

Clinical profile, multimodal imaging, and treatment response in macular serpiginous choroiditis

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Purpose: To describe the clinical profile, multimodal imaging, and treatment response in macular serpiginous choroiditis (MSC). **Methods:** Clinical records of 16 eyes (14 patients) with MSC presenting to a tertiary eye care institute between 2015 and 2019 were analyzed retrospectively. **Results:** Mean age of 14 patients presenting with MSC was 33 ± 13 yrs with 64% males and 36% females. Mean visual acuity of the eyes with MSC at presentation was 0.43 ± 0.46 (logMAR) improving to 0.16 ± 0.28 (logMAR) at final visit. Thirteen eyes (81.3%) had active lesion at presentation. Mantoux test was positive in seven patients (50%) and QuantiFERON TB gold test positive in 10 patients (71%). HRCT chest showed latent tuberculosis in seven patients (50%). All patients underwent multimodal imaging. All patients received oral steroids as treatment therapy; 11 patients also received immunosuppressives, nine patients received additional anti-tubercular therapy (ATT). Mean duration of follow-up for the patients was 18 ± 10 months. A total of eight (50%) eyes had recurrence of lesions after an average duration of 14 ± 14 (3-36) months and were restarted on the treatment as per the requirement. At final follow-up, all eyes showed a good response to treatment and had healed lesions. Comparing the final BCVA to the initial BCVA, 38% (n = 6) showed improvement, 56% (n = 9) remained stable, and 6% (n = 1) eyes worsened at the final follow-up. **Conclusion:** Clinical profile and presentation of MSC is similar to that of CSC, and combination treatment with intravenous methyl prednisolone (IVMP), steroids, immunosuppressives, and ATT can salvage vision. A high suspicion of associated tuberculosis in endemic regions should be kept in mind.

Key words: Fundus fluoroscein angiography, Indocyanin green angiography, macular serpiginous choroiditis, multimodal imaging, optical coherence tomography angiography, serpiginous choroiditis, tuberculosis

Serpiginous choroiditis (SC) is an asymmetric, usually bilateral disease with chronic, recurrent, and progressive course affecting the retinal pigment epithelium (RPE), choriocapillaris, and choroid. Active SC presents with grayish-yellow, cream-colored lesions at the RPE with overlying retinal edema, spreading to the periphery in a contiguous fashion.^[1-3]

Based upon etiology of the disease, Vasconcelos-Santos *et al.*^[4] have classified SC as Classic Serpiginous Choroiditis (CSC) and Serpiginous-like Choroiditis (SLC). CSC is the term used to denote autoimmune, non-infectious variety of SC. It is often idiopathic in origin. SLC is often associated with infective etiology, mostly associated with tuberculosis, especially in endemic countries like India.^[5-8]

However, the exact etiology in many cases with SC remains elusive. This takes precedence in Indian population where large number of cases are thought to be tubercular or viral in origin, thus giving rise to the term SLC.

Most commonly, the lesions are seen in peripapillary region, although the macular involvement is seen in about 5.9% of the cases. Earlier involvement of the foveal center as well as increased risk of choroidal neovascular membrane formation are associated with a poorer visual prognosis in eyes with macular SC (MSC).

A higher proportion of misdiagnosed cases due to lesions similar to age-related macular degeneration also results in delayed institution of treatment with subsequent larger macular scarring.

As SC presents with varied clinical manifestations in different geographical locations, it becomes imperative to differentiate and detect the etiology early because instituting immunosuppressive therapy can have devastating consequences on the concomitant tuberculosis, if present. Also, delaying the anti-tubercular therapy (ATT) in undetected cases may result in recurrent choroiditis lesions resulting in larger macular involvement and poorer visual prognosis.

Therefore, we believe that it is important to understand the morphological and clinical features in eyes with MSC especially in Indian population with a large number of cases with tuberculosis. We undertook this study isolating the cases with macular involvement without any peripheral manifestations to ascertain if these eyes behave in a fashion different from the ones with CSC. MSC can affect visual prognosis grossly because of early involvement of macula. This study reports the clinical profiling of a large series of MSC with multimodal imaging and treatment response.

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Methods

This was a retrospective, observational case series approved by the Institutional Review Board of Vision Research Foundation, Chennai, India. The study was carried out in a tertiary eye care center in South India. The medical records of patients with a confirmed diagnosis of MSC presenting between January 2015 and October 2019, with a minimum follow up of 1 year were analyzed retrospectively. Patients with lesions exclusively in the macular region, confined within the vascular arcades were included in this study. The other variants of SC (peripapillary and multifocal serpinginous choroiditis) and patients with less than 1 year of follow-up were excluded. The study was approved by the Institutional Review Board and adheres to the tenets of the Declaration of Helsinki.

