A Study to Analyse the Presentation of Autoimmune and Idiopathic Serpiginous Choroiditis in Indian Population

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ABSTRACT

BACKGROUND

Serpiginous choroiditis (SC) is an intraocular inflammatory disorder displaying a geographic pattern of choroiditis, extending from the juxtapapillary choroid and intermittently spreading centrifugally. It involves the overlying retinal pigment epithelium (RPE), the outer retina including the choriocapillaries and the choroid.[1,2,3] Infectious diseases like tuberculous (TB) uveitis, herpes simplex virus (HSV) uveitis whose fundus changes mimic SC are termed as serpiginous-like choroidopathy (SLC). On slit lamp examination, anterior segment usually appears quiet, non-granulomatous anterior uveitis with mild vitritis and / or fine pigmented cells within the vitreous can be seen. The pattern of fundus involvement varies between the two groups. Fundus fluorescein angiography and indocyanine green angiography (FFA and ICG) are important modalities of investigation that help in differentiating the pattern of involvement and confirming clinical findings. The duration of follow up, reactivation of lesions and complications vary. Hence, it is important to differentiate between SC and SLC for proper diagnosis and appropriate management. The aim of this study is to highlight important features of serpiginous choroiditis and serpiginous like choroidopathy that will aid in the correct diagnosis of these two entities.

METHODS

This is a retrospective study of 40 patients. Following variables were analysed - age, gender, laterality, visual acuity, and intraocular inflammation through slit lamp examination, pattern of involvement, choroidal-neovascularization, reactivation, clinical investigations and diagnosis.

RESULTS

32 patients had serpiginous choroiditis (SC) and eight patients had serpiginous like choroiditis (SLC). Mean age was 50 and 51 years (SC and SLC respectively). Males were predominantly affected (65.5 % in serpiginous choroiditis and 62.5 % in serpiginous like choroiditis). Bilaterality was 80 % in SC-group and 46 % in the SLC-group. Vitreous haze was lesser than or equal to 1 + in SC group. The juxtapapillary-area was involved in 90 % in SC eyes and 0 % in SLC-group. Midperiphery of fundus was involved in 54 % of SLC-group. Reactivation is more common in SLC group than in SC group in a follow up period of one year. Choroidal-neovascularisation was found in two patients only in SLC-group.

CONCLUSIONS

In cases where vitreous haze is greater than 1 + with unilateral involvement and disease free peripapillary area is present, an infectious aetiology has to be strongly suspected, as an immunomodulatory therapy could have severe consequences.

KEYWORDS

Serpiginous Choroiditis, Serpiginous like Choroiditis, Autoimmune

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BACKGROUND

Historical Perspective

Hutchison originally described this disease entity as multifocal choroiditis secondary to tuberculous aetiology but then with recent molecular advancement studies, an auto immune association has been established. These diseases are synonymous with peripapillary choroiditis, helicoid peripapillary choroidal sclerosis, helicoid peripapillary chorioretinal degeneration, geographic helicoid peripapillary choroidopathy, geographic helicoid choroidopathy, serpiginous choroidopathy, and recently serpiginous-like choroiditis (SLC).[1] The term SLC and multifocal serpiginoid choroiditis (MSC) were used by Gupta and associates in 2003 and 2012, respectively, to differentiate SC due to tubercular ethology from classic SC (CSC).[2,3] Although the lesions in SC are not typically multifocal, they are often included in spectrum of white dot syndrome by many authors[4]

Serpiginous choroiditis (SC) is an intraocular inflammatory disorder displaying a geographic pattern of choroiditis, extending from the juxtapapillary choroid and intermittently spreading centrifugally.[5] It involves the overlying retinal pigment epithelium (RPE), the outer retina including the choriocapillaries and the choroid.[6,7,8] Infectious diseases like TB uveitis, HSV uveitis whose fundus changes mimic SC are termed as serpiginous-like-choroidopathy (SLC). It is important to differentiate between SC and SLC for proper diagnosis and appropriate management. SC is bilateral, chronic, progressive but asymmetrical affecting middle age men, associated with human leukocyte antigen (HLA-B27), with no familial or ethnic predisposition.[1] The trigger provoking ocular immune response with involvement of uvea and retina due to molecular mimicry remains unknown in idiopathic SC.[8,9] Infectious agents are also suspected to incite such localized immune response leading to SLC.[10,11,12]

