SD-06: Preventing Stroke in Your Patients

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Wednesday, October 24, Room 121 A-C, 2:00 – 3:00 PM

I. Stroke/ Cerebrovascular Accident (CVA)
   A. Definition
      1. Loss of brain function due to sudden interruption in blood flow through the brain or thrombus of a blood vessel in the brain
   B. Classification
      1. Ischemic (approx 87% of strokes)
         - Interruption of blood supply to area of brain
         - Thrombosis – obstruction of BV by local clot
         - Embolism – obstruction due to emboli from elsewhere in body
         - 3 hours, brain suffers irreversible injury, death of tissue
         - Tissue plasminogen activator (TPA)
      2. Hemorrhagic
         - Headache, vomiting
         - Rupture of BV or abnormal vascular structure
         - Intra-axial hemorrhage – blood inside brain
         - Extra-axial hemorrhage – blood outside brain but inside skull
            a. Epidural, subdural hematoma
            b. Subarachnoid hemorrhage
         - Blood compresses adjacent areas and can distort/injure tissue
   C. Clinical Symptoms
      1. Weakness on side of body in face, arm, and/or leg
      2. Slurred speech
      3. Confusion
      4. Dizziness, loss of balance
      5. Paresthesias
      6. Headache
      7. Ocular
         - Blurred vision in 1 or both eyes
         - Partial vision loss/ field defect
         - Double vision
   D. Risk Factors
      1. Hypertension
      2. Diabetes
      3. High Cholesterol
      4. TIA
      5. Smoking
      6. Atrial Fibrillation
   E. Imaging

1. Computed Tomography (CT)
2. Magnetic Resonance Imaging (MRI)

F. Treatment
1. Thrombolysis with TPA – dissolves clot
2. Antiplatelet drugs
3. Blood pressure medications
4. Statins
5. Carotid Endarterectomy
6. Anticoagulants

G. Correlation to the Eye
1. Blood flow to eye, Ophthalmic via carotid artery
2. Retinal blood vessels share similar anatomic