AN OCULAR GRAND ROUNDS TOUR OF ORLANDO

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OBJECTIVES
1. To learn about the differential diagnosis of the cases presented.
2. To learn to utilize ancillary testing as part of the diagnostic process for these cases.
3. To learn additional information about the diagnosis of the cases presented.
4. To learn about the management of the cases presented to improve the practitioner's care of similar patients.

CASE 1
38 / AA / F
CC: No changes in vision, no ocular comfort problems
Oc Hx: LEE 18 mos, unremarkable
Med Hx: +DM (gestational, still on meds), +HTN, anemia
Fam Hx: unremarkable
Soc Hx: -tobacco, -alcohol
BVA: 20/20 OD, 20/20 OS -450-075x025, -475-075x155
PREMIS: FTFC OD OS, FROM, Normal Pupils, No APD
SLE OD corneal scar, OS normal
IOP: 18/18 mm Hg @ 1p
DFE See Photos

DDX OF ONH EDEMA
ONE POSSIBILITY
PAPILLEDEMA
BILATERALLY SWOLLEN OPTIC NERVES CAUSED BY INCREASED INTRACRANIAL PRESSURE
CAUSES OF INCREASED INTRACRANIAL PRESSURE
INTRACRANIAL MASS LESION
CEREBRAL EDEMA
DECREASED CSF ABSORPTION
OBSTRUCTIVE HYDROCEPHALUS
OBSTRUCTION OF VENOUS OUTFLOW
MALIGNANT HYPERTENSION
IDIOPATHIC INTRACRANIAL HYPERTENSION

EMERGENCY ROOM
MRI LOOKING FOR
INTRACRANIAL MASS LESION
OBSTRUCTION OF VENOUS OUTFLOW
OBSTRUCTIVE HYDROCEPHALUS
DECREASED CSF ABSORPTION
INCREASED CEREBROSPINAL FLUID
* CT IF MRI DELAYED OR CONTRAINDICATED*

MRV LOOKING FOR
VENOUS OBSTRUCTION IN DURAL SINUSES AND NECK
IF NORMAL (NOT BEFORE)
LUMBAR PUNCTURE
OPENING PRESSURE AND ANALYSIS
IF > 25 CM (250 MM H2O) THAT’S ELEVATED, 20-25 IS EQUIVOCAL

DIAGNOSIS
IIH / PSEUDOTUMOR CEREBRI
INCIDENCE
1-2/100,000
HIGHER IN WOMEN AGE 15-44
4-8X MORE LIKELY THAN MEN
MEDIAN AGE IS 30
OTHERS?

RISK FACTORS
WOMEN OF CHILDBEARING AGE WOMEN
OVERWEIGHT
>10% OVER IDEAL BODY WEIGHT = 13X MORE LIKELY TO DEVELOP IIH
> 20% = 19X MORE LIKELY

MEDICATIONS
BCPS, TETRACYCLINES, HORMONE REPLACEMENT, ACCUTANE, VITAMIN A
HGH, ORAL STEROIDS, SYNTHROID, ISONIAZID, LITHIUM, NITROGLYCERIN

FAMILY HISTORY?

SYMPTOMS
HEADACHE 84-92%
LATERALIZED, THROBBING, PULSATILE
WORSE WITH POSTURE CHANGES
TRANSIENT VISUAL OBSCURATIONS 68-72%
INTRACRANIAL NOISES (PULSATILE TINNITUS) 52-60%
PHOTOPSIA 48-54%
BACK PAIN 55%
RETROBULBAR PAIN 44%
DIPLOPIA 18-38%
SUSTAINED VISUAL LOSS 26-32%

SIGNS
SWOLLEN OPTIC NERVES
  BILATERAL, ASYMMETRIC OR UNILATERAL
VISUAL LOSS
  VA < 20/20 10-29%
VF LOSS
  TYPICALLY PERIPHERAL, CENTRAL LATE
6th NERVE Palsy
  UNILATERAL OR BILATERAL
OTHER CN INVOLVEMENT
  I, III, IV, V, VII, VIII

OTHER SIGNS?
POSSIBLY
  FLATTENING OF THE POSTERIOR SCLERA
  TORTUOSITY OF THE OPTIC NERVE SHEATH
  EMPTY SELLA SYNDROME
  STENOSIS OF THE TRANSVERSE VENOUS SINUSES

MECHANISM
NOT REALLY KNOWN
THEORIES
  INCREASED PRODUCTION OF CSF
  INCREASED BLOOD FLOW TO THE BRAIN OR INCREASE IN THE BRAIN TISSUE
  RESTRICTED VENOUS DRAINAGE FROM BRAIN

IIH TREATMENT OPTIONS
MEDICAL
  WEIGHT LOSS
  SERIAL LUMBAR PUNCTURES
  CORTICOSTEROIDS
  DIURETICS
    ACETAZOLAMIDE
    FUROSEMIDE
SURGICAL
  LUMBOPERITONEAL SHUNT
  VENTRICULOPERITONEAL SHUNT
  VENTRICULOATRIAL SHUNT
  OPTIC NERVE SHEATH FENESTRATION
  SHOULD ONLY BE PURSUED IF
    SEVERE VISION LOSS
    PROGRESSIVE VISION LOSS
    INTRACTABLE HEADACHES

...BACK TO THE PATIENT

CHIARI MALFORMATIONS

EPIDEMIOLOGY
TRUE FREQUENCY IS UNKNOWN
MORE SINCE 1985 DUE TO MRI USAGE
TYPE I PREVALENCE 0.1 TO 0.5%

ETIOLOGY
USUALLY CONGENITAL
  NOT NECESSARILY INHERITED, MUTATION OR DELETION, EXOGENOUS TERATOGEN
EXACT CAUSE IS UNKNOWN BUT THERE ARE THEORIES
  SMALL POSTERIOR FOSSA / CROWDING THEORY
  MOLECULAR GENETIC THEORY
  HYDRODYNAMIC PULSION THEORY
  OLIGO-CEREBROSPINAL FLUID THEORY

HOW DIAGNOSED
OPISTHION-BAISON LINE
  LOWER LIMIT OF POSTERIOR FOSSA
HEALTHY ADULTS
  CEREBELLAR TONSILS RARELY 3MM BELOW FORMAMEN MAGNUM
TYPE I
  TONSILS 5MM BELOW
CHIARI TYPE I

