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Commotio Retinae With Underlying Active Choroiditis And Chronic Choroidal Neovascular Membrane Signs: A Case Report

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Abstract
Commotio retinae is a well-recognised consequence of blunt ocular trauma characterised by transient cloudy swelling and greyish discolouration of the sensory retina. When co-existing with active choroiditis and a chronic choroidal neovascular membrane (CNVM), the clinical picture becomes considerably more complex, mandating prompt diagnosis and a nuanced management strategy. We report a 45-year-old male daily-wage worker who presented with a 15-day history of painless, progressive diminution of vision in the left eye following blunt trauma to the head and eye with a bamboo stick. Anterior segment examination revealed sluggish pupillary reaction, conjunctival congestion, and early immature cataracts bilaterally. Fundus examination of the left eye demonstrated cloudy media, absent foveal reflex, dilated and tortuous blood vessels, and diffuse multiple choroidal lesions with pigmentary changes, consistent with commotio retinae superimposed on active choroiditis with a pre-existing chronic CNVM. The differential diagnoses considered included choroidal rupture, traumatic macular hole, and retinal detachment. After respiratory medicine clearance to exclude tuberculous aetiology, the patient was commenced on oral prednisolone (Tab Wysolone 40 mg once daily). This report highlights the importance of recognising coincidental choroidal pathology in patients presenting with post-traumatic visual loss and underscores the role of systematic clinical evaluation in formulating an appropriate treatment plan.

Keywords: *Commotio retinae; Choroiditis; Choroidal neovascular membrane; Ocular trauma; Blunt trauma; Retinal oedema*

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I. Introduction

Commotio retinae, first described by Berlin in 1873, refers to the transient grey-white opacification of the sensory retina that follows non-penetrating blunt trauma to the globe or the periorbital region. The cloudy swelling most frequently involves the temporal fundus and, when it involves the macula, may produce a cherry-red spot closely resembling that seen in central retinal artery occlusion. In severe cases, intra-retinal haemorrhages further compromise macular function. At the ultrastructural level, the injury is localised principally to the photoreceptor outer segments and the retinal pigment epithelium (RPE), with disorganisation of the outer segment disc membranes being the hallmark histopathological finding.

Choroiditis denotes inflammation of the choroid, the richly vascularised middle coat of the eye situated between the retina and the sclera. In India, granulomatous infectious aetiologies—most notably ocular tuberculosis—remain important considerations and must be actively excluded before initiating immunosuppressive therapy. Choroidal neovascularisation (CNV) represents an aberrant angiogenic response in which new blood vessels arising from the choriocapillaris breach Bruch's membrane and proliferate into the sub-RPE or subretinal space. The resultant chronic CNVM may lead to persistent subretinal fluid, lipid exudation, and progressive photoreceptor loss.

The concurrence of commotio retinae with active choroiditis and a pre-existing chronic CNVM in the same eye is an uncommon and diagnostically challenging clinical scenario. Traumatic oedema may mask or mimic the underlying choroidal inflammatory lesion, and the vascular tortuosity associated with active choroiditis can confound the assessment of trauma-related haemodynamic changes. We present such a case, discussing the differential diagnoses, investigations, and management approach.

II. Case Report

Patient Demographics and Chief Complaint

A 45-year-old male daily-wage worker, a Hindi- and Marathi-speaking resident, presented to the outpatient department of Ophthalmology, with a chief complaint of diminution of vision in the left eye of 15 days' duration. The onset was gradual, the course progressive, and the nature entirely painless.

History of Presenting Illness

Fifteen days prior to presentation, the patient sustained blunt trauma to the left side of the head and the left eye from a bamboo stick while at work. Following the injury, he noticed a progressive decline in visual acuity in the left eye. He denied associated ocular pain, redness, photophobia, loss of visual field, watering, or perception of floaters. There was no history of spectacle usage, previous ocular trauma, prior ocular surgery or procedure, or known systemic illness (specifically hypertension, type 2 diabetes mellitus, asthma, or thyroid disorder). There was no history of drug allergy. A significant social history included tobacco consumption for the preceding 15 years.

