shown in monkey fovea that parafoveal Muller cells have long outer trunks in the outer plexiform layer which accompanies and unsheathes the Henle's fibres on their course from the foveal center to the foveal periphery.[8] This anatomical characteristics of the fovea helps in maintaining the cohesion of Henle's layer with the inner retina. Our case did show a neural split on the lateral edge of the macular hole. A serial OCT could have possibly demonstrated these several changes. Haouchine et al [6] have observed initially a split in the foveal center, in the inner part of the foveal tissue and subsequently an outer foveal opening resembling a centrifugal separation of the photoreceptors. They postulated that it might be the result of damage to the foveal and parafoveal Muller cells resulting in dehiscence of the neural tissue from the foveola. Probably, our case developed a pseudocyst and then macular hole due to initial insult to the foveolar Muller cells during vitreous surgery. Since complete PVD was created during primary vitrectomy, the role of posterior vitreous in the formation of macular hole can be ruled out in our case. The various steps of vitreous surgery probably are the inciting factors in stimulating the glial or Mullers cells activation. However, we do not have any other valid explanation for the possible harmful effect of surgery on the macula.

Foveal pseudocyst with progression to full thickness macular hole was not noted earlier in a vitrectomised eye. Since there was no evidence of epiretinal membrane and posterior hyaloid stripping was done in the primary surgery, we hypothesise that Muller cell insult is a probable hypothesis of macular hole formation in a vitrectomised eye. We are aware that a single case report do not confirm the pathomechanism, which is possible only by histopathology.

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Congenital Glaucoma Associated with 22p+ Variant in a Dysmorphic Child

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A case of congenital glaucoma with developmental delay and several dysmorphic features showing 22p+ chromosomal variant is reported.

Key Words: Congenital glaucoma, dysmorphia, chromosomal abnormality

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The association of congenital glaucoma with chromosomal abnormalities has been reported previously.[1,2] We examined a patient with suspected congenital glaucoma who also had developmental delay and several dysmorphic features. Hence chromosomal and molecular analysis was performed.

Case report

The patient was a two-month-old infant referred with bilateral buphthalmos and other extraocular manifestations. The main phenotypic features were mongoloid slanting of the eyelid fissures, low nasal bridge, hypertelorism, hypotonia, brachycephalic head with a flat occiput and mental retardation (Figure 1). The visual acuity could not be recorded as the child had severe photophobia. Ocular examination under anaesthesia showed severe corneal oedema with horizontal corneal diameter of 17 mm in both eyes. Intraocular pressure (IOP) was 28 mm Hg in both eyes. A clinical diagnosis of congenital glaucoma was

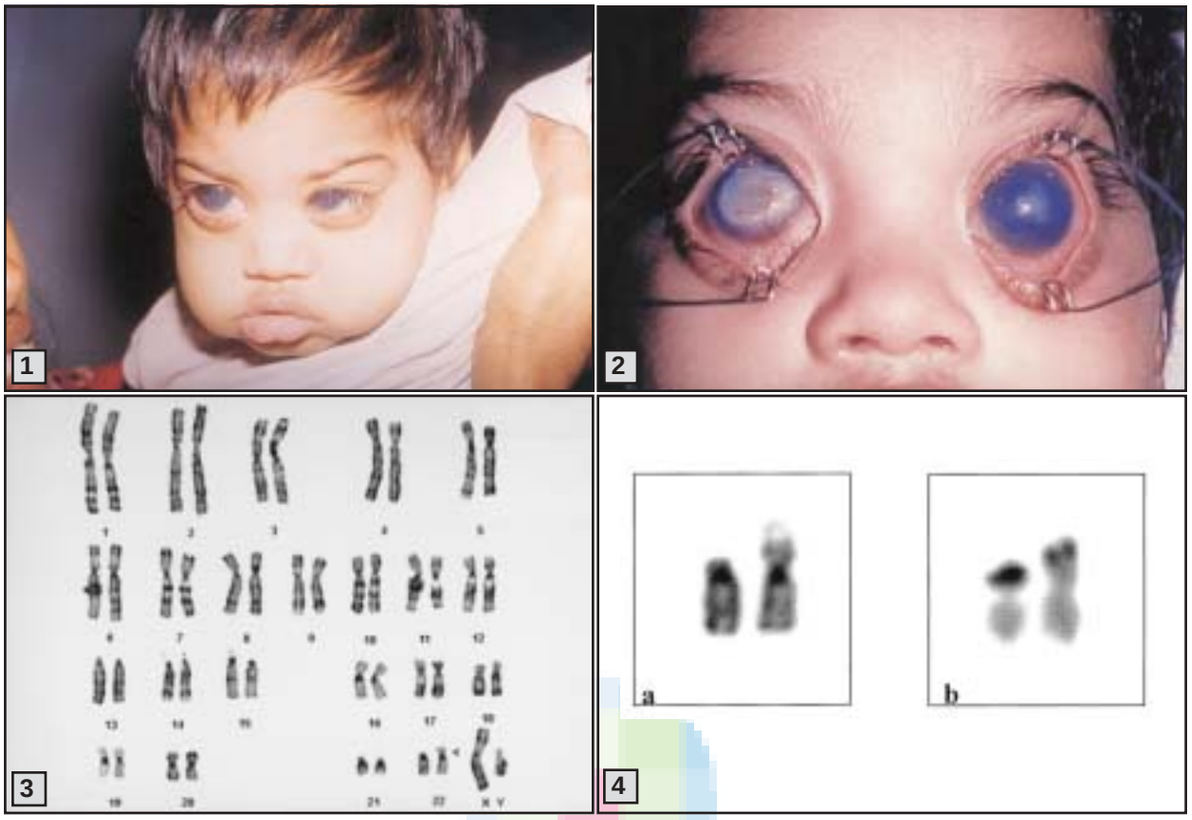

Figure 1. Clinical photograph of the patient showing dysmorphic features; **Figure 2.** Bilateral megalocornea with vascularised corneal scar; **Figure 3.** Representative G-banding karyotype of patient showing 46, XY, 22p+. 22p+ is marked by arrow; **Figure 4.** Partial karyotype of chromosome 22 **a)** G-banding **b)** Nucleolar Organizing Region (NOR) staining showing 22p+ and NOR above the Giemsa staining.

