TUBERCULOUS SCLERITIS A CHALLENGING DIAGNOSTIC

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Manuscript Info

Manuscript History
Received: 28 October 2022
Final Accepted: 30 November 2022
Published: December 2022

Key words:
Scleritis, Tuberculosis, Mantoux Test

Abstract

Scleritis is a severe painful inflammatory process localized in the sclera. Scleritis may be the revealer of many systemic diseases so that it is important to exclude multisystem disease. We present a case of non-necrotizing antero-posterior scleritis revealing a systemic tuberculosis which is an uncommon manifestation of tuberculosis. Diagnosing and managing the disease can be difficult. However, a good anamnesis, a careful clinical evaluation, an adequate interpretation of the investigations and an appropriate management allow a good prognosis.

Introduction:-

Scleral inflammation can be the initial manifestation of various systemic diseases; Scleritis in its posterior or anterior form is a relatively rare form of ocular inflammation.

Posterior scleritis is associated with a systemic disorder in 10-20% of patients and rarely described as a result of an infectious process. However, the process of systemic infectious disease is not uncommon and it is important to exclude a possible infectious origin, since it may be similar to a systemic autoimmune origin [1,2]. This article reports a case of scleritis secondary to tuberculosis as well as the difficulties in diagnosing and managing such cases.

Case Report:-

A 36-year-old female patient presents to our hospital for a painful loss of visual acuityin the right eye evolving for 15 days. Shehad consulted an ophthalmologist who put her on topical corticosteroids during the previous 5 days. moreover, the patient reports the notion of tuberculosis contagion with a family member suffering from active pulmonary tuberculosis.

On ophthalmological examination, her best corrected visual acuity (BCVA) was 4/10in the right eye and10/10in the left eye; Slit-lamp examination of the right eye revealed aconjunctival hyperemia, quiet anterior chamber, no cells in vitreous**(Figure 1)**,fundus examination showed an oedematous optic disc with surrounding subretinal fluidand vascular tortuosity**(Figure 3)**. Pupils were normal and reactive in both the eyes. Slit-lamp and fundus examination of the left eye was without abnormalities. Ultrasound B-scan of the right eye demonstrated sclerochoroidal thickening with widening of sub-Tenon space with scant fluid and elevation of optic papilla**(Figure 2)**. Fundus fluorescein angiography of the right eye showed early disc staining andleaks in the early phase of angiogram which gradually showed pooling around the disc in late phases**(Figure 4)**.

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She was widely investigated for the cause of her scleral inflammation. Her erythrocyte sedimentation rate was elevated, and serum angiotensin converting enzyme was normal. Anti-nuclear antibody, rheumatoid factor, anti-cytoplasmic antibodies, serologies for syphilis were negative. Her Mantoux test was strongly positive (20mm) **(Figure 5)**, QuantiFERON (QTB) was positive; Detection of acid-fast bacilli on smear and Mycobacterium tuberculosis genome by PCR were negative and a pulmonary radio with chest CT show evidence of healed pulmonary tuberculosis**(Figure 6)**.

A collegial decision including pulmonologists was in favor of a presumed tuberculous origin in front of strong clinical and paraclinical arguments, for that we started anti-tubercular treatment (ATT), rifampicin, isoniazid, pyrazinamide and ethambutol for 9 months withoral corticosteroids 60 mg/day in tapering schedule.

She was controlled after a month, her BCVA in right eye improved to 7/10; Slit-lamp examination showed a quiet anterior chamber and anterior vitreous**(Figure 7)**; Fundus examination of the right eye showed resolving papilledemawith surrounding subretinal fluid**(Figure 8)**.

After 3 months, her BCVA improved to 10/10; Fundus examination of the right eye revealed complete resolution of optic disc edema, resolution of surrounding subretinal fluid. A repeat Ultrasound B-scan of the right eye demonstrated reduction in sclerochoroidal thickening and resolution of the papilledema**(Figure 9)**. She was advised to continue ATT andshe under clinical and paraclinical controlfor last 6 months and till now she did not develop any recurrence.

Discussion:-

Infectious scleritis, which accounts for 8% of all scleritis cases, is rarely caused by Mycobacterium tuberculosis, its mechanism may be direct invasion of the sclera (due to hematogenous spread of pulmonary tuberculosis) or an immune-mediated reaction to circulating mycobacterial antigens from a distant focus in the absence of bacteria in the eye; It can rarely occur by extension from adjacent tissues or after trauma [3].

