Normal-tension Glaucoma: Evaluation and Treatment

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OBJECTIVES
✓ DEFINE NORMAL TENSION GLAUCOMA
✓ PATHOPHYSIOLOGY IN COMPARISON TO POAG
✓ DEFINE CLINICAL PROBLEMS OF MIMICKERS OF LTG
✓ DIFFERENTIAL DIAGNOSIS AND EVALUATION
✓ TREATMENT OF LTG

Historical background
1857 Von Graefe noted-
✓ “Nerve head excavation without a palpable increase in IOP”
NORMAL TENSION GLAUCOMA- how do you define it?
✓ Progressive optic neuropathy
✓ Significant disc cupping
✓ Significant visual field loss
✓ Open angle, IOP less than 21 mm Hg
✓ No other systemic or ocular disorders

TO MAKE THE DIAGNOSIS
✓ Optic nerve assessment
✓ Retinal nerve fiber layer assessment
✓ Computerized visual field
✓ Gonioscopy
✓ Diurnal pressure curve

You should ask:
✓ Is there a history of shock, steroids, trauma?
✓ Migraine history is helpful
✓ Blindness in family? Family history often positive in 20% of cases

WHO GETS IT?
✓ 6th or 7th decade of life
✓ No racial predilection
Women greater than men
Inheritance polygenic or multifactorial

Prevalence of Open-Angle Glaucoma in the United States
- 0.4% <40 Y.O.
- 1.3% 60-69 Y.O.
- 4.7% 70-79 Y.O.
- 11.4% > 80 Y.O.
Mean IOP in the white population 15.6% mm Hg with 97% <24 mm Hg

Typical presentation
- Unlike POAG, IOP is not the tip off
- Damage to the visual field or disc usually occurs before detection by the M.D.
- Most pts with NTG are diagnosed late in the disease

HOW COMMON IS NTG IN THE POPULATION
- Baltimore Eye Study 1991 - 10,444 eyes
- 194 cases open angle glaucoma
- 114 (58.8%) had IOP<21 mm Hg.
- Sommers 1996 concluded (conservative estimate) 20-30% have IOP<21 mm Hg.
- Other investigators- 50% have LTG

Is the optic nerve in NTG different?

OPTIC NERVE HEAD
- Can you tell LTG from POAG just by looking at the disc?
- CAPRIOLI & SPAETH noted
  Inferotemporal and temporal rims thinner in LTG

ARE ACQUIRED PITS OR NOTCHES MORE COMMON IN NORMAL TENSION GLAUCOMA?
Javitt et al
- Odds of an acquired pit in the optic nerve is 16X greater with NTG
- Acquired pits are more common with NTG
  - 74% NTG vs. 15% POAG

ACQUIRED PIT OF THE OPTIC NERVE
Urgurlu and Caprioli et al, 1997
- Pits more common inferiorly than superiorly (76% vs. 24%)
- 64% of patients with pits progress (follow up 7.9 years)
- Visual field progression was common in 56% of study group
- Disc hemorrhage in 40% of study group

ARE DISC HEMORRHAGES MORE COMMON WITH NORMAL TENSION GLAUCOMA?
- Bjerrum described disc hemorrhage first in 1899
- Drance 1972 - 20% disc hemorrhages with LTG
- Kitazowa et al 2001- 30.5% incidence of disc hemorrhages in Japan
- USA-Disc hemorrhage normal population 0.33%
DISC HEMORRHAGES
- Located in the pre-laminar area of the optic disc within the superficial retinal layer.
- More common upper & lower poles.
- More common in the inferotemporal quadrant.