SYMPTOMS
ASYMPTOMATIC / RESOLVES
OR
HEADACHE, NECK PAIN
BALANCE PROBLEMS
DIZZINESS
MUSCLE WEAKNESS
NUMBNESS
DIFFICULTY SWALLOWING
RINGING / BUZZING IN EARS
HEARING LOSS
VOMITING
INSOMNIA, DEPRESSION

OCULAR PROBLEMS
DIPOPIA
PHOTOPHOBIA
BLURRED VISION
NYSTAGMUS
PAIN BEHIND THE EYES
MAY WORSEN WITH
PHYSICAL ACTIVITY
COUGHING
LAUGHING
SNEEZING

COMPLICATIONS
HERNIATED TISSUES COMPRESS THE BRAINSTEM, BLOCKS FLOW OF NORMAL CSF
SYRINGOMYELIA
BUILDUP OF FLUID IN SPINAL CORD
SYRINX IS A FLUID FILLED CYST
LOSS OF MUSCLE MASS, MUSCLE WEAKNESS, NUMBNESS, SCOLIOSIS, ATAXIA, CHRONIC PAIN
HYDROCEPHALUS
BUILDUP OF FLUID IN THE BRAIN

MANAGEMENT
ASYMPTOMATIC
MONITORING AND SURVEILLANCE WITH MRI
6 MOS THEN YEARLY
SYMPTOMATIC / LARGE / PROGRESSIVE SYRINX
SURGERY
POSTERIOR FOSSA DECOMPRESSION
SYMPTOMS IMPROVE IN 80-95%,
20% COMPLICATION RATE
SPINAL LAMENECTOMY
SHUNTING PROCEDURES

DID THE LP DO THIS?

IIH AND CHIARI TYPE I
RELATIONSHIP IS POORLY UNDERSTOOD
ELEVATED INTRACRANIAL PRESSURE IN IIH MAY CAUSE CEREBELLAR TONSILS TO HERNIATE THROUGH FORAMEN MAGNUM
OR
PATIENTS WITH CHIARI TYPE I HAVE ABNORMAL CSF DYNAMICS WHICH MAY PREDISPOSE PATIENTS TO ELEVATED
INTRACRANIAL PRESSURE AND IIH

...BACK TO THE PATIENT

CASE 2
45 / W / F
CC: loss of vision and floaters in left eye hours after visiting a theme park
OCULAR HX:
LEE 1yr, lattice degeneration ou, ERM od, no h/o surgery
MED HX: unremarkable OU
MEDS: none
FAM HX: unremarkable OU
SOCIAL HX: -etoh -tobacco

BVA:
cc 20/20 -650DS
cc 20/200, ph NI -675DS
SLIT LAMP: unremarkable OU
IOP: 15/16 @ 815a
DFE:
OD lattice degeneration, a few holes, no tears/detachments
OS see photo, lattice degeneration, a few holes, vitreous hemorrhage
DIFFERENTIAL DIAGNOSIS
VITREOUS DETACHMENT
RETINAL HOLE
RETINAL TEAR
RETINAL DETACHMENT

RETINAL DETACHMENT
OCULAR EMERGENCY
SIGHT THREATENING
INCIDENCE 1 IN 10,000
RESULT OF SEPARATION OF NEUROSENSORY RETINA FROM THE RPE

TYPES
EXUDATIVE
TRACTIONAL
RHEGMATOUS
BREACH IN NEUROSENSORY RETINA
SEEPAGE OF FLUID INTO SUBRETINAL SPACE

RISK FACTORS
LATTICE DEGENERATION
PERIPHERAL RETINAL BREAKS
PATHOLOGIC MYOPIA
HISTORY OF OCULAR SURGERY
TRAUMA
PREVIOUS RETINAL DETACHMENT
FAMILY HISTORY
OTHER
CONTACT SPORTS, HIGH RISK ACTIVITIES

HIGH RISK ACTIVITY?
AMUSEMENT PARK ACCIDENTS
MOST COMMON
HEAD, NECK, BACK INJURIES
DEATH
STROKE
TRAUMATIC BRAIN INJURY
BRAIN ANEURYSMS
LACERATIONS, BROKEN BONES, TORN LIGAMENTS
DROWNING

CAUSES
MECHANICAL FAILURE OF RIDE
IMPROPER OPERATION OF RIDE
PASSENGER MISUSE OR FAILURE TO FOLLOW INSTRUCTIONS
INHERENT NATURE OF RIDE

PREVIOUSLY REPORTED ROLLER COASTER SEQUELAE
NEUROLOGIC
SUBDURAL HEMATOMA
SUBARACHNOID HEMORRHAGE
INTRAPARENCHYMATOUS HEMORRHAGE
CERIVOCEPHALIC ARTERIAL DISSECTION
CAROTID ARTERY THROMBOSIS
POSTTRAUMATIC MIGRAINE

OCULAR
BLURRED VISION
RETINAL ARTERY OCCLUSION
RETINAL / MACULAR HEMORRHAGE
LENS DISLOCATION
SUBLUXATION IN MARFAN’S LEAD TO GLAUCOMA
INTRAOCULAR LENS DISLOCATION
REPOSITIONING AFTER ROLLER COASTER

MACULAR HEMORRHAGE
THEORETICAL CAUSE
HIGH HYDROSTATIC PRESSURE FROM G FORCES
MAY DISRUPT VASCULAR FLOW TO HEAD AND EYE
SUDDEN INCREASED CEREBRAL PRESSURE
LEADS TO INCREASED RETINAL VENOUS PRESSURE

RETINAL ARTERY OCCLUSION
THEORETICAL CAUSE
COMPLEX AND ABRUPT CHANGES IN MOTION MAY LEAD TO HYPEREXTENSION, HYPERFLEXION OF NECK AND CAUSE INTIMAL TEARS OF CAROTID OR VERTEBRAL
RETINAL ARTERY OCCLUSION NOT FROM EMBOLUS BUT FROM HYPOPERFUSION

ROLLER COASTER RD MECHANISM
RAPID ACCELERATION AND DECELERATION
PRESSURE CHANGES
WHIPLASH LIKE HEAD MOVEMENTS CAUSE SHEAR STRESS THAT MAY EXCEED STRENGTH OF RETINAL ADHESIONS
SHEARING OF NEURAL PARENCHYMA AND VASCULATURE