Clinical Features

Patient presents with unilateral blurring of vision, photopsia / metamorphopsia and central / paracentral scotoma, which can be either absolute or relative.[13,14] On slit lamp examination, anterior segment usually appears quiet, non-granulomatous anterior uveitis with mild vitritis and / or fine pigmented cells within the vitreous can occur. Intraocular pressures are normal.[15,16] Infectious SLC is mostly associated with underlying causative agents such as *Mycobacterium tuberculosis*, Herpes simplex or *Treponema pallidum*.

Histopathology

Histopathological reports of eyes with SC remain sparse. An inflammatory reaction, localized primarily in choroid with extensive infiltration of choroid by lymphocytes, has been described by Wu et al.[17] This infiltration was relatively higher at the margins of the atrophic scars. The scarring was characterized by the loss of the RPE and photoreceptor layers with focal defects of the underlying Bruch's membrane. Fibro glial tissue was observed over the inner surface of Bruch's membrane and some part of the fibro glial tissue was noted to invaginate into the choroid through the breaks in Bruch's membrane.

Clinical Varieties of SC

Classic or peripapillary geographic SC is most common type involving 80 % cases of SC. The lesion begins with ill-defined patches of greyish / creamy yellow sub retinal infiltrates starting at the peripapillary area, progressively involving the periphery in a serpentine fashion. Retinal oedema due to secondary spread may result in serous retinal detachment.[18]

Macular serpiginous choroiditis has the worst prognosis due to macular involvement frequently complicating to choroidal neovascular membrane. These patients usually have symptoms early in the course of the disease, resulting in less extensive involvement of the posterior retina and choroid.[19] Macular SC lacks the characteristic geographic atrophic scars, and juxta papillary choroid may not be involved. Ampiginous choroiditis is a rare variety with multifocal lesions involving the periphery. It was first thought as a recurrent form of acute posterior multifocal placoid pigment epitheliopathy (APMPPE) resembling SC in its bilateral and recurrent nature. Knowledge on HPE findings learnt from studying clinically inactive lesions in chronic cases suggest that there are focal aggregates of lymphocytes with moderate mononuclear inflammatory cell infiltrating the choroid, especially the margins of lesions. Fibrotic choroidal lesions are surrounded with variable degrees of RPE hyperplasia. Presence of defects in Bruch's membrane with atrophic choriocapillaris, RPE, and photoreceptors has also been elicited.

On fundus auto-fluorescence, lesions appear as hyper and hypo-auto fluorescence patches with sharp margins. With further progression they appear as hyper-auto fluorescence patches with hypo-auto fluorescent margins, finally speckled or granular hyper-autofluorescence.[20] Indocyanine green angiography (ICG) is more sensitive than FFA in determining choroidal lesions. It shows two patterns hypo-fluorescent areas beginning from early to late phase with ill-defined margins indicating non-perfusion of the chorio-capillaries.[21,22]

Secondly it may appear as early hypoautofluorescence with increased fluorescence toward the late phases with faint edges revealing areas of delayed filling, late perfusion of choriocapillaris. Optical coherence tomography (OCT) reveals increased retinal thickness with hyper reflectivity of outer retina and choroid (waterfall effect) in active lesions and loss of outer retinal architecture inner segment / outer segment and external limiting membrane (IS / OS and ELM loss) with thinning of retina in healed lesions. Visual field testing will demonstrate dense scotoma, corresponding in size, shape, and location to active lesions becoming less dense scotoma as disease activity subsides.[23,24]

Electroretinogram (ERG) and electrooculogram (EOG) is normal except in extensive diseases involving macula. Choroidal neovascularization with secondary haemorrhage, exudation, and serous retinal detachment has been described to occur in approximately 13 % - 20 % of eyes

with SC in long term studies.[25] Few cases may complicate to serous retinal detachment during the active phase. Shallow exudative detachment of the retina resolves as disease activity subsides. Other complications include RPE detachment, cystoid macular oedema occur in active macular serpiginous choroiditis.[26,27] Retinal vasculitis, sub-retinal fibrosis, retinal vascular occlusions, inflammation and neovascularization of the optic disc have also been seen uncommonly in later phases if left untreated.