All the patients underwent a complete ophthalmic examination including detailed medical and ophthalmic history and demographic profiling, active TB or a contact with a patient with active TB. All the study participants had undergone multimodal imaging including color fundus photography, fundus autofluorescence (FAF), and fundus fluorescence angiography (FFA) at their initial visit. Patients were advised additional imaging procedures such as indocyanine green (ICG) angiography, optical coherence tomography (OCT), optical coherence tomography angiography (OCTA), and Humphery Visual Field (HVF) at their initial and at follow-up visits, whenever necessary. All the patients underwent routine hemogram, Mantoux test, QuantiFERON TB gold test, chest X-ray, and high resolution computed tomography (HRCT) of chest as part of the choroiditis panel study. Patients were advised to evaluate their central vision of both the affected and the contralateral eye with home Amsler's chart.

After a detailed evaluation and confirmed diagnosis, these patients were started on systemic corticosteroids as a mainstay of therapy. Patients with active, serious sight-threatening lesions were treated with intravenous methyl prednisolone (IVMP) 1 gm per day for three consecutive days initially, followed by a course of oral steroids (tab prednisolone 1 mg/kg of body weight) in tapering dose. Immunosuppressives were used as an adjunctive therapy or as a steroid sparing therapy in recalcitrant cases. Standard 4-drug regimen antituberculosis treatment (ATT) was advised for 9 months in all patients with suggestive laboratory, radiologic and systemic findings of tuberculosis, given alongside the immunosuppressive therapy. A pulmonologist's review was sought prior to starting ATT in all these patients.

Visual outcomes, number of recurrences, response to treatment, and complications developed during the follow-up till 1 year were the main outcomes of the study. Best corrected visual acuity (BCVA) was assessed using Snellen chart in all patients at initial and final visit and was converted to logMAR units for the purpose of analysis. Visual outcome, defined as 'improved' if the change in visual acuity of more than or equal to two lines at their final follow-up, 'worsen' if the deterioration of more than two lines, and 'stable' if it remained unchanged, was noted. Statistical analysis was done using Statistical Package for the Social Sciences version 20.0. Results were considered significant if $P < 0.05$.

Results

A total of 16 eyes of 14 patients with clinical diagnosis of MSC were included in the study. There were 64% males ($n = 9$) and 36% females ($n = 5$). Age of presentation ranged from 15 years to 52 years (mean 33 ± 13 years). A total of nine patients (64%) had bilateral SC with seven patients having classic/multifocal type of SC in other eye and two patients having bilateral MSC, and five patients had unilateral involvement.

Most common chief complaint of patients at presentation was blurring of vision ($n = 9$), whereas five patients presented with central scotoma. Table 1 shows the complete clinical profile of the patients with MSC. Four patients presented de novo to the outpatient department with no history of prior treatment and 10 patients had some treatment at other centers and were referred for further course of management. All the eyes had quiet anterior chamber (no cells/flare), whereas one patient had vitreous cells at presentation. Thirteen eyes had active disease at presentation, whereas three eyes had healed/inactive presentation.

Mantoux test was positive in seven (50%) patients. QuantiFERON Tb gold test was positive in 10 patients (71%). HRCT demonstrated lesions that were consistent with latent tuberculosis in seven patients (50%). Based on these investigations, nine (64%) patients were started on standard 4 drug regime of anti-tubercular drugs (rifampicin, isoniazid, ethambutol, and pyrazinamide) for initial 2 months and two drugs rifampicin and isoniazid for 7 months was continued. Remaining patients were started on steroids/IVMP/ immunosuppressive therapy, as discussed earlier.

Funduscopically, these patients had lesions similar to CSC but located initially in macular area with peripapillary sparing. The active lesions were sharply demarcated, discoid/confluent translucent patches with serpentine-like borders, gray-yellowish in color and located deep to the retina, with overlying edematous retina in the macular area within the arcades. The eyes with healed lesions presented with large chorioretinal scars at the macula, ranging in size from 2.5-disc area to 6-disc area, and a healthy retina between the scar and optic nerve. None of the eyes presented with choroidal neovascular membrane.

In this study, all patients underwent color fundus photo and fundus autofluorescence (FAF) imaging. FAF features [Fig. 1] included hypo-FAF lesions due to accumulation of melanin pigments, with hyper-FAF margins showing activity at the corresponding margins of the lesions. Healed lesions showed complete hypo-FAF with sharp irregular margins. Autofluorescence has recently emerged as an investigation of choice due to its non-invasive nature and ability to diagnose active lesions in SC.^[9]

Fundus fluorescein angiography (FFA) was done in all 14 patients. FFA [Fig. 2a and c] showed early phase hypo-fluorescence of the lesion due to blocked fluorescence secondary to the overlying edematous retina and inflamed RPE. In the mid phase, lesions showed hyperfluorescent borders due to leakage from choriocapillaris at the margin of the lesion. The late-phase angiogram showed stippled hyper-fluorescence of the lesions and later the entire lesion became hyperfluorescent with irregular borders due to diffuse leakage from larger choroidal vessels. Healed lesions showed marked hypo-fluorescent areas with sharply delineated margins in early phases of angiogram due to the extensive destruction of choriocapillaris. Later-phase films showed hyper-fluorescence due to diffuse staining of these lesions as the dye diffused into the scarred area from surrounding healthy choriocapillaris. All the FAF lesions were seen to be larger than those seen with FFA.

