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Case Report

Central Serous Chorioretinopathy in A Young Female with Leukocytoclastic Vasculitis on Colchicine Therapy: Manifestation of the Drug or the Disease?

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Abstract

Central Serous Chorioretinopathy (CSCR) is a disease characterized by serous detachment of neurosensory retina at the macula. It occurs in middle aged adults with a male preponderance and is predisposed by psychological stress, pregnancy, obstructive sleep apnoea, Systemic Lupus Erythematosus (SLE) and drugs including corticosteroids and Rowatinex. Leukocytoclastic Vasculitis (LCV) is a small-vessel vasculitis which presents as a palpable purpuric rash or ulceration affecting lower extremities. Colchicine, an anti-inflammatory drug is of proven efficacy in LCV. A 33 year old female with biopsy proven LCV on colchicine therapy for 15 days, presented with sudden decrease in vision in right eye. Visual acuity was 6/18, Amsler's grid showed central metamorphopsia and photostress recovery time was delayed. Fundus examination, Optical Coherence Tomography and Fundus fluorescein angiography confirmed CSCR with pigment epithelial detachment (PED). Colchicine was withdrawn and switched to Dapsone, followed by complete resolution of CSCR with normal visual acuity in four weeks. We speculate that colchicine by itself, or in conjunction with LCV is the plausible causal association. The propounded mechanism underlying vasculitis and drug induced CSCR may be focal choroidal vascular compromise and disruption of retinal pigment epithelial tight junctions. To the best of our knowledge, there are no reports relating LCV or colchicine use with CSCR. Clinicians should bear in mind that CSCR is a potential complication of colchicine therapy in LCV. Prompt withdrawal of offending agent hastens resolution.

Keywords: Central Serous Chorioretinopathy; Leukocytoclastic Vasculitis; Colchicine; Drug induced CSCR

Abbreviations

CSCR: Central Serous Chorioretinopathy; LCV: Leukocytoclastic Vasculitis; PED: Pigment Epithelial Detachment; OCT: Optical Coherence Tomography; FAF: Fundus Autofluorescence; FFA: Fundus Fluorescein Angiography; SLE: Systemic Lupus Erythematosus; ARMD: Age Related Macular Degeneration.

Introduction

Central Serous Chorioretinopathy (CSCR) is a disease characterized by serous detachment of neurosensory retina from the retinal pigment epithelium. The occurrence is common among middle aged adults with a male preponderance [1]. Psychological stress and increased levels of corticosteroids of exogenous and endogenous origin can precipitate CSCR [2]. Small-vessel vasculitis can cause CSCR [3,4]. Leukocytoclastic Vasculitis (LCV) is a small-vessel vasculitis which presents as a palpable purpuric skin rash or ulcer, predominantly affecting the lower extremities [5]. It has an incidence rate of 30 cases per million per year [6]. Colchicine, an anti-inflammatory drug has been found to be effective in the treatment of cases with LCV. Gastrointestinal side effects are common with colchicine use. However, ocular side effects have not been reported [7]. We report a case of unilateral CSCR in a patient with LCV treated with colchicine.

Case Report

33yr old female patient presented to the outpatient department with complaints of sudden decrease in visual acuity in the right eye for four days. The patient had fever, joint pain and a painful skin ulceration in the lower limb for two weeks (Figure 1). The diagnosis of LCV was made following punch biopsy of skin lesion in lower limb. Histopathological examination unveiled dense perivascular leukocytic infiltration in the dermis and fibrinoid necrosis of small vessel walls, with neutrophilic debris and epidermal ulceration co-

vered with exudate (Figure 2a and b). The patient was treated with colchicine for 15 days. On examination, visual acuity in right eye was 6/18 and left eye was 6/6. Anterior segment examination was normal in both eyes. Fundus examination of right eye showed a large ovoid area of dome shaped serous elevation of neurosensory retina involving the entire macula (Figure 3). Left eye examination was unremarkable. On further investigation, Amsler grid showed a large area of metamorphopsia in the right eye. Photo-stress recovery time was delayed. Optical coherence tomography (OCT) showed a serous elevation of neurosensory retina with an underlying pigment epithelial detachment (PED) in the right eye (Figure 4). Fundus autofluorescence (FAF) revealed larger area of serous detachment, than was clinically obvious (Figure 5). Fundus fluorescein angiography (FFA) showed focal areas of "ink-blot" leak at the posterior pole, gradually increasing in size and intensity in later phases (Figure 6). A diagnosis of unilateral CSCR with PED was made. Colchicine intake was suspected to be the inciting event. Colchicine was withdrawn and substituted by Dapsone. Four weeks later, there was complete resolution of CSCR with the visual acuity improving to 6/9.