Optical coherence tomography angiography (OCT-A) is relatively a new modality of non-invasive investigation which can produce depth-resolved, high-resolution images of retinal and choroidal vasculature by detecting intravascular blood flow based on split-spectrum amplitude-decorrelation angiography without injecting the dye. OCTA in central serous chorioretinopathy (CSC) demonstrated decreased vascularity on choriocapillaris but intact retinal vascularity El Amin and Herbert compared the OCT-A and ICG images in SC and observed that the hypofluorescent, hypo perfused areas on ICG correspond to the dark areas seen in the choriocapillaris layer of OCT-A. The authors found ICG more preferable because of its ability to delineate choriocapillary lesions more clearly than OCT-A.

Treatment

Serpiginous choroiditis is usually treated with high dose corticosteroids and immune suppressants. Immunosuppressive agents such as methotrexate, azathioprine, cyclosporine, chlorambucil, or cyclophosphamide can help to attain longer period of disease inactivity and reduce the risk of potential side effects associated with high-dose systemic steroids. However, immunosuppressive agents usually take longer time to attain the desired level of therapeutic concentration of the drug and thus cannot be used to treat acute exacerbations. Immunosuppressive treatment with alkylating agents (chlorambucil and cyclophosphamide) has been found to be associated with long-term drug-free remission of SC.

Intravitreal corticosteroids aid in providing an inflammation free period of close to 8 months and are essential in the management of autoimmune choroidal diseases. The use of intravitreal drugs also provides adequate concentration of steroids for prolonged drug release. They reduce posterior pole manifestations and reduce macular signs. SLC is treated with anti-tuberculosis treatment (ATT). Many patients on ATT alone is not enough and may need anti-inflammatory therapy as supplementation. The use of anti-inflammatory agents and steroids must be done with caution as many of these patients may develop adverse effects due to the use of these drugs and develop complications.

We wanted to analyse the features of serpiginous and serpiginous like choroiditis.

METHODS

A retrospective descriptive analysis of all patients diagnosed with serpiginous choroiditis and serpiginous like choroidopathy was done between October 2017 - December 2019. A total of 40 patients were diagnosed to have fundus findings that met our defining criteria. We included only those patients who had proven to have SLC or SC on fundus examination that was confirmed by FFA and ICG and who had a follow up period of at least 1 year. We excluded all those patients who had other causes and those who had a follow up period of less than 1 year.

Demographic data were gathered from the medical records of all patients and compared. Variables analysed were age, gender, laterality, visual acuity, intraocular inflammation, pattern of involvement, choroidal-neovascularization, reactivation, clinical investigations and diagnosis.

The patients were divided on basis of diagnosis and confirmatory tests into serpiginous choroiditis group and serpiginous like choroidopathy group and all the demographic data and clinical variance was analysed to look at the variability in presentation.

Slit Lamp bio-microscopy was done with a 78 D lens, Intra ocular inflammation was documented and graded on slit lamp examination in the anterior segment using the standardization of uveitis nomenclature (SUN) classification system. Vitreous haze was graded and documented with Grade 1 being mild and Grade 4 being severe using the Nussenblatt classification. Presence of choroidal neovascularization and pattern of involvement was confirmed using FFA and ICG. All the observational data was analysed and compared with the overall population and the data is represented in tables as follows.