RD TREATMENT OPTIONS
1950s SCLERAL BUCKLE
1970s VITRECTOMY
1986 PNEUMATIC RETINOPEXY

HOW TO TREAT RHEGMATOGENOUS RD?
2013 METANALYSIS
7 REPORTS REVIEWED
836 EYES PPV
670 EYES SB

RESULTS
PHAKIC
NO SIGNIFICANT DIFFERENCES IN REATTACHMENT
SB EYES HAD BETTER VISION (LESS CATARACTS)
PSEUDOPHAKIC
NO DIFFERENCES IN REATTACHMENT VS BCVA AT 6 MONTHS

HOW TO PREVENT COASTER PROBLEMS
IMPROVE SAFETY
RIDER EDUCATION
BETTER RESTRAINTS
SHORTENING THE RIDE
REDUCES TIME AN INJURED PERSON SUFFERS

WHAT SHOULD OPTOMETRISTS DO?
EDUCATE AT RISK PATIENTS
ADVISE POTENTIALLY PREDISPOSED PATIENTS ABOUT THEIR OCULAR RISKS OF HIGH RISK ACTIVITIES (NOT JUST ROLLERCOASTERS)
HAVE EYES CHECKED IMMEDIATELY IF
FLASHES OF LIGHT
FLOATING SPOTS
ANYTHING APPEARING LIKE A CURTAIN IN FRONT OF THE VISION
LOSS OF PERIPHERAL VISION

...BACK TO THE PATIENT

CASE 3
89 / W / M
CC: distance blur x 1yr, no near problems, no comfort problems
Oc Hx:
LEE 2yrs: trauma os 1944, h/o strab surgery, pseudophakic with ac/iol OS
Oc Meds: none
Med Hx: +HTN, +Heart (A fib)
Fam Hx: unremarkable
Soc Hx: -etoh, -tobacco
BVA: cc 20/25-2, cc 20/40+1
Prelim: FROM, FFTC OD OS, surgical pupil OS, no APD
SLE: see photos
IOP: 20/20 @ 1259p
DFE: see photos

DIFFERENTIAL DIAGNOSIS
MEDICATIONS
ANTIMALARIALS, MINOCYCLINE, AMIDOARONE, CHLORPROMAZINE
HEMOCHROMATOSIS
POLYCYPHEMA VERA
ADDISON’S DISEASE
MELANOSIS FROM METASTATIC MELANOMA
HEAVY METALS
GOLD, MERCURY, BISMUTH
CYANOSIS
CENTRAL OR PERIPHERAL
BLUE NEVUS
OCHRONOSIS
ARYRIA

ARGYRIA
SIGNS
HYPERPIGMENTATION IN SUN-EXPOSED AREAS OF SKIN
FOREHEAD, NOSE, HANDS
ENTIRE SKIN MAY BE SLATE BLUE-GRAY COLOR
SCLERA, NAIL BEDS, MUCOUS MEMBRANES MAY BE HYPERPIGMENTED
VISCERA SHOW BLUE DISCOLORATION
SPLEEN, LIVER, GUT
CAUSE
PROLONGED CONTACT WITH OR INGESTION OF SILVER

RISK FACTORS
RISK
INDIVIDUAL VARIABILITY
DUE TO LENGTH OF EXPOSURE AND TOTAL DOSE NEEDED
INCREASED RISK
WORKERS
SILVER MINERS / REFINERS
SILVERWARE / GLASS / CHINA MANUFACTURING
PHOTOGRAPHIC PROCESSING

INCREASED RISK
SILVER SALT IRRIGATION
URETHRAL / NASAL MUCOUS MEMBRANES
EYE DROPS
WOUND DRESSING
SMOKING REMEDY
SURGICAL AND DENTAL PROCEDURES
ACUPUNCTURE
EARRING SITES
DIETARY SUPPLEMENTS
COLLOIDAL SILVER

WHAT IS COLLOIDAL SILVER?
ONCE AVAILABLE IN OTC DRUG PRODUCTS
1999 FDA RULED NOT SAFE AND EFFECTIVE
STILL BEING SOLD
AS HOMEOPATHIC REMEDIES AND DIETARY SUPPLEMENTS
MAKE YOUR OWN

COLLOIDAL SILVER
SERIOUS COMPLICATIONS
GI PROBLEMS
TISSUE WASTING
UREMIA
ALBUMINURIA
FATTY DEGENERATION
LIVER, KIDNEY, HEART
THROMBOCYTOPENIA
FLUIDITY OF THE BLOOD
CHRONIC BRONCHITIS
LOSS OF COORDINATION
DECREASED NIGHT VISION
TASTE DISTURBANCE
VESTIBULAR IMPAIRMENT
GRAND MAL SEIZURES
PARALYSIS OF RESPIRATORY SYSTEM
DEATH

OCULAR COMPLICATIONS
CONJUNCTIVAL ARGYRIA
BLACK TEARS RARELY OCCUR
SCLERAL HYPERPIGMENTATION
DECREASED NIGHT VISION

HOW DOES IT HAPPEN
COLORLESS SILVER
REDUCED BY SUNLIGHT TO BROWNISH-BLACK SILVER SULPHIDES AND SELENIDE WITHIN TISSUE
SILVER
STIMULATES TYROSINASES ACTIVITY OF MELANOCYTES TO PRODUCE MORE MELANIN
THEREFORE
MOST PROMINENT IN SUN-EXPOSED AREAS

TREATMENT OPTIONS
DEPIGMENTATION PREPARATIONS
SOME REPORT 5% HYDROQUININE MAY WORK
LASER
TO REMOVE PIGMENTED SKIN / CELLS
CHELATION UNSUCCESSFUL
SUNSCREEN MAY PREVENT WORSENING

PROGNOSIS
PERMANENT, IRREVERSIBLE METALLIC TINGE
...BACK TO THE PATIENT

CASE 4

65/ W / M
CC: no changes in vision, blue top drop stings, dry mouth from purple
Oc Hx: LEE 4 mos, dx/tx Glaucoma x 13yrs, highest IOP off meds 26/27, h/o Lasik OU, dry eyes OU, DM without Ret OU, Early Cataracts OU
Oc Meds: Latanoprost qhs ou, cosopt q12h ou, brimonidine 0.2 q12h ou
Med Hx: +DM, +HTN, +Chol
Fam Hx: unremarkable
Soc Hx: -ethoh, -tobacco