OPTIC DISC HEMORRHAGES
  Blue Mountain Eye Study; Australia
  - Optic disc hemorrhages are present in
    - 13.8% of open angle glaucoma patients
    - 25% of low tension glaucoma patients
    - 8% of high tension glaucoma patients
    - 1% of people with a normal optic nerve
  - Risk factors for hemorrhage: increased IOP, PSXF, diabetes, increased vertical cup

Drance in 1973
- Found that 3 months after a hemorrhage of the optic nerve, a notch occurred in the same place

What else is associated with disc hemorrhage?
- Localized damage to both the rim and RNFL
- Where? In the inferotemporal sector more commonly

Liou et al, Morphologic characteristics of optic disc with disc hemorrhage in NTG, AJO 2001, 132:618-625

Peripapillary defects—what are they?
- They represent the absence of RPE adjacent to the disc
- Question: is peripapillary atrophy more common in NTG patients?
- Answer: controversial

What about peripapillary atrophy in NTG---is it important?
- Jonas et al, Arch Ophthalmol, 1992
  Peripapillary atrophy enlarges with optic nerve damage
  Clinical point: that’s why optic nerve photos are so important

  Peripapillary retinal blood flow in normal-tension glaucoma
  Chung, Harris, Kagemann et al, Br J Ophthalmol 1999;83:466-469

NTG is characterized by reduced blood flow in the peripapillary retina, suggesting that blood flow deficits may accompany and contribute to disease development.

Summary of peripapillary defects
- Peripapillary Defects are Not More Common in Normal-Tension Glaucoma.
  However, Enlargement of Atrophic Zones is Suggestive of Continuing Disease

What About the Neuroretinal rim Loss in NTG?
- Jonas et al, AJO1998:125;137-144
Patients with NTG had thinner neuroretinal rims and more pronounced optic nerve damage than eyes with higher IOP glaucoma (98 eyes with color stereo discs).

Summary of optic disc appearance

- Cannot differentiate high tension from low tension glaucoma on optic disc appearance alone
- BUT
- Optic nerve pits or notches more common in normal-tension glaucoma
- Optic disc hemorrhage more common in normal-tension glaucoma and more common at the inferior and superior poles

Are there findings that help identify a normal tension glaucoma disc?

- Pts with NTG tend to have more focal loss of the rim, more peripapillary atrophy and more disc hemorrhages.
- Caprioli, Spaeth, Arch Ophthalmol 1985,103;1145-1149.

What about the pattern of visual field loss in normal vs. high tension glaucoma?

Pattern of Visual Field Defects in Normal-tension glaucoma

- Cannot differentiate high versus low tension glaucoma based on visual field, however normal-tension glaucoma visual field defects are
  - More localized, closer to fixation, denser in superior nasal quadrant
  - May be more prominent in lower hemifield

Does IOP play a role in visual field loss in NTG?

- Controversial :
  - Cartwright & Anderson
    - There is greater visual field loss in the eye with the highest pressure
  - Orgul and Flammer
    - There is no relationship between the eye with a higher IOP and visual field loss in glaucoma
  - It has been my clinical experience that the eye with the higher IOP tends to have more severe disease.

NTG patients with unilateral field loss are at risk of developing field loss in the eye with an initial normal visual field

- What are the risk factors to predict visual field loss?
- Severity of the visual field damage in the contralateral eye at presentation

Frequency doubling perimetry

- Frequency doubling technology (FDT) detected abnormalities in POAG cases more sensitively than in NTG cases
- Other studies have found FDT equal to or superior to Humphrey visual field in detecting early defects in NTG
Is Progression of Visual Fields in Normal-Tension Glaucoma common?
✓ Answer: Yes
✓ Levene 1980 - 41% of eyes progressed
✓ Anderson 1985 - 40% progressed in a 10.5 year period
✓ NTG patients with unilateral field loss are at higher risk of developing field loss
  in the eyes with an initial normal visual field

Does NTG Progress More than HTG?
✓ Yes! Glicklich and Spaeth, 1988
✓ 5 year follow up,
✓ 62% NTG had visual field progression compared to 42% of POAG
  IOP was lowered 20% either with meds or surg.