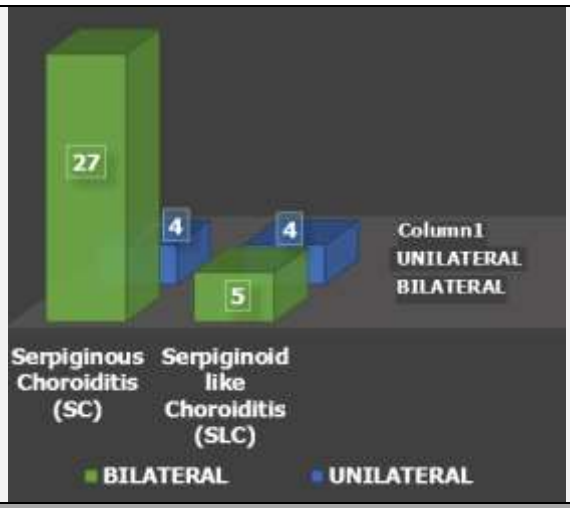
RESULTS

Criteria	Serpiginous Choroiditis (SC)	Serpiginoid Choroidopathy (SLC)
Total number of cases	32	8
Mean age	50	51
Gender - Males	21	5
Females	11	3
Laterality - Unilateral	27	4
Bilateral	5	4
Vitreous haze	Less than or equal to 1+	More than 2+
Juxtapapillary area involvement	29 cases	0 cases
Involvement of mid periphery	0 cases	4 cases
Reactivation of lesions	8 / 32	6 / 8
Choroidal neovascularization	0 cases	2 cases

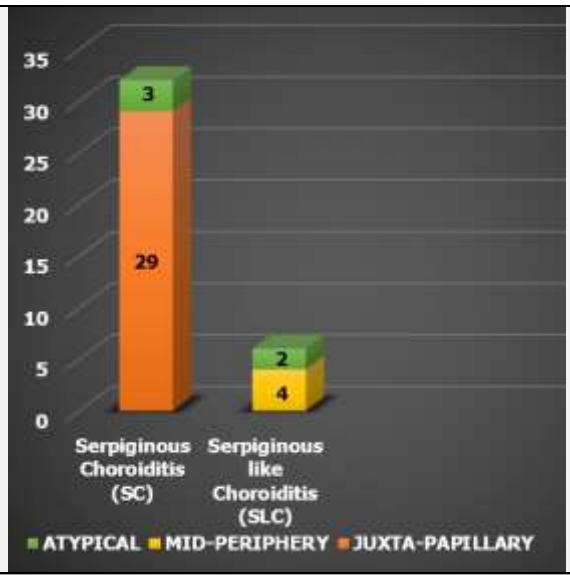
Table 1. Clinical Profile of SLC versus SC

A total of 40 patients were included in our study. Thirty-two patients had SC and eight patients had SLC. Mean age was 50 years for serpiginous choroiditis patients and 51 years for serpiginous like choroiditis patients. Males were predominantly affected. 21 males had SC whereas only 5 males had SLC. Laterality of presentation was analysed in both groups, bilaterality was found in 27 cases in SC-group and 4 cases in the SLC-group. Fundus changes were analysed, and vitreous haze was documented. Vitreous haze was lesser than or equal to 1 + in SC group whereas in the SLC group it was 2 +.

The juxtapapillary area was involved in 29 cases in SC eyes and whereas none of the patients in SLC-group had juxtapapillary involvement.



Graph 1. Laterality



Graph 2. Pattern of Involvement

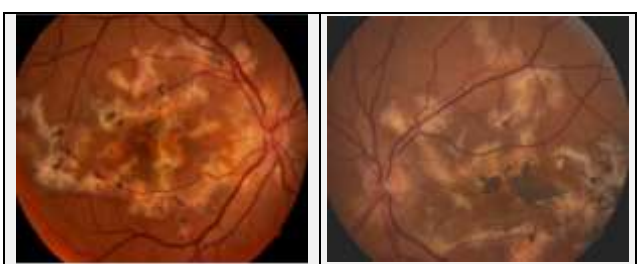


Figure 1. Serpiginous Choroiditis

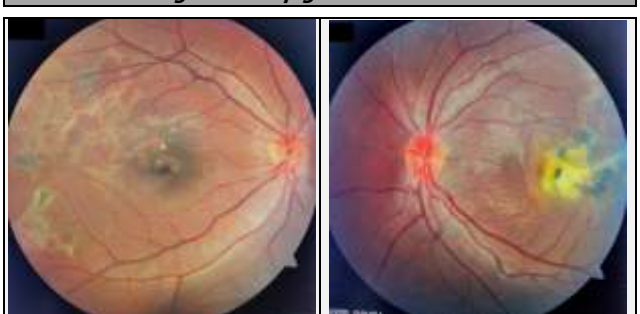


Figure 2. Serpiginous Like Choroiditis

Midperiphery of fundus was involved in 4 cases in the SLC group whereas none of the patients in the SC group had mid periphery involvement. Reactivation is more common in SLC group because 6 / 8 patients had reactivation of lesions when followed up than in SC group where only 8 off the 32 patients had a reactivation in a follow up period of one year. Choroidal neovascularization was found in two patients only in SLC-group whereas this was not a feature in the SC group.