BVA:
cc 20/25-2, cc 20/25-1+2
Prelim: FROM, FTFC OD OS, No APD
SLE: see photos
IOP: 14/14@ 625a, pachym: 547/548
GONIO: ou open to CBB 360, no PAS/recess/nv, trace pigment
DFE: see photos

WGA CONSENSUS STATEMENTS
Progression of Glaucoma, World Glaucoma Association, 2011 Kugler Publications

ONCE THE DIAGNOSIS OF GLAUCOMA HAS BEEN MADE THE MOST IMPORTANT REMAINING QUESTION IS…
WHETHER THE DISEASE IS STABLE AND THE THERAPY / COMPLIANCE ARE SUFFICIENT
OR
WHETHER THE DISEASE IS PROGRESSIVE AND THE THERAPY IN RELATION TO LIFE EXPECTANCY HAS TO BE INTENSIFIED

HAS THIS PATIENT PROGRESSED?
WGA CONSENSUS STATEMENTS
Progression of Glaucoma, World Glaucoma Association, 2011 Kugler Publications
CURRENTLY, NO SPECIFIC TEST CAN BE REGARDED AS THE PERFECT STANDARD FOR DETERMINATION OF PROGRESSION
BOTH ONH STRUCTURE AND FUNCTION SHOULD BE EVALUATED FOR DETECTION OF PROGRESSION

YOUR PATIENT IS GETTING WORSE. WHAT ARE YOU GOING TO DO NOW?
IOP LOWERING OPTIONS
LASER
SLT
SURGERY
CATARACT SURGERY
MIGS
ISTENT
TRABECULECTOMY
TUBE

LASER TRABECULOPLASTY
ALT OR SLT
MECHANISM IS NOT CLEAR
MECHANICAL
CELLULAR
BIOCHEMICAL

PROS
GLT: ALT AS GOOD AS TIMOLOL
EFFECTIVE

CONS
IOP SPIKE, A/C REACTION
SLT NEEDS DEEP ANGLE, PIGMENTED TM
NOT EFFECTIVE ON EVERYONE
MAY WEAR OFF

SLT IN CLINICAL TRIALS
SLT VS ALT
SIMILAR EFFECT
EFFICACY
20-30% IOP REDUCTION

UNCONTROLLED OAG MAX MEDS, PRIOR FAILED ALT
70% DECREASED 3 mmHg
24% SPIKED ≥ 5 mmHg

SLT MED STUDY
Katz LJ, et al. 2011
SLT (58 EYES)
100 APPLICATIONS, 360 DEGREES (REPEATED IF ABOVE TARGET)
26.4% IOP REDUCTION

MEDICATION (36 EYES)
MED CHANGED IF ABOVE TARGET
27.8% IOP REDUCTION
ADDITIONAL IOP TREATMENT NEEDED IN MED GROUP
CATARACT SURGERY
32 STUDIES REVIEWED
POAG, PXG, PACG
POAG / NTG (ON 1-2 MEDS)
REDUCES IOP 13%, MEDS BY 12%
COMPLICATIONS
17.5% CHANCE OF IOP SPIKE
UNSURE ABOUT PATIENTS WITH UNCONTROLLED POAG
OHTS RESULTS
17% IOP REDUCTION 3YRS AFTER
OTHER STUDIES
2-4 mmHg IOP REDUCTION

CATARACT SURGERY
BALTIMORE VAMC 2006-2008
RETROSPECTIVE STUDY, 115 PTS
RESULTS
HIGHER PREOPERATIVE IOP ASSOCIATED WITH GREATER IOP LOWERING
PHACO TIME ASSOCIATED WITH IOP REDUCTION
MECHANISM
NOT WELL UNDERSTOOD
THEORIES
HYPOSECRETION OF AQUEOUS DUE TO FREE RADICAL
IMPROVED TM OUTFLOW DUE TO DEEPENING ANTERIOR CHAMBER
STRESS REMODELING FROM THE ULTRASONIC VIBRATIONS ACTIVATES CYTOKINE PATHWAY

MIGS MINIMALLY INVASIVE GLAUCOMA SURGERY
FROM THE AMERICAN GLAUCOMA SOCIETY
REDUCE IOP BY IMPROVING OUTFLOW
APPROACHED FROM
INSIDE (AB INTERNO) or OUTSIDE THE EYE (AB EXTERNO)
LIMITED SURGICAL MANIPULATION OF THE SCLERA
LITTLE MANIPULATION OF THE CONJUNCTIVA
DOES NOT PRECLUDE POSSIBILITY OF TRADITIONAL SURGERY

BENEFITS
HIGHER SAFETY PROFILE VS TRABECULECTOMY
SHORTER SURGERY TIME
FEWER COMPLICATIONS
FASTER RECOVERY TIME
DECREASE IOP AND/OR PATIENT’S NEED FOR MEDICATION

MIGS
WHO IS A CANDIDATE?
MILD-MODERATE OPEN-ANGLE GLAUCOMA
PSEUODEXFOLIATION GLAUCOMA
PIGMENTARY GLAUCOMA
UNCONTROLLED GLAUCOMA ON MAXIMUM TREATMENT OR BARRIERS TO PREVENT ADEQUATE DOSING
>18 CATARACT?

CONTRAINDICATIONS
ANGLE CLOSURE
MODERATE-ADVANCED SECONDARY GLAUCOMA
SEVERELY UNCONTROLLED GLAUCOMA
PREVIOUS GLAUCOMA SURGERY
PREVIOUS REFRACTIVE PROCEDURES
MONOCULAR PATIENTS

iSTENT
FDA APPROVED 2012
MECHANISM
BYPASSES THE TRABECULAR MESHWORK
PROCEDURE
120 um DIAMETER TITANIUM IMPLANT
PRELOADED APPLICATOR
INSERTED INTO SCHLEMM’S CANAL TO ALLOW AQUEOUS TO DRAIN DIRECTLY FROM A/C
2ND / 3RD GENERATIONS IN TRIALS

iSTENT WITH CATARACT SURGERY
PROS
NO BLEB THEREFORE PRESERVES CONJ FOR TRAB
NO IRIDECTOMY NEEDED
CONS
- IOP does not reach single digits (limited by EVP)
- Surgical complications
- Touching the iris
- Failure to implant
- Endothelium contact
- Malposition
- $1000 apiece

STUDY RESULTS
FDA report
116 ISTENT with cataract surgery vs 123 cataract surgery alone
Results
- 68% combined IOP <21
- 50% cataract only

ISTENT ALONE
2015 ARMENIAN STUDY
119 patients followed 18 months
- 38 (1 stent) vs 41 (2 stents) vs 40 (3 stents)
- IOP 15.9 vs 14.1 vs 12.2
- Unmedicated 64.9% vs 85.4% vs 92.1%
Health insurance generally only reimburses for one stent
FDA has approved stent in conjunction with cataract surgery but not as stand-alone procedure

FUTURE MIGS?