Is progression of NTG common?
✓ 160 patients randomized to no treatment
✓ 50% showed confirmed visual fields deterioration by 7 years — but the change was slow

What is the Relationship between Corneal Thickness and Curvature in NTG?
Morad et al, AJO 1998:125;164-168
✓ Corneal thickness is significantly reduced in NTG compared with POAG (p=.002) and normals
✓ This may lead to underestimation of IOP and misdiagnosis in some pts.

The Ocular Hypertensive
Treatment Study
✓ Patients who developed POAG
  – Mean corneal thickness 553 +/- 38 microns
✓ Patients who did not develop POAG
  – Mean corneal thickness 574 +/- 37 microns

What are other associated findings in Normal-tension glaucoma?
✓ Phelps and Corbett (1985) found:
✓ 48% of NTG patients had migraine
✓ Other investigators:
  (2001) found:
  24% of patients with NTG had migraine

What are other risk factors for progression?
✓ Myopes have a higher chance of progressing within 10 years with early cecocentral defects and a distinct chance of visual acuity loss
✓ The association between myopia and NTG was strong
Vasospasm as a risk factor-what is the relationship?
NTG patients appear to have a higher prevalence of vasospastic disorders, a higher prevalence of acute vascular collapse, and greater blood pressure drops during a 24-hour period.

Gasser and Drance found that vasospasm made NTG worse
- Abnormal capillary response to cold in the digital circulation (hands)
- One hand of a patient placed in cold water, the visual field was repeated—the visual field got worse
- Higher incidence of Raynaud's disease in this population

OTHER RISK FACTORS
- Migraine
- Raynaud's disease
- Prinzmetal's angina
- Sleep apnea
- High resistance wind instruments
- WEIGHTS

PATHOGENESIS: ISCHEMIC THEORY
- Elevated IOP causes the ischemia
- Ischemia causes retinal ganglion cell death by neurotrophic deprivation
- Poor optic nerve perfusion initiates the cascade that ends in cell death

Pathogenesis: Mechanical theory
- Increased IOP distorts the lamina cribrosa
- Get compression of the axons
- This interferes and disrupts axoplasmic flow
- This may lead to cell death
  - What predisposes to this? Abnormal lamina cribrosa, structural factors, abnormal connective tissue bundles?

Hypoperfusion theory
  Either:
  - Postural hypotension
  - Nocturnal hypotension with optic nerve hypoperfusion
  - Sleep apnea contributing to optic nerve hypoperfusion
  - Increased resistance to blood flow in the ophthalmic and retinal arteries

Hypoperfusion theory
- The optic nerve has inadequate perfusion pressure and the optic nerve blood flow is compromised
- You get ischemia to the optic nerve even at normal IOP
- Poor auto-regulation?
Role of systemic hypotension
✓ 24 hour ambulatory monitoring
✓ There is a greater nocturnal decrease and lower level of diastolic blood pressure in the NTG patients
✓ Those pts on anti-hypertensives who had greater systolic dips in BP, tended to have more deterioration of visual fields
✓ Greater nocturnal fluctuation with BP dips may disturb the microcirculation. This may damage the optic nerve resulting in increasing visual field loss in NTG.

Theory of APOPTOSIS
✓ Ischemia causing a chain of events leading to programmed cell death.

Can there be an immune basis for NTG?
✓ 9 of 10 patients with NTG studied
✓ No other signs of connective tissues.
✓ Positive elevated serum immunoreactivity bacterial heat shock protein 60
✓ Confirmed by Kitazawa, 2001

IS THERE A GENETIC BASIS FOR NTG?
✓ OPA 1 GENE
✓ GENOTYPE IVS+4 C/T;32T/C
✓ May be strongly associated with NTG

Neuro-ophthalmologic ddx: objective
To prevent erroneous diagnosis of glaucoma in patients with neuro-ophthalmologic disorders requiring other treatments

Why consider neuro-ophthalmic disease?
✓ IOP is normal
✓ Visual field defects may be identical
✓ Cupping occurs in optic neuropathies