made and primary combined trabeculotomy and trabeculectomy was performed for control of IOP. Two months after surgery the patient injured the right eye with his fingernail and presented with pain, redness and watering. Examination under anaesthesia showed corneal ulceration in the right eye; he was treated with intensive topical antibiotics (fortified cefazolin and fortified gentamicin drops) and anti-inflammatory (1% atropine eye drops) agents. The corneal ulcer healed in three weeks resulting in a vascularised corneal scar in the right eye (Figure 2). The patient was followed up for 30 months. At the last follow-up visit, his visual acuity was no light perception in the right eye and fixing and following light in the left eye. IOP in the right eye could not be recorded because of corneal scarring, and it was 14 mm Hg in the left eye.

Chromosomal analysis was done by blood lymphocyte culture and Giemsa banding technique. Fifteen metaphases were analysed and the karyotype of the patient was found to be 46, XY, 22p+ (Figure 3 and 4a). The variant 22p+ was confirmed by Nucleolar Organizing Region (NOR) staining (Figure 4b).

Discussion

The patient represents an unusual case of congenital glaucoma with several extraocular manifestations (dysmorphic features) and 22p+ variant (amplification of 'p' arm of chromosome 22). Although phenotypically the patient had several dysmorphic features along with congenital glaucoma, the chromosomal analysis did not reveal any gross cytogenetic defect. However, the karyotype revealed that the patient has an unusual 22p+ chromosome, which is considered a normal variant in some cases.[3] Chromosome 22p+ is also associated with late onset Alzheimer's disease,[4] psychomotor retardation,[5] spontaneous abortion and gonadal dysgenesis.[6] Since the predominant cause of primary congenital glaucoma is mutations in the *CYP1B1* gene[7] (cytochrome P450), the coding region was amplified and sequenced, but disease causing mutations were not found. To our knowledge, the congenital glaucoma manifested along with this unusual extraocular phenotype has not been reported earlier, and the cytogenetic variant present here might provide a clue in identifying the underlying genetic defect. The phenotype seen in this patient seems to be both clinically and genetically heterogeneous. Although the association of 22p+ with congenital glaucoma is novel, it is not clear whether it is coincidental or causal.

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Presumed Ocular Toxoplasmosis Presenting as Papillitis

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Unilateral papillitis is a rare manifestation of ocular toxoplasmosis. However, other causes of papillitis need to be ruled out before concluding the diagnosis.

Key Words: Toxoplasmosis, papillitis, chorioretinitis

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Toxoplasmosis is a common infection of man and animals. The disease is widespread and has a worldwide distribution.[1, 2] In the eye, the retina is the primary site of infection. It manifests as a necrotizing retinitis with subjacent choroidal inflammation.[2] Optic nerve involvement such as papillitis has been reported before, and can occur due to a lesion at the disc, or secondary to a subjacent retinal lesion.[3, 4]

We report the case of a young woman who presented with sudden loss of vision in her right eye due to papillitis secondary to direct involvement of the optic disc.

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Case report

A 32-year-old woman presented to the ophthalmic emergency department with a history of loss of vision in her right eye of 10 days duration. This was preceded a few hours by a right-sided headache that responded to systemic analgesics. There was no associated ocular pain. Her medical history was otherwise non-contributory.

On examination the best-corrected visual acuity in the right eye was 6/36 (-0.50 Dsph/ -0.25Dcyl@125°) and 6/6 (-0.25 Dsph) in her left eye. External ocular examination, extraocular muscle movements and slitlamp examination was normal in both eyes. There was an afferent pupillary defect in the right eye. The intraocular pressure (IOP) was 12 mmHg in the right eye, and 14 mm Hg in the left eye. Colour vision (Ishihara'sTests, Kanehara & Co, Ltd, Japan. 38 Plates Edition, 1985) was normal in both eyes. Ophthalmoscopic examination revealed oedema and hyperaemia of the optic disc in the right eye. There was a yellow infiltrate at the upper nasal border of the disc. Healed patches of chorioretinitis were present, close to the temporal arcades in both eyes. In the left eye, a pigment clump was noted in the center of the healed chorio-retinitis patch (Figures 1a and 1b). There was no vitritis in either eye. Visual field (Goldmann Perimeter) was normal in the left eye but showed enlargement of blind spot in the right eye and two relative scotomas to object size I2e and I4e, in the inferior field between the 10° and 20° isopter (Figure 2a).

Pattern visually evoked potential was normal in both the eyes. Fundus flourescein angiography revealed hyperfluorescene at the disc in the right eye due to leakage of the dye. There was also hyperfluorescence corresponding to the healed patches of chorioretinitis in both the eyes with blocked fluorescence corresponding to the pigment clump in the left eye (Figure 2b).

Laboratory investigations revealed normal blood counts, erythrocyte sedimentation rate, and C reactive protein. Mantoux test was negative. X ray chest and magnetic resonance imaging of the brain and orbit were normal. Serology was negative for syphilis, cytomegalovirus, retrovirus, herpes simplex virus and rubella. Ig G was positive for toxoplasmosis by the ELISA technique (4 IU / ml) (Normal 2- 3 IU/ ml), but Ig M was