DISCUSSION

Recent publications highlight that intraocular tuberculosis may present with features simulating serpiginous choroiditis.[28,29] In the present study, we compared distinctive clinical aspects of SLC with classic SC. It is always very important to differentiate between these 2 entities because the immunosuppressive treatment which is given for SC has several adverse effects and may lead to reactivation of tuberculosis infection or even death.[30] On the other hand, the toxicity of antituberculosis treatment is very significant.[31] The incidence of serious adverse effects in patients receiving these drugs approaches 10 % in routine clinical practice and which is worse in elderly patients,[32,33] who are more often affected by SC.

Patients with SC were found to have autoimmune background, whereas patients with SLC had infective aetiology, with tuberculosis being commonly associated. Tuberculosis aetiology is suspected in cases of SC since many years.[28] In patients from areas endemic for tuberculosis, Gupta and colleagues[34] described SLC of presumed tubercular aetiology that mimicked SC. Previously, evidence of tuberculosis infection was either direct or indirect, and response to antimicrobial therapy was pursued to distinguish between SC due to infectious and non-infectious causes. Later, in few recent studies, some distinctive features of SLC and classic SC were compared and analysed. In our study sample, vitreous inflammatory cell infiltration was a notable feature of our patients with presumed SLC, but vitreous haze was less than or equivalent to 1 + in classic SC.

Moreover, patients with SC and SLC differed with regards to the distribution of choroidal lesions. Choroidal lesions in SLC were mostly unilateral, multifocal involving the mid periphery, while bilateral involvement was seen in patients with classic SC, with comparatively larger lesions extending from the juxtapapillary area. Ustinova and colleagues[35] compared the clinical features of SC with cases of established tuberculosis chorioretinitis. They aimed to prove that the former had no association with tuberculous aetiology. Tuberculosis aetiology was hypothesized in the 32 cases in their first category, but this was subsequently ruled out by further workup and empirical treatment. Interestingly, some patients with SC had evidence of pulmonary tuberculosis, similar to patients with tuberculous chorioretinitis.[35] Their peripapillary geographic choroiditis cases were mostly bilateral, extending from the juxtapapillary area, similar to the classic SC cases in our research. This trend differed from that found with tuberculosis chorioretinitis in their cases. However, their cases of tuberculosis chorioretinitis, showed solitary or numerous small lesions that are seen in multifocal choroiditis and are less likely to be clinically confused with classic SC.

The pathophysiology of presumed SLC is unknown. There is speculation that it might be associated with a hypersensitivity to microbial antigens (*M. tuberculosis*, syphilis, herpes) clinically manifesting as an inflammatory involvement of the RPE, choriocapillaris, and choroid that mimics SC.[32,33] However, it is not easy to rule out the related infectious components in these situations. This site may be suggested as a potential reserve for dormant bacilli due to the existence of tuberculous bacilli at the level of RPE, as well as the similarity between these cells and alveolar macrophages. Such local inflammation may also result either from reactivation of dormant bacilli or often, choroidal seeding due to reactivation anywhere else in the body. This hypothesis of an infectious component is reinforced by the favourable response only after initiation of anti-tuberculosis treatment.[33,34] Entities such as toxoplasmosis,[36] herpes zoster infection, syphilis,[37] and sarcoidosis [38] have also been re-ported to mimic SC, and it may be that other conditions with extensive involvement of the RPE and choriocapillaris could produce a similar picture. Both SC / SLC can have different aetiologies, and the presenting characteristics imply that site of tissue damage may contribute to such varied clinical features for different infectious agents.