Nonglaucomatous optic atrophy
✓ 20% of optic atrophy eyes have cupping
✓ 6% have typical glaucomatous cupping
  — Trobe, arch ophth, 1980

Non-glaucomatous cupping
✓ Congenital
✓ Hereditary optic neuropathy
✓ Inflammatory optic neuropathy
✓ Compressive optic neuropathy
✓ Ischemic optic neuropathy
Toxic optic neuropathy
Radiation optic neuropathy
Degenerative disease

Cupping: glaucoma vs. Nonglaucoma
Slides reviewed by 2 glaucoma specialists and 1 neuro-ophthalmologist

- 44% of nonglaucoma misdiagnosed by at least 1 examiner
  - Trobe, arch ophth, 1980

Glaucoma vs. Nonglaucoma
- Rim pallor 94% specific for nonglaucoma
- Focal or diffuse rim obliteration 87% specific for glaucoma
- Rim thinning only 47% specific for glaucoma

Is it glaucoma?
Red flags
- Atypical history
- Atypical exam
- Atypical optic disc
- Atypical visual field

29 yo man
- Followed as unilateral glaucoma
- History of pain o.d. With progressive visual loss over days
- Stable for 8 months
- MRI normal

Neuro-ophthalmic exam
- VA: 20/200 o.d., 20/20 o.s.
- Color: 1/13 o.d., 13/13 o.s.
- Pupils: 3+ apd o.d.
- IOP: 8 mm hg o.d., 10 mm hg o.s.

Evaluation
- CXR: hilar adenopathy
- ACE level: 100
- Bronchoscopy: non-caseating granulomas

Treatment
- Pulsed solumedrol
- Oral prednisone taper
- No improvement

38 y o woman
- Followed as glaucoma suspect because of cupping
Multiple sclerosis diagnosed for 3 years
Recurrent optic neuritis o.u.
Iop always in teens

Neuro-ophthalmic exam
Va 20/30 o.d., 20/200 o.s.
Color: 3/13 o.d., 0/13 o.s
Pupils: 2 + r.l o.d., 1+ o.s., 2+ apd o.s.

Vf: central scotoma o.s., superotemporal defect o.d. (junctional pattern)

Inflammatory optic neuropathy
Optic neuritis
Sarcoidosis
Syphilis

Optic neuritis: typical presentation
Sudden painful visual loss
Young adult
Va, color, field loss, apd
normal or swollen disc
Eventual optic pallor
Rarely cupping

75 yo woman with possible glaucoma
Abrupt visual loss o.u. 2 years ago

Va 20/200 o.u.
Color: 0/13 isihara
Pupils 1+ reactive o.u.
Vf: central and inf. Altitudinal defects o.u.
Iop never above 20 mm hg
Cupping + pallor

Past medical history
Polymyalgia rheumatica
Headache
Biopsy (+) for giant cell arteritis
Stable on prednisone 5 mg/d

Cupping in nonarteritic aion
Hayreh, 1974: 2/14
Quigley, 1977: 6/61
Radius, 1979: 1/25 (large contralateral cup)
Danesh-meyer et al, 2001: 2%

cupping in arteritic aion
Hayreh, 1974: 11/11
Quigley, 1977: 5 (2 with elevated iop, 3 large cup in fellow eye)
Sebag, 1986: 5 with (-) glaucoma evaluation
Danesh-meyer et al, 2001: 92%

Anterior ischemic optic neuropathy: typical presentation
Painless, sudden visual loss
Older adult
Decreased acuity, color, vf, apd
Swollen optic disc
Eventual pallor, cupping in gca

Ischemic
Aion - giant cell arteritis
Aion - non-arteritic
Shock induced ntg
Central retinal artery occlusion

70 yo woman with ltg
Neuro-ophth referral for headache
Iop always <19 mm hg

Past medical history
Diabetes
Coronary artery disease
S/p cabg and carotid endarterectomies
Medications
Insulin, mevacor, zantac, betagan, pilocarpine, neptazane

