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Sir,

Transient amaurosis with intracameral lidocaine

Intracameral lidocaine is often used to augment topical anesthesia during ocular surgery. This is a safe and effective method of anesthesia that eliminates discomfort caused by tissue manipulation and thus improves patient cooperation. Although rare, complications can occur. We describe a case of a rare complication with intracameral lidocaine.

Case report

We report a patient who underwent peripheral iridectomy under topical anaesthesia, augmented by intracameral lidocaine 1%. He had vitrectomy for retinal detachment years ago, and vision was 20/70. He underwent a recent repeat vitrectomy with secondary anterior chamber intraocular lens inserted for dislocation of posterior chamber intraocular lens. The surgery was uneventful, but when the dressing was removed an hour later, he noted complete loss of vision. Vision was perception of light with the presence of a relative afferent pupillary defect (APD). Anterior and posterior segment was normal, with no evidence of disc swelling or cherry red spot. Carotid examination revealed no bruit. He reported gradual return of vision over 4 h. Within 20 h, vision improved to counting fingers at 3 m with resolution of APD. Two weeks later, vision recovered to 20/30 with no residual defects.

Comment

Intracameral lidocaine (1%) can result in transient visual loss.[1,2] The recovery period here was similar to the case of Lincoff *et al*,[3] where inadvertent intraocular injection of lidocaine showed improvement in retinal function 4 h later and recovery in 16 h. This is rare but can occur especially in cases of communication with the posterior segment, for example, ruptured posterior capsule and aphakia, where anaesthesia can diffuse readily into the vitreous cavity coming into direct contact with the retina and optic nerve. Visual recovery is complete with no apparent functional damage. Electrophysiological studies in animals injected with intraocular lidocaine[4] show b-waves demonstrated a decrease in the amplitude and an increase in the implicit time. Electroretinogram responses recovered within 24 h.

In the absence of a definite anaesthesia history, only after adequate investigation to rule out vascular or neurological complications, one can attribute amaurosis to intracameral lidocaine.

This case illustrates that intracameral lidocaine 1% is safe to use to augment topical anaesthesia, even when posterior capsule is not intact. The surgeon should be aware that transient amaurosis may occur. Patients can be reassured that it is reversible, although this may take up to several hours to days. In patients with a deficient capsule, or one-eyed patients, other alternatives such as subtenon or peribulbar anaesthesia can be considered.

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Sir,

New fundus findings in a case of Kabuki syndrome

Kabuki syndrome is a multiple congenital anomalies/mental retardation syndrome of unknown cause. Its five cardinal manifestations are characteristic facies, skeletal anomalies, dermatoglyphic anomalies, mental retardation, and short stature.[1–4]

We report a case of tortuous retinal vessels and prepapillary gliosis in Kabuki syndrome.

Case report

A 21-year-old man diagnosed with Kabuki syndrome by the medical geneticist was referred with peculiar optic discs and macula irregularities.

He was born by caesarean section following fetal distress. He was noted to have cleft soft palate, micrognathia (Figure 1), and umbilical hernia. He developed jaundice after birth and was treated with phototherapy. He also had a hypoglycaemic seizure in

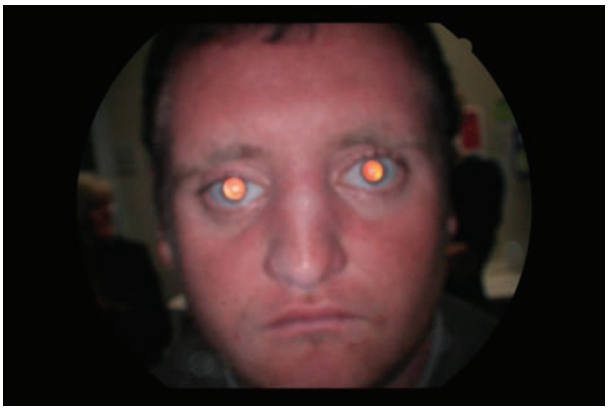

Figure 1 Photograph shows long palpebral fissures, mild ptosis, arched eyebrows, and micrognathia.

