Low -Tension Glaucoma
Review of Risk Factors, Treatment and Developing Research Project

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PURPOSE

Review LTG with attention to:
  • Risk factors for prevalence and progression
  • Common treatment practices

Introduce New Research Project on LTG
  • Dr. George Rosanelli and Dr. William Blackshear
LOW-TENSION GLAUCOMA DEFINED

Similar optic nerve and visual field damage as in POAG, but with IOP less than or equal to 21 mm Hg (1,2)
DIFFERENCES IN PRESENTATION BETWEEN POAG AND LTG?

Clinical Features

• Appears to be greater likelihood of optic disc hemorrhages \(^{(1)}\)

• Some believe visual field defects are denser, more localized, and closer to fixation. \(^{(1,3)}\)

• A dense nasal paracentral defect is typical \(^{(1,3)}\)
In any individual patient, there is no characteristic abnormality of the optic disc or visual field that distinguishes normal-tension glaucoma from POAG with higher IOPS \(^{(3)}\)
ELEPHANT IN THE ROOM

Whether normal-tension glaucoma represents a distinct disease entity or whether it is simply POAG with IOP within the average range
Glaucoma is a Multi-Factorial Disease

Evidence That Ischemia Of The Optic Nerve Head Is Responsible For The Damage

Whether Ischemia is Mechanically Induced In LTG By IOP Or Is Due to Vascular Pathology Has Been Heavily Studied
KNOWN AND HYPOTHESESIZED RISK FACTORS FOR LTG

- Elevated IOP (4)
- Vasospastic disorders (5, 6, 7)
  - Migraine Headaches
  - Raynaud phenomenon
- Age (8)
- Hemodynamic crises (9)
- Low systemic blood pressure (10)
- Nocturnal dips in blood pressure (11, 12)

- Ischemic Vascular diseases (13)
- Autoimmune Diseases (3)
- Coagulopathies (3)

RISK FACTORS IDENTIFIED:

HOW DOES THIS INFLUENCE MANAGEMENT AND TREATMENT OF THIS DISEASE?
Purpose was to determine if IOP plays a part in the pathogenic process of normal tension glaucoma.

Compared Glaucomatous progression between untreated patients with Normal-Tension Glaucoma and patients whose IOP was therapeutically reduced by 30% from baseline.
CONCLUSIONS

• IOP is part of the pathogenic process in NTG

• Therapy that is effective in lowering IOP would be expected to be beneficial in patients who are at risk of disease progression.

Standard practice to reduce IOP in patients who have Low-Tension Glaucoma (1)
COLLABORATIVE NORMAL TENSION GLAUCOMA STUDY

WHAT THE CRITICS SAY...

Over half of those not treated showed no discernible visual field progression over 5 to 7 years of careful follow up.

The disease continues in 20% of eyes even though the IOP has been substantially lowered.

**There may be cases in which pathogenic factors damage the optic nerve independently of the level of IOP reduction**
RISK FACTORS FOR PROGRESSION IN NORMAL-TENSION GLAUCOMA (COLLABORATIVE NORMAL-TENSION GLAUCOMA STUDY GROUP)

<table>
<thead>
<tr>
<th>Continuous Variables</th>
<th>Discrete Variables</th>
<th>Discrete Variables</th>
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<tbody>
<tr>
<td>Age</td>
<td>Asthma</td>
<td>Low Blood Pressure Tendency</td>
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<tr>
<td>Pulse</td>
<td>Renal Stones</td>
<td>Vomiting or diarrhea</td>
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<tr>
<td>Diastolic Blood Pressure</td>
<td>Cardiac arrhythmias</td>
<td>Fatigue or weakness</td>
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<tr>
<td>Systolic Blood Pressure</td>
<td>Major cardiovascular crisis</td>
<td>Muscle Weakness</td>
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<tr>
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<td>-hypotension</td>
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<tr>
<td>Refraction cylinder power</td>
<td>- blood transfusion</td>
<td>Anxiety</td>
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<tr>
<td>Disc Size</td>
<td>- major surgery</td>
<td>Mental Depression</td>
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<tr>
<td>Cup/Disc Ratio- horizontal</td>
<td>Cardiovascular Disease</td>
<td>Disorientation</td>
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<td>Cup/Disc Ratio-vertical</td>
<td>- HTN, Angina, MI</td>
<td>Family History</td>
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<tr>
<td>IOP</td>
<td>Diabetes</td>
<td>- Glaucoma</td>
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<td>Mean Deviation Index</td>
<td>Malignancy</td>
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<td>Nonmigraine Headaches</td>
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<tr>
<td></td>
<td>Migraine</td>
<td>-Disc Hemorrhage</td>
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<td>Anemia</td>
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Natural progression of LTG is faster in those with:

1. Disc Hemorrhages
2. Migraine (As a possible surrogate for vasospasm or vascular dysregulation)
3. Female Gender
4. Specific Racial Heritage
   • African Americans > Caucasians > Asians
RESEARCH IN VASOSPASM AND LTG

1. 1985 Phelps and Corbett (7)
   - High incidence of migraine in low-tension glaucoma patients
   - Suggested for the first time that vasospastic events might play a role in the optic nerve changes in low-tension glaucoma

2. 1986 Gasser and associates (15)
   - They noted that visual field defects became aggravated by the immersion of a hand in cold water and that the scotoma often improved after they received a calcium channel blocker

3. 1988 Drance and associates (6)
   - Measured blood flow in the finger of normal subjects and in subjects with low-tension glaucoma
   - They found that the mean baseline flow and the flow after exposure to cold was significantly lower in the patients with low tension glaucoma than in the normal subjects
SO WHAT???????????
RESEARCH USING CALCIUM CHANNEL BLOCKERS FOR PATIENTS WITH LTG

1989 KITAZAWA (16)

• Conducted a prospective study to evaluate the effects of Calcium channel blockers on the visual fields in LTG.
• Also analyzed the effects on
  • Systemic Blood Pressure
  • Reactivity of Peripheral Blood Vessels
  • Pulse Rate

CONCLUSIONS:

In some cases of low-tension glaucoma, vasospasm plays a significant role in the development of visual-field defects and Calcium antagonists may be effective in improving the visual field by reversing the vasospastic events.
Ongoing Research → Present 2015

WHERE DOES THAT LEAVE US?
TREATING PATIENTS WITH LTG

LOWERERING IOP

• CNTGS: lowering IOP plays an important role in preventing progression (1)

MODIFICATION OF CARDIOVASCULAR RISK FACTORS

• Ischemia to the optic nerve head may play a role in the pathogenesis of low-pressure POAG. Modification of cardiovascular risk factors is appropriate in managing general health, but has not proven beneficial in managing glaucoma. (1)
TREATING PATIENTS WITH LTG

VASOSPASM

• May play a role in pathogenesis of LTG
• Some investigators advocate the use of systemic CCB in the treatment of this condition (1)

CALCIUM CHANNEL BLOCKERS ARE NOT CURRENTLY STANDARD TREATMENT FOR LTG
Retrospective Study
Comparison of glaucomatous progression in Low-Tension Glaucoma between untreated patients with vasospasm and patients treated with Nifedipine.

Hypothesis
Patients with LTG who have vasospasm and are treated with nifedipine have a more stable course than those not treated with Nifedipine.
Dr. Rosanelli and Dr. Blackshear

Possible Low Tension Glaucoma Patient
  ↓
 Modified Diurnal Curve To Confirm Diagnosis
  ↓
 Vasospasm Work Up With Dr. Blackshear
  ↓
 If Positive-Plan for Nifedipine
  ↓
 Baseline Blood Pressure Readings: 10 different measurements on different days
  ↓
 Initiate Nifedipine 30mg PO daily: Patient records BP measurements on 10 different days
  ↓
 If any decrease in the average diastolic blood pressure, Nifedipine is discontinued. If no change in diastolic blood pressure, Nifedipine treatment is continued.
  ↓
 Patient undergoes repeat vasospasm testing. If condition improves, keep Nifedipine at 30mg. If vasospasm has not responded, continue to titrate the dose to 60mg -90mg until vasospams results improve.
Nifedipine Research

Experimental Group
Those diagnosed with vasospasm and treated with Nifedipine

Control Group
Those diagnosed with vasospasm and not treated with Nifedipine

Comparing the survival of patients with this disease.
To be continued......

Plan to report data at our next conference: May 2016 AOCOO-HNS Scottsdale, AZ

May 2016 The Association for Research in Vision and Ophthalmology conference in Seattle Washington
REFERENCES


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