from inadvertent intraocular injection of gentamicin. Six months after his initial retinal reattachment surgery visual acuity remained counting fingers but the intraretinal haemorrhages had resolved.

The value of periocular antibiotics is now well established and gentamicin is one of the commonest drugs used.[1] The toxic effects of gentamicin have been well documented in the animal model[3,4] and in humans;[5,6] toxicity depends on the dosage and the route of administration. Recently McDonald reported five cases of severe retinal ischaemia associated with gentamicin injection, in three of which massive doses of gentamicin had been inadvertently injected into the eye.[5] In each case the clinical picture was similar with intraretinal haemorrhages, retinal infarction and arteriolar narrowing in the early stages. Retinal pigmentary degeneration, optic atrophy, rubeosis iridis and neovascular glaucoma ensued. The similarity of the clinical picture in our patients suggests that gentamicin was the causative factor. However, we are not aware of exudative detachment (case 1) having been previously observed in cases of gentamicin toxicity. Baum and Peyman reported retinal haemorrhages in the same quadrant as periocular injection of gentamicin but without any of the other features of gentamicin toxicity.[7] In our first case there were large areas of exposed uveal tissue through which high concentrations of gentamicin might have diffused into the intraocular tissues and caused such profound effects. In case 2 gentamicin was injected in the region of a silastic sponge at the site of inadvertent sclerostomy and may have entered the eye through this route.

These cases suggest that care should be taken when administering potentially toxic antibiotics in the region of open wounds or near deficient sclera through which they might diffuse into the intraocular tissues. The possibility also exists that episcleral silastic sponges may act as a reservoir for drugs in these circumstances, increasing the danger of excessive intraocular penetration and toxicity.

Your sincerely.

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SIR—A case is presented of a child born with the rare form of craniostenosis known as cloverleaf or Kleeblattschädel syndrome. Very severe bilateral proptosis occurred with complete dislocation of the globe on one side, due to extreme shallowing of the orbital fossae. Bilateral tarsorrhaphies were performed to protect the dislocated globe and prevent exposure to the other eye.

Case history

A four-day-old boy with the cloverleaf form of craniostenosis was referred by the Department of Paediatric Surgery for an urgent ophthalmological opinion. The mother was 37 years old with three other children: one healthy, one with Perthe's disease and one with Perthe's disease and a mild form of haemophilia. Normal delivery took place at full term with Apgar scores of eight at one minute and ten at five minutes.

The appearance at birth was essentially the same as when seen by the Eye Department. The child showed many features of the Kleeblattschädel syndrome with a cloverleaf shaped head, low set ears and bilateral syndactyly of second and third toes. There was complete dislocation of the left globe from the orbit, with the eyelids obscured by conjunctival chemosis. The right globe was proptosed to a lesser degree (Figs.1–3). Both corneas showed epithelial loss and central corneal oedema. The pupils reacted to bright direct light. CT scans and skull X-rays showed hydrocephalus and shallow truncated orbits (Fig. 4).