Neuro-ophthalmic exam
Va: 20/20 o.u.
Color: 7/7 isihara o.u.
Pupils: miotic from pilocarpine
Vf: temporal > nasal loss

Treatment
Stereotactic radiosurgery
Stable for 1 year
Died from cardiac disease

Compressive lesions
- 16/250 patients with structural lesions
  - 8 pituitary adenoma
  - 5 meningioma
  - 2 chiasmal glioma
  - 1 aneurysm
    - Kupersmith, ann ophthalmol, 1984

Compressive lesions
- 5/16 misdiagnosed as glaucoma
- All 16 with normal iop
- 1/7 with borderline outflow on tonography
- C/d for color > c/d for contour
  - Kupersmith, ann ophthalmol, 1984

Cupping in compression
- Retrospective masked review
- C/d 0.37 compared with 0.10 in controls
- Intereye difference 0.13 vs. 0.04 in controls – showing it was acquired
  - Bianchi-marzoli s et al, ophthalmol 1995

Ntg and neuroimaging
- Studied pts. With ntg who had neuroimaging over a 10 yr period
- Control group of those with intracranial masses, with c/d at least 0.4
- 31 pts. 2 excluded, 1 with homonymous hemianopsia, 1 with 6th nerve paresis

Ntg and neuroimaging
- No ntg pts. Had a related tumor
- 2 ntg pts. Had clinically silent tumors unrelated to vf defect (6.9%)

Ntg eyes
- Better va
- Vertical cup elongation
- Splinter hemorrhages
- Peripapillary atrophy
- Vf defects that were nerve fiber bundle or bordering horizontal meridian

Specific for glaucoma
- Splinter disc hemorrhage 100%
Family hx of glaucoma 96%
Nerve fiber bundle vf defect 84%
Vertical loss of neuroretinal rim 77%

Specific for nonglaucomatous
Neuroretinal rim pallor 90%
Vf defect bordering vertical meridian 81%
Visual acuity < 20/40 77%
Age < 50 years 93%

Compressive lesions:
  typical presentation
Progressive visual loss
Nerve, chiasmal, or homonymous vf loss
Headache, diplopia or other symptoms
Disc normal, swollen or pale, occasional cupping

Hereditary optic neuropathy
Leber’s optic neuropathy
Familial optic neuropathy (mendelian)

Dominant optic atrophy
  • focal temporal triangular cupping
    • Kline lb, glaser js, arch ophthalmol, 1979

Leber’s hereditary optic neuropathy
  0.7 - 0.9 c/d in 7 patients after visual loss
  - Ortiz, ajo, 1992

Lhon mutation in ntg
  Screened 54 ntg patients for lhon mtdna mutations at nucleotides 3460, 11778 and 14484
  None had lhon mutations
    • Opial d et al, graefes arch…., 2001

Congenital cupping
  Colloboma
  Optic pit
  Myopia
  Physiologic cupping

Physiologic cupping
  Large discs
Normal rim area
Horizontal cup > vertical cup
Jonas, ajo, 1989

Toxic optic neuropathy
Methanol
Stelmach, aust-n-z-ophth, 1992

Is glaucoma more common in alzheimer’s disease?
112 pts. With probable ad
Found vf defects or cupping in 29 (26%)
Ocular hypertension was not seen
Propose that on less resistant than normal in ad
Bayer au et al, eur neurology, 2002

Degenerative
Perhaps cupping and vf are an optic neuropathy from ad and not glaucoma
Tsai, arch ophth, 1991

Thyroid disease
6/25 ntg patients had thyroid disease
Thyroid group more hyperopic
Postulate risk factor for glaucoma or optic neuropathy mimicking ntg
Jamsen, acta ophth scand, 1996
6.5% prevalence of ntg in japanese pts. With graves (2.0% expected)
Ohtsuka, ajo, 2000
0/100 hypothyroid pts. Had findings of glaucoma
Karadimas et al, ajo, 2001