ICG angiography (ICGA) [Fig. 2b and d] was done in 10 patients and it showed blockage of dye in both early and late phase in the area of lesions due to non-perfusion of choriocapillaris and choroidal vessels as well as blocked fluorescence due to inflamed RPE and retina. The extent of the area involved delineated by ICG was seen to be larger than that marked by fluorescein angiography in all the active cases.

Optical coherence tomography (OCT) imaging was done in 13 patients. OCT imaging [Fig. 3] of active lesions in MSC

Table 1: Clinical profile of the patients with macular serpiginous choroiditis

Case no	Presenting complaint	Duration of symptoms	Age (in years)/ Gender	Laterality	Eye	Visual acuity		Disease status		Treatment	Recurrence	Complications
						Initial visit	Final visit	Initial visit	Final Visit			
1	Blurred vision	8 Months	48/M	B/L	L	6/6	6/7.5	Active	Healed	Oral steroids, Azathioprine	Yes	NA
2	Central scotoma	1 Week	20/M	U/L	L	6/9	6/6	Active	Healed	Oral steroids, ATT	Nil	NA
3	Blurred vision	1.5 Years	23/M	B/L	L	6/36	6/60	Active	Healed	Oral steroids, Azathioprine	Nil	NA
4	Blurred vision	4 Years	46/M	B/L	R	6/18	6/18	Active	Healed	Oral steroids, IVMP, Azathioprine, ATT	Nil	NA
5	Central scotoma	1 Week	42/M	B/L	L	6/45	6/6	Active	Healed	Oral steroids, PST, IVMP, Azathioprine	Nil	NA
6	Blurred vision	1 Week	52/F	U/L	R	6/6	6/6	Active	Healed	Oral steroids, IVMP, ATT	Nil	Cataract
7	Central scotoma	1 Year	20/M	B/L	R	6/6	6/6	Healed	Healed	Oral steroids, Azathioprine	Nil	NA
8	Central scotoma	1 Month	36/M	U/L	R	6/6	6/6	Active	Healed	Oral steroids, MMF, ATT	Yes	NA
9	Blurred vision	2 Months	39/F	B/L	R	6/18	6/6	Active	Healed	Oral steroids, IVMP, MMF, ATT	Yes	CNVM
10	Blurred vision	3 Months	39/M	U/L	L	2/60	6/12	Active	Healed	Oral steroids, MMF	Nil	CNVM
11	Blurred vision	4 Months	26/F	U/L	R	6/12	6/9	Active	Healed	Oral steroids, IVMP, Azathioprine, ATT	Nil	NA
12	Blurred vision	2 Weeks	15/F	B/L	R	6/18	6/6	Healed	Healed	Oral steroids, ATT	Yes	NA
					L	6/18	6/9	Healed	Healed		Yes	NA
13	Blurred vision	3 Days	11/F	B/L	R	CF@2m	3/60	Active	Healed	Oral steroids, IVMP, PST, Azathioprine, ATT	Yes	NA
					L	6/6	6/6	Active	Healed		Yes	NA
14	Central scotoma	1 Week	42/M	B/L	L	6/36	6/6	Active	Healed	Oral steroids, PST, IVMP, Azathioprine, Cyclosporine, ATT	Yes	NA

R: Right eye, L: Left eye, M: Male, F: Female, B/L: Bilateral, U/L: Unilateral, IMT: Immunosuppressive therapy, IVMP: Intravenous methyl prednisolone, PST: Posterior sub tenon injection, ATT: Anti tubercular therapy, CNVM: Choroidal neovascular membrane, NA: Not applicable, MMF: Mycophenolate mofetil

