Single Cotton Wool Spot as a Late Manifestation of Head Trauma

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Purpose: To report a patient with a single cotton wool spot (CWS) following head trauma.

Case Report: A 37-year-old male electrician presented with painless paracentral blurred vision in his left eye of one month duration together with three transient episodes of obscuration of vision in the same eye lasting for 10 minutes. He reported blunt head trauma due to a fall 40 days before referral. Fundus examination at presentation was normal but after 4 months, revealed the appearance of a white spot along the superotemporal arcade in the absence of other fundus lesions. A comprehensive systemic workup was performed revealing no specific findings. Ophthalmoscopic examination after 6 weeks disclosed resolution of the CWS with no intervention. The patient’s complaint of paracentral visual field defect improved and subsequent optical coherence tomography showed retinal thinning in that area.

Conclusion: Herein, we report a patient with a single CWS near the optic disc four months after head trauma along with normal systemic and paraclinical evaluations. Head trauma may thus be considered as a cause of CWS.

Keywords: Retinopathy; Head Injury; Retina; Eye

INTRODUCTION

Cotton wool spots (CWSs) are comprised of localized accumulations of axoplasmic debris within adjacent bundles of the unmyelinated ganglion cell axons. These lesions resolve over a period of 4 to 12 weeks, leaving an area of inner ischemic retinal atrophy.1 These white, fluffy, small and superficial lesions are most commonly observed in diabetic and hypertensive retinopathy. CWSs can be detected in many systemic diseases, including connective tissue diseases, valvular heart disease, metastatic cancer and infectious diseases such as AIDS; however, occasionally no underlying etiology may be present.2,3 CWSs obscure underlying blood vessels4 and are clinically evident only in the post-equatorial retina, where the nerve fiber layer is of sufficient thickness to make them visible.

Herein, we report a single cotton wool spot as a late manifestation of head trauma and describe spectral domain optical coherence tomography (SD-OCT) changes from presentation to resolution.

CASE REPORT

A 37-year-old male electrician attended our ophthalmology clinic complaining of painless blurred vision in the paracentral region of his left eye for one month; he also reported 3 episodes of transient obscuration of vision lasting for 10 minutes. He recalled blunt head trauma due a fall
40 days before, but had no history of ophthalmic or systemic disease, use of medications, smoking and high risk sexual behaviour. Family history was unremarkable.

Corrected visual acuity was 20/20 in both eyes. Fundus examination at presentation was normal, but after 4 months revealed a white spot along the superotemporal arcade (Figure 1), in the absence of any other fundus lesions. Systemic examination was normal and his blood pressure was 120/75 mmHg.

A hematologic, rheumatologic and infectious disease workup was performed including complete blood counts, urine analysis, urea, creatinine, fasting blood glucose, erythrocyte sedimentation rate, C-reactive protein,

Figure 1. Fundus examination 4 months after initial visit revealed a white spot along the superotemporal arcade.

Figure 2. Visual field assessment using the Swedish Interactive Threshold Algorithm (SITA) 24-2 standard test demonstrated a scotoma compatible with the location of the cotton wool spot.
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rheumatoid factor, anti-double-stranded deoxyribonucleic acid, antinuclear antibody, C-anti-neutrophil cytoplasmic antibodies, peripheral anti-neutrophil cytoplasmic antibodies, anti-cyclic citrullinated peptide, toxoplasma immunoglobulin M (IgM), serum anti-human immunodeficiency virus, purified protein derivative, sputum specimen for tuberculosis, all with normal results. Cardiologic evaluations by electrocardiography and echocardiography revealed normal findings as well.

Fundus fluorescein angiography was performed and images were consistent with the ophthalmoscopic findings and did not show any other abnormalities. Visual field test using the Swedish Interactive Threshold Algorithm (SITA) 24-2 standard test demonstrated a scotoma on the pattern deviation plot corresponding to the location of the CWS (Figure 2).

Optical coherence tomography (OCT) employing Spectralis SD-OCT (Heidelberg Engineering Inc., Vista, CA, USA), was also performed at presentation which revealed marked retinal thickening along with increased reflectivity of the nerve fiber layer and inner neurosensory retina in the region of the CWS (Figure 3A).

After 6 weeks the CWS resolved without any therapeutic intervention. The patient’s complaint of paracentral field defect was improved and subsequent OCT showed retinal thinning in the same area (Figure 3B).

DISCUSSION

The most common cause of CWSs is diabetes mellitus followed by the acquired immunodeficiency syndrome (AIDS) or systemic arterial hypertension. Occasionally a healthy patient has a CWS and systemic evaluation is may be entirely normal. Brown et al found that undiagnosed diabetes mellitus and systemic hypertension were the most common etiologies of CWS in 24 patients. Other causes included cardiac valvular disease, radiation retinopathy, severe carotid artery obstruction, dermatomyositis, systemic lupus erythematosus, polyarteritis nodosa, leukemia, AIDS, Purtscher’s retinopathy, metastatic carcinoma, intravenous drug abuse, partial central retinal artery obstruction and giant cell arthritis. In one patient no underlying cause was revealed in spite of systemic workup. Isolated cases of CWS have also been reported as the presenting sign of multiple myeloma.