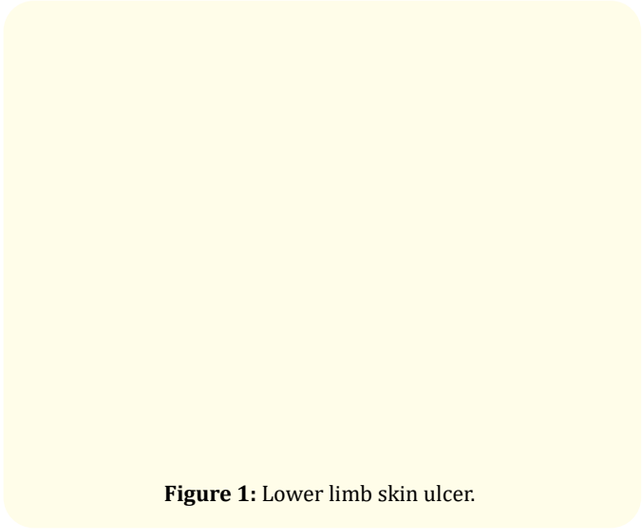

Figure 1: Lower limb skin ulcer.

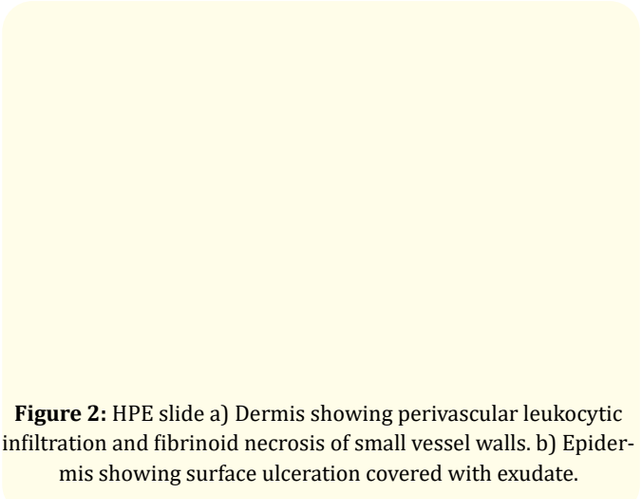

Figure 2: HPE slide a) Dermis showing perivascular leukocytic infiltration and fibrinoid necrosis of small vessel walls. b) Epidermis showing surface ulceration covered with exudate.

Figure 3: Fundus photograph of showing gross elevation of neurosensory retina (dotted lines) that includes entire macula and reaches close to temporal border of disc.

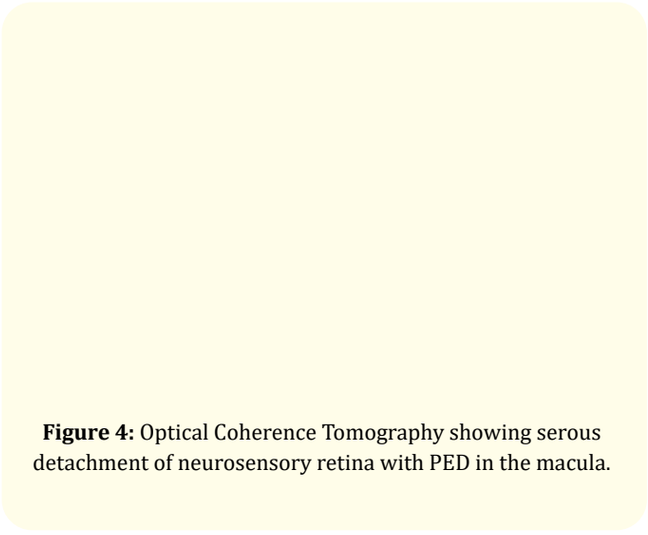

Figure 4: Optical Coherence Tomography showing serous detachment of neurosensory retina with PED in the macula.