Is it glaucoma?
Red flags
Atypical history
Atypical exam
Atypical optic disc
Atypical visual field

History favoring neurologic
Abrupt visual loss
Rapid progression
Headache, ocular pain
Ocular motility defects
Neurologic symptoms/signs

Visual acuity: glaucoma vs. Neurologic
Poor va compared to c/d in neurologic
✓ Good va compared to c/d in glaucoma

Visual field favoring neurologic
✓ Central or cecocentral loss
✓ Hemianopic field loss
✓ Inferior altitudinal defect

Disc features: glaucoma vs. Neurologic
• Pallor of preserved rim is neurologic
✓ Obliteration of rim is usually glaucoma
✓ Cupping is a late finding in neurologic
✓ Retinal arteriolar narrowing in vascular or trauma

Whom to work up?
✓ Young age
✓ Decreased acuity
✓ Asymmetry of color or apd
✓ Neuro symptoms
✓ Neuro field defects
✓ Rim pallor
✓ Atypical course of progression

Evaluation of ntg
directed by individual situation
✓ Cat scan or mri scan
✓ Serologic evaluation
✓ Chest x-ray
✓ Csf examination

Evaluation of ntg:
neuroimaging
✓ Mri preferred, especially for chiasm
✓ Cat scan is a good second choice
✓ Crucial to obtain appropriately focused study

Evaluation of ntg:
serologic tests
✓ Cbc, b12
✓ Ana, esr
✓ Ace level
✓ Rpr, fta, lyme
✓ Mitochondrial dna studies
✓ ? Thyroid function tests
Cxr
✓ Rule out sarcoidosis

Lumbar Puncture
✓ Rule out meningeal process
  – Inflammatory
  – Neoplastic
  – Infectious

How do we treat NTG?
✓ What are the guidelines?
Collaborative Normal-Tension Glaucoma Study Group
✓ AJO 1998;126:487-492
✓ Answer the following questions
  – To what extent is IOP involved in the mechanism of nerve damage?
  – Should we be aggressively lowering the IOP in NTG?

The Collaborative Glaucoma Study
✓ Followed patients until progression
✓ Randomized to treatment or no treatment
✓ If treatment, the end point was a 30% drop in IOP

The Collaborative Glaucoma Study
✓ Results
  – 140 eyes of 140 patients
  – 61 treated (meds, ALT, surgery)
  – 79 controls (no treatment)

RESULTS
8-Year Follow UP
✓ 35% of untreated eyes progressed by VF criteria and disc progression
✓ 12% of treated eyes progressed
✓ This demonstrates that IOP is part of the pathogenesis of NTG

Collaborative Low Tension Glaucoma Study
✓ 14% developed cataracts in the untreated group
✓ 38% developed cataracts in the treated groups. (Higher in those after filtering surgery)

Low IOP Slows or Halts Vision Loss
in Normal-Tension Glaucoma

How Do We Treat NTG?
The study showed unequivocally that once an IOP reduction of 30% was achieved, there was a slower rate of progression of visual field defects than in untreated eyes AND

30% IOP reduction was achieved with meds, laser or both in 50% of the patients


TREATMENT OPTIONS
New commitment: lower the IOP 30%
but how???

Current Medical Treatment to Lower Intraocular Pressure
Listed in order of US prescription volume†:
• Prostaglandin derivatives
• β-Adrenergic antagonists (β-blockers)
• Adrenergic agonists (sympathomimetics)
• Carbonic anhydrase inhibitors
• Cholinergic agonists (miotics)

Pathways to Lower Intraocular Pressure
Current strategies:
✓ Start with a prostaglandin or beta blocker
✓ Switch before adding, keeping number of drops to a minimum to improve compliance
✓ Alpha agonist or topical CAI consider next
✓ Keep adding and subtracting until the drops are efficacious and tolerated

What happened to neuroprotection?
✓ Betoptic S - calcium channel blocker
✓ Alphagan – up regulates growth factors and secondary neuroprotection to cells at risk
✓ Prostaglandins- may be neuroprotective by just lowering the IOP 30%

