CASE REPORT

EVOLUTION OF PLACOID INFLAMMATORY LESIONS OF TUBERCULOUS SERPIGINOUS-LIKE CHOROIDITIS ON OPTICAL COHERENCE TOMOGRAPHY (OCT) AND BLUE AUTOFLUORESCENCE (BAF)

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Mycobacterium tuberculosis infection can affect the eyes, causing Serpiginous-like choroiditis (SLC) characterized by multifocal choroidal lesions at the level of Retinal Pigment Epithelium (RPE) that coalesce into a serpiginous pattern. It is managed with antitubercular treatment. On the other hand, Serpiginous Choroiditis (SC) is a chronic recurrent inflammatory disorder affecting the RPE layer with choriocapillaries as the primary target, and is managed with immunosuppressants. Differentiating SLC from SC is very important, since treating SLC with immunosuppressants can have devastating consequences for a TB-infected patient. We present a case of Tuberculous SLC in a 22-year-old male describing the evolution of placoid inflammatory lesion of the posterior pole. In addition, the role of BAF imaging in the management of the disease along with the correlation of BAF and OCT image findings has also been discussed.

Keywords: Placoid lesions; Tuberculous; Serpiginous-like Choroiditis; OCT; BAF; Choroiditis

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INTRODUCTION

Serpiginous-like choroiditis (SLC) is caused by infection with *Mycobacterium tuberculosis* resulting in multifocal choroidal lesions at the level of Retinal Pigment Epithelium (RPE) that coalesce into a serpiginous pattern.[1] In contrast, Serpiginous Choroiditis (SC) is a chronic recurrent inflammatory disorder affecting the RPE layer with choriocapillaries as the primary target.[2] Fundus autofluorescence is a non-invasive, novel imaging technique to map lipofuscin pigment deposition in the retinal pigment epithelium layer. Hyperautofluorescence indicates increased metabolic activity of the RPE cells, which is a predictor of dysfunction and hypoautofluorescence signifies loss of photoreceptor cells. Fundus autofluorescence (FAF) provides more information compared to conventional fundus photographs, fluorescein angiography, and optical coherence tomography. FAF also helps monitor disease activity and guide management.[3] Recently, FAF imaging has been introduced into the clinical practice to provide a qualitative assessment of the structure and function of the RPE layer in chorioretinal inflammatory diseases.[4] We present a case of Tuberculous Serpiginous-like choroiditis in a 22 years old male describing the evolution of placoid inflammatory lesion of the posterior pole. In addition, the role of BAF in the management of the disease along with the correlation of BAF and OCT image findings has also been discussed. In our setup, we employed Blue Autofluorescence (BAF) in Heidelberg Engineering (HRA+OCT SPECTRALIS) for imaging.

CASE REPORT

A 22 years old male, with a previous best corrected VA of 6/6 in both eyes, presented with a sudden painless decrease in vision and floaters in the right eye for the past one month. On examination, the patient had Visual Acuity (VA) of 6/18p (right) that improved to 6/12p (right) with pinhole. VA in left eye was 6/6. Examination of the right vitreous revealed 1+ cells and fundoscopy revealed pale yellow lesions at the posterior pole. Workup for decreased vision was initiated.

On one-week follow-up, the VA worsened to 6/24, improving to 6/18 with pinhole. VA in left eye remained normal. Patient was investigated for underlying disease. The patient had a Positive QuantiFERON Gold test, but all other laboratory investigations were unremarkable (Table-1).

Fundus Autofluorescence was used to image and map the retinal pigment epithelium of the patient which showed an acute lesion of SLC, the results of which have been described in a series of images below in chronological order.

In an acute lesion on BAF, there was a well-defined area of increased hyperautofluorescence around the placoid lesion (Figure 1).

Patient was referred to pulmonologist and was advised Anti-Tuberculous Treatment (ATT) and he was started on Tab Rifampicin (150mg) + Isoniazid (75mg) 4 tabs per day.

On one-month follow-up, the VA improved to 6/12 with pinhole in right eye and remained unchanged with glasses (6/12). VA in left eye remained normal. The patient was advised to continue with ATT.

