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COMMON OPTIC NEUROPATHIES THAT CAN PRESENT WITH DISC SWELLING

- ANTERIOR ISCHEMIC OPTIC NEUROPATHY
- OPTIC NEURITIS
- PAPILLEDEMA
- PSEUDOTUMOR CEREBRI
- PSEUDO-PAPILLEDEMA
- INFILTRATIVE OPTIC NEUROPATHIES
- COMPRESSIVE OPTIC NEUROPATHIES
- TOXIC OPTIC NEUROPATHIES
- HEREDITARY OPTIC NEUROPATHIES

ANTERIOR ISCHEMIC OPTIC NEUROPATHY (AION)

- PATIENTS OVER THE AGE OF 40
- PAINLESS
- SUDDEN UNILATERAL LOSS OF VISION
- REDUCED COLOR VISION
- AFFERENT PUPILLARY DEFECT
- ALTITUDINAL FIELD LOSS

FUNDUS APPEARANCE OF AION

- PALE DISC SWELLING
- OFTEN SECTORIAL (SUPERIOR)
- PERIPAPILLARY HEMORRHAGES
- ATTENUATED RETINAL ARTERIOLES
- DELAYED CHOROIDAL FILLING ON FA
- DEVELOPMENT OF OPTIC ATROPHY (4-6 WEEKS)

SYSTEMIC ASSOCIATION OF AION

- ARTERITIC
  GIANT CELL ARTERITIS (GCA) (6%)

- NON-ARTERITIC
  HYPERTENSION (40%)
  DIABETES (17%)
  ARTERIOSCLEROSIS (14%)
  MIGRAINE (2%)
  IDIOPATHIC (27%)

ARTERITIC AION (GCA)

- PATIENTS OVER THE AGE OF 70
- F>M
MORE COMMON IN CAUCASIANS
OVER 50% WITH 20/200 OR WORSE VA
65% RISK TO FELLOW EYE IF UNTREATED
ELEVATED SEDIMENTATION RATE/POSITIVE C REACTIVE PROTEIN
POSITIVE TEMPORAL ARTERY BIOPSY

SYSTEMIC SIGNS OF GCA

HEADACHES
SCALP TENDERNESS
SWOLLEN TEMPORAL ARTERIES
JAW CLAUDICATION
WEIGHT LOSS
MUSCLE STIFFNESS

OCULAR SIGNS OF GCA

AION
AMAUROSIS FUGAX
OPHTHALMOPLEGIA
CENTRAL RETINAL ARTERY OCCLUSION

NON-ARTERITIC AION

LACK SYSTEMS OF GCA
YOUNGER (40-65)
NO PREFERENCE TO SEX OR RACE
45% WITH 20/40 OR BETTER VA
SMALL DISC - NO CUP
NORMAL ESR/C-REACTIVE PROTEIN
12-40% RISK TO FELLOW EYE OVER 3-5 YEARS
UP TO 43% OF PTS MAY SHOW A SIGNIFICANT IMPROVEMENT IN VA

MANAGEMENT

Differentiate arteritic from non-arteritic AION
Evaluate for signs and symptoms of GCA
Order ESR and C-reactive protein
Evaluate size of the fellow optic nerve

TREATMENT

ARTERITIC
HIGH DOSE SYSTEMIC CORTICOSTEROIDS
MANAGED BY A RHEUMATOLOGIST

NON-ARTERITIC
NO PROVEN EFFICACY WITH STEROIDS?
RECENT HAYREH PUBLICATION SUGGESTS THERE MAY BE A BENEFIT WITH ORAL STEROIDS, HOWEVER NOT A RANDOMIZED, DOUBLE BLIND PROSPECTIVE STUDY
REFER TO THEIR INTERNIST (HTN, DM, MI)
GREATER RISK OF CVA AND MI – REC ASA THERAPY
613 patients with NA-AION
• Patient self selection of high dose oral steroids or observation
• Treatment 80 mg oral prednisone with taper over 2-3 months until optic nerve swelling resolves
• Patients followed with serial VA and VFs
• Median follow up 3.8 years

• Patients with initial VA 20/70 or worse and started on steroids within 2 weeks of onset, VA improved in 70% of treated group vs 41% of untreated group. VF improved in 40% of treated patients vs 25% of untreated group.
• Improvement occurred within first 6 months and not much after.
• Some patients learned to eccentrically fixation which could result in improved visual acuity
• Eliminated eccentric fixation, 55% in the treat group vs 28% in the untreated group improved
• Less benefit of steroids in patients with 20/60 or better vision
• Study criticism: not double blind, not randomized, no ETDRS acuities
• High dose steroids over 2-3 months have potential side effects

ISCHEMIC OPTIC NEUROPATHY DECOMPRESSION TRIAL

• 244 PATIENTS WITH NON ARTERITIC AION WITH VA 20/64 OR WORSE
• RANDOMIZED TO DECOMPRESSION SURGERY VS OBSERVATION
• AT 6 MONTHS, 33% SURGERY VS 43% OBSERVE IMPROVED 3 LINES VA
24% SURGERY VS 12% OBSERVE HAD DECLINED 3 LINES VA
• CLINICAL TRIAL SHOWED OPTIC NERVE SHEATH DECOMPRESSION HAS NO BENEFIT IN THE TREATMENT OF NON-ARTERITIC AION AND MAY BE HARMFUL.