CWSs usually fade within a few weeks, but may remain longer in patients with diabetes or may have a shorter half-life in AIDS retinopathy. Following resolution, visual function usually does not recover, visual acuity in our patient also did not change after the CWS resolved within 6 weeks.

Cotton wool spots due to retinal ischemia appear as regions of increased reflectivity...
in the retinal nerve fiber layer and inner neurosensory retina on cross sectional OCT images. Toxoplasma chorioretinitis and CWSs are two differential diagnoses of retinal hyper-reflective lesions. Toxoplasmic chorioretinitis on OCT produces a distinctive inner retinal hyperreflectivity which can be diagnostic. Presumably this is due to the proliferation of tachyzoites and infiltration of inflammatory cells at a stage in which the lesion is not white yet. Cystoid bodies (cotton wool patches) also display inner retinal hyper-reflectivity, but ordinarily are distinguishable from toxoplasmic chorioretinitis by other features such as lack of vitreous inflammation and feathery retinal whitening corresponding to the nerve fiber layer.

Purtscher’s retinopathy was first described in patients with severe head trauma. In a study by Agrawal et al on Purtscher’s retinopathy following trauma in patients with blurred or lost vision, acute retinal findings such as CWSs, retinal hemorrhage and Purtscher flecken were observed in 26% of patients over a one month period which resolved after 6 months in all patients. The most common chronic findings in this study were optic disc pallor and RPE atrophy. After 4.5 months on average, half of the patients had at least 2 Snellen chart lines of spontaneous improvement and 23% gained at least 4 Snellen chart lines. Cotton wool spots following multiple trauma has been reported in other case reports.

In summary, the patient presented herein, was a 37-year-old man complaining of paracentral blurred vision in whom a single cotton-wool spot was seen near the optic disc. He had a history of head trauma 4 months before and all systemic and paraclinical evaluations were normal, so it seems that the single cotton-wool spot was due to head trauma.

Conflicts of Interest
None.

REFERENCES
1. McLeod D. Why cotton wool spots should not be regarded as retinal nerve fibre layer infarcts. Br J Ophthalmol 2005;89:229-237.
2. Mansour AM, Jampol LM, Logani S, Read J, Henderly D. Cotton-wool spots in acquired immunodeficiency syndrome compared with diabetes mellitus, systemic hypertension, and central retinal vein occlusion. Arch Ophthalmol 1988;106:1074-1077.
3. Brown GC, Brown MM, Hiller T, Fischer D, Benson WE, Magargal LE. Cotton-wool spots. Retina 1985;5:206-214.
4. Bek T, Lund-Andersen H. Cotton-wool spots and retinal light sensitivity in diabetic retinopathy. Br J Ophthalmol 1991;75:13-17.
5. Schmidt D. The mystery of cotton-wool spots - a review of recent and historical descriptions. Eur J Med Res 2008;13:231-266.
6. Shami MJ, Uy RN. Isolated cotton-wool spots in a 67-year-old woman. Surv Ophthalmol 1996;40:413-415.
7. Chui TY, Thibos LN, Bradley A, Burns SA. The mechanisms of vision loss associated with a cotton wool spot. Vision Res 2009;49:2826-2834.
8. Mansour AM, Rodenko G, Dutt R. Half-life of cotton-wool spots in the acquired immunodeficiency syndrome. Int J STD AIDS 1990;1:132-133.
9. Ioannides A, Georgakarakos ND, Elaroud I, Andreou P. Isolated cotton-wool spots of unknown etiology: management and sequential spectral domain optical coherence tomography documentation. Clin Ophthalmol 2011;5:1431-1433.
10. Puliafito CA, Hee MR, Lin CP, Reichel E, Schuman JS, Duker JS, et al. Imaging of macular diseases with optical coherence tomography. Ophthalmology 1995;102:217-229.
11. Lieb DF, Scott IU, Flynn HW Jr, Davis JL, Demming SM. Acute acquired toxoplasma retinitis may present similarly to unilateral acute idiopathic maculopathy. Am J Ophthalmol 2004;137:940-942.
12. Purtscher O. Unknown findings after head trauma. Berl Dtsch Ophthal Ges 1910;36:294-301. [Article in German]
13. Agrawal A, McKibbin M. Purtscher’s retinopathy: epidemiology, clinical features and outcome. Br J Ophthalmol 2007;91:1456-1459.
14. Meyer CH, Callizo J, Schmidt JC, Mennel S. Functional and anatomical findings in acute Purtscher’s retinopathy. Ophthalmologica 2006;220:343-346.
15. Watkins RC, Hambrick EL, Martin M, Washington M. Purtscher’s retinopathy: a case of visual impairment associated with multiple trauma. J Natl Med Assoc 1993;85:557-559.