

Fig. 2. The lid 2 months after argon laser treatment, showing the more cosmetically acceptable appearance.

Comment

We have found that the cosmesis of the lid margin is improved if the conjunctiva is not sutured to the skin at the initial surgery, thereby allowing the superior edge to retract behind the lid margin following division of the flap. Rogers² suggests that the skin should be divided 4 mm higher than the normal lid margin so that as the skin retracts the conjunctiva remains behind and unexposed. There is no doubt that with time many of these cases settle down; however, if there is a persistently hyperaemic conjunctival margin argon laser is effective. We do not have histological evidence but propose that the cosmetic improvement is produced by the destruction of the hyperaemic vessels, metaplastic change in the conjunctiva and the destruction of the goblet cells.

This treatment is also effective in the management of conjunctivalised lid margins following cryotherapy for trichiasis. Treatments can be reapplied with further therapeutic effect.

References

1. Hughes WL. A new method for rebuilding a lower lid. Arch Ophthalmol 1937;17:1008–17.

2. Rogers PA. Letter. Aust NZ J Ophthalmol 1991;19:160-1.

Mr P. Heyworth, FRCOphth 🖂 Moorfields Eye Hospital City Road London EC1V 2PD, UK

Mr G. J. Crawford, FRACO Centre for Ophthalmology and Visual Science University of Western Australia and The Lions Eye Institute Nedlands Perth, Western Australia 6009

Sir,

Conjunctivitis – **sometimes more than meets the eye!** Children with red eyes are frequently encountered in general practice, eye clinics, and accident and emergency departments. We describe the case of a child with Kawasaki disease who presented with conjunctivitis.

Case report

A 3¹/₂-year-old girl was referred to the eye clinic by her general practitioner with conjunctivitis not responding to 4 days of topical treatment with fusidic acid. Her mother gave a history of an unwell child, who had 5 days previously developed fever followed by bilateral red eyes and a sore throat. She had also noted the appearance of a rash on both upper and lower limbs on the morning of the hospital visit.

On examination the child was alert, but ill looking with a temperature of 38.8 °C. There was bilateral diffuse conjunctival hyperaemia without any discharge. Ocular examination was otherwise unremarkable. Systemic examination revealed lymphadenopathy involving the right preauricular, upper deep cervical and both submandibular groups. She had dry cracked lips, two petechiae on her palate, a few mouth ulcers and pharyngeal hyperaemia. A macular skin rash involving upper limbs, lower limbs and upper chest was also noted. Her pulse was 120/min, and regular. Heart sounds were normal and on auscultation an ejection flow murmur was heard in the aortic area.

On the basis of the patient's systemic findings a clinical diagnosis of Kawasaki disease was made and she was referred to the Paediatric Department. Both electrocardiography and echocardiography were normal, as was the full blood count. The erythrocyte sedimentation rate was 32 mm/h and the blood cultures were negative. Conjunctival swabs for adenovirus, Chlamydia and bacteria were also negative. The child was treated with intravenous gammaglobulin 2 g/kg and aspirin 30 mg/kg per day. Within 24 h of beginning treatment the temperature had returned to normal and all systemic signs and symptoms were rapidly resolving. A day later mild desquamation of the feet was noted, but the child was otherwise well. After 3 days the patient was discharged on aspirin for follow-up as an outpatient with serial echocardiograms. To date she remains well without any cardiac sequelae.

Discussion

Kawasaki disease, or mucocutaneous lymph node syndrome, is a generalised vasculitis of unknown aetiology. Its frequency is increasing worldwide¹ and it is now the commonest cause of acquired heart disease in children in developed countries. It typically affects children aged less than 5 years, the incidence in Britain being 3.4 per 100 000.²

A high index of suspicion is necessary for its diagnosis, as it mimics many common childhood infections. There is no single diagnostic test and diagnosis is based on the clinical findings (Table 1).³ Conjunctival injection is first noted shortly after the onset of the pyrexia and is typically bilateral, painless and non-exudative. Cardiac involvement with its potential for long-term morbidity and mortality is the most important manifestation. Coronary arteritis leading to formation of aneurysm occurs in 20–30% of untreated patients.⁴ Thrombosis within an aneurysm, myocardial ischaemia,

Table 1. Diagnostic criteria for Kawasaki disease³

Fever lasting for at least 5 days Presence of *four* of the following *five* conditions:

- Changes in peripheral extremities including acute, i.e. oedema and/or erythema of hands or feet, and convalescent, i.e membranous, usually periungual desquamation.
- Polymorphous skin rash, primarily truncal; but non-vesicular
- Bilateral painless non-purulent conjunctival injection
- Changes in lips and oral cavity such as erythema and cracking of lips, strawberry tongue, and diffuse injection of oral and pharyngeal mucosae
- Cervical lymphadenopathy

infarction or rupture of aneurysm may occur during the acute phase of the illness. Pericarditis, myocarditis, endocarditis, valvular insufficiency, heart failure and arrhythmia may also occur. The differential diagnosis includes scarlet fever, measles and other exanthems, toxic shock syndrome, leptospirosis, rickettsial disease, juvenile rheumatoid arthritis, drug reactions, Stevens–Johnson syndrome, vasculitides and adenoviral infections. There are no specific diagnostic tests, but the acute phase reactants are usually markedly elevated.