Tuberculous scleritis usually presents as an anterior nodular scleritis rarely a posterior form, which when not diagnosed early, can progress to scleral abscess eventually leading to scleral perforation as seen in many studies. It can occur in isolation or involve the adjacent peripheral cornea causing what is called sclerokeratitis [4].

The diagnosis of tuberculous scleritis is a clinical challenge due to the varied presentations, it is often presumptive based on a bundle of arguments includinga rigorous anamnesis, guiding clinical signs, positive tuberculin skin test (> 15 mm or more of induration) or QTB, evidence of healed or active pulmonary or extrapulmonary tuberculosis, exclusion of other causes of scleritis, and a favorable therapeutic response to anti-tuberculosis treatment. Definitive diagnosis is based on the detection of acid-fast bacilli on smears, growth on culture medium, demonstration of the Mycobacterium tuberculosis genome by PCR and histopathological evidence; The majority of the cases described occur in isolation without any detectable systemic involvement of tuberculosis[5].

For Bouza E et al. ocular tuberculosis was found in 18% of the 100 patients with proven tuberculosis, important ocular findings being choroiditis, and other ocular lesions including papillitis, retinitis, vitritis, vasculitis, dacryoadenitis, and scleritis[6]. In a study recently reported at Srilanka [2], ocular tuberculosis was diagnosed in 23 patients of the total 2130 tuberculosis patients, among whom episcleritis and inflammatory scleral nodule were observed in 2 patients. Donahue found 1.4% ocular tuberculosis in 10 000 patients with primary pulmonary TB who were evaluated in the eye clinic of the Mattapan Sanatorium, Massachusetts, USA [1].

A study by Wroblewski suggested that longstanding tuberculous ocular infections can produce corneoscleral perforations[7]. Shoughy et al. treated all the 8 patients having scleritis with a minimum of 6 months of antitubercular drugs without concomitant use of corticosteroid and the complete resolution was seen [8].

Patel et al. studied mycobacterial ocular disease in a series of 17 patients in which two cases of necrotizing nodular scleritis resulted in perforation and enucleation despite systemic ATT and immunosuppressive therapy [9]. Chansangpetch et al.also reported an atypical case of MTB uveitis preceding nodular scleritis in which scleral pus showed acid-fast bacilli onsmear, grew MTB in both solid and liquid culture media, andwas also positive for Mycobacterium tuberculosis complex onreal-time PCR [10]. **(Table1)**

The patients who were treated with immunosuppressives besides corticosteroids had devastating outcomes like enucleation of the eyeball. The diseaseis frequently misdiagnosed at the beginning and treated withonly corticosteroids and immunosuppressives as seen in manypublished cases [11,12]. The management of tuberculous scleritis is tough with a balance between the direct infection and immune-related effects.

Thetreatment normally includes the first line antitubercular therapy, rifampicin, isoniazid, ethambutol, pyrazinamide, and streptomycin withor without systemic corticosteroids [13];The use of oral corticosteroids is a controversially discussedissue in tuberculous scleritis [14,16].

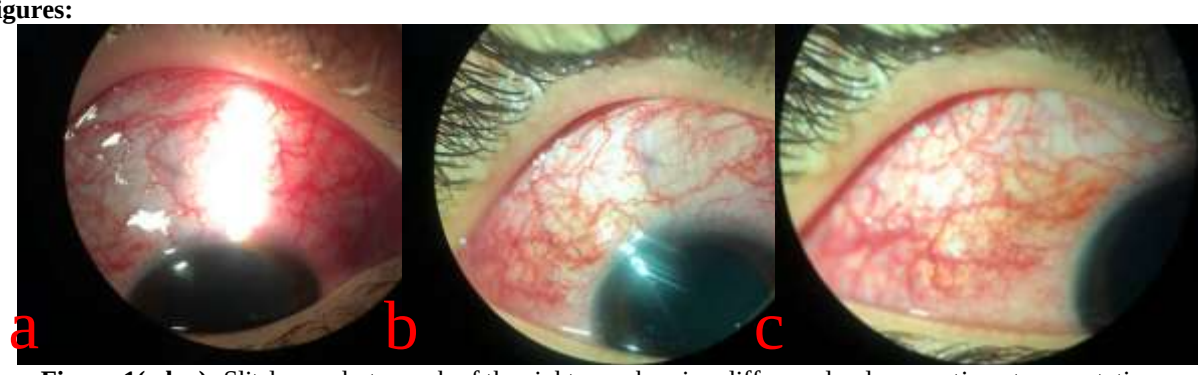
Conclusion:-

Tuberculous scleritis, extremely rare, can present with posterior and/or anterior scleral involvement. High suspicion, clinical evaluation and appropriate investigations can help make the diagnosis of tuberculosis and treat it early and appropriately.

