Herpes zoster ophthalmicus with ocular involvement in overtreated hyperthyroidism

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A 25-year-old man, with a history of hyperthyroidism presented with herpes zoster ophthalmicus (HZO) with neurotrophic ulcer and superadded infection 4 weeks after a sudden decrease in his thyroid hormones, caused by overtreatment of hyperthyroidism and radioactive iodine therapy. He underwent lateral tarsorrhaphy and was started on conservative treatment for the ulcer. The ulcer had resolved after 2 months. HZO is caused by reactivation of varicella-zoster virus by a decline in cell-mediated immunity. The decrease in thyroid hormones can also reactivate varicella-zoster by immunosuppression. This case highlights the importance of stability in thyroid hormones in a patient with thyroid disease and history of varicella.

Key words: Herpes zoster ophthalmicus, herpes zoster in thyroid disease, reactivation of herpes zoster

Herpes zoster ophthalmicus (HZO) is characterized by a unilateral, dermatomal vesicular rash with radicular pain; caused by reactivation of varicella-zoster virus (VZV) lying dormant in the trigeminal or dorsal nerve root ganglia after primary chickenpox infection. The decrease in cell-mediated immunity in immunocompromised individuals leads to reactivation of VZV.[1] Its prevention and early diagnosis in susceptible individuals is essential to reduce morbidity by devastating complications such as keratitis, glaucoma, acute retinal necrosis, or optic nerve atrophy.[2] Herein, we report a case of HZO in a young male with hypothyroidism caused by aggressive treatment of hyperthyroidism.

Case Report

A 25 years old male presented to us with a 2-week history of pain, redness, and decreased vision in the right eye with ipsilateral painful blisters on his forehead. Systemic history was remarkable for hyperthyroidism; being treated with carbimazole 5 mg/day for 2 years. Medical records mentioned flare-up of hyperthyroidism previous month, for which he received radioactive iodine-131 (RAI) therapy and was then started on propranolol 20 mg/day with continuation of carbimazole 5 mg/day, causing sudden decrease in triiodothyronine (T3) and thyroxine (T4) (T3-40 ng/dL, T4- 2.4 μg/dL, reference range 100–200 ng/dL; 5–11.5 μg/dL, respectively; pretreatment T3- 350 ng/dL, T4- 15.5 μg/dL). After a week, he developed painful vesicular eruptions and fluid-filled blisters on the right side of his forehead respecting the midline, with fever and axillary lymphadenopathy; followed by redness in the right eye for 2 weeks after RAI. At the time of his presentation to us (4 weeks after appearance of vesicles), his vision was hand movements right eye (OD) and 20/20 left eye (OS), with multiple hemorrhagic blisters on the right side of his forehead along the distribution of V1 of trigeminal nerve, with significant edema, crusting, and scarring of upper eyelid causing lagophthalmos in the right eye [Fig. 1]. Slit-lamp examination of the right eye revealed diffuse conjunctival congestion, a triangular epithelial defect (8 × 4.5 mm) with rolled-out margins in the inferior half of cornea, suggestive of neurotrophic ulcer. There were also full-thickness corneal infiltrates with a creamy base, diagnostic of superadded microbial infection [Fig. 2]. Corneal sensations and blink reflexes were remarkably decreased. Ultrasonography of the right eye and examination of the left eye was unremarkable. Since the aggregation of blisters was dermatomal with the involvement of ipsilateral eye, clinical diagnosis of right HZO with severe corneal ulcer with neurotrophic keratitis, secondary to exposure keratopathy was made. Retrospective history revealed a history of chickenpox at the age of 5 years. Corneal scraping revealed gram-positive cocci on gram stain. Therapeutic debridement of the ulcer base and temporary lateral tarsorrhaphy were done. The patient was started on oral valacyclovir 1 g thrice a day, hourly fortified vancomycin 5%, and tobramycin 1.4% eye drops, cycloplegic and frequent lubricants to treat the ocular surface. HIV, hepatitis B and C were negative with decreased T3, T4 and increased thyroid-stimulating hormone (TSH) (T3- 42 ng/dL, T4- 3.2 μg/dL; TSH- 5.8 mIU/L, reference range 0.4–4 mIU/L). Since the patient was hypothyroid for 4 weeks, propranolol was discontinued by the endocrinologist in view of current hypothyroidism. After 2 months, his thyroid hormone profile was stable (T3-178 ng/dL, T4- 8.8 μg/dL, TSH- 3.3 mIU/L) and corneal ulcer had healed.

