Severe steroid-induced psychosis following systemic corticosteroid therapy for idiopathic serpiginous choroiditis: a multidisciplinary learning case

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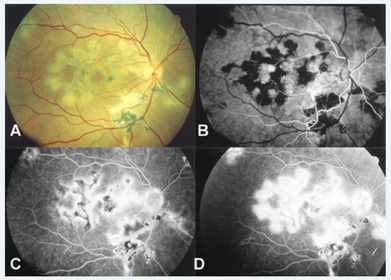
Background

Idiopathic serpiginous choroiditis is a sight-threatening, recurrent posterior uveitis that often requires prolonged, high-dose systemic corticosteroid therapy.

While severe psychiatric reactions to systemic corticosteroids are highly dose-dependent and well documented in rheumatology and internal medicine, they remain dangerously under-recognised among ophthalmologists, who are frequently the prescribing clinicians. When severe psychiatric adverse effects occur, they can precipitate life-threatening cascades.

The central learning aim of this case is to highlight that ophthalmologists prescribing high-dose corticosteroids must actively screen for psychiatric complications using validated tools, rather than relying on unstructured questioning, and must plan steroid-sparing therapy early to mitigate cumulative patient risk.[1]



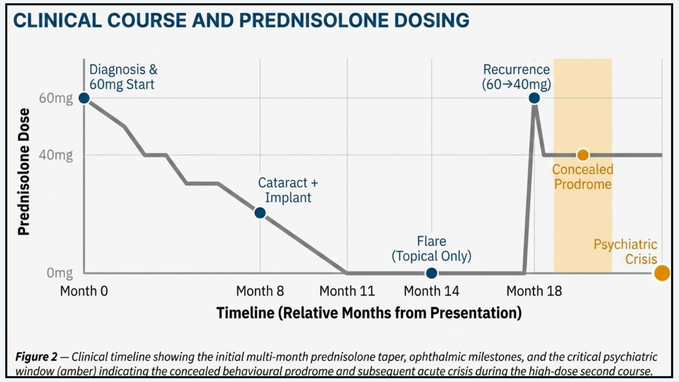


Figure 2 *— Clinical timeline showing the initial multi-month prednisolone taper, ophthalmic milestones, and the critical psychiatric window (amber) indicating the concealed behavioural prodrome and subsequent acute crisis during the high-dose second course.*

Case Narrative

A man in his late 50s with hypertension, type 2 diabetes mellitus (T2DM), chronic kidney disease (CKD; stage 3B), and a single remote resolved depressive episode presented with profound vision loss and active choroiditis. Following a negative infective and inflammatory screen, idiopathic serpiginous choroiditis was diagnosed.[2]

His first oral prednisolone course (60 mg induction, ~11-month taper) included intercurrent cataract surgery with an intravitreal dexamethasone implant (700 mcg); this prolonged systemic exposure remained psychiatrically uneventful. A subsequent interim anterior flare was successfully managed with topical dexamethasone alone.

Approximately four months later, a second severe recurrence catastrophically reduced his **right-eye best-corrected visual acuity (BCVA) to 1/60.** Oral prednisolone was restarted at 60 mg and maintained at 40 mg. Despite explicit verbal counselling regarding potential mood disturbances, he disclosed nothing at subsequent clinical reviews. Family members observed a profound behavioural prodrome approximately four to six weeks into the second course, which the patient actively concealed due to cultural stigma, lack of insight, and severe occupational threat to his professional driving licence. By weeks six to eight, he developed severe command auditory hallucinations, precipitating acute systemic complications of self-harm requiring critical-care admission and emergency surgical intervention.

Outcomes

Psychiatric — Hallucinations resolved; insight returned; stable on aripiprazole 10 mg once daily with community follow-up; no inpatient psychiatric admission required.

Surgical — Recovered from emergency surgical intervention; one re-admission for a sub-phrenic collection managed with antibiotics ± interventional radiology.

Ophthalmological — Disease quiescent; **systemic corticosteroids permanently contraindicated**; future flares to be managed with steroid-sparing therapy.

Occupational — DVLA fitness-to-drive assessment pending; return to safety-critical work not yet possible.

Discussion

The dose-response relationship for steroid-induced psychiatric reactions is well-documented: incidence is approximately 1.3% at prednisolone-equivalent doses below 40 mg/day, rising sharply to approximately 5% at doses of 40 mg/day or higher.[1] In this case, profound symptom concealment occurred due to an occupational threat to the patient's safety-critical licence, deep cultural stigma, acute financial pressures, and insufficient verbal counselling.

The resulting cascade of life-threatening systemic complications must be explicitly framed as a foreseeable and preventable sequence, rather than a rare anomaly. Because the core prescribing decision sits with ophthalmology, the ophthalmologist must act as the central multidisciplinary team (MDT) coordinator. This responsibility mandates the active use of structured, validated screening tools (such as the PHQ-9 and GAD-7) at every high-dose review, proving that verbal screening alone is inadequate.[2] Proactive induction of steroid-sparing agents[3,4] must occur before catastrophic toxicity forces intervention. While limited inherently by being a single case, this report unequivocally demonstrates the peril of unmonitored high-dose prescribing.

Agent class	Merit	Demerit
Systemic corticosteroids	Rapid control of sight-threatening inflammation	Dose-dependent psychiatric, metabolic, ocular and bone toxicity
Mycophenolate mofetil	Steroid-sparing; reasonable tolerability	Slow onset (weeks to months); GI and haematological monitoring
Methotrexate	Established uveitis evidence; weekly dosing	Hepatotoxicity; teratogenicity; folate co-prescription
Tacrolimus	Useful in steroid-resistant disease	Nephrotoxicity; drug-level monitoring
Adalimumab (anti-TNF)	Strong RCT evidence in non-infectious posterior uveitis (VISUAL-I)	Cost; infection screening required; injection-site reactions

References

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