GL-12: Under Pressure: Ocular Perfusion, Nocturnal IOP and Eye Disease

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Saturday, October 27, North Ballroom, 9:00-10:00 AM

I. Ocular Perfusion Pressure

A. Difference between arterial BP and the intraocular pressure (IOP)

1. Mean Perfusion Pressure (MPP)=

2/3 [diastolic BP + 1/3 (systolic BP – diastolic BP)] - IOP

2. Systolic PP (SPP)= Systolic BP – IOP

3. Diastolic PP (DPP)= DBP - IOP

B. Alterations can cause ischemia and poor irrigation of tissues in optic nerve

1. Can be due to low BP or relatively high IOP

2. During nocturnal period, potential for both low BP and high IOP

C. Ocular Perfusion Pressure: risk factor for development of new glaucoma and progression of established glaucoma

1. Barbados Eye Study- low OPP (< 55 mm Hg) associated with 3 times increased risk of glaucoma

2. Baltimore Eye Survey – low DPP (<30 mm Hg) associated with 6 times higher risk than DPP > 50 mm Hg

3. Egna-Neumarket Study – low DPP (< 68 mm Hg) associated with marked, progressive increase in frequency of glaucoma

4. Early Manifest Glaucoma Trial – low SP, lower SPP, and cardiovascular disease at baseline progressed faster than counterparts

5. Los Angeles Latino Eye Study – both low diastolic and high systolic BP associated with increased prevalence of open-angle glaucoma

6. Rotterdam Study – only patients on antihypertensive medications, 4.68 times greater chance of developing glaucoma in patients with low DPP < 50 mm Hg compared to DPP > 65 mm Hg

7. Thessaloniki Eye Study – cup area, C/D ratio were increased and rim area decreased in subjects with lower DBP resulting from treatment compared to high DBP and untreated normal DBP groups

II. Ocular Perfusion Pressure: Nocturnal influences on IOP

A. Management Dilemma

1. Glaucoma patients with advanced disease and/or progression in spite of low pressures during office visits
2. Compliance
3. Diurnal curve
    i. Serial Tonometry, IOP checks in AM or PM

B. Nocturnal IOP

1. 24 hour IOP monitoring
    i. Highest IOPs often measured during period where patient is not in office.
    ii. Greater fluctuation of IOPs
2. Both healthy and glaucoma patients experience increase in IOP overnight
3. Office readings may not give accurate clinical picture
    i. Exercise caution when extrapolating results of in-office IOP measurements when making glaucoma treatment decisions
4. Sleeping on back- Supine position
    i. IOP increases – due to ncreased episcleral venous pressure?
    ii. Nocturnal IOP increase still seen even when all IOP measurements performed around the clock in supine position; more than just supine position involved
5. Glaucoma patients greater IOP rise when change from upright to horizontal vs. normals
    i. Greater IOP variation with more advanced disease
    ii. Worsening of visual field in NTG associated with IOP in supine position and magnitude of IOP elevation with postural change

6. Sleeping on side
    i. Larger C/D ratio on preferred side for sleeping
7. Sleeping on stomach – Prone position
    i. Significantly higher IOP than supine position
    ii. Angle closure risk
8. Management Pearls
    i. Elevating head by using pillow neutralized IOP increase
    ii. Supine position better for IOP than prone position, especially in patients with potential for angle closure

C. Obstructive Sleep Apnea

1. Pauses in breathing or abnormally low breathing during sleep
2. Characterized by snoring, restless sleep, daytime drowsiness
3. Continuous Positive Airway Pressure (CPAP)
    i. Keeps airway open during sleep
    ii. Also increases IOP, but believed that oxygen delivery more important to glaucoma than IOP increase
4. Glaucoma
    i. Higher prevalence of glaucoma in OSA patients than general population
    ii. Ischemic Mechanism: disrupted blood flow from either less blood reaching optic nerve or less oxygen in blood that does reach optic nerve

D. IOP lowering treatment

1. Beta blockers and alpha 2 agonists not as effective, natural 50% aqueous production decrease over night
2. Prostaglandin analog have sustained 24-hour IOP lowering effect
3. Carbonic anydrase inhibitors shown to be better than beta blockers and alpha 2 agonists at controlling nocturnal IOP
4. SLT showed benefit in nocturnal IOP measurements even if did not show response during the day

5. Trabeculectomy controlled 24-hour IOP better than maximum medications

III. Ocular Perfusion Pressure: Nocturnal Hypotension

1. Hypertension

    i. One of most important risk factors for cardiovascular morbidity and mortality

    ii. Risk of cardiovascular mortality doubles with each 20 mm Hg rise in systolic BP and each 10 mm HG rise in diastolic BP