WHAT’S NOT KNOWN?
Longterm safety or effectiveness data

PATIENTS IN TROUBLE
- Patient presenting with severe damage
- Based on optic nerve, nerve fiber layer, visual field
- Patient who is young
- Patient who is African American
- Patient who is an IOP spiker
- Patient with a thinner cornea
- Patient with family member (sibling) blind from glaucoma
- Patient blind in one eye from glaucoma
- Patient who is non-compliant
- Patient who shows progression despite treatment

TRABECULECTOMY
First described in 1968
Mechanism
- Creates a drainage channel (a hole in the eye) from the anterior chamber to external surface of the eye under the conjunctiva
- Aqueous seeps into bleb which is slowly absorbed

TUBE / SHUNT (GLAUCOMA DRAINAGE DEVICE)
Mechanism
- Placement of tube / valve to facilitate aqueous outflow
Types
- Little resistance to aqueous (Baerveldt, Molteno)
- Unidirectional valve (Ahmed, Krupin)

TUBE vs TRAB (TVT) STUDY
Prospective study (17 centers, 212 eyes of 212 patients)
107 in tube group, 105 in trab / MMC group
Patients
- Uncontrolled glaucoma, s/p CE/IOL and / or failed TRAB
5 year results (Gedde SJ, et al. March 3, 2011 AGS Meeting, California)
- IOP: 14.2 +/- 6.3 mmHg TUBE vs 12.8 +/- 5.8 mmHg TRAB
- Probability of failure: 26% TUBE vs 45% TRAB (P = 0.002)
- Late complications: 34% TUBE vs 37% TRAB (P = 0.67)
- Endophthalmitis / blebitis: TUBE 0 vs TRAB 4.8%

CONCLUSIONS
- Tube shunts are a good alternative in those who have had prior surgery
- Total costs of tube were higher than trab
- Was not a study on “fresh” patients

TUBE vs TRAB
Fresh patients

BACK TO THE PATIENT...

CASE 5
55 / B / M
CC: no vision complaints, would like new NVO, some dryness
OCULAR HX:
LEE 1yr, trauma to left eye while fishing
MED HX: +DM, +Chol
FAM HX: unremarkable OU
SOCIAL HX: -etoh -tobacco
BVA: sc 20/20-1, sc hm at 2 feet
PRELIMS: 3/5 bright, 5/5 dim, 3+/0, ROUND/IRREGULAR
NO APD BY REVERSE
SLIT LAMP: OD unremarkable, OS See photo
IOP: 20/18 @ 945a
DFE: OD unremarkable, OS no view

OCULAR TRAUMA UNITED STATES
2.5 MILLION OCULAR INJURIES PER YEAR
40,000-60,000 CASES OF BLINDNESS PER YEAR
25% RELATED TO SPORTS / RECREATION
~100,000 SPORTS RELATED EYE INJURIES / YR WARRANT VISIT TO ER OR DOCTOR’S OFFICE

FISHING-RELATED OCULAR TRAUMA STUDY
CAUSES
FISHHOOKS, WEIGHTS, LURES, RODS
DEMOGRAPHICS
6-68 YEARS OLD, 39% < 19 YO
79% MALE
OTHER DATA
BYSTANDERS IMPACTED IN 24.48%
2% INVOLVED ALCOHOL
PROGNOSIS
21% < 20/200
57% CLOSED GLOBE > 20/40
11% OPEN GLOBE > 20/40

WHAT HAPPENED HERE?

PREVIOUSLY
CORNEAL PENETRATION / LACERATION AND ANTERIOR CAPSULAR DISRUPTION
LENS TRAUMA
POSSIBILITIES
BLUNT TRAUMA
PENETRATING TRAUMA
CATARACT FORMATION
TRAUMA TO LENS CAPSULE OR INJURY TO LENS PUMP
INCREASED PERMEABILITY
INFLUX OF SODIUM AND WATER FROM AQUEOUS
INTRACELLULAR / EXTRACELLULAR SWELLING OF EPITHELIAL CELLS
LENSES PROTEOLYSIS, AGGREGATION, CONFORMATIONAL CHANGES
LENS OPACIFICATION

TRAUMA CONSIDERATIONS
ZONULAR DIALYSIS
POSTERIOR CAPSULAR TEAR
IRIS TRAUMA
CORNEA TRAUMA
POOR VISUALIZATION FROM MEDIA HAZE

SURGICAL CONSIDERATIONS
SYNECHIAE
NONDILATING PUPIL
INTRAOPERATIVE DIALYSIS
NUCLEUS DROP
VITREOUS LOSS
HYPHEMA

ZONULAR DEHISCENCE
WHAT CAUSES IT
INHERITED OR ACQUIRED
HIGH MYOPIA
PSEUDEXFOLIATION, RETINITIS PIGMENTOSA, MARFAN SYNDROME, HOMOCYSTINURIA
BLUNT AND PENETRATING TRAUMA
S/P MULTIPLE VITRECTOMIES, SILICONE OIL
IDIOPATHIC

PREVENTION
KNOW YOUR PATIENTS
DEMOGRAPHIC
HOBBIES
RECOMMEND
PROTECTIVE LENSES
POLYCAR OR TRIVEX
REDuces RISK of INJURY BY 90%
OTHER: UV, POLARIZED
IF INJURY OCCURS...
BASIC FIRST-AID
DON'T REMOVE FISH HOOK

