Anterior uveitis and diffuse scleritis after pamidronate infusion

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A 58-year-old, bisphosphonate-naive woman with a history of Hashimoto thyroiditis was admitted to hospital for hypercalcemia (2.81 [normal 2.20–2.60] mmol/L) due to primary hyperparathyroidism. She received 60 mg of intravenous pamidronate. Two days later, she developed right eye pain, photophobia and blurred vision, with progressive redness and periorbital swelling. She presented to an emergency eye clinic 6 days later and had a visual acuity of 20/30 in the left eye and only light perception in the right eye, with proptosis, an adduction deficit, corneal edema and a hypopyon (Figure 1A). The posterior segment view was obstructed but no vitritis was seen on B-scan ultrasonography. Computed tomography of the orbits revealed right-sided diffuse scleral thickening (Figure 1B). She was treated empirically with topical moxifloxacin, corticosteroids and cycloplegics. Infectious and inflammatory work-up was negative, apart from an elevated C-reactive protein (16.4 [normal < 3] mg/L).

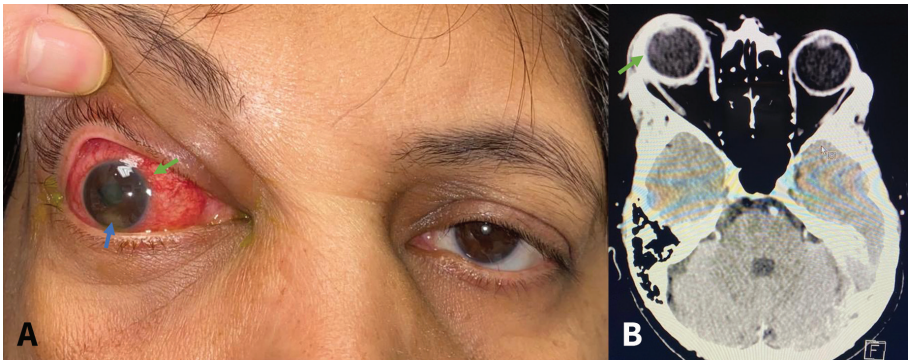

Figure 1: A) Pamidronate-induced anterior uveitis and diffuse scleritis in a 58-year-old woman. Proptosis, hypopyon (blue arrow) and scleral injection and edema (green arrow) began within 48 hours of pamidronate infusion. B) A computed tomography scan of the orbits showed right-sided proptosis and circumferential scleral thickening and hypervascularity with adjacent edema (green arrow), consistent with diffuse scleritis.

We saw the patient a week later and diagnosed drug-induced anterior uveitis with scleritis from the pamidronate infusion. Her intraocular inflammation resolved after starting oral corticosteroids with a slow taper. At the 7-week follow-up visit, her visual acuity in the right eye had improved to 20/50. The patient eventually received a parathyroidectomy, which resolved her hypercalcemia.

The differential diagnosis of anterior uveitis with scleritis includes infectious, autoimmune, traumatic, neoplastic and drug-induced causes. For this patient, neoplasm was ruled out as she had no posterior segment involvement. Infectious and autoimmune disorders were considered and empirically treated pending investigations, which later returned as negative. Drug-induced uveitis or scleritis may be seen with use of parenteral or oral bisphosphonates, with incidence rates of 7–114 per 10 000 and 8–63 per 10 000 people per year, respectively.[1,2] Onset of uveitis or scleritis tends to be faster after parenteral (1–7 d) than oral (days to months) bisphosphonates.[2] If patients develop red eye associated with pain, photophobia, blurred vision, or periorbital swelling within a week of new medication exposure, the medication should be stopped immediately. Suspected drug-induced uveitis is managed empirically with topical corticosteroids and antibiotics with close follow-up by an ophthalmologist.[3]

References

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