Discussion

Advancing age and immunosuppression due to HIV, post-renal transplant, and diabetes are major causes of reactivation of VZV.[3] Corneal involvement in HZO can be as neurotrophic, pseudodendritic, or stromal keratitis. VZV is a neurotrophic virus causing corneal hypoesthesia. Corneal nerves ensure

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Figure 1: Photograph of the face showing multiple blisters on the right side of the forehead, respecting the midline, with ipsilateral upper lid edema and crusting; suggestive of herpes zoster ophthalmicus

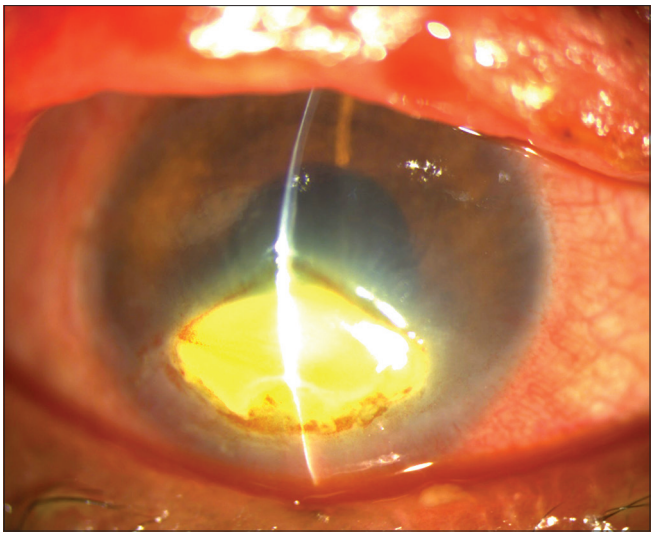

Figure 2: Anterior segment photograph of the right eye showing diffuse conjunctival congestion with a neurotrophic ulcer with full-thickness corneal infiltrates in the inferior half of eye

proper blink reflex and release of trophic factors such as substance *P* for epithelial maintenance.[4] Oral antiviral treatment with valacyclovir, famciclovir, or acyclovir is started within 72 h to decrease viral shedding, duration, and severity of the disease. In our patient, lagophthalmos and decreased blink reflex caused neurotrophic keratopathy.[5]

Thyroid hormones play an essential role in the immune system, nervous system, and cell signal transduction; along with gene expression, silencing, latency, and replication of herpes virus. A laboratory study has shown reduced herpes simplex virus (HSV) replication and releases by treatment of infected neuron-like cells with T3; and after removal of T3, the virus retained the ability to replicate and reactivate. It has been hypothesized that disrupted thyroid balance can cause VZV reactivation, by a similar mechanism, since both belong to alpha-herpes virus family with a high degree of genome homology, similar protein functionality, and virion structure.[6] Retrospective studies reveal a three-fold increase in reactivation of VZV by disrupted thyroid balance.[7] Disseminated zoster has been reported in a patient with extensive burns, hypothesizing burn-related immunosuppression as the cause. Similarly, thyroid-related immunosuppression can be a cause of HZO in our patients.[8]

Hypothyroidism has been reported in 57% of patients after RAI. We believe that sudden hypothyroidism caused by overtreated hyperthyroidism and RAI in our patient-led to HZO.[9] Various live-attenuated or HZ recombinant adjuvant vaccines are available to prevent zoster in older adults. These vaccines have substantially reduced incidence and morbidity with HZ in all age groups.[10]

Conclusion

To the best of our knowledge, this is the first case report documenting HZO after the overtreatment of hyperthyroidism and RAI. Patients with thyroid disease and history of varicella-zoster need stability in thyroid disease to prevent reactivation.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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