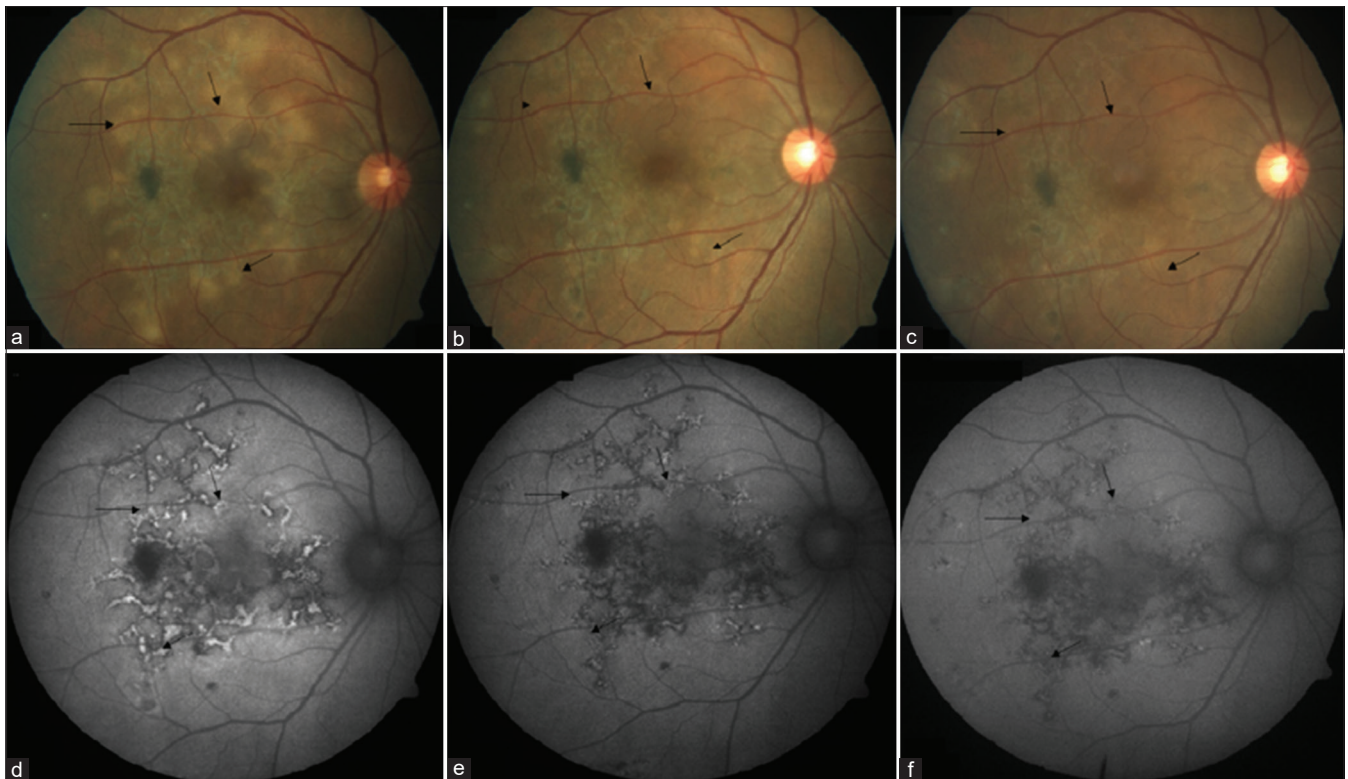


Figure 1: (a-c): Color fundus photograph of posterior pole of the right eye showing progressive resolution of lesions (a) (baseline), (b) (1 month) and (c) (3 months) on treatment, with active lesions ((a), black arrows) seen as yellowish lesions with fuzzy margins and retinal edema, with overlying vitreous haze; healing lesions (b) show more indistinct margins, and resolving retinal edema; and inactive lesions (c) showing remnant retinal pigment epithelial (RPE) atrophy with resolved retinal edema. (d-f): Corresponding Fundus autofluorescence (FAF) images of the same eye. Active lesions ((d) black arrows) showing a central hypo FAF with surrounding hyper FAF; healing lesions (e) show a linear hypo FAF surrounding central but reduced hyper FAF; and healed lesions (f) showing a uniform hypo FAF corresponding to the region of RPE atrophy