On three-month follow-up, the patient's VA improved to 6/9 right eye and remained the same in left eye. Clinically, the lesions were enlarging. On repeat BAF, the placoid lesions evolved into multifocal pattern with hyperautofluorescent margins (Figure 2). Although, the patient VA had improved, due to the increasing size of the lesion patient was offered systemic steroid therapy. Patient was advised tab Prednisolone on 1mg/kg dose, tapering 5mg on weekly basis with ATT.

On 7-month follow-up, the VA was 6/6 corrected in both eyes. By this time, the patient had uninterruptedly used ATT for 7 months and oral steroids (tapering dose) for 3 months. He was asked to discontinue both ATT and systemic steroids.

On 18-month follow-up, the VA remained stable at 6/6 in both eyes with glasses, with no recurrence (Table-2 for VA and management summary).

The SD-OCT showed localized area of hyperreflectivity in the outer retinal layers involving the RPE, photoreceptors (PRs), external limiting membrane (ELM), and the outer nuclear layer (ONL). The lesion was localized external to the outer plexiform layer. There was also a sliver of subretinal fluid below the macula on SD-OCT (Figure-3 a,b,c).

As the lesions started to heal, they became multifocal over a period of 6 months and were hypoautofluorescent with isolated central areas of hyperautofluorescence on BAF. The SD-OCT scan through the hyperautofluorescent area revealed the hyperreflective elevations in the outer retinal layers. The RPE, PRs and the ELM could not be distinguished. The ONL was distorted (Figure 4 a,b).

As the lesions healed over the next 1 year, they became predominantly hypoautofluorescent with stippled appearance. The SD-OCT scan showed focal loss of RPE, PRs and ELM. As the lesions start to heal, BAF shows increased hypoautofluorescence signifying loss of RPE cells. OCT at this stage shows knobby elevations, which suggests hypertrophy and clumping of RPE cells at these points (Figure 5 a, b, c)

Table-1: - Investigations performed for decreased vision

Test	Result
Full blood count	Hb= 14.5 g/dl, WBC= 4,800 /mm^3 Rest of parameters within normal range
ESR (erythrocyte sedimentation rate)	15 mm/1sthr (cut-off limit= 20 mm/1sthr)
Qualitative C- Reactive protein (CRP)	Negative
HBsAg, Anti-HCV, and Anti-HIV 1 & 2	Negative
Urine R/E	Within normal parameters
TORCH serology	Rubella IgG and IgM= Positive Rest of serology was negative
Mantoux Test	Negative (induration of less than 5 mm) in 72 hrs
QuantiFERON GOLD	Positive
MRI Brain	Normal Study

Table-2: Follow-up VA and management summary

Time Period	VA		PH		Glasses		Treatment given
	Rt	Lt	Rt	Lt	Rt	Lt	
Initial visit	6/18p	6/6	6/12p	6/6	6/18p	6/6	-
1 week follow-up	6/24	6/6	6/18		6/24	6/6	ATT started
1-month	6/24	6/6	6/12	-	6/12	6/6	ATT continued
3-months (Acute Flare)	6/9	6/6	6/9	6/9	6/9	6/6	Oral steroids started ATT continued
7-months	6/6	6/6			6/6p	6/6	CST with ATT and oral steroid
18-month	6/6	6/6			6/6	6/6	ATT stopped

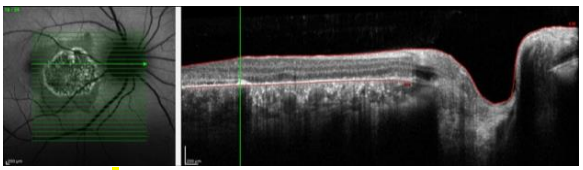

Figure-1: BAF image, right eye. Well-defined area of increased hyperautofluorescence around the placoid lesion.

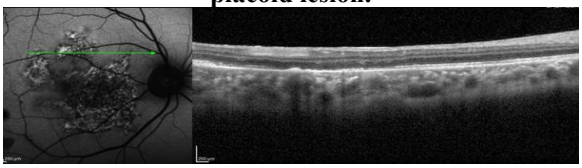

Figure-2: BAF image, right eye. Placoid lesions

have evolved into multifocal pattern with hyperautofluorescent margins.