List Of Figures:

1. **Figure 1** (a,b,c): Slit-lamp photograph of the right eye showing diffuse scleral congestion at presentation.
2. **Figure 2:** Ultrasound B-scan of the right eye showing (a)increased choroidal thickness and a 'T' sign (b) widening of sub-Tenon space with scant fluid and (c) elevation of optic papilla (Red arrow).
3. **Figure 3:** (a)fundus examination showing an oedematous optic disc with surrounding subretinal fluid and vascular tortuosity with (b,c) papillary and macular OCT.
4. **Figure 4:** Fluorescein angiography (FA) of both eyes showing in the right early disc staining which gradually increase and persists in late phases; FA normal in the left.
5. **Figure 5:** Mantoux test positive (a) intradermal injection, (b) after 48 h, (c) after 72 h (>15 mm).
6. **Figure 6:** (a)pulmonary radio with (b) chest CT showing evidence of healed pulmonary tuberculosis, (Red arrow) bronchiectasis with (black arrow) precarious centimetric mediastinal adenopathy.
7. **Figure 7 :** (a,b,c) Slit-lamp photograph of the right eye showing regression of scleral congestion after treatment.
8. **Figure 8 :** (a) fundus examination showing regression of the papilledema, subretinal fluid and vascular tortuosity, (b) normal macular OCT.
9. **Figure 9 :** (a,b) Ultrasound B-scan of the right eye demonstrated reduction in sclerochoroidal thickening and resolution of the papilledema.
10. **Table 1:** Demographics, clinical features, investigations, and treatment in tubercular scleritis.

Figures:



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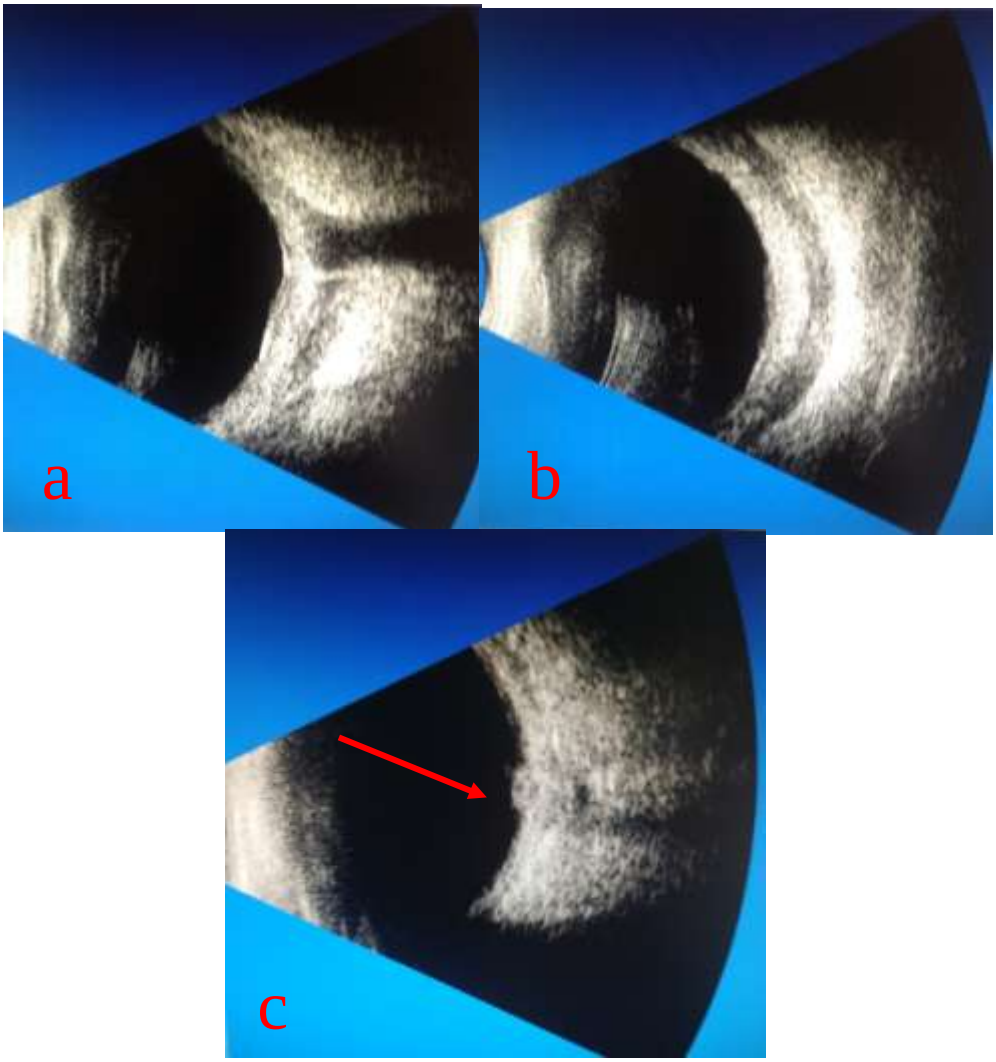

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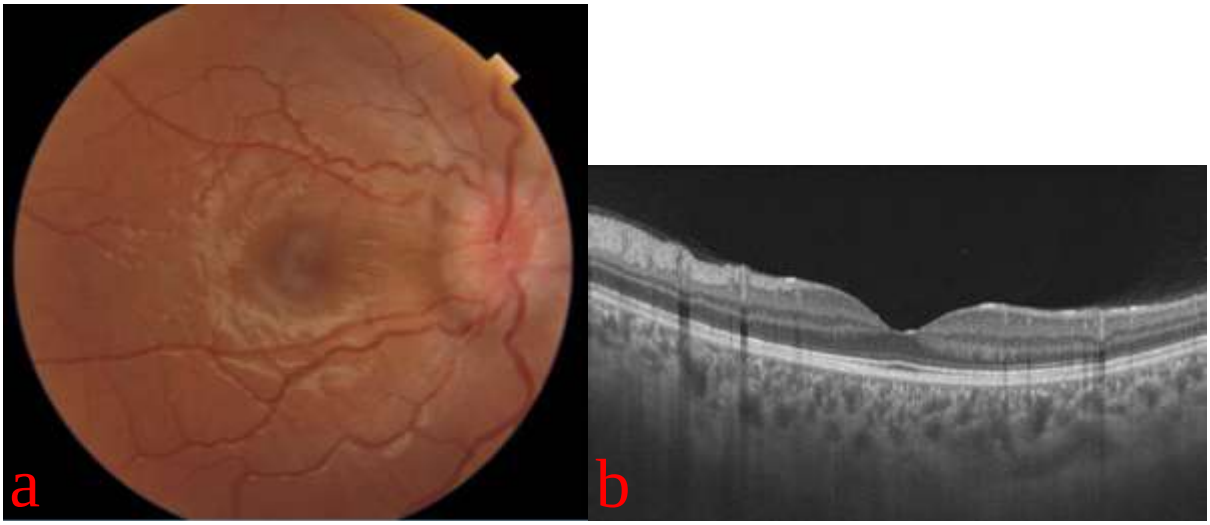

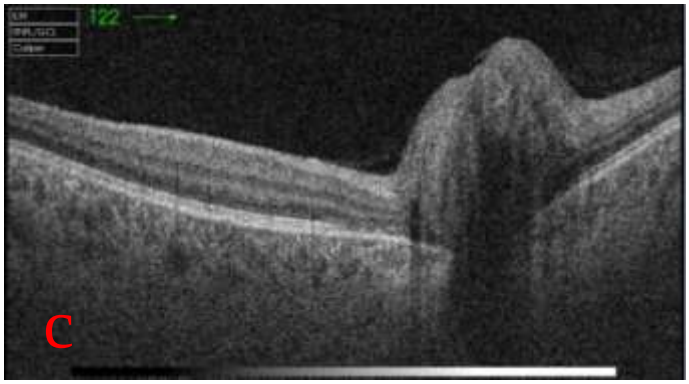

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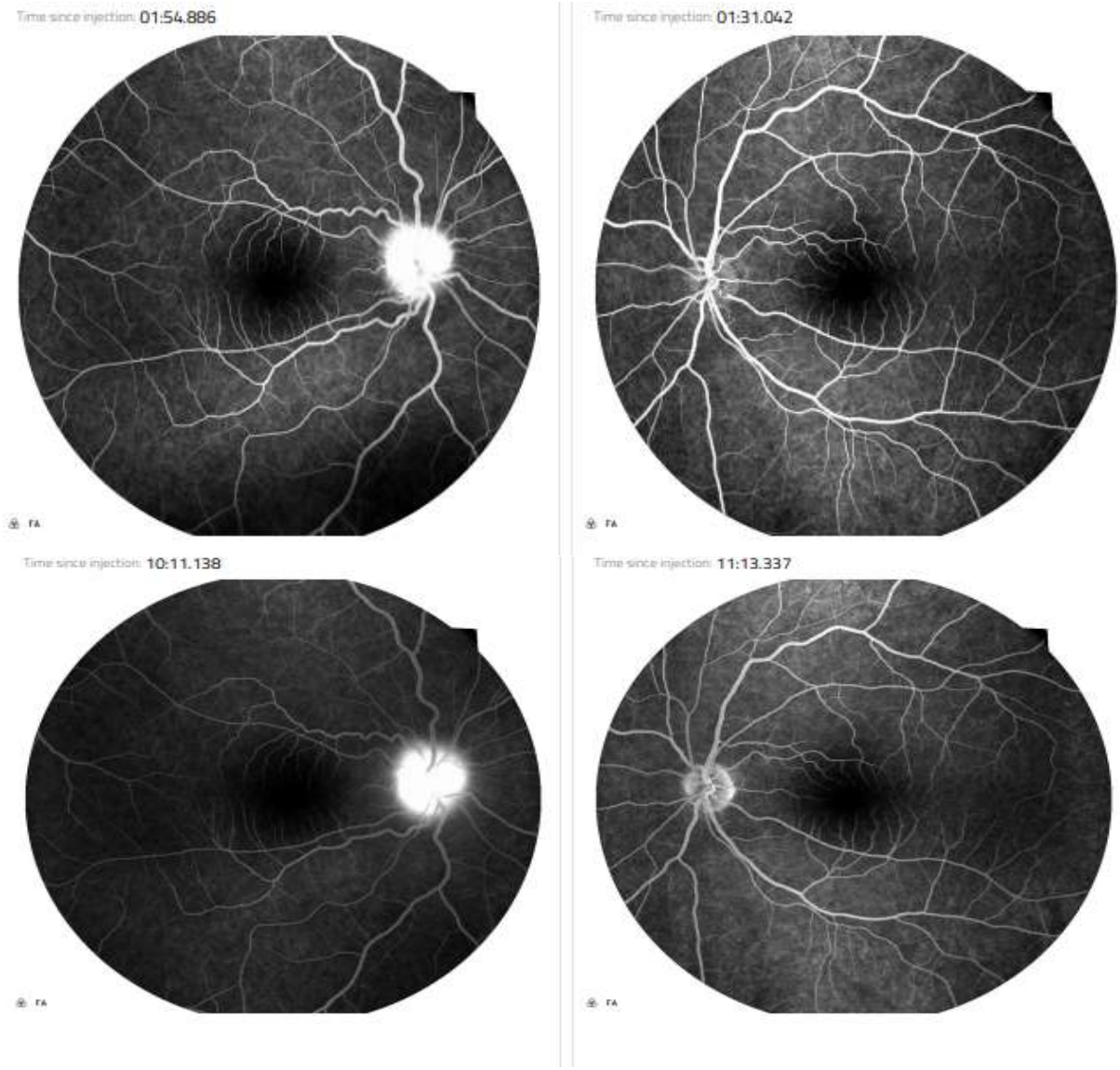

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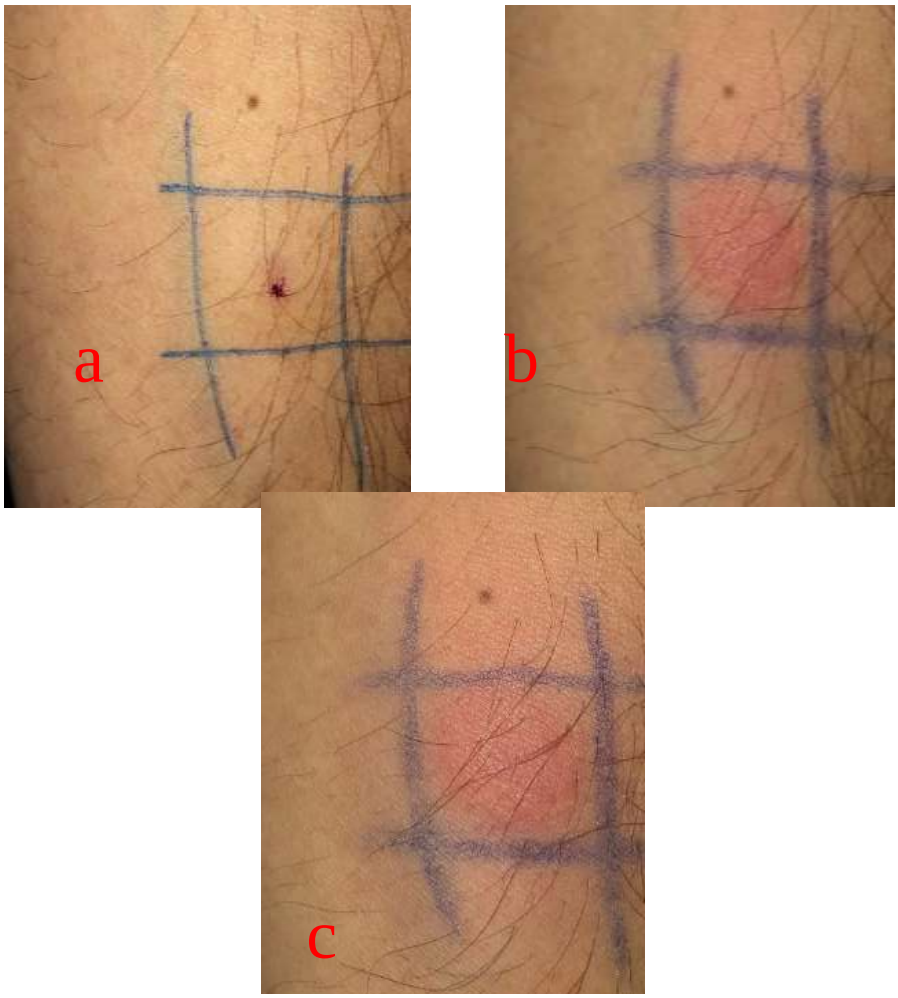

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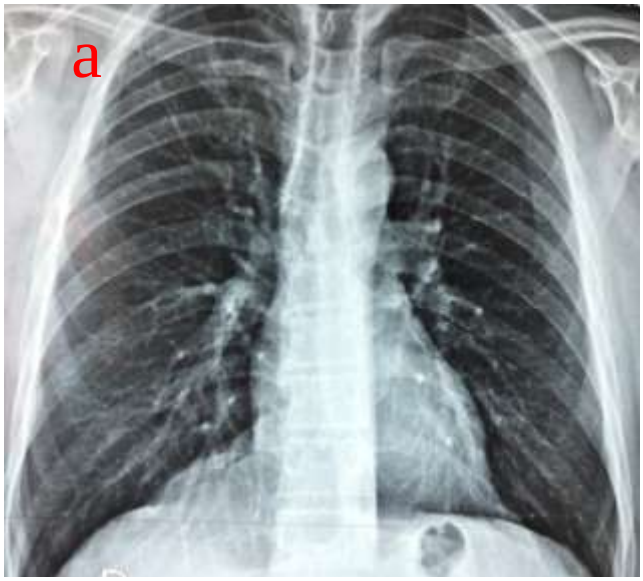

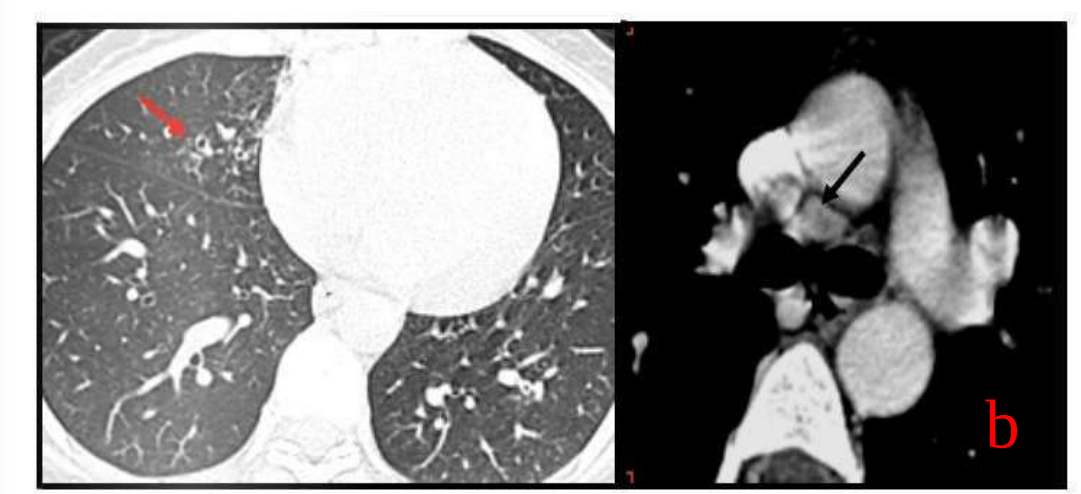

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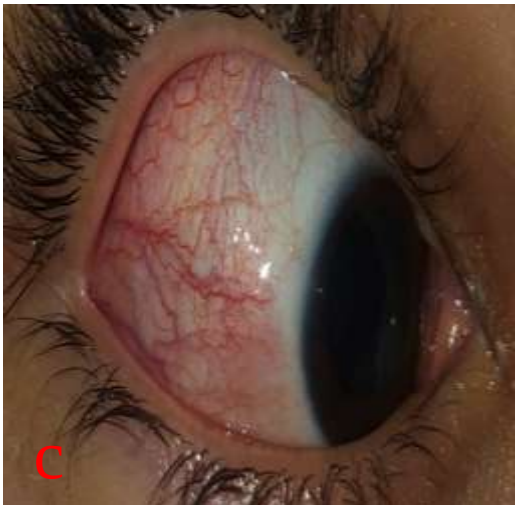

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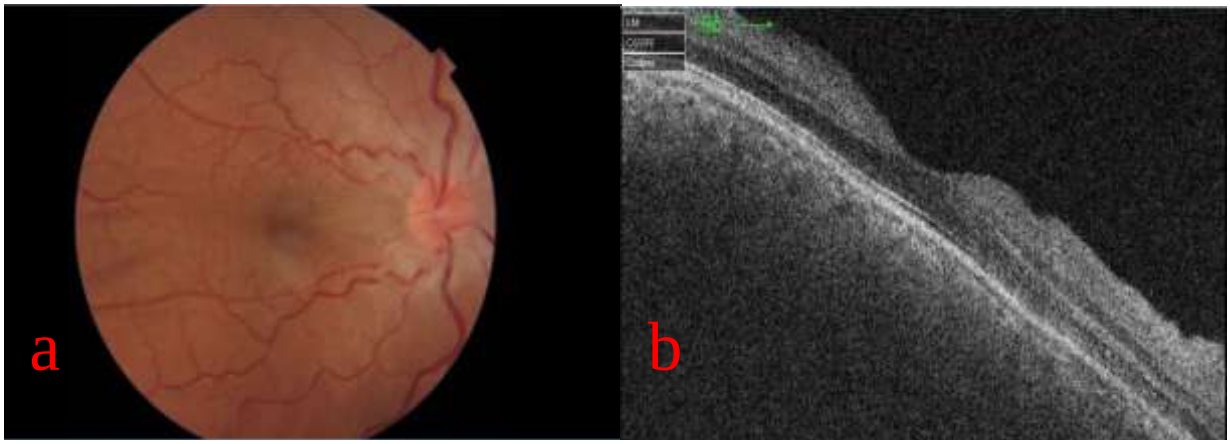

Figure 8:- (a) fundus examination showing regression of the papilledema, subretinal fluid and vascular tortuosity, (b) normal macular OCT.

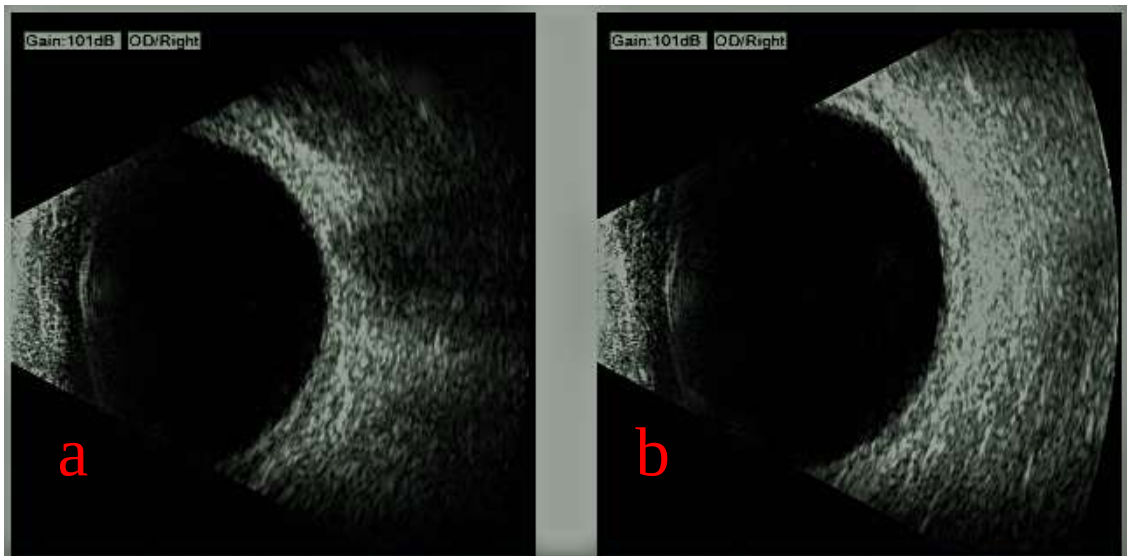

Figure 9:- (a,b)Ultrasound B-scan of the right eye demonstrated reduction in sclerochoroidal thickening and resolution of the papilledema.

Table 1:- Demographics, clinical features, investigations, and treatment intubercular scleritis.

No.	Authors	Age/ Gender/ Immune status	Diagnosis	Positive tests	Negative or Normal tests	Systemic involvement	Presumed or Confirmed TB	Failed treatment	Current Treatment
1	Sharma et al. (2010) [15]	30/F/IC	NNS	Mx 18 mm	CXR Sputum	Absent	Presumed	Topical steroid	Oral steroids Topical steroid ATT
2	Taki et al (2011) (3 cases) [19]	29/F/IC 72/F/IC 68/M/IC	NNS	Mx QTB	CXR	Absent	Presumed	Topical steroid	ATT
3	Lhaj et al. (2016) [17]	43/F/ IC	NNS	Mx – 20 mm QTB	CXR CT chest	Absent	Presumed	Oral NSAIDS Topical steroid	Oral NSAID Topical steroid ATT
4	Parchand et al. (2016) [20]	60/F/IC	NS	Z-N stain PCR MTB IS6110	CXR Mx-6 mm	Absent	Confirmed	Topical steroid	Topical steroid Topical antibiotic ATT
5	Majumder et al. (2018) [21]	23/F/IC	NNS	Mx – 20 mm QTB HRCT	PCR (aqueous) MPB64, IS6110. LN biopsy	Absent	Presumed	Oral steroids Topical steroid Topical antibiotics	Oral steroid ATT
6	Paul et al. (2019) [22]	9/F/IC	NNS	Mx−22x20mm HRCT	-	Absent	Presumed	-	Oral steroids Topical steroid ATT
7	Kariyawasam et al. (2020) [23]	63/M/IS HIV+	NS	Z-N stain HPE Cervical LN biopsy Sputum GeneXpert HRCT	Mx	Present (Lungs)	Confirmed	Oral steroids Topical steroid Systemic antibiotics	ATT HAART
8	Agarwal et al. (2021) [5]	28/F/IC	NS	Z-N stain Culture PCR HPE HRCT, MRI	-	Present (Lungs) (Brain)	Confirmed	Oral steroids Topical steroid Azathioprine	Second line ATT Enucleation
9	Our case	36/F/IC	NNS	MX 30 mm QTB CT chest HRCT	PCR	Present (Lungs)	Presumed	Topical steroid	Oral steroid ATT

M: male, F:female, IC:immunocompetent, IS: immunosuppressed, NNS: non-necrotizing scleritis, NS:necrotizing scleritis,Mx: Mantoux test, CXR: chest X-ray, HRCT: high resolution computed tomography of chest, ZN: Ziehl – neelson, AFB :acid-fast bacilli, , HIV:human immunodeficiency virus, HAART:highly active anti-retroviral therapy.

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