What Drugs Effect Optic Nerve Blood Flow?
✓ Beta blockers- (nonspecific) have no effect
✓ Betaxolol -decreases resistance to flow
✓ Latanoprost - improves ocular perfusion
✓ Brimonidine - no effect on flow
✓ Dorzolamide - accelerates inferotemporal retinal dye transit with increased pulsatile blood flow
✓ Carteolol-with sympathomimetic activity may decrease vascular resistance in NTG

What is available systemically?
✓ Memantine
NMDA antagonist that blocks glutamate and should block the apoptotic event
Multicenter 5 year study
Patients get the drug or placebo, same glaucoma meds
Outcome measures: visual field and optic nerve change. This drug does not lower IOP

What else can you use systemically?

- **Calcium channel blockers**

  Do we have the correct one?
  
  - Nimodipine?
  - Brovincamine?
  - Nilvadipine?
  - Verapamil?

  **Commonly used in Japan, not in the USA**

  - Oral calcium channel blocker stabilize the visual fields in NTG
    - Pts on 2mg nilvadipine with laser-Doppler flowmetry, showed decreased vascular resistance in 12 pts. avg
    - 57 yo Gifu Univ. Japan

How do calcium channel blockers work?

- **Calcium channel blockers relax the blood vessel walls thereby decreasing vascular resistance**

  Blood Flow = Blood Pressure – IOP

  Vascular Resistance

Why Not Use Calcium Channel Blockers in Everyone?

- Routine use of Nifedipine or any other Calcium channel blocker has not been shown to provide uniform improvement in visual function or retrobulbar hemodynamic response in this country

- Routine use of Nifedipine or Cardiazem for NTG is not advantageous

**LASER OPTIONS FOR NTG**

Laser trabeculoplasty for treatment of NTG

- **Argon**
  - 50 micron
  - 0.1 second
  - 514 nm wavelength

- **Nd:YAG (SLT)**
  - 400 micron
  - 3 nanosecond
  - 532 nm

  1 argon “application” carries 6,000 times the energy of one SLT “shot”

Why consider a laser?

- Good results for the majority of patients
- 80% of patients will experience a 20% drop in IOP
50% of patients will experience a sustained IOP drop for the next 5 years
The rate of decay of failure is 10% per year

OTHER OPTIONS THAT HAVE BEEN TRIED
Optic nerve sheath decompression but with poor results
Carbon dioxide rebreathing - causes vasodilation
CO₂ inhalation may predict who will do well with certain Ca# channel blockers.
  – Niwa et al J Glaucoma, Vol 9, No3, 2000

DOES EARLY SURGERY HELP NTG PROGRESSION?
Early surgery appears to slow or halt visual field loss in patients with progressive NTG compared with controls

Glaucoma surgery with or without adjunctive antiproliferatives in NTG

  – Reduction of IOP 20-30% slows progression
  – Patients followed by progresor VF analysis
  – Conclusion: MMC is associated with greater risk of VF progression despite a greater fall in IOP
  – Explanation: late postoperative complications with MMC compared with 5-FU

TREATMENT OF NTG SHOULD INCLUDE:
Insure IOP is normal over 24 hours, if not- treat
Rule out other causes of treatable optic neuropathy
Frequent visual fields to show progression and if so, be more aggressive
HRT or GDX to follow the nerve fiber layer
Rule out hypotension at night, hyperviscosity
Lower the pressure 30%
Consider antimetabolites

TREATMENT OPTIONS
Aggressive topical therapy
Argon or Selective laser trabeculoplasty
Calcium channel blockers for selective use only
C-Pap for sleep apnea Kremer et al, Klin Monatobl Aug 2001
Watch for dips in B.P. at night by alerting the internist about over zealous anti-hypertensive Rx
Aggressive filtration surgery
References


References for the Neuro-Ophthalmologic DDX of Glaucoma