Management

Bilateral medial and lateral tarsorrhaphies

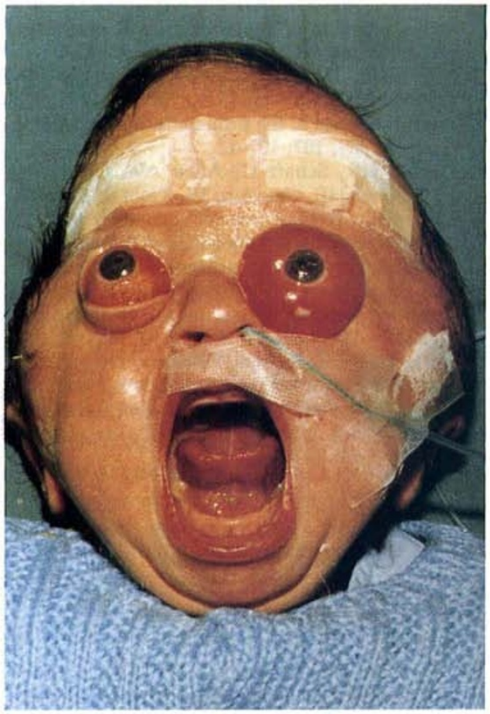

Fig. 1

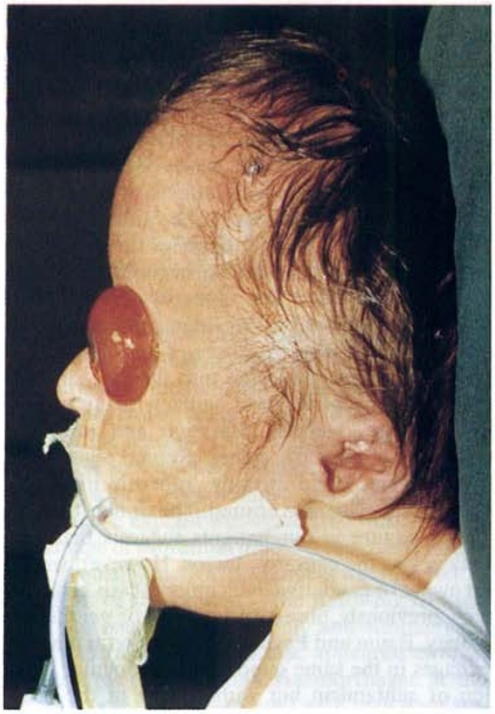

Fig. 2

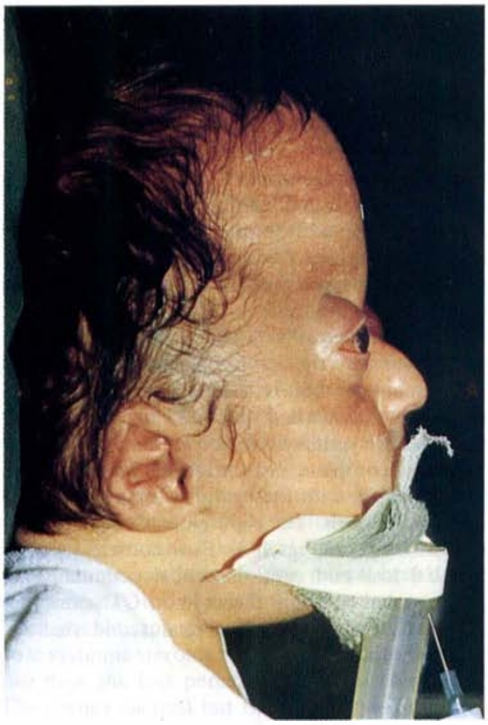

Fig. 3

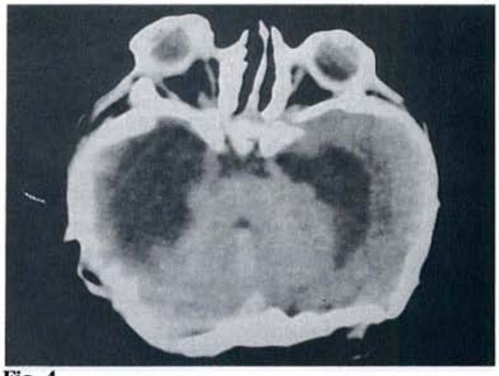

Fig. 4

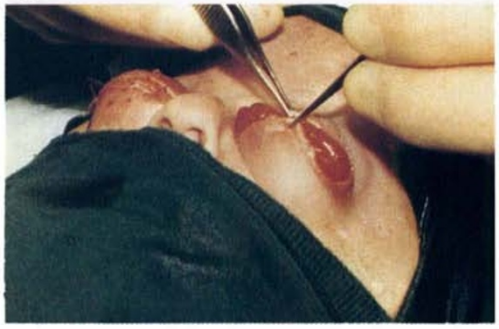

Fig. 5

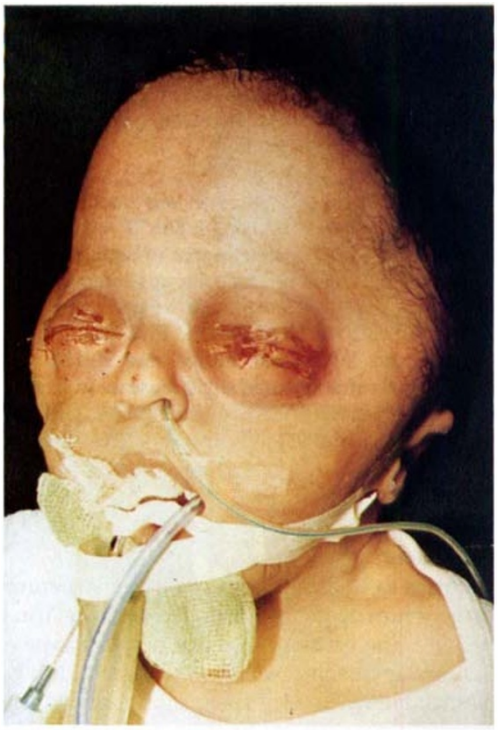

Fig. 6

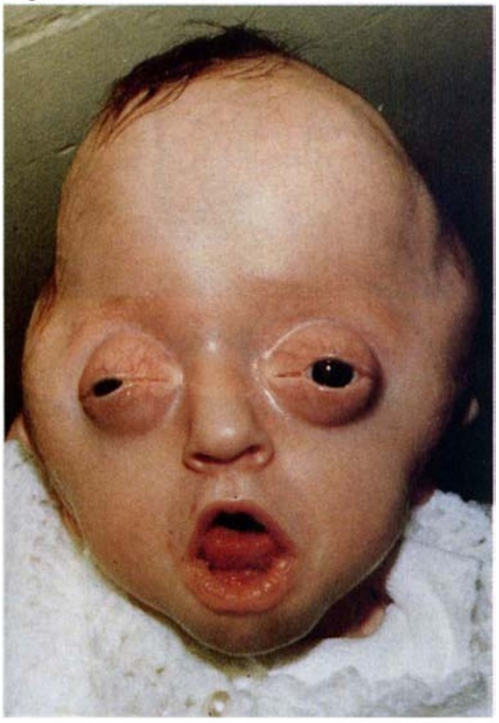

Fig. 7

were performed to protect the globes. The chemosed conjunctiva was irrigated during the operation with a weak solution of adrenaline which aided lid closure (Figs. 5–7). Three weeks later, the left tarsorrhaphy was opened centrally and an examination under anaesthetic performed. Fine central corneal opacities with deep anterior chambers, round pupils and red reflexes were present, right and left. No fundal details were visible. At one month the child showed no response to a light, or to the Catford drum. There was little change at three months although the pupils remained active. The child's general condition improved initially, following the insertion of ventriculoperitoneal shunt, but later deteriorated and he died aged four months on supportive therapy but without further surgical intervention.