CASE 6
45 / W / F
CC: floaters os, no ocular comfort problems, no vision problems
OCULAR HX: LEE few years ago, no surgery, no trauma/cataracts/glaucoma
MED HX: unremarkable
MEDS: none
FAM HX: unremarkable
SOCIAL HX: unremarkable
BVA: sc 20/20, sc 20/20
PRELIMS
FTFC OD OS, FROM
NORMAL PUPILS, NO APD
SLIT LAMP: unremarkable
IOP: 12/13 @ 719a
DRE: see photos

DIFFERENTIAL DIAGNOSIS
CHOROIDAL NEVUS
RPE HYPERPLASIA
CONGENITAL HYPERTROPHY OF THE RPE
MELANOCYTOMA OF CHOROID
METASTATIC CARCINOMA
MALIGNANT MELANOMA
HAMARTOMA
ADENOMA
ADENOCARCINOMA

RPE HYPERTROPHY APPEARANCE
FLAT, ROUND OR OVAL
WELL DERMARcATED OR SCALLOPED MARGINS
88% PIGMENTED
PRESENT AT BIRTH
½ BROWN / 1/3 BLACK / GRAY / RARELY NO PIGMENT
½ HAVE LACUNAE (ATROPHIED WINDOW DEFECTS)
PIGMENTED OR NONPIGMENTED HALO
LACK OF LIPOFUSCIN
PROGRESSIVE LOSS OF OVERLYING PHOTORECEPTORS (SEEN ON OCT)

RPE HYPERTROPHY BACKGROUND
0.3-40% PREVALENCE
FEMALES > MALES 2:1
2-3 SUBTYPES
SOLITARY CHRPE
GROUPED (BEAR TRACKS) AND / OR MULTIPLE

RPE HYPERTROPHY PATHOLOGY
FOCAL AREA OF TALLER RPE CELLS
MORE DENSELY PACKED WITH MELanosomes
MELanosomes ARE LARGER AND SPHERICAL COMPARED TO NORMAL RPE (SMALLER, ELLIPTICAL)
INTENSELY HYPERTROPHIED RPE CELLS
PROGRESSION
75-80% SLOWLY GROW IN DIAMETER
MAY DEVELOP AN ELEVATED NODULE
REPRESENTS ADENOMA OR ADENOCARCINOMA

BEAR TRACKS
A CHRPE VARIATION
CONGENITAL
APPEARANCE
CLUSTER OR GROUPS OF LESIONS
SMALL, SHARPLY CIRCUMSCRIBED
DIFFERENT THAN CHRPE?
MORE ELLIPTICAL

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)
POTENTIAL SIGN
MULTIPLE CHRPE LESIONS (BEAR TRACKS) IN BOTH EYES
70-75% OF FAP PATIENTS HAVE THESE LESIONS
DIFFERENT THAN A CHRPE?
SOME THINK SO
PIGMENTED OCULAR FUNDUS LESIONS OF FAP (POFLs)
DEVELOPMENT OF NEW BILATERAL LESIONS
STRONGLY ASSOCIATED WITH FAP
SENSITIVITY 65-84%, SPECIFICITY >94%

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)
AUTOSOMAL DOMINANT
CHARACTERISTICS
ADENOMATOUS POLYPOSIS OF LARGE AND SMALL INTESTINE
HUNDREDS TO THOUSANDS OF POLYPS
HAMARTOMAS OF SKELETON
VARIOUS SOFT TISSUE TUMEFACIONS

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)
VARIANTS
ATTENUATED FAP
MILDERS FORM, LATER AGE / CANCER
GARDNER SYNDROME
CHRPE LIKE LESIONS IN 66-75%
COLONIC ADENOMATOUS POLYPOSIS, BONE CYSTS, HAMARTOMAS, SOFT TISSUE TUMORS (DESMOID TUMORS)
TURCOT SYNDROME
RARE
NEUROEPITHELIAL TUMORS OF CNS
MEDULLOBLASTOMAS AND/OR ASTROCYTOMAS

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)
FATALITY RATE IS 100% IF UNTREATED BY AGE 50
1% OF ALL COLORECTAL CANCERS
5% OF FAMILIAL COLORECTAL CANCER
PREVALENCE
1 IN 11300 TO 1 IN 37600
MANIFESTS BY LATE TEENS TO TWENTIES
MALE = FEMALE
NO SYMPTOMS UNTIL POLYPS ARE LARGE

SYMPTOMS
RECTAL BLEEDING
IRON-DEFICIENCY ANEMIA
GI COMPLAINTS
CHANGE IN BOWEL HABITS
CONSTIPATION
DIARRHEA
ABDOMINAL PAIN

MULTIPLE CHRPE AND / OR FAMILIAL ADENOMATOUS POLYPOSIS (FAP) MANAGEMENT
IF 4 OR MORE CHRPE LESIONS
PROBE PATIENT HISTORY AND / OR FAMILY HISTORY
ABOUT POLYPS, COLON CANCER, COLORECTAL SURGERY
REFER TO GASTROENTEROLOGIST / GENETICIST
CONSIDER COLONOSCOPY
SURGICAL REMOVAL OF POLYPS
COLON SURGERY
CANCER TREATMENT
GENETIC TESTING
ISOLATED TO ADENOMATOUS POLYPOSIS COLI (APC) GENE ON CHROMOSOME 5q21-q22

...BACK TO OUR PATIENT

CASE 7
51 / W / M
CC:
establishing VA care, decreased vision OU x many years
OC Hx:
LEE 3 weeks privately
+legally blind, +h/o bleeding OU, h/o laser OU, h/o injections OU (last avastin 3 wks ago OS)
Med Hx: +chol, +arthritus
Fam Hx: -dm, -glaucoma, -blind
Soc Hx: -eth, -tobacco
BVA CF @ 4f, CF @ 2 ft
Prelims FTFC OD OS, FROM, No APD
Facial Amsler: central loss OU
SLEU/nremarkable
IOP 16/16 @ 1259p
DFE OU trace NS
CNVM CAUSES
AGE RELATED MACULAR DEGENERATION
MYOPIC DEGENERATION
OCULAR HISTOPLASMOSIS SYNDROME
CHOROIDAL RUPTURE
OPTIC NERVE DRUSEN
ANGIOID STreakS
CHOROIDAL MELANOMA
MULTIFOCAL CHOROIDITIS
CHOROIDAL OSTEOMA
HISTORY OF LASER
OTHERS
IDIOPATHIC

