EYE - DAWN
DR.AGARWALS
NEWSLETTER
RISE WITH KNOWLEDGE

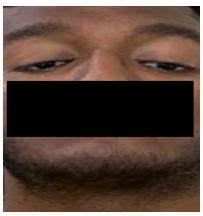
APRIL 2025
BY POST GRADUATES

EYE-CONIC CASE
Glimpse of an enticing case scenario

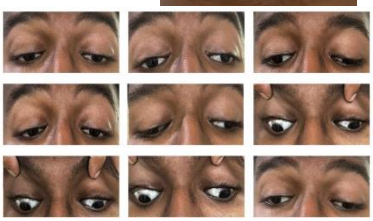
A CASE OF CONGENITAL FIBROSIS OF EXTRA OCULAR MUSCLE (CFEOM)

- DR. SIKHA BINCE

A 22 year old male presented with complaints of drooping of upper eyelids and inability to move eyes since birth . H/o similar complaints in mother, but siblings were unaffected. No consanguinity in parents. On examination, best corrected visual acuity in RE was 6/9, LE was 6/12, IOP measured in RE 13 mm hg , LE 14 mm hg. Patient had marked chin elevation, frontalis overaction, bilateral ptosis with poor bells and minimal lagophthalmos.



Left eye exotropia with both eye hypotropia in primary position. Both eyes there was marked elevation deficit 5- and adduction deficit 3- ,other movements were also restricted. Pupil was sluggishly reacting to light in both eyes, anisocoria noted with RE miotic pupil. Otherwise anterior and posterior segment examination was within normal limits.



Clinical findings were suggestive of Congenital Fibrosis of Extra ocular muscle, probably CFEOM Type 2 since pupil is involved and exotropia is noted. Genetic counseling given to the patient and was advised to use tear supplements to prevent exposure keratopathy and advised refractive correction by glass prescription.

Periodic follow up explained to patient.

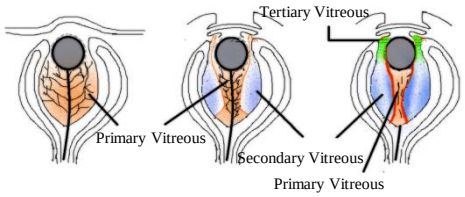
Ptosis surgical correction was deferred due to poor bells.

.CFEOM is a rare congenital syndrome characterized by non progressive unilateral or bilateral restrictive strabismus with or without ptosis. There is a fibrosis of extra ocular muscle causing restricted ocular motility, especially elevation. Genes are implicated in CFEOM: COL25A1 ,KIF21A, PHOX 2A, TUBA1A TuBB2B, TuBB3. Mutations are most commonly inherited in Autosomal Dominant and Autosomal Recessive forms. Different Types of CFEOM include CFEOM 1 , CFEOM 2, CFEOM 3; CFEOM 4 (Tukel Syndrome) CFEOM 5. Diagnosis is made clinically and identifying specific form of CFEOM through genetic testing. Differential diagnosis: Monocular elevation deficit, Congenital third Nerve Palsy, Brown syndrome Congenital Myasthenia Syndrome . Management : Non Surgical management include Refractive error correction ,Amblyopia treatment ,Lubricant eye drops ,Crutch glasses Surgical Management: Ptosis Correction and squint correction which is very challenging.

EYE-OPENER
Lets brush-up our basics

DEVELOPMENT OF VITREOUS

- DR. AMAN GUPTA



The development of the vitreous is a succession of three distinct phases.

At 4 weeks of gestation - Mesodermal cells invade optic cup cavity and forms hyaloid artery and branches (vasa hyaloidaie propria) - occupies space between lens and neural retina .

1) Primary vitreous - Between 4th and 5th weeks, fibrillar material, mesenchymal cells and vascular cells fills up retrolental space.