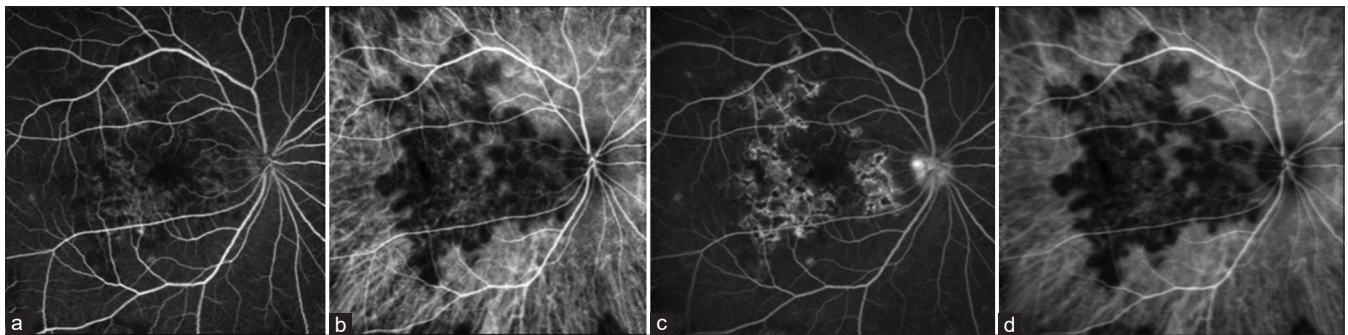


Figure 2: Fundus fluorescein angiography (FFA) and indocyanine green angiography (ICG) images of the corresponding right eye images as in Fig. 1. FFA and ICG angiography done at presentation. (a) (FFA) and (b) (ICG) early phase angiography images of the same patient showing hypofluorescence of the lesion. (c) Late phase FFA showing hyperfluorescence of the lesions due to diffuse leakage from the lesions. Optic disc leakage is also seen in late phase. (d) Late phase ICG angiography continues to show hypoperfusion of choriocapillaris and choroidal vessels

showed inflammation of RPE and outer retinal layers, seen as hyperreflective bands in outer retina with loss of ellipsoid zone and IS-OS junction. Healed lesions showed retinal thinning and decreased subfoveal choroidal thickness in the areas of chorioretinal scarring. Two eyes had developed choroidal neovascular membrane during the course of follow-up, and were managed with intravitreal injections of anti-VEGF agents.

OCT angiography (OCTA) is a recently introduced non-invasive imaging technique, which can detect the changes in retinal and choroidal vasculature without the use of dye injection. In this study, 10 patients underwent OCTA imaging [Fig. 4], which showed decreased vascularity

in choriocapillaris slab and choroidal region and relatively retained inner retinal vascularity. Over the course of treatment, the flow voids seen in choriocapillaris layer reduced in areas and was replaced with irregular capillary like network. Thus, OCTA can be used to explain the loss of RPE and photoreceptor layer that is supplied by outer choroidal vasculature.

HVF using 30-2 protocol [Fig. 5] was done in 11 patients. Although this investigation is not indicated in routine cases, especially with macular involvement, we carried out the HVF for analyzing the changes taking place with effective treatment. Visual fields evaluation showed dense absolute and/or relative scotoma in active MSC, which corresponds to the size, shape,

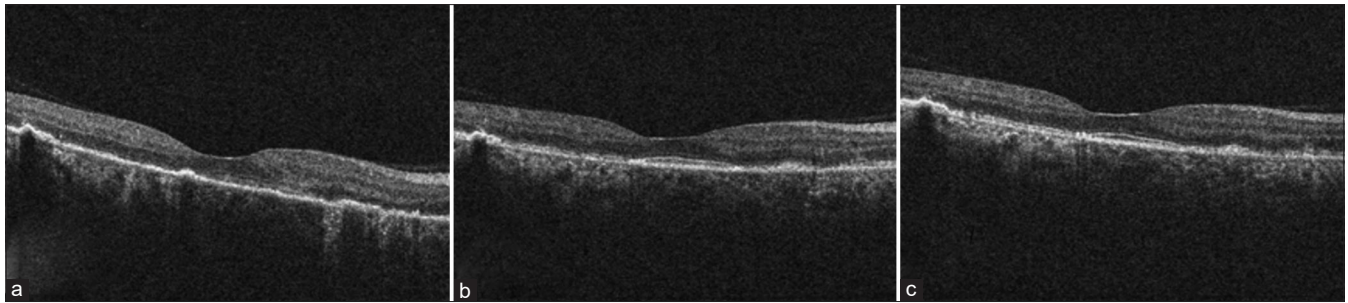


Figure 3: OCT images taken at presentation (a), 1 month (b), 3 months (c). (a) showing the hyperreflective outer bands due to destruction of RPE, photoreceptor layers and outer retinal layers with loss of Ellipsoid Zone (green arrow), increased retinal thickness adjacent to fovea (orange arrow) due to activity. (b) and (c) Inactive lesions show reduced retinal edema at corresponding location, and relative preservation of Ellipsoid zone at macula (blue arrow). Also, RPE layer backscattering is seen more during the healing phase

