certain patients about this rare, under-reported and embarrassing complication of β-blockers, along with the more commonly encountered adverse effects.

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Sir,

Disciform keratitis and optic disc swelling in Kawasaki disease: an unusual presentation

Kawasaki disease (KD) or mucocutaneous lymph node syndrome was first described by Tomisaku Kawasaki in 1967.[1] It is a disease of unknown aetiology and diagnosis is based on characteristic clinical features. These include fever persisting for 5 days and at least four of the following five principal clinical features:

1. erythema and fissuring of lips, tongue, buccal, and pharyngeal mucosa,
2. polymorphous exanthema,
3. erythema and oedema followed by desquamation of the skin of palms and soles,
4. bilateral nonexudative conjunctival injection,
5. acute nonpurulent cervical lymphadenopathy.

Diagnosis can be made by the presence of fever and fewer than four principal symptoms when coronary artery disease is detected on echocardiography or coronary angiography.[2,3]

Case report

An 11-year-old Caucasian male was admitted in the paediatric ward with a 5-day history of fever and rash on wrists and ankles. The rash became more widespread, involving his palms and soles and he also developed erythematous blisters on his lips and tongue with bilateral nonpurulent conjunctivitis. Laboratory investigation showed lymphocytosis (9.7×10^9 cells/l) and raised CRP (25 mg/l). No organism was identified on blood and urine culture, and anti-streptolysin titres as well as antibody screen for virus and mycoplasma were negative. He was managed as suspected KD and systemic signs responded well to treatment with intravenous immunoglobulin (IVIG) and high-dose aspirin. Echocardiogram showed normal function and no aneurysm was detected.

At ophthalmic referral on presentation, his visual acuity was noted as 6/6 in the right eye and 6/5 in the left eye. Anterior segment examination revealed diffuse bilateral nonpurulent conjunctival congestion, without any follicles or papillae and mild punctuate corneal staining. No anterior chamber or vitreous reaction was present and fundoscopy was normal.

After 3 weeks, he complained of cloudy vision in both eyes without any associated pain, redness, or photophobia. Vision was reduced to $<6/60$ in both eyes. He had no relative afferent papillary defect, and colour vision, measured with Ishihara plates, was intact. There was no conjunctival injection or discharge in either eye. Bilateral central stromal oedema with localised keratic precipitates and mild anterior chamber reaction characteristic of disciform keratitis was noted. Intraocular pressure was 10 mmHg OD and 12 mmHg OS by applanation. Fundoscopy revealed bilateral disc swelling without any other signs of inflammation in the posterior segment (Figure 1). A full neurological assessment excluded signs of increased intracranial

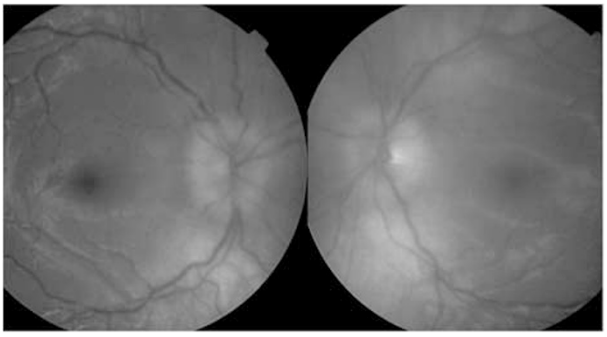

Figure 1 Fundus photographs at 3 weeks showing bilateral optic disc swelling.

pressure or neurological deficit. Lumbar puncture was deferred as ocular signs seemed to be of localised nature. CT scan of brain and orbits was normal.

He was started on oral acyclovir 200 mg five times a day, topical steroids every 2 h, and cyclopentolate 1% twice a day. Ocular signs showed rapid resolution, and vision improved to 6/6 in the right eye and 6/5 in the left eye in the next 3 weeks. Treatment was tapered and stopped after 6 weeks, on complete resolution of anterior segment inflammation and disc swelling.

Comment

KD most frequently affects children under 5 years of age and is more common in males. It is currently the leading cause of acquired heart disease in children.[2,3] The pathology of acute KD reveals a panvasculitis of the small- and medium-sized vessels. Mortality rate is higher among male patients with cardiac sequelae at 2.35%, while female patients with sequelae and those without sequelae do not seem to have more risk than the general population.[4] Case fatality rate has been reduced to 0.2–0.4% with use of IVIG and aspirin and prevention of coronary aneurysm.[5]

Clinical and epidemiological features suggest an infectious etiology for KD but no single organism has been consistently identified. Role of a super-antigen toxin similar to staphylococcal and streptococcal toxins has also not been supported by isolation of such a toxin. The possibility of multiple agents leading to a common cascade of host responses in genetically predisposed individuals is being considered.[3,6]

Although bilateral nonpurulent conjunctivitis is a diagnostic criteria, other ocular features that have been reported are superficial punctuate keratitis, anterior uveitis, vitreous opacities, optic disc swelling, retinal ischemia (presenting as cotton wool spots), vascular occlusions, and periorbital vasculitis.[7–9] Only one case of KD with disciform keratitis has been reported previously.[10] Our case highlights the importance of a detailed ocular examination in children diagnosed with KD.

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