2) Secondary Vitreous - Starts forming between boundary of primary vitreous and retina. Composed of more compact type 2 collagen fibrils

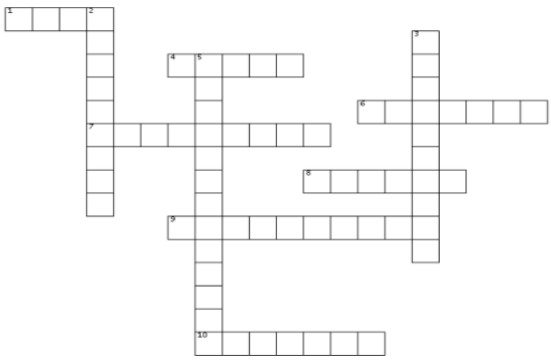
3) Tertiary Vitreous- At 12th week, Condensation of thicker collagen fibres of secondary vitreous occurs and gets attached firmly to internal limiting membrane of optic cup at its rim and extends to equator of lens, forms the vitreous base.

Applied Anatomy- Persistent Fetal Vasculature (PFV) caused by the failure of the primary vitreous and hyaloid vasculature to regress properly during development.

CROSSWORD

DR. DHIVYA M

EYE QUEST
To tease your brain a little



ACROSS

1. Bilateral congenital cataract, Posterior lenticonus, Loss of deep tendon reflexes, Fanconi syndrome
4. Triad of BRAO, Sensorineural hearing loss, encephalopathy
6. Craniosynostosis, Maxillary hyperplasia, Exorbitism, Hypertelorism
7. Ipsilateral abduction deficit, Facial pain, CSOM
8. Non progressive nyctalopia, Golden yellow retina that turns normal red on prolonged dark adaptation
9. X linked, Congenital cataract-Y sutural, Microcornea, Microphthalmia
10. Corpus callosum agenesis, Infantile spasms, Chorioretinal lacunae defects, coloboma

DOWN

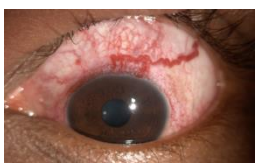
2. Postop inflammation, Secondary glaucoma, Iris transillumination defects, Needs surgical repositioning/ IOL exchange
3. Periorbital pain, restricted extraocular movements, facial hypoesthesia, Scintillating scotoma,
5. Fixed and dilated pupil, Diffuse iris atrophy following penetrating keratoplasty

STURGE WEBER SYNDROME – BLOOD IN SCHLEMMS CANAL

- Dr. NEETHU

EYE-WORTHY SNAP
Captured clinical findings

Gonioscopy showing blood in schlemm's canal



Episcleral hemangioma

A 22 year old male patient came with complaints of pain in left eye. Patient had reddish lesion on left forehead with mild redness in left eye since birth. On examination, Portwine stain was noted on left forehead. Best corrected visual acuity is 6/6 in BE, IOP measured with Goldmann applanation tonometer RE was 16mm hg and LE was 40mmhg and LE had prominent episcleral vessels, s/o Episcleral hemangioma. Gonioscopy of the angles revealed blood in schlemm's canal in LE. Fundus examination showed 0.8:1CDR and bipolar thinning of neuroretinal rim in LE. No evidence of Choroidal hemangioma noted. Features are suggestive of Sturge Weber syndrome with secondary open angle glaucoma.

EYE APPRECIATE

Congratulations Dr. Sara and Dr. Sikha for winning 3rd prize in "Silent theif hunt: Glaucoma Quiz" conducted by Regional Institute of Ophthamology -GH , Chennai on 16th March 2025.

EYE QUEST Answers January 25

1. Brushfield spot – Downs syndrome 2. Zentmayers line 3. Hummelsheim repair 4. Knies sign 5. Nettleship punctum dilator 6. Seidels test 7. Marc Amsler

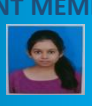
STUDENT LEAD


DR. HRUDYA


DR. SARA

STUDENT MEMBERS


DR. DIVYA . M


DR. DHIVYA . R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