ANGIOID STreakS
FIRST REPORTED IN 1889 BY DOYNE
DESCRIPTION
BILATERAL, NARROW, IRREGULAR LINES DEEP TO RETINA IN RADIATING PATTERN FROM ONH ONE TO MULTIPLE ORANGE, RED, GREY OR BROWN
ETIOLOGY
BRUCH’S MEMBRANE BECOMES CALCIFIC AND THICKENED CRACK-LIKE BREAKS DEVELOP CAN DEVELOP SPONTANEOUSLY OR FROM BLUNT TRAUMA

DIFFERENTIAL DIAGNOSIS
LACQUER CRACKS FROM PATHOLOGIC MYOPIA
CHOROIDAL RUPTURE
OTHERS

SYMPTOMS
ASYMPTOMATIC
VISUAL IMPAIRMENT
IF CNVM OR SUBRETIINAL HEMORRHAGE

SYSTEMIC ASSOCIATIONS
PSEUDOXANTHOMA ELASTICUM (85% HAVE ANGIOID STreakS)
EHLErS-DANLOs SYNDROME
PAGET’S DISEASE
SICKLE CELL DISEASE
IDIOPATHIC
MARFAN’S SYNDROME
OTHER:
ABETALIPOPROTEINEMIA, ACROMEGALY, DIABETES, FAcIAL ANGIOMATOSIS, HYPERCALCINOSIS, HEMOCHROMATOSIS, HEMOLYTIC ANEMIA, HEREDITARY SPHEROCYTOSIS, HYPERPHOSPHATEMIA, LEAD POISONING, MYOPIA, NEUROFIBROMATOSIS, SENILE ELASTOSIS, STURGE-WEBER SYNDROME, TALASSEMIA, TUBEROUS SCLEROSIS

WORK-UP
COMPLETE PHYSICAL WITH REVIEW OF SYSTEMS
PSEUDOXANTHOMA ELASTICUM
SKIN BIOPSY
EHLErS-DANLOs SYNDROME
LOOK FOR LOOSE JOINTS, FRAGILE / STRETCHY SKIN, FAMILY HISTORY
GENETIC TESTING
PAGET’S DISEASE
SERUM ALKALINE PHOSPHATASE, CALCIUM AND PHOSPHATE
X-RAY
BONE SCAN
SICKLE CELL
HEMOLOBIN ELECTROPHORESIS

PSEUDOXANTHOMA ELASTICUM
BACKGROUND
FIRST DESCRIPTION 1896
CAUSE
GENETIC DISEASE
AUTOSOMAL RECESSIVE MUTATIONS IN ABCC6 GENE ON CHROMOSOME 16
PREVALENCE
1:25,000
FEMALES 2X > MALES
RESULT
FRAGMENTATION AND MINERALIZATION OF ELASTIC FIBERS

MOST COMMON PROBLEMS
SKIN
EYES
BLOOD VESSELS
PREMATURE ATHEROSCLEROSIS
SKIN SIGNS
- SMALL, YELLOWISH PAPULAR LESIONS
- CUTANEOUS LAXITY OF NECK, AXILLA, GROIN, INSIDE OF ELBOWS / KNEES
- REDUNDANT AND LAX SKIN
- DIAGONAL GROOVES OF CHIN

OCULAR SIGNS
- ANGIOID STREAKS
- CNVM
- PEAU D’ORANGE
  - “ORANGE PEEL SKIN OR SKIN OF AN ORANGE”
  - Dimples
- ONH DRUSEN
- COMET TAIL LESIONS
- CHORIORETINAL ATROPHY
- SALMON SPOTS

DIAGNOSIS
- SKIN TESTING

TREATMENT
- NONE
- EDUCATION ABOUT POSSIBLE COMPLICATIONS
- PROPHYLACTIC MEASURES
- LIFESTYLE ADJUSTMENTS
- GENETIC COUNSELING
- SKIN
  - PLASTIC SURGERY FOR EXCESSIVE SKIN
- CARDIOVASCULAR
  - TREAT LIKE NORMAL, AVOID BLOOD THINNERS / ANTI-INFLAMMATORIES

CNVM FROM ANGIOID STREAKS
TREATMENT
- LASER
  - RECURRENCE IN 77%
- PDT
  - RETREATMENT 2.9-3.4 X IN 1YR
- ANTI-VEGF
  - 11-33% RECURRENCE
  - MAY DEVELOP AT ALTERNATE SITES (20%)
  - SERIAL INJECTIONS NEEDED UNTIL CNVM QUIESCENCE
  - AVASTIN, LUCENTIS, EYLEA HAVE ALL BEEN TRIED

...BACK TO THE PATIENT

CASE 8
64 / W / M
CC: eye infection x 2 weeks, treated by primary with polytrim
Oc Hx: LEE 10yrs, no surgery/trauma/cataracts/glaucoma
Oe Meds: polytrim qid od
Med Hx: hyperlipidemia, HTN, leukemia, h/o skin cancer
Fam Hx: dm (mother), glaucoma (mgf)
Soc Hx: -etoh, h/o smoking
BVA: 20/20, 20/20-
Prelim: FROM, FTFC OD OS, no APD
SLE: see photos
IOP: 16/18 @ 1250p
DFE: unremarkable

DIFFERENTIAL DIAGNOSIS
- CORNEAL PANNUS
- PAPILLOMA
- PTERYGIUM
- PINGUECULA
- MALIGNANT MELANOMA
- NEVUS
- MOOREN’S ULCER
- KERATOACANTHOMA
- MALIGNANT MELANOMA
- PYOGENIC GRANULOMA
- CONJUNCTIVAL LYMPHOMA
- DYSKERATOSIS
- SQUAMOUS CELL CARCINOMA
- OCULAR SURFACE SQUAMOUS NEOPLASIA (OSSN)
- BENIGN DYSPLASIA
- PAPILLOMA
- PSEUDEPITHELIOATOUS HYPERPLASIA
- BENIGN HEREDITARY INTRAEPITHELIAL DYSKERATOSIS
PREINVASIVE OSSN
CONJUNCTIVAL / CORNEAL CARCINOMA IN SITU
INVASIVE OSSN
SQUAMOUS CARCINOMA
MUCOEPIDERMOID CARCINOMA