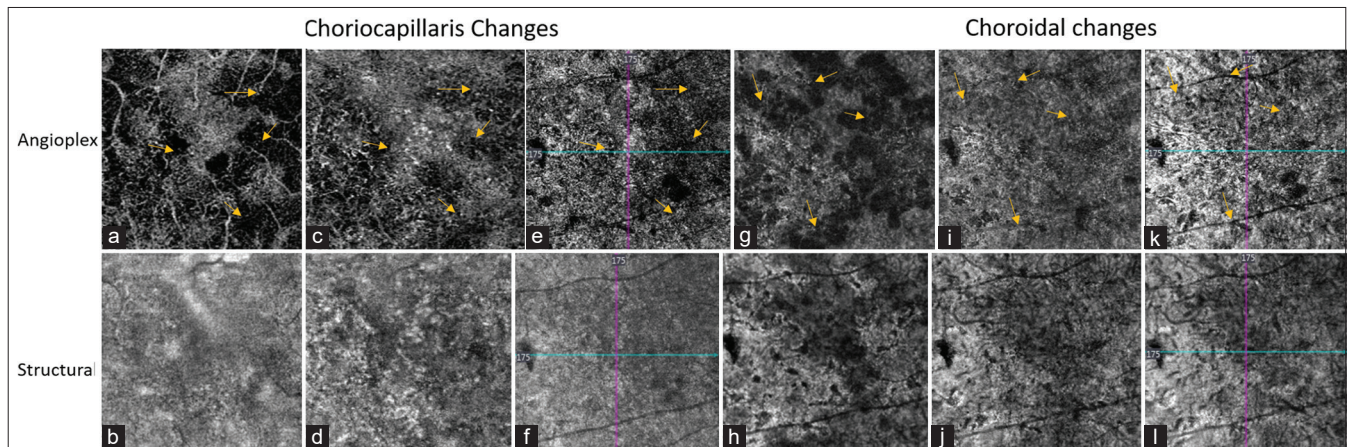


Figure 4: Optical coherence tomography Angiography done at baseline (a, b, g, h), 1 month (c, d, i, j) and 3 months (e, f, k, l). OCTA shows decreased vascularity in choriocapillary ((a), orange arrows) and choroidal region ((g), yellow arrows) seen as dark areas. The dark areas seen in choriocapillaris reduced in area (c and e) and was replaced with irregular capillary-like network as the lesions showed improvement with treatment at subsequent visits

and location of the lesions, and mostly appearing as central scotoma. With the treatment, the central scotoma became less dense and also showed a decrease in size corresponding to the resolution of the lesion.

Systemic corticosteroids were given in all patients. Seven (50%) patients with severe vision-threatening disease required IVMP. Posterior sub tenon (PST) injection was administered in three (18.8%) eyes. Immunosuppressive agents were used in 11 (79%) out of 14 patients. Azathioprine (n = 8, 57%) was the commonest immunosuppressive used. Mycophenolate mofetil was used in three patients (21.4%). One patient (7%) was treated with azathioprine initially, but due to multiple recurrent lesions was changed to cyclosporine. As mentioned earlier, nine patients were started on ATT at presentation based on the investigations.

Patients were advised to have regular home Amsler's chart monitoring and review immediately if they notice any enlargement of scotoma or metamorphopsia. A total of eight (50%) eyes had recurrences after an average duration of 14 ± 14 (3–36) months and were restarted on the treatment (steroids + immunosuppressants) tailored to each patient. Three patients (19%) in our cohort developed ocular complications of which, one eye developed cataract (6%), and two eyes (13%) developed choroidal neovascularization membrane. Resolution of lesions was noted in all the eyes at the final visit. Mean duration of follow-up

was 18 ± 10 months (range 8–43 months). Mean visual acuity at initial presentation was 0.43 ± 0.46 logMAR units and 0.16 ± 0.28 logMAR units at final follow-up. Regarding visual outcome, we noted a statistically significant improvement in vision ($P = 0.032$). While comparing the final BCVA to the initial BCVA, 38% (n = 6) showed improvement, and 56% (n = 9) remained stable and 6% (n = 1) eyes worsened.

Discussion

In this study, we analyzed the clinical profile, multi-modal imaging, and response to treatment in eyes with isolated MSC. The results indicate that there is not much difference between patients presenting with MSC in terms of clinical profile, imaging as well as response to therapy. However, a high suspicion of an associated systemic focus of tuberculosis, especially in the endemic regions should be kept in mind by the clinicians. Although 50% eyes in our study showed recurrence of the disease, the response to treatment at a follow-up period of 1 year was satisfactory in all. We believe that this entity should be detected early as differentials of MSC include eyes with wet form of age-related macular degeneration, toxoplasmosis and so on in the active phase, whereas healed cases may be confused with scarred choroidal neovascular membrane, and other forms of macular scar like trauma, toxoplasmosis and so on. Correct diagnosis with an early investigation of latent tuberculosis can result in early