The research was too limited to attempt statistical analysis, even though it was retrospective in nature. However, this comparative sequence has raised the important issue of clinically separating SLC from classic SC, both for diagnostic purposes and for subsequent appropriate therapeutic and prognostic reasons. Further immunologic evidence of tuberculosis infection is required to support our current research, as well as a therapeutic response, and further follow up is also needed.

CONCLUSIONS

In our study, patients with SLC commonly showed unilateral involvement, with multifocal lesions mostly involving the mid-periphery, sparing the juxtapapillary choroid, along with inflammatory cells in the vitreous indicating significant vitritis. Whereas, in classic SC patients, bilateral involvement with larger solitary geographic or serpentine lesions, extending from the juxtapapillary region was more common, with no or minimal vitritis. In order to choose the most appropriate mode of management, it is very important to clinically distinguish between the two conditions. Those patients with SLC require treatment with appropriate antimicrobial agents along with steroids, whereas those with SC are usually managed with immunosuppressive drugs. There are various serious adverse effects associated with both modalities of treatments. To further validate the proposed differences between SLC and SC, further prospective studies in large cohorts are required.

Data sharing statement provided by the authors is available with the full text of this article at jebmh.com.
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REFERENCES

[1] Hutchinson J. Serpiginous choroiditis in scrofulous subjects: choroidal lupus. Arch Surg (Lond) 1900;11:126-135.
[2] Gupta V, Gupta A, Arora S, et al. Presumed tubercular serpiginouslike choroiditis: clinical presentations and management. Ophthalmology 2003;110(9):1744-1749.
[3] Bansal R, Gupta A, Gupta V, et al. Tubercular serpiginous-like choroiditis presenting as multifocal serpiginoid choroiditis. Ophthalmology 2012;119(11):2334-2342.
[4] Gupta V, Agarwal A, Gupta A, et al. Clinical characteristics of serpiginous choroidopathy in North India. Am J Ophthalmol 2002;134(1):47-56.
[5] Jumper JM, McDonald R, Johnson RN, et al. Serpiginous choroiditis. In: Ryan SJ, edr. Retina. Chap-10. St. Louis: Elsevier Mosby 2006: p. 1811-1820.
[6] Schatz H, Maumenee AE, Patz A. Geographic helicoid peripapillary choroidopathy: clinical presentation and fluorescein angiographic findings. Trans Am Acad Ophthalmol Otolaryngol 1974;78:747-761.
[7] Hamilton AM, Bird AC. Geographical choroidopathy. Br J Ophthalmol 1974;58(9):784-797.
[8] Lim WK, Buggage RR, Nussenblatt RB. Serpiginous choroiditis. Surv Ophthalmol 2005;50(3):231-244.
[9] Wu JS, Lewis H, Fine SL, et al. Clinicopathologic findings in a patient with serpiginous choroiditis and treated choroidal neovascularization. Retina. 1989;9(4):292-301.
[10] Arantes TE, Matos K, Garcia CR, et al. Fundus autofluorescence and spectral domain optical coherence tomography in recurrent serpiginous choroiditis: case report. Ocul Immunol Inflamm 2011;19(1):39-41.
[11] Gass JDM. Stereoscopic Atlas of macular diseases: a funduscopic and angiographic presentation. St. Louis: CV Mosby Co, 1970: p. 66.
[12] Laatikainen L, Erkkila H. Serpiginous choroiditis. Br J Ophthalmol 1974;58(9):777-783.
[13] Masi RJ, O'Connor GR, Kimura SJ. Anterior uveitis in geographicor serpiginous choroiditis. Am J Ophthalmol 1978;86(2):228-232.
[14] Gass JDM. Inflammatory diseases of the retina and choroid. In: Stereoscopic atlas of macular diseases: diagnosis and treatment. Vol. 2. 4th edn. St. Louis: Mosby 1987: p. 710.
[15] Hardy RA, Schatz H. Macular geographic helicoid choroidopathy. Arch Ophthalmol 1987;105(9):1237-1242.
[16] Mansour AM, Jampol LM, Packo KH, et al. Macular serpiginouschoroiditis. Retina 1988;8(2):125-131.
[17] Abrez H, Biswas J, Sudharshan S. Clinical profile, treatment and visual outcome of serpiginous choroiditis. Ocul Immunol Inflamm 2007;15(4):325-335.
[18] Mansour AM, Jampol LM, Packo KH, et al. Macular serpiginous choroiditis. Retina 1988;8(2):125-131.

