Chaker, et al.: Herpes zoster ophthalmicus

Commentary

In this issue of the Journal of Neuroscience in Rural Practice, Chaker *et al.*^[1] describe the case of an elderly patient presenting with diplopia, resulting from sixth nerve palsy, following acute infection of herpes zoster ophthalmicus (HZO). The authors describe the transient, self-limiting nature of cranial nerve (CN) VI palsy with favorable prognosis.

HZO occurs more commonly in patients over 50 years of age due to age-related decline in immunity. Cranial

nerve palsy following HZO has been reported with ocular-motor (cranial nerve (CN III), trochlear (CN IV) and abducens (CN VI) nerves most commonly affected.^[2] Other causes of CN palsies include intracranial space-occupying lesions, micro-vascular infarctions, trauma or inflammation. The most common cause of CN VI palsy in the elderly is micro-vascular ischemia associated with diseases such as diabetes mellitus and hypertension. Due to diverse causality, a comprehensive history should be obtained and a thorough ocular and cranial nerve examination should be performed to direct the need for further investigations such as magnetic resonance imaging or angiogram (MRI or MRA), erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and cerebrospinal fluid polymerase chain reaction (PCR), particularly in the absence of zosteriform rash.^[3]

Oral valacyclovir (1000 mg) or famciclovir (500 mg) three times daily for seven days are the recommended antiviral therapies for HZO, due to significant reductions in risk of developing post-herpetic neuralgia compared to oral acyclovir (800 mg five times daily for seven days).^[4] Adjunct corticosteroids have been reported to improve diplopia associated with CN palsy following HZO,^[3] although researchers are still unravelling the pathophysiology of this HZO complication. The addition of corticosteroids in patients with active infection should be carefully considered, with the aim to avoid potentiating existing infection. However, cranial nerve palsies associated with HZO are often reported approximately one week following HZO symptom onset and after antiviral therapy commencement, thereby decreasing this risk. Of interest, Nithvanandam et al.^[5] report a comparable HZO complication rate in HIV infected patients when antiviral therapy is instigated within 72 h.

Of note, CN palsies in HZO can sometimes present atypically. Czyz *et al.*^[6] reported a case of complete paralytic mydriasis as the only symptom of partial CN III involvement. While Babu *et al.*^[7] described a patient with CN III and VI involvement presenting with nodular scleritis and nummular keratouveitis. The importance of comprehensive slit lamp examination of the eye including dilated fundus examination, as well as cranial nerve assessment in all cases of HZO cannot be overemphasised.

Zoster vaccination aims to decrease the severity and duration of herpes zoster and minimize complications of infection. Tseng *et al.*^[8] conducted a large cohort study investigating zoster vaccine safety and reported no increased risk of Bell's palsy or Ramsey-Hunt syndrome following vaccination in patients over the age of 50. However, recurrent CN VI palsy has been reported in a child following vaccination against varicella, measles, mumps and rubella.^[9] Although the live attenuated zoster vaccine has been noted to be not as effective in patients

with reduced immune response, and is contraindicated in immunosuppressed patients, it is specifically these patients that require the most protection. Therefore, an efficacious-inactivated VZV vaccine is also needed to protect patients most at risk.^[10]

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