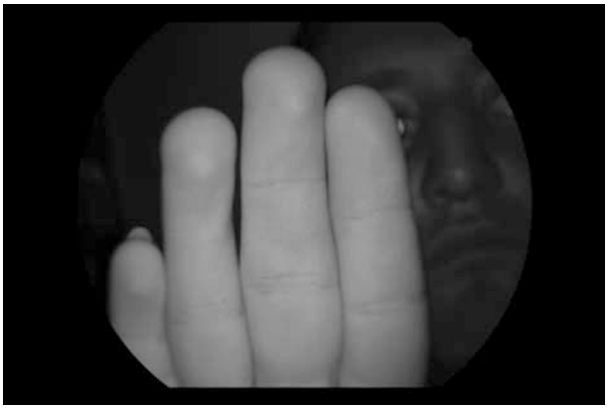

Figure 2 Photograph shows fetal fingertip pads.

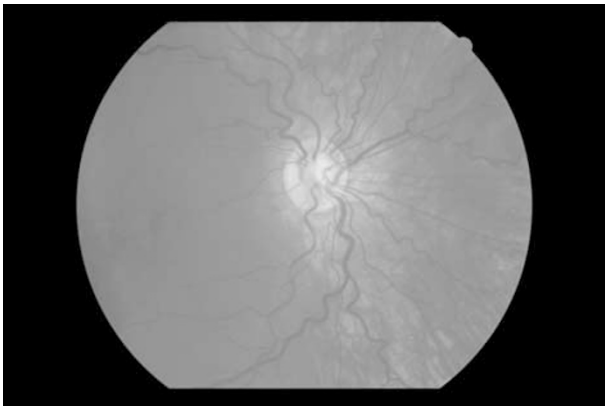

Figure 3 The fundus photograph of right eye shows prepapillary gliosis, tortuous retinal vessels, foveal irregular pigmentation, and tilted disc.

early neonatal period. He was fed with cow's milk and egg-free diet as he developed severe immediate hypersensitivity reaction to them. Chromosomal studies showed a normal 46 XY pattern with no evidence of

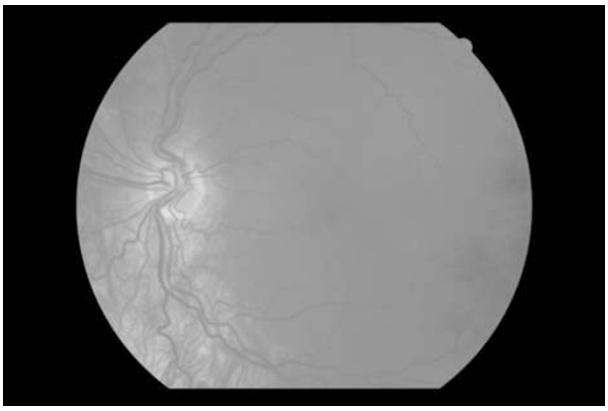

Figure 4 The fundus photograph of left eye shows tortuous retinal vessels, foveal irregular pigmentation, and tilted disc.

Table 1

Published Kabuki syndrome features	*Presence of the features in this patient*
Amblyopia	No
Refractive error	Yes
Strabismus	Yes
Nystagmus	No
Coloboma	No
Microcornea	No
Cornea opacity	No
Blue sclera	No
Cataract	No
Nasolacrimal duct obstruction	No
Jaw-winking ptosis	No
Caruncle lipoma	No
Cornea pannus	No
Retinal telangiectasia	No
Retinal pigmentation	Yes
Arched eyebrows	Yes
Prominent eyelashes	Yes
Lateral lower lid eversion	Yes
Long palpebral fissures	Yes
Long malformed ears	Yes
Brachydactyly	Yes
Fetal fingertip pads	Yes
Intellectual disability	Yes
Short stature	Yes

fragile X syndrome. Molecular and cytogenetic studies revealed no abnormalities. There was no history of consanguinity or background of mental disability or peculiar facies. Other features included short stature, moderate learning, and coordination difficulties and general developmental delay. Previously, he had squint surgery to correct left inferior oblique overaction, cleft palate repair, bilateral myringoplasty, and left tympanoplasty . He went to speech therapy once yearly.