A single high dose of intravenous gammaglobulin, 2 g/kg, given over 12 h and commenced before the tenth day of the illness, significantly reduces both the incidence and severity of coronary artery abnormalities.^{5,6} Therapy with high-dose aspirin, 80–100 mg/kg per day, is also advised during the febrile stage and is continued at a single antiplatelet dose for 6–8 weeks or longer if definite coronary vessel abnormalities exist.⁷ Careful and serial follow-up evaluations are warranted for children with significant residual coronary sequelae.

In summary, Kawasaki disease is a serious and potentially life-threatening condition. Prompt recognition and treatment will, in most cases, reduce the morbidity and mortality associated with this disease. One of the principal criteria for diagnosis is a red eye – a common presentation for many conditions in both the ophthalmic and general practice settings. As the incidence of Kawasaki disease increases worldwide, ophthalmologists and all other healthcare professionals should be particularly aware of the significance of this often innocuous sign. We would therefore recommend a detailed systemic examination in any febrile child with conjunctivitis, looking in particular at skin, hands and feet, lips, oral cavity and lymph nodes to rule out Kawasaki disease.

References

- 1. Taubert KA. Epidemiology of Kawasaki disease in the United States and worldwide. Prog Pediatr Cardiol 1997;6:181–5.
- Dhillon R, Newton L, Rudd PT, Hall SM. Management of Kawasaki disease in the British Isles. Arch Dis Child 1993;69:631–6.
- 3. Dajani AS, Bisno AL, Chung KJ, Durack DT, Gerber MA, Kaplan EL, *et al.* Diagnostic guidelines for Kawasaki disease: American Heart Association Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease. Am J Dis Child 1990;144:1218–9.

- 4. Kato H, Akagi T, Sugimura T, Sato N, Hashino K, *et al.* Kawasaki disease. Coron Artery Dis 1995;6:194–206.
- 5. Newburger JW, Takahashi M, Beiser AS, Burns JC, Bastian J, Chung KJ, *et al.* A single intravenous infusion of gamma globulin as compared with four infusions in the treatment of acute Kawasaki syndrome. N Engl J Med 1991;324:1633–9.
- Terai M, Shulman ST. Prevalence of coronary artery abnormalities in Kawasaki disease is highly dependent on gamma globulin dose but independent of salicylate dose. J Pediatr 1997;131:888–93.
- Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. Diagnosis and therapy of Kawasaki disease in children. Circulation 1993;87:1776–80.

Girish G. Kamath ⊠ David Kent Louis G. Clearkin Department of Ophthalmology Arrowe Park Hospital Wirral CH49 5PE, UK Tel: +44 (0)151 678 5111 Fax: +44 (0)151 604 7152

Sir,

Keratoconjunctivitis sicca associated with lichen sclerosus et atrophicus

Many causes are known for dry eye. Among them are autoimmune diseases, infectious diseases, tumours, genetic disorders and response to medication or radiation. We present a case of bilateral keratoconjunctivitis sicca associated with lichen sclerosus et atrophicus (LSA).¹

Case report

A 58-year-old woman was admitted to our centre complaining of irritation, itching and foreign body sensation in both her eyes. On ocular examination positive fluorescein staining of the cornea and of the bulbar conjunctiva with rose bengal was observed. A decrease in tear production (1 mm in a Schirmer I test) and a tear break-up time of less than 10 s was caused by a degree of meibomitis and meibomian gland inspissation. No other abnormal ocular findings were demonstrated. A diagnosis of keratoconjunctivitis sicca was made.

Treatment with tear substitutes was insufficient for management, and occlusion of the puncta with collagen plugs was performed. Symptoms of dry eye persisted and the patient underwent an uneventful bilateral tarsorraphy with marked improvement.

Dermatological examination revealed asymptomatic porcelain-white papules and plaques, 0.5–10 cm in diameter, located on the neck, trunk, hips and eyelids. Histological findings from one of the plaques showed an atrophic epidermis with keratotic plugging. The upper dermis was oedematous with homogenisation of collagen fibres and mild perivascular lymphocytic infiltrate.