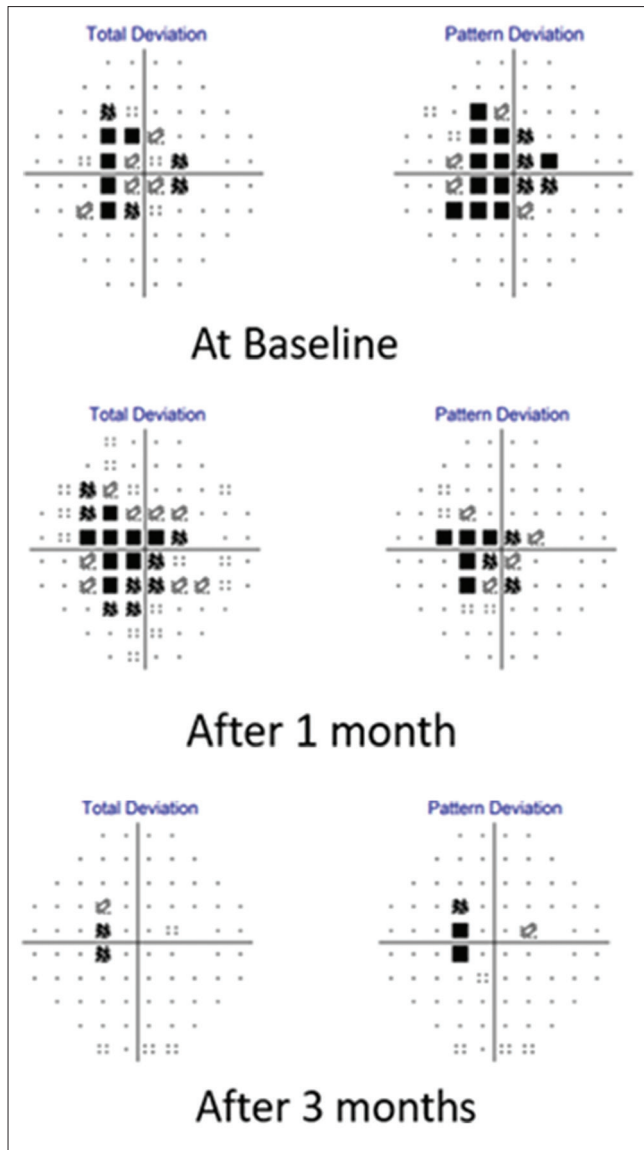


Figure 5: Right eye HVF using 30-2 protocol showed involvement of central visual field at presentation and showed improvement with treatment over the subsequent follow-ups

institution of ATT with an early resolution of a dreaded disease. In terms of ophthalmic perspectives, this study carries great significance. Early involvement of macular region and a possible development of choroidal neovascular membrane can cause significant visual impairment in such eyes, and should be treated early. A high index of suspicion for recurrent episodes translates into a good communication between the clinician and the patient, with earlier presentation if any visual distortion is noted by the patient.

Hardy and Schatz^[10] were the first to describe macular geographic helicoid peripapillary (GHPC) choroidopathy in eight patients as a separate clinical entity with clinical profile similar to typical GHPC but with early macular involvement. Masour *et al.*^[11] described similar macular variant of SC in four patients with extensive vision loss. Sahu DK and Rawoof^[12] reported MSC in nine eyes of six patients with mean age of 30.5 years and reported that MSC can mimic other diseases with posterior pole involvement like APMPE, idiopathic

subretinal neovascular membrane, pigment epithelitis, and posterior scleritis.

This study included the patients with SC, which had involvement of macular region and the disease was confined to the vascular arcades, and there was sparing of peripapillary region. As compared to the CSC where the patients often report late in the course of disease, when the macula is involved. In MSC the patients tend to present early with vision loss with macular involvement. The visual prognosis is also not good in MSC if not treated aggressively. The clinical profile of MSC is similar to CSC in terms of age at presentation, no gender or laterality predilection, good response to steroids and immunosuppressants, similar progressive nature of disease with recurrences.

The clinical investigations, including OCT and OCTA, were helpful in studying the structural and angiographic changes in the disease. The findings were consistent as done by few authors previously. Agarwal *et al.*^[13] studied the choroidal vascular changes in SC using extended depth imaging protocol in OCT and found significantly increased choroidal thickness in active lesions, whereas healed lesions showed decreased choroidal thickness and total choroidal area. OCT also helps in observing the follow-up of the patient for the development of choroidal neovascular membrane, which appears as sub retinal or sub RPE neovascular membrane. OCTA findings in our study are consistent with the findings of Ahn *et al.*^[14] and El Ameen *et al.*^[15] who studied the case reports on ICG angiography and OCTA in cases of SC. All such eyes may not need every investigation listed in the paper, and a tailored approach should be followed for each eye. As noted, the FAF and ICGA were able to detect the lesion in its entire dimension, much larger than FFA could pick up. This is not surprising, as FFA would allow imaging of the innermost retinal layers, whereas FAF and ICGA target the deeper retinal and choroidal layers better. There are a few studies in which multi-modal imaging has been applied to study such eyes, and the results are concurrent with our study.^[16,17]

The disease has natural course with possible recurrence after a period of latency, and newer lesions tend to develop at the active borders of healed lesions. In this study 50% cases (n = 8) had recurrent episodes of the disease with average time of recurrence being 14 ± 14 months. Recurrent episodes of disease were also managed similarly with steroids and immunosuppressants as per the dosage required.