OPTIC NEURITIS

• PATIENTS UNDER THE AGE OF 45
• FEMALES: MALES RATIO 2:1
• PAIN ON EYE MOVEMENTS
• VARIABLE ACUTE VISUAL ACUITY OR FIELD LOSS
• REDUCED COLOR VISION
• AFFERENT PUPILLARY DEFECT
• ANY VF DEFECT POSSIBLE
• VISION IMPROVES IN 2-8 WEEKS (SOMETIMES LONGER) AFTER INITIAL EVENT
• DISC PALLOR MAY DEVELOP

DISC APPEARANCE IN OPTIC NEURITIS

• 40% DISC SWELLING (PAPILLITIS)
• 50% WITHOUT DISC SWELLING (RETROBULBAR)
• 10% WITH DISC PALLOR (RECURRENT)
CAUSES OF OPTIC NEURITIS

- DEMYELINATING DISEASE (MS)
  - 74% FEMALE VS 34% MALE AFTER 15 YEARS
- IDIOPATHIC
- COLLAGEN VASCULAR DISEASE
- SYPHILIS

THE FULL WORK UP OF OPTIC NEURITIS

- MRI OR CT
- LP
- ANA
- CXR
- FTA-ABS

TREATMENT OF OPTIC NEURITIS

THE OPTIC NEURITIS TREATMENT TRIAL

- 2X GREATER INCIDENCE OF RECURRENCE WITH ORAL STEROIDS
- IV STEROIDS FOLLOWED BY ORAL STEROIDS - FASTER RECOVERY, BUT LITTLE LONG TERM BENEFIT IN VA COMPARED TO PLACEBO
- PATIENTS WITH UBO'S ON MRI HAVE A GREATER RISK OF MS
- IV STEROIDS MAY PREVENT OR DELAY THE ONSET OF MS FOR FIRST TWO YEARS

PAPILLEDEMA

- ANY AGE
- NORMAL VA/COLOR VISION *
- NO AFFERENT PUPILLARY DEFECT
- ENLARGED BLIND SPOT
- HEADACHES, NAUSEA AND VOMITING
- FOCAL NEUROLOGICAL SYMPTOMS
- TRANSIENT OBSCURATIONS OF VISION
- BILATERAL VI NERVE PALSY

DISC APPEARANCE IN PAPILLEDEMA

- BILATERAL HYPEREMIC DISC SWELLING
- PERIPAPILLARY HEMORRHAGES\EXUDATE
- PATON’S LINES
- LOSS OF SUPERFICIAL VENOUS PULSE
- SEVERE OR CHRONIC - MAY LEAD TO PERMANENT DAMAGE

MANAGEMENT OF PAPILLEDEMA

- CHECK BLOOD PRESSURE
- CT / MRI
- LP
- DISC DOCUMENTATION
- TREAT UNDERLYING CAUSE - MONITOR VA, CV AND VF
PSEUDOTUMOR CEREBRI

- YOUNG OBESE FEMALES
- NORMAL IMAGING STUDIES (CT OR MRI)
- NORMAL OR SMALL CEREBRAL VENTRICLES
- INCREASED INTRACRANIAL PRESSURE (LP)
- NORMAL CSF COMPOSITION (LP)
- 20-25% DEVELOP VISUAL LOSS OVER COURSE OF THEIR DISEASE

MANAGEMENT OF PSEUDOTUMOR CEREBRI

- R/O EXOGENOUS AGENTS (VIT A, STEROIDS, ACCUTANE, BIRTH CONTROL)
- WEIGHT LOSS
- DIAMOX (1-4 GRAMS/DAY)
- MONITOR VA, CV, VF AND DISC
- SHUNT PROCEDURES
- OPTIC NERVE SHEATH DECOMPRESSION

OPTIC DISC DRUSEN

- HYALIN BODIES
- ELEVATED DISC (NO CUPPING)
- ANOMALOUS DISC VESSELS
- FAMILIAL
- MAY DEVELOP VISUAL LOSS
- NFL DROPOUT

CAUSES OF PSEUDO-PAPILLEDEMA

- OPTIC NERVE HEAD DRUSEN
- HIGH HYPEROPIA
- ANOMALOUS DISCS
- MEDULLATED NERVE FIBERS
- PERIPAPILLARY CHOROIDAL NEOVASCULAR MEMBRANE (CNVM)

MANAGEMENT OF AN ANOMALOUS DISC

- PATIENT HISTORY
- MONITOR VA, CV, VF
- PHOTO DOCUMENTATION
- ULTRASONOGRAPHY
- FLUORESCEIN ANGIOGRAPHY
- CT / MRI

INFLTRATIVE OPTIC NEURPHTY

- ANY AGE
- PROGRESSIVE VA LOSS
- REDUCED COLOR VISION
- VARIOUS VISUAL FIELD DEFECTS
- WITH OR WITHOUT DISC SWELLING
- VITREOUS CELLS
- SYSTEMIC ILLNESS
CAUSES OF INFILTRATIVE OPTIC NEUROPATHY

- LEUKEMIA
- LYMPHOMA
- SARCOID
- METASTASIS

MANAGEMENT OF INFILTRATIVE OPTIC NEUROPATHY

- SYSTEMIC WORK-UP
- LOCAL RADIATION
- CHEMOTHERAPY

COMPRESSIVE OPTIC NEUROPATHY

- WITH OR W/O DISC SWELLING
- PROGRESSIVE VA LOSS
- LOSS OF COLOR VISION
- AFFERENT PUPILLARY DEFECT
- OPTOCILIARY SHUNT VESSELS
- PROPTOSIS
- EVENTUAL DISC PALLOR

CAUSES OF COMPRESSIVE OPTIC NEUROPATHY

- ORBITAL TUMORS
- DYSTHYROID OPTIC NEUROPATHY
- OPTIC NERVE GLIOMA
- OPTIC NERVE SHEATH MENINGIOMA
- PITUITARY TUMORS
- CRANIOPHARYNGIOMAS

MANAGEMENT OF COMPRESSIVE OPTIC NEUROPATHY

- CT/MRI
- THYROID FUNCTION TESTS