General Examination

The patient was well-built and well-nourished, oriented to time, place, and person. Vital signs recorded were pulse 86 beats per minute, blood pressure 140/90 mmHg, respiratory rate 16 breaths per minute, and temperature afebrile. The mildly elevated blood pressure was noted but did not meet criteria for hypertensive retinopathy at this level.

Anterior Segment Examination

PARAMETER	RIGHT EYE	LEFT EYE
Visual Acuity (Distance/Near)	6/9 → 6/6 | N6	FC at 1M → NI | <N36
Amsler Grid	Normal	Distorted
Extra-Ocular Movements	Free, full, painless	Free, full, painless
Lids & Adnexa	Normal	Normal
Conjunctiva	Normal	Normal
Cornea	Clear	Clear
Corneal Sensations	Present	Present
Sclera	Normal	Congestion present
Anterior Chamber	Normal depth	Normal depth
Pupil	3 mm, RRR, RAPD absent	3–4 mm, sluggish RTL, RAPD absent
Lens	Early IMC	Early IMC

Table 1. Anterior Segment Examination Findings

Posterior Segment Examination

PARAMETER	RIGHT EYE	LEFT EYE
Media	Clear	Cloudy
Optic Disc – Colour	Pink	Pink
Optic Disc – Margins	Well-defined	Well-defined
Cup-to-Disc Ratio	0.4:1	0.3:1
Blood Vessels	Normal	Dilated, increased tortuosity
Foveal Reflex	Present	Absent
Macula	Normal	Diffuse, multiple choroidal lesions; pigmentary changes

Table 2. Posterior Segment Examination Findings

The fundus of the left eye demonstrated cloudy media, dilated and tortuous blood vessels, absent foveal reflex, and diffuse multiple choroidal lesions with pigmentary changes spanning the macular and peri-macular zones. These features were consistent with acute commotio retinae superimposed upon an active choroidal inflammatory process. The pigmentary changes and the morphology of the choroidal lesions were in keeping with a pre-existing, chronic choroidal neovascular membrane. The right eye fundus was unremarkable.

III. Investigations And Differential Diagnosis

Proposed Investigations

Given the clinical findings, the following investigations were indicated:

- Optical Coherence Tomography (OCT) macula of the left eye: to characterise the degree of retinal oedema, outer segment disruption, and to delineate the extent and activity of the CNVM.
- Fundus Fluorescein Angiography (FFA): to assess choroidal vascularity, detect active leakage from the CNVM, and identify areas of choroidal ischaemia or rupture.
- B-scan ultrasonography: to evaluate the posterior segment in the setting of cloudy media.
- Haematological workup: complete blood count, erythrocyte sedimentation rate, Mantoux test, QuantiFERON-TB Gold, chest X-ray / HRCT thorax to exclude tuberculous choroiditis.
- Serum ACE and VDRL to exclude sarcoidosis and syphilitic choroiditis, respectively.
- Blood glucose, HbA1c, and lipid profile for metabolic risk stratification.

Differential Diagnosis

The following diagnoses were considered in the context of post-traumatic macular pathology:

- Choroidal rupture – direct full-thickness disruption of the choroid, Bruch's membrane, and RPE by the traumatic force, typically appearing as a crescent-shaped, concentric arc to the optic disc.
- Traumatic macular hole – full-thickness defect of the neurosensory retina at the fovea resulting from the centrifugal shearing forces generated by blunt trauma.
- Traumatic retinal detachment – separation of the neurosensory retina from the RPE consequent upon giant retinal tear or dialysis at the ora serrata.
- Purtscher's retinopathy – cotton-wool spots and haemorrhages secondary to microemboli or complement activation following remote trauma.
- Active tuberculous choroiditis with CNVM – an important infective aetiology in the Indian subcontinent necessitating exclusion prior to corticosteroid initiation.

Management

Given the constellation of clinical findings—active choroidal inflammation with multiple choroidal lesions—and the geographic prevalence of tuberculosis in India, a respiratory medicine consultation was sought prior to initiating systemic immunosuppression. This referral was specifically to exclude active or latent pulmonary tuberculosis as a potential underlying aetiology for the choroiditis, thereby minimising the risk of dissemination of a mycobacterial infection under corticosteroid cover.