To the best of our knowledge, there are very limited studies on MSC and with the advancement of multi modal imaging modalities, the disease entity has been studied in more detail. With timely and more aggressive intervention, the study noted a significant difference in baseline visual acuity and in final visual acuity, which could be attributed to the initiation of ATT along with systemic corticosteroids in cases with tuberculous etiology. The treatment requires a much personalized and tailored approach, and long-term steroids and immunosuppressants may be required to have better control on the disease. The retrospective nature of the study may be one of the limiting factors for the study, but it highlights the importance of multimodal imaging in early diagnosis of the disease and early intervention can salvage useful for the patient.

Conclusion

To conclude, MSC is a vision-threatening, clinically distinct variant of SC with presumed tubercular etiology in endemic countries like India. IVMP should be used in such cases if presented in active stage of disease. Multimodal imaging helps to identify the activity status of the disease and extent of the disease, and helps to diagnose the recurrence early with

regular follow-up, so that the disease can be controlled with a minimal damage.

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Conflicts of interest

There are no conflicts of interest.

References

- Schatz H, Maumence AE, Patz A. Geographical helicoid peripapillary choroidopathy: Clinical presentation and fluorescein angiographic findings. *Trans Am Acad Ophthalmol Otolaryngol* 1974;78:747-61.
- Hamilton AM, Bird AC. Geographical choroidopathy. *Br J Ophthalmol* 1974;58:784-97.
- Weiss H, Annesley WH Jr, Shields JA, Tomer T. The clinical course of serpiginous choroidopathy. *Am J Ophthalmol* 1979;87:133-42.
- Vasconcelos-Santos DV, Rao PK, Davies JB, Sohn EH, Rao NA. Clinical features of tuberculous serpiginouslike choroiditis in contrast to classic serpiginous choroiditis. *Arch Ophthalmol* 2010;128:853-8.
- Gupta V, Gupta A, Arora S, Bambery P, Dogra MR, Agarwal A, *et al.* Presumed tubercular serpiginouslike choroiditis: Clinical presentations and management. *Ophthalmology* 2003;110:1744-9.
- Bansal R, Gupta A, Gupta V, Dogra MR, Sharma A, Bambery P, *et al.* Tubercular serpiginouslike choroiditis presenting as multifocal serpiginoid choroiditis. *Ophthalmology* 2012;119:2334-42.
- Gupta V, Agarwal A, Gupta A, Bambery P, Narang S. Clinical characteristics of serpiginous choroidopathy in North India. *Am J Ophthalmol* 2002;134:47-56.
- Nazari Khanamiri H, Rao NA. Serpiginous choroiditis and infectious multifocal serpiginoid choroiditis. *Surv Ophthalmol* 2013;58:203-32.
- Gupta A, Bansal R, Gupta V, Sharma A. Fundus autofluorescence in serpiginouslike choroiditis. *Retina* 2012;32:814-25.
- Hardy RA, Schatz H. Macular geographic helicoid choroidopathy. *Arch Ophthalmol* 1987;105:1237-42.
- Mansour AM, Jampol LM, Packo KH, Hrisomalos NF. Macular serpiginous choroiditis. *Retina* 1988;8:125-31.
- Sahu DK, Rawoof A, Sujatha B. Macular serpiginous choroiditis. *Indian J Ophthalmol* 2002;50:189-96.
- Agarwal A, Agrawal R, Khandelwal N, Invernizzi A, Aggarwal K, Sharma A, *et al.* Choroidal structural changes in tubercular multifocal serpiginoid choroiditis. *Ocul Immunol Inflamm* 2018;26:838-44.
- Ahn SJ, Park SH, Lee BR. Multimodal imaging including optical coherence tomography angiography in serpiginous choroiditis. *Ocul Immunol Inflamm* 2017;25:287-91.
- El Ameen A, Herbolt CP Jr. Serpiginous choroiditis imaged by optical coherence tomography angiography. *Retin Cases Brief Rep* 2018;12:279-85.
- Cozuba R, Ungureanu E, Instrate SL, Alexandrescu C, Nanu RV, Carstocea L, *et al.* Macular serpiginous choroiditis-case report. *Rom J Ophthalmol* 2018;62:217-21.
- Dutta Majumder P, Biswas J, Gupta A. Enigma of serpiginous choroiditis. *Indian J Ophthalmol* 2019;67:325-33.