[19] Piccolino FC, Grosso A, Savini E. Fundus autofluorescence in serpiginous choroiditis. Graefes Arch Clin Exp Ophthalmol 2009;247(2):179-185.
[20] Altan-Yaycioglu R, Akova YA, Akca S, et al. Inflammation of the posterior uvea: findings on fundus fluorescein and indocyanine green angiography. Ocul Immunol Inflamm 2006;14(3):171-179.
[21] Bouchenaki N, Cimino L, Auer C, et al. Assessment and classification of choroidal vasculitis in posterior uveitis using indocyanine green angiography. Klin Monbl Augenheilkd 2002;219(4):243-249.
[22] Salati C, Pantelis V, Lafaut BA, et al. An 8 months indocyanine angiographic follow-up of a patient with serpiginous choroidopathy. Bull Soc Belge Ophtalmol 1997;265:29-33.
[23] Masi RJ, O'Connor GR, Kimura SJ. Anterior uveitis in geographic or serpiginous choroiditis. Am J Ophthalmol 1978;86(2):228-232.
[24] Chisholm IH, Gass JD, Hutton WL. The late stage of serpiginous (geographic) choroiditis. Am J Ophthalmol 1976;82(3):343-351.
[25] Weiss H, Annesley WH Jr, Shields JA, et al. The clinical course of serpiginous choroiditis. Am J Ophthalmol 1979;87(2):133-142.
[26] Gupta V, Gupta A, Arora S, et al. Presumed tubercular serpiginouslike choroiditis. Ophthalmology 2003;110(9):1744-1749.
[27] Gupta V, Gupta A, Rao NA. Intraocular tuberculosis—an update. SURV Ophthalmol 2007;52(6):561-587.
[28] Jabs DA, Rosenbaum JT, Foster CS, et al. Guidelines for the use of immunosuppressive drugs in patients with ocular inflammatory disorders: recommendations of an expert panel. Am J Ophthalmol 2000;130(4):492-513.
[29] Cordero-Coma M, Benito MF, Hernandez AM, et al. Serpiginous choroiditis. Ophthalmology 2008;115 (9):1633.
[30] Yee D, Valiquette C, Pelletier M, et al. Incidence of serious side effects from first-line antituberculosis drugs among patients treated for active tuberculosis. Am J Respir Crit Care Med 2003;167(11):1472-1477.
[31] Tostmann A, Boeree MJ, Aarnoutse RE, et al. Antituberculosis drug-induced hepatotoxicity: concise up-to-date review. J Gastroenterol Hepatol 2008;23(2):192-202.
[32] Ustinova EI, Zhuravleva LV, Bataev VM, et al. Experience in the differential diagnosis of peripapillary "geographic" choroid disease and tuberculous chorioretinitis [in Russian]. Vestn Oftalmol 1990;106(6):43-46.
[33] Mahendradas P, Kamath G, Mahalakshmi B, et al. Serpiginous choroiditis- like picture due to ocular toxoplasmosis. Ocul Immunol Inflamm 2007;15(2):127-130.
[34] Gass JD, Braunstein RA, Chenoweth RG. Acute syphilitic posterior placoid chorioretinitis. Ophthalmology 1990;97(10):1288-1297.
[35] Edelsten C, Stanford MR, Graham EM. Serpiginous choroiditis: an unusual pre- sentation of ocular sarcoidosis. Br J Ophthalmol 1994;78(1):70-71.
[36] Kawali A, Emerson GG, Naik NK, et al. Clinicopathologic features of tuberculous serpiginous-like choroiditis. JAMA Ophthalmology 2018;136(2):219-221.
[37] Majumder PD, Biswas J, Gupta A, et al. Enigma of serpiginous choroiditis. Indian Journal of Ophthalmology 2019;67(3):325-333.
[38] El Ameen A, Herbort CP Jr. Serpiginous choroiditis imaged by optical coherence tomography angiography. Retin Cases Brief Rep 2018;12(4):279-285.