Following respiratory medicine clearance, the patient was commenced on oral prednisolone (Wysolone) 40 mg once daily, tapered as per clinical response. Close ophthalmic follow-up was planned to monitor visual acuity, control of choroidal inflammation, CNVM activity, and potential development of trauma-related complications.

Anti-vascular endothelial growth factor (anti-VEGF) therapy with intravitreal bevacizumab or ranibizumab was considered for management of the active CNVM component, particularly if OCT confirmed significant subretinal fluid or haemorrhage. The timing of anti-VEGF injection relative to the acute inflammatory phase was to be individualised based on disease activity.

IV. Discussion

This case is notable for the co-existence of three distinct but inter-related pathological processes in a single eye: acute commotio retinae, active choroiditis, and a chronic CNVM. The temporal relationship between the traumatic event and the onset of symptoms, combined with the fundoscopic evidence of acute retinal oedema, strongly implicates trauma as the immediate precipitant of the visual loss. However, the multifocal choroidal lesions with pigmentary changes and dilated, tortuous blood vessels are indicative of an antecedent chronic inflammatory and neovascular process that was likely subclinical prior to the traumatic injury.

The pathophysiology of commotio retinae is thought to involve the disruption of photoreceptor outer segment disc membranes by the rapid deformation of the globe at the moment of impact. This mechanical distortion impairs the function of the outer retinal layers and alters RPE phagocytic capacity, resulting in the clinical appearance of greyish-white opacification. The cherry-red spot, observed in severe macular involvement, reflects the relative preservation of the foveal RPE and choroidal vasculature through which the reddish-orange reflex of the choroid is transmitted against a background of surrounding oedematous, pale retina.

Active choroiditis superimposed on commotio retinae represents a therapeutically challenging scenario because the standard management of commotio retinae is expectant (watchful waiting), whereas active choroidal inflammation typically warrants systemic immunosuppression. In patients residing in tuberculosis-endemic regions such as Maharashtra, empirical anti-tubercular therapy (ATT) is often administered alongside corticosteroids once infective aetiology cannot be excluded—a practice endorsed by the published literature on presumed ocular tuberculosis.

The presence of a chronic CNVM further complicates the prognosis. Trauma-related choroidal rupture, when it heals, may itself stimulate neovascularisation; however, in this case the pigmentary changes and choroidal

lesion morphology suggested a pre-existing inflammatory CNVM rather than a new post-traumatic CNV. The elevated blood pressure (140/90 mmHg) also warrants attention as hypertension may contribute to vascular tortuosity and can adversely affect the course of CNVM.

V. Complications And Prognosis

Potential complications in the acute and chronic phases include:

- Retinal tears and giant retinal dialysis
- Choroidal rupture with secondary haemorrhage
- Lens subluxation from zonular disruption
- Traumatic hyphema
- Late-onset secondary glaucoma
- Traumatic cataract acceleration
- Chorioretinal atrophy at the site of commotio retinae
- Progression of CNVM with subretinal fibrosis and permanent macular damage

The prognosis for commotio retinae in isolation is generally favourable; most cases resolve spontaneously within 3 to 6 weeks with restoration of near-normal visual acuity, provided the macula is not severely involved. However, in patients with concomitant active choroiditis and CNVM, the prognosis is guarded. Persistent sub-foveal fluid, photoreceptor loss, and RPE atrophy may result in a permanent central scotoma and significant reduction in reading vision. Early institution of anti-inflammatory therapy and anti-VEGF injections may mitigate some of the long-term sequelae.

VI. Conclusion

This case underscores the importance of a thorough fundoscopic evaluation in patients presenting with post-traumatic visual loss, as underlying choroidal pathology may be unmasked or exacerbated by the traumatic insult. The concurrent presence of commotio retinae, active choroiditis, and a chronic CNVM demands a systematic, stepwise approach to investigation—with particular emphasis on excluding infective aetiologies—before initiating systemic corticosteroids. Multidisciplinary collaboration between ophthalmologists and respiratory/internal medicine specialists is essential in the Indian clinical setting. Long-term follow-up with serial OCT and clinical assessment remains pivotal to monitoring disease activity and guiding timely therapeutic interventions.

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