Aided visual acuities were 6/6 with normal orthoptic assessment. There were long malformed ears, long palpebral fissures, lower palpebral eversions, mild

ptosis, arched eyebrows with lateral thinning of eyebrows, and prominent eyelashes (Figure 1). There were also fetal fingertip pads and abnormally short fifth digits in both hands (Figure 2). The anterior segment examination was normal. The fundus examination showed right prepapillary gliosis, bilateral tortuous retinal vessels, foveal irregular pigmentation, and tilted discs (Figures 3 and 4). The axial lengths of right and left eyes were 24.42 and 24.67 mm, respectively. The updated refraction showed −2.00 −0.50 at 100 (right eye) and −2.50 −1.00 at 100 (left eye).

Comment

Amblyopia, refractive errors, strabismus, nystagmus, colobomas, microcornea, corneal opacities, blue sclera, cataracts, nasolacrimal duct obstruction, jaw-winking ptosis, caruncle lipoma, cornea pannus, retinal telangiectasia, and retinal pigmentation have all been reported in Kabuki syndrome.[5–9] To the best of our knowledge, prepapillary gliosis and tortuous retinal vessels have not been reported (Table 1).

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Sir,

Corneal epithelial dysmaturation: case report

Corneal epithelial dysmaturation is clinically similar to primary corneal epithelial dysplasia or corneal intraepithelial neoplasia without a prominent limbal lesion. We report a case of successful treatment of an unusual case of unilateral central corneal epithelial dysmaturation.

Case report

A 67-year-old man visited the ophthalmology department for an opaque corneal opacity in his right eye, which had developed 1 year before and had gradually increased in size. Slit-lamp examination showed an individual island of opalescent corneal epithelial lesion, 2 × 2.4 mm in size, located in the central cornea, without neoplastic fibrovascular corneal pannus (Figure 1). Best-corrected visual acuity was 20/40 in the right eye and 20/20 in the left eye. Other ocular findings were normal. A decision was made to remove the lesion and subject it to cytological examination. The lesion was carefully removed by simple superficial excision with a knife. Cytological examination of the removed lesion showed normal nuclear/cytoplasmic ratio and hyperplastic epithelium with no atypia (Figure 2). Corneal epithelial dysmaturation was diagnosed based upon the clinical and cytological results. Topical antibiotic and steroid were used for 4 weeks with tapering of the steroid. Visual acuity improved to 20/20

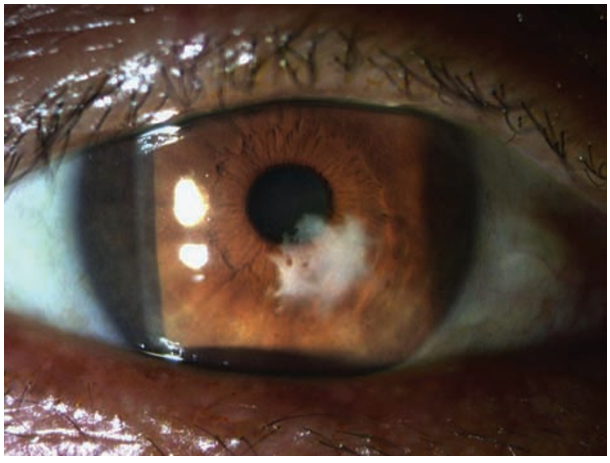

Figure 1 Right anterior segment view at the initial examination. It shows an individual island of opalescent corneal epithelial lesion, 2 × 2.4 mm in size, located at central cornea, without neoplastic fibrovascular corneal pannus.