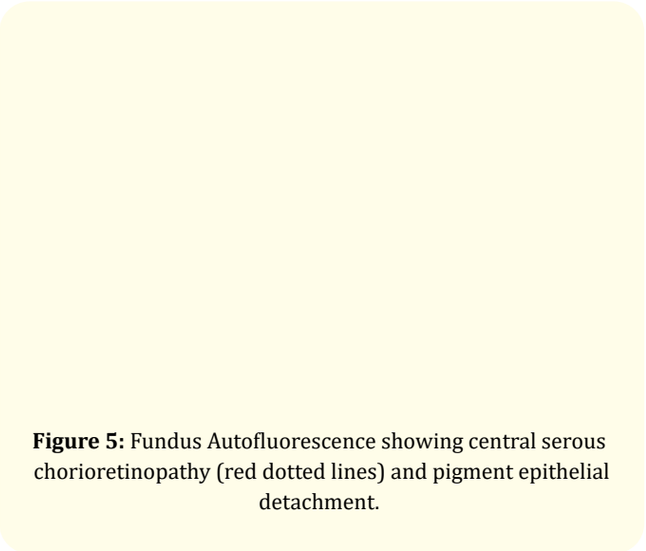

Figure 5: Fundus Autofluorescence showing central serous chorioretinopathy (red dotted lines) and pigment epithelial detachment.

Figure 6: Fundus Fluorescein Angiography showing focal areas of "ink blot" leak at posterior pole (arrows).

Discussion

CSCR occurs due to leakage of fluid from hyperpermeable choriocapillaries through a focal defect in retinal pigment epithelium, which collects in the subretinal space to cause the neurosensory detachment [1]. However the pathophysiology of CSCR is incompletely understood. Blurring of vision, metamorphopsia, central and paracentral scotomas can occur in patients with CSCR when the macula is involved [8]. Type A personality, pregnancy, emotional lability, hysteria, Helicobacter pylori and drugs such as corticosteroids have been suggested as predisposing factors for CSCR [2,8].

Systemic lupus erythematosus (SLE) can cause CSCR secondary to small vessel vasculitis [3]. Central serous chorioretinopathy has been reported in a patient with Weber-Christian disease possibly due to vasculitis involving choriocapillaris [4] LCV, also known as hypersensitivity vasculitis, is a small vessel vasculitis which can occur in any age group with no gender preference. Circulating immune complexes are involved in the pathogenesis of LCV [5]. Etiological triggers include drugs, food, medications and autoimmune disorders. Patients typically present with a painful burning purpuric rash involving the lower limbs, and less frequently trunk and upper extremities [6]. Other manifestations include skin ulcers, bullae, papules, livedo reticularis and arthritis of knees or ankles [5]. Skin ulcer in the lower limb is a manifestation of LCV in this case. Ocular manifestations have not been reported in LCV. In this patient, LCV could be the probable cause of CSCR, the underlying pathophysiological basis being similar to other vasculitis causing CSCR. There is no evidence in literature associating LCV to central serous chorioretinopathy.

Colchicine is an anti-inflammatory drug used primarily in the treatment of gout and Familial Mediterranean Fever [7]. Colchicine is of proven benefit in LCV [6]. Colchicine acts by binding to tubulin dimers and disrupting microtubule polymerization [7]. Since the patient was not on treatment with drugs such as corticosteroids or Rowatinex which are established causes of CSCR, colchicine therapy is hypothesized as a possible cause of CSCR [1,2]. Ocular side effects have not been reported with colchicine use. There is no data available in literature relating colchicine use to CSCR. The proposed mechanism by which steroids cause CSCR include increased permeability of choriocapillaris and disruption of retinal pigment epithelial tight junctions, which constitute the outer blood retinal barrier [6]. It can be speculated that colchicine could have acted via similar mechanism to cause CSCR.

CSCR, age related macular degeneration(ARMD), ischemic and inflammatory chorioretinal diseases have been associated with pigment epithelial detachment [9]. The PED in the right eye of this patient is secondary to CSCR. CSCR usually has a good prognosis. Both drug and vasculitis induced CSCR resolve spontaneously [1-4]. Recovery of vision may be poor, with guarded prognosis in vasculitis induced CSCR [3,4]. Since complete resolution of CSCR was observed on cessation of colchicine therapy, we suggest that colchicine treatment alone or in conjunction with LCV may have played a role in the development of CSCR. This is probably the first report explaining the causal association.

Conclusion

We speculate that colchicine by itself, or in conjunction with LCV is the plausible causal association for CSCR in this patient. The propounded mechanism underlying vasculitis and drug induced CSCR may be focal choroidal vascular compromise and disruption of retinal pigment epithelial tight junctions. To the best of our knowledge, there are no reports relating LCV or colchicine use with CSCR. Clinicians should bear in mind that CSCR is a potential complication of colchicine therapy in LCV. Prompt withdrawal of offending agent hastens resolution.

Acknowledgements

Nil

Conflict of Interest

Nil

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