2. Hypotension – not ideal either

    i. Stroke, heart attack, renal damage, end-organ damage

    ii. "White Coat Syndrome"

    iii. Orthostatic Hypotension: flashes and floaters with postural changes

    iv. During sleep, decrease in sympathetic nervous system – leads to nocturnal dip in blood pressure ~ 10-15%

        i. 10-15% = dippers, <10% = non dipper, <15% = over dipper

3. Glaucoma and Blood Pressure

    i. Systolic BP increase of 10 mm HG is equivalent to 0.23 – 0.32 mmHg higher IOP. Diastolic BP increase of 10 mm Hg equivalent to 0.19 to 0.60 increase in IOP

    ii. Low BP and nocturnal over-dipping associated with increased probability of VF deterioration in glaucoma patients

    iii. Excessive reduction of OPP in overdippers may lead to short term ischemia followed by reperfusion damage and glaucomatous damage

    iv. Rotterdam Study: increased risk of glaucoma with calcium channel blockers compared to beta blockers

    v. Hayreh Study: if diastolic BP decreased below critical level, beneficial effect is lost and mortality/morbidity rates increase again. Avoid excessive reduction of BP overnight

    vi. If not contraindicated, consider changing blood pressure medication dosage time to morning

4. Questions still remain
5. Cause or the effect? Is decreased blood flow involved in etiology of glaucoma or secondary from loss of retinal ganglion cells and consequent decrease in metabolic demand for oxygen and nutrients
6. Is increased blood flow beneficial for glaucoma patients? No evidence at this time to support the idea that increasing optic nerve perfusion would protect retinal ganglion cells from damage.
7. Difficult to use a treatment strategy for glaucoma that seeks to increase OPP by increasing blood pressure due to risk of cerebrovascular morbidity and mortality

IV. Ocular Perfusion Pressure and Ischemic Optic Neuropathy

A. Non-Arteritic Ischemic Optic Neuropathy (NAION)

1. Temporary hypoperfusion or nonperfusion of anterior optic nerve circulation
2. Predisposing risk factors
    i. Systemic: HTN, nocturnal hypotension, diabetes, hyperlipidemia, atherosclerosis
    ii. Optic Nerve Head: absent or small cup, location of watershed zones
3. Precipitating risk factors, “last straw”: Nocturnal Hypotension
    i. Nocturnal hypotension: Normal drop in blood pressure during sleep; if falls below critical auto-regulatory range in susceptible person, can develop NA-AION
    ii. Wary of night time dosage of blood pressure medications causing even greater drop in perfusion pressure
    iii. Patients typically develop vision loss during sleep and discover it on waking in the morning, ~ 80%
    iv. Erectile dysfunction medications
4. Watershed Zones
    i. Border between 2 territories of 2 end arteries are areas of comparatively poor vascularity
    ii. Decrease in ocular perfusion pressure makes areas vulnerable to ischemia

B. Posterior Ischemic Optic Neuropathy

1. Decreased blood flow in posterior part of nerve
2. Surgery
    i. Prolonged, major surgical procedure
    ii. Severe hypotension – prolonged general anesthesia, surgical trauma, massive blood loss
    iii. Reduced blood flow to optic nerve and/or ischemia
3. Less common than anterior ischemic optic neuropathy
    i. PCA is end-arterial system without anastomoses with surrounding arteries
    ii. Posterior part of nerve is supplied by pial vascular plexus, NOT an end-arterial system because various collateral branches supplying it anastomose freely with one another in pial plexus

V. Management

A. Remember that IOPs fluctuate and tend to be highest over night

B. Consider alternate etiologies for glaucoma progression in presence of low IOPs in office

1. Consider low perfusion pressure in patients with hypotension and glaucoma
2. Blood pressure readings as part of routine eye exam
3. Medical history for sleep apnea (CPAP)
4. Sleep position: supine better than prone position for IOP, sleeping with pillows may help IOP as well
5. Beta blocker and alpha 2 agonists have shown less efficacy at controlling over-night IOPs compared to carbonic anhydrase inhibitors and prostaglandin analogues

C. Avoid excessive lowering of BP at night in patients with low perfusion. Consult with PCP

1. NA-AION and advanced glaucoma, consider change night time dosage of blood pressure medications to morning if no medical contraindication
2. Discontinue Viagra in patients with NA-AION

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