ophthalmic care, and lengthy in-patient psychiatric care may subsequently be needed. These patients are at high risk for further self-mutilation and precautions must be taken to prevent these.

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Sir,

Orbital vasculitis following varicella. A case report
Eye (2004) **18,** 432–433. doi:10.1038/sj.eye.6700678

Chicken pox is a common childhood disease caused by the varicella zoster virus. Although most cases of varicella infection resolve without any sequelae, complications have been described.[1,2] To our knowledge, this is the first case report of an orbital vasculitis following chicken pox infection in an immunocompetent patient.

Case report

A 7-year-old Filipino girl presented to the paediatric department with a 'swollen right eye', 6 days following chicken pox infection. On examination, she was systemically unwell and pyrexial. There was a right-sided axial proptosis with conjunctival chemosis and generalized restriction of extraocular movements. There was no relative afferent pupillary defect (RAPD) and her fundi looked healthy. She had neutrophilia and the C-reactive protein (CRP) level was raised. An urgent contrast-enhanced CT scan showed inflammatory changes within the superior retro-orbital fat and opacification of the right maxillary sinus (Figure 1a and b). The other paranasal sinuses were clear. A diagnosis of bacterial orbital cellulitis was made and treatment with intravenous benzylpenicillin and flucloxacillin was commenced.

After 4 days, she was referred to the ophthalmic adnexal service for management as the proptosis had worsened (Figure 2a). The globe was displaced downwards and there was a mild right RAPD with mydriasis of the right pupil. Fundal examination remained unremarkable. Systemically, she was getting better with improvement in her neutrophilia and CRP levels.

An urgent repeat contrast-enhanced CT scan revealed prominent orbital blood vessels with surrounding extensive inflammatory changes. The maxillary sinusitis had improved with no evidence of an abscess or cavernous sinus thrombosis in the right orbit (Figure 1c and d).

A noninfective inflammatory orbital disease affecting the orbital vasculature was suspected and high-dose oral prednisolone (2 mg/kg) was commenced under the cover of intravenous acyclovir (250 mg/m^2 t.i.d.). There was a rapid improvement in her orbital

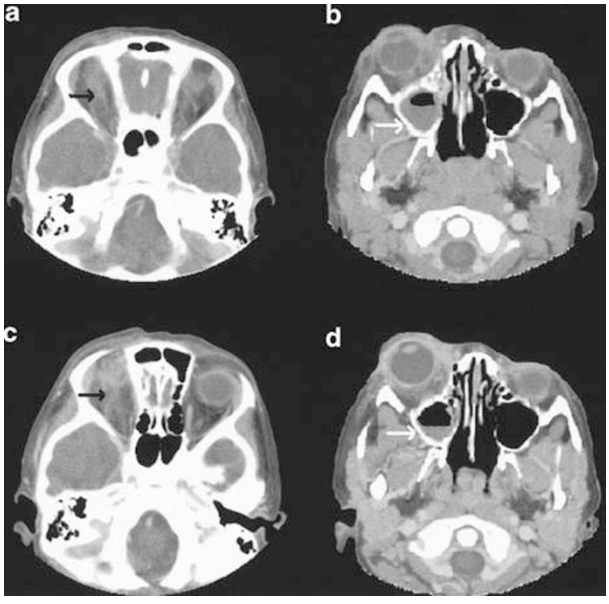

Figure 1 Contrast-enhanced CT scan of the orbits and paranasal sinuses on presentation (a, b) and 4 days later (c, d). Black arrows point to the dilated right superior ophthalmic vein. White arrows point to the opacified right maxillary sinus.

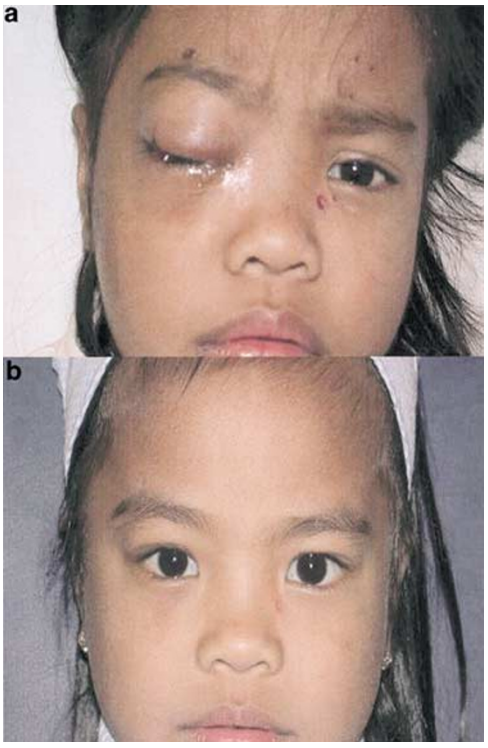

Figure 2 Colour photographs of the patient on presentation to the ophthalmic adnexal unit (a) and 1 month following treatment with systemic steroids (b).

inflammation and corresponding signs, and the acyclovir was discontinued 2 days later. The steroid dosage was tapered off over 2 months.

On review 1 month after presentation, there was complete resolution of her orbital inflammation without any complications (Figure 2b).

Comment

This case illustrates the potential diagnostic pitfalls that can occur in the management of a 'hot' orbit. The clinical presentation was suggestive of bacterial orbital cellulitis with subsequent abscess formation causing globe displacement. However, it is uncommon to develop bacterial orbital infection from maxillary sinus disease with an intact orbital floor. The repeat CT scan did not confirm the presence of an abscess or cavernous sinus thrombosis as potential causes for the worsening proptosis. Instead, the orbital blood vessels were found to be distended with marked surrounding inflammatory changes. The systemic improvement of the patient in the face of worsening orbital inflammation led us to suspect the possibility of a noninfective orbital inflammatory disorder affecting the orbital vasculature. The diagnosis was supported by the rapid response to high-dose systemic steroids. Unfortunately, a magnetic resonance angiogram that may have helped confirm the diagnosis of vasculitis was not performed.[3]

Varicella-associated vasculopathy is a complication of chicken pox infection with potentially serious consequences. This has been described in the central nervous system, kidney, and retina. The pathology ranges from small vessel vasculitis with lymphocytic infiltration of the vessel wall to giant cell arteritis involving the larger blood vessels.[1,4–6] Fortunately, our patient experienced no permanent sequelae from the vasculitis.

In conclusion, this case demonstrates that orbital vasculitis can occur following chicken pox infection. A high index of suspicion is needed to make the correct diagnosis as orbital vasculitis can mimic orbital cellulitis. Treatment with high-dose systemic steroids results in a rapid resolution of the inflammation.

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