Comments

Cloverleaf or Kleeblattschädel syndrome is a rare form of craniostenosis first described in 1960 by Holtermuller and Wiedermann.[1] Fusion of the coronal and lambdoidal sutures is thought to occur early in intrauterine life, possibly at 6 to 8 weeks gestation.[2] The result is a vertical skull protrusion separated from bulges in the temporal region and face by a furrow, forming the cloverleaf shaped head.[3] Internally, the skull vault becomes trilobed with a bony shelf (corresponding to the external furrow) separating a frontal vesicle of the brain from two temporal vesicles. The eyeballs protrude because of shortening of the orbital fossae. Hydrocephalus is characteristic.

The major craniofacial manifestations are:[4,5]

1. Cloverleaf skull.
2. Severe exophthalmos, corneal ulcerations and shallow orbits. Hypertelorism, divergent strabismus and antimongoid eye slant.
3. Downward displacement of the ears (occasionally reaching a position parallel to the shoulders).
4. Beaked nose with a depressed nasal bridge.
5. Premaxillary and maxillary hypoplasia, with mouth open at rest and a high arched palate.

In addition, ankylosis of the fingers and joints and syndactyly have been reported. The condition is related to Apert's syndrome and Crouzon's disease, although there is no evidence that it is an inherited disorder. Survival to fourteen years has been reported. Recent studies suggests that total, subtotal craniectomy within the first few weeks of life may improve the otherwise dismal prognosis.

Yours sincerely,

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SIR—A case is presented in which a seven-year-old boy sustained a blunt injury to one eye. On examination, the lens of this eye was found to have the appearance of a posterior lenticonus, associated with a rupture of the posterior capsule. Refraction revealed a high degree of astigmatism induced by the change in shape of the lens. The rapid development of a dense cataract was anticipated, but in this case only minimal opacities formed with little deterioration in visual acuity. A mature cataract was eventually formed eight months post trauma.

Case report

A seven-year-old Asian boy attended the Ophthalmic Casualty Department, having been kicked in the left eye four days previously. He has complained of diminished vision since that time.

Visual acuity was found to be hand movements with accurate light projection in the left eye and 20/15 in the right. There was no external evidence of trauma, but the lens showed a dramatic change in shape (Figs. 1 & 2). This had the appearance of a large posterior lenticonus associated with rupture of the posterior lens capsule and presumably, herniation of cortex into the defect. There were no other abnormalities, apart from some minimal punctate lens opacities posteriorly.

The child was known to have a slightly amblyopic left eye with documented previous refraction of:

Right Plano = 20/15
Left Plano = 20/40

Refraction on this occasion was unchanged on the right, but was $-7.00/+6.00\times70^{\circ} = 20/200$ on the left, revealing a high degree of astigmatism in concordance with the lenticular appearance.

Three weeks later the left visual acuity had improved to 20/60 with pinhole. Five months post-trauma the left visual acuity was 20/120 with unchanged refraction and slight increase in size and density of the lens opacities (Figs. 3 & 4). A mature cataract eventually formed eight months post-injury.

Comment

Injuries to the lens and supporting structures from blunt trauma are common. The usual result is the formation of one or other type of lenticular opacity, which may or may not be associated with a visible capsular tear. A large

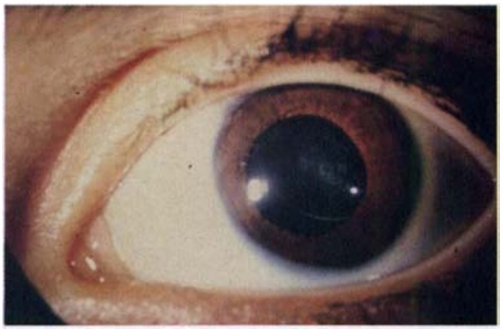

Fig. 1

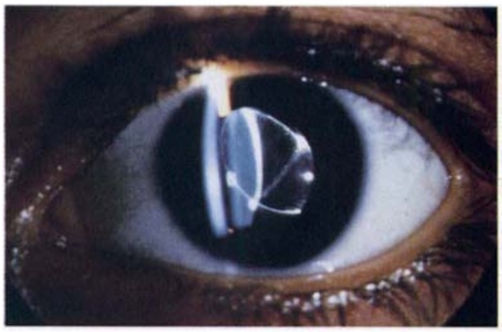

Fig. 2