OSSN HISTOPATHOLOGY
BENIGN DYSPLASIA
MILD = < 1/3 OF THICKNESS WITH ATYPICAL CELLS
MODERATE = ¼ THICKNESS WITH ATYPICAL CELLS
SEVERE = FULL THICKNESS WITH ATYPICAL CELLS

PREINVASIVE OSSN
ABOVE AND LOSS OF NORMAL SURFACE LAYER
INVASIVE OSSN
ABOVE AND WHEN BASAL EPITHELIAL LAYER IS BREACHED AND SUBSTANTA PROPIA IS INVADED

OCULAR SURFACE SQUAMOUS NEOPLASIA (OSSN)
CLINICAL PRESENTATION
RARELY AFFECTS VISION
ASYMPTOMATIC TO CHRONICALLY IRRITATED RED EYE
INVOLVES CORNEA, CONJUNCTIVA OR BOTH
95% ORIGINATE AT LIMUS (3-9 OCLOCK)
COLOR
PEARLY GRAY TO REDDISH BROWN
MAY OR MAY NOT HAVE FEEDER VESSELS
INITIALLY MOBILE, LATER FIXED IF SCLERA INVOLVED

APPEARANCE
GELATINOUS
LEUKOPLAKIC
PAPILLOMATOUS

DIAGNOSIS
ROSE BENGAL CAN IDENTIFY EXTENT OF LESION
DIFFICULT TO DIAGNOSE AT SLIT LAMP
ONLY 40% ACCURATELY DIAGNOSED
TISSUE SPECIMEN IS NEEDED
EXCISIONAL BIOPSY DUE TO POSSIBLE MALIGNANCY
PUNCH OR INCISIONAL WEDGE IF LARGE
SPREAD TO
SCLERA (37%)
INTRAOCULARLY (13%)
INTO ORBIT (11%)
USE ULTRASOUND, GONIOSCOPY, MRI OF ORBIT WITH GADOLINIUM
RARE TO CAUSE REGIONAL OR DISTANT METASTASES

OSSN RISK FACTORS
OLDER AGE (50-75)
MALE
FAIR SKIN, PALE IRISES, PROPENSITY TO SUNBURN
H/O SKIN CANCER
EXPOSURE TO SOLAR UV RADIATION
HPV, HIV (13X RISK), TRACHOMA, VITAMIN A DEFICIENCY
XERODERMA PIGMENTOSUM, CHRONIC IRRITANTS
IMMUNOSUPPRESSION (ORGAN TRANSPLANTS, AIDS)
WITHIN 30 DEGREES OF EQUATOR
SMOKING
CHEMICAL EXPOSURE
CHRONIC INFLAMMATION
PEMPHIGOID, ECZEMA
BLEPHAROCONJUNCTIVITIS

OSSN TREATMENT
GOAL
COMPLETE ERADICATION, MINIMIZE SPREAD OF DISEASE
OPTIONS
EXCISION
FOLLOWED BY CONJUNCTIVAL AUTOGRAFT OR AMNIOTIC MEMBRANE
CRYOTHERAPY
CHEMOTHERAPY
COMBINATION OF ABOVE
RARELY RADIOTHERAPY (EXTERNAL BEAM OR PLAQUE)
RARELY ENUCLEATION, EXENTERATION
EXCISION
GOLD STANDARD
SIDE EFFECTS
LIMBAL STEM CELL DEFICIENCY
SURFACE IRREGULARITY
SCARRING

CONS
MAY NOT REMOVE ALL OF LESION
8-40% RECURRENCE
2-4 MM BEYOND CLINICALLY EVIDENT MARGIN

CRYOTHERAPY
AT EDGE TO FREEZE RESIDUAL CELLS

CHEMOTHERAPY
RATIONALE
TREATS ENTIRE OCULAR SURFACE
TREATING MICROSCOPIC / SUBCLINICAL DISEASE
AVOIDS EXTENSIVE INCISION
AVOID SURGICALLY INDUCED LIMBAL STEM CELL DEFICIENCY
MAY WORK ON INEXCISABLE LESIONS
HELPFUL IN RECURRENT OR DIFFUSE LESIONS

INDICATIONS
MULTIFOCAL TUMOR WITH INDISTINCT MARGIN
RECURRENT TUMORS
HIV+ WITH LARGE TUMOR OR BILATERAL INVOLVEMENT

MITOMYCIN C
ALKYLATING AGENT
BINDS TO DNA DURING ALL PHASES OF CELL CYCLE
LEADS TO CROSS-LINKING AND INHIBITS NUCLEOSIDE SYNTHESIS
0.04% QID 4 DAYS A WEEK FOR 4 WEEKS
SUCCESS RATE FROM 87-100%
SIDE EFFECTS
ALLERGIC REACTION 34%, EPIPHORA FROM PUNCTAL OCCLUSION (14%)
BLEPHAROSPASM
PEK

5-FLUOROURACIL
PYRIMIDINE ANALOGUE, ANTIMETABOLITE
INHIBITS THYMIDYLATE SYNTHETASE DURING S PHASE OF CELL CYCLE
PREVENTS DNA / RNA SYNTHESIS
TREATMENT
QID X 1 WEEK WITH PUNCTAL OCCLUSION
OFF FOR 1 MONTH OR CONTINUOUS
SIDE EFFECTS
LESS THAN MMC
EPITHELIAL DAMAGE
CONJUNCTIVAL INFLAMMATION AND IRRIGATION
7.3% RECURRENCE

INTERFERON ALPHA 2B
GLYCOPROTEIN MOLECULES, ACT AT CELL SURFACE RECEPTORS
PRODUCE ANTIVIRAL AND ANTITUMOR ACTIVITIES
THEORY: INDIRECT ANTIPROLIFERATIVE EFFECT FROM PROMOTING HOST IMMUNE RESPONSE AND CYTOTOXIC
EFFECTOR CELLS
TREATMENT
1-3 MIL IU/ML QID X 3 MOS
SUBCONJ INJECTION
SIDE EFFECTS
MINIMAL TO TOPICAL
FLULIKE FOR INJECTION
3.7% RECURRENCE

DIAGNOSE AND MONITORING
ULTRAHIGH RESOLUTION OCT

...BACK TO THE PATIENT