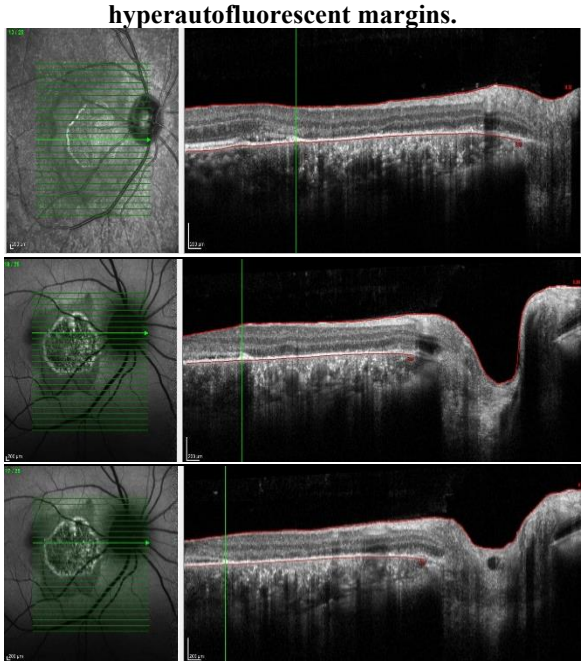

Figure 3(a): SD-OCT showing sliver of subretinal fluid along with localized area of hyper-reflectivity in the outer retinal layers involving the RPE, PR, ELM, and ONL (b,c): Well defined area of hyperautofluorescence around placoid lesion on BAF imaging

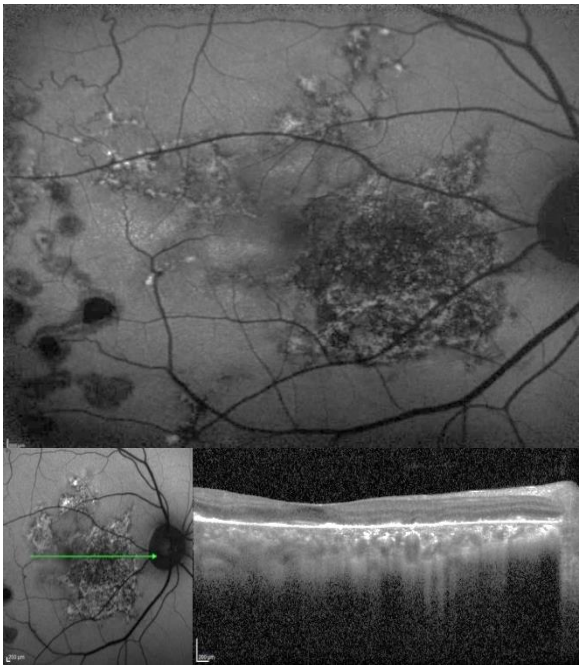

Figure-4: (a) On BAF,a multifocal hypoautofluorescent placoid lesion observed with isolated central areas of hyperautofluorescence. (b) SD-OCT shows hyperreflective elevations in outer retinal layers. The RPE, PRs and ELM indistinguished.ONL is distorted.

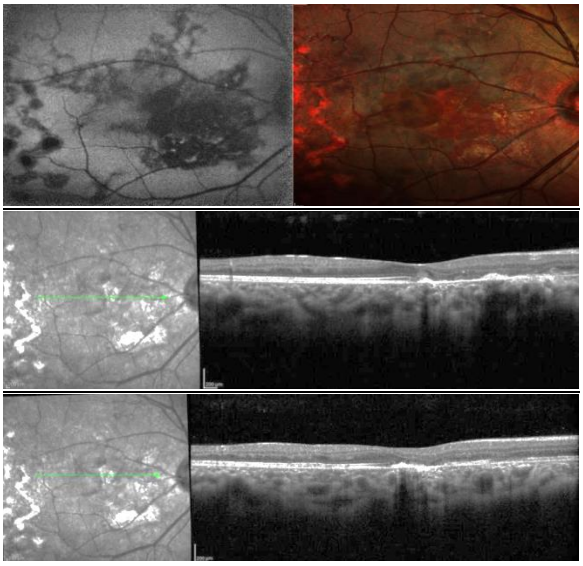

Figure 5: (a) BAF image on Left; colour fundus photo on right. Hypoautofluorescent stippled appearance is noted.
(b,c)SD-OCT shows focal RPE, PRs and ELM loss. Knobby elevations signifying RPE hypertrophy and clumping, are present.

DISCUSSION

A possible association between Serpiginous choroiditis and tuberculosis was first described in 1952.[5] Recently, Intraocular tuberculosis has been described as a separate disease from the conventional Serpiginous choroiditis.[6,7] Making a diagnosis of Serpiginous-like choroiditis can be a daunting task. Labelling a case as SLC requires suggestive clinical features, a positive PPD or QuantiFERON Gold test after excluding other causes. Moreover, isolating *Mycobacterium* DNA from ocular fluids confirms the diagnosis. In the absence of the aforementioned findings, response to antitubercular treatment can also suggest a diagnosis of SLC. Usually, due to the absence of enough objective evidence, a suspected case can be categorized as presumed intraocular tuberculosis.[8] To differentiate SLC from SC is of utmost importance, since the treatment of SLC with immunosuppressants can be devastating.[9] Tuberculous SLC cases are usually from endemic areas and have a positive PPD or QuantiFERON Gold test. Clinically, SLC can be differentiated from SC by the presence of vitreous inflammation and involvement of the posterior pole usually unilaterally with sparing of the juxtapapillary region. On the other hand, the lesions in SC are bilateral, involve the optic disc, and also spread to the macula.[8]

The pathophysiology of SC involves damage

localized to the RPE cells and choriocapillaries.[10]The pathophysiology of SLC is unknown. One theory is that SLC is a hypersensitivity reaction to *Mycobacterium* and involves the RPE and choriocapillaries.[1]Another probable hypothesis is that infectious damage involving the RPE cells is responsible for the disease manifestations. The hypothesis is reinforced by isolation of *Mycobacterium* from the RPE cells and resemblance of RPE cells to alveolar macrophages.[11] Moreover, resolution of symptoms with anti-tuberculous therapy also supports the theory of infectious damage by *Mycobacterium tuberculosis*.[12]

SLC lesion can also resemble relentless placoid chorioretinitis which is characterized by numerous multifocal lesions.[13] In our case, the positive QuantiFERON GOLD test and a favourable response to antituberculous medications favoured Tuberculous SLC. Other infectious etiologies like Toxoplasma and herpes can also mimic SC due to the involvement of RPE and choriocapillaries.[14–16]

BAF imaging provides insight into disease activity and the transition from the acute stage to the stage of healing. In the acute stage, lesions are predominantly hyperautofluorescent. As the healing ensues, lesions become predominantly hypoautofluorescent. Changes in the outer retinal layers on OCT correlated with corresponding changes on BAF. Hyperautofluorescence in the acute stage signifies increased metabolic activity of the RPE, which corresponds to retinal oedema of the outer retinal layers on OCT scan. The retinal oedema is a sign of acute inflammation involving the RPE cells. As the lesions starts to heal, BAF shows increased hypoautofluorescence signifying loss of RPE cells. OCT at this stage shows knobby elevations, which indicates hypertrophy and clumping of RPE cells at those points.[3]

The RPE, PRs, Inner Segment-Outer Segment junction and ELM are also indistinguishable at this stage on OCT scan. As the lesions heal completely, BAF is predominantly hypoautofluorescent, the SD OCT shows complete loss of RPE-photoreceptor complex.[17]

It is possible that after starting treatment with ATT, there is paradoxical worsening of symptoms and signs on OCT and BAF. According to one study, alteration in the fluorophores of the RPE cells can be altered by inflammation via certain pro-oxidative pathway, which could lead to the finding of hyper-autofluorescence on BAF.[18] Our case showed new areas of hyperautofluorescence after initiating treatment. Findings of new hyperautofluorescent areas after initiating treatment could signify acute inflammatory response following treatment with anti-tuberculous drugs. Another study reported continued progression of the disease in 14% of patients with SLC despite being on ATT. Autoimmune damage similar to the Jarisch-Herxiheimer reaction seen in syphilis maybe at play here. The paradoxical worsening of the symptoms was controlled by starting the patients on corticosteroids.[19]

CONCLUSION

Blue autofluorescence is a novel imaging technique that can be used to evaluate tuberculous serpiginous-like choroiditis and assess response to treatment. BAF can show enlargement of the initial lesions after starting anti-tuberculous therapy due to an autoimmune response induced by dying *Mycobacteria*. This can be prevented by giving antitubercular therapy under the cover of steroids.

The purpose of this study was to observe the evolution of placoid lesions in SLC using BAF and OCT imaging as guiding modalities in the management of SLC and in differentiating SLC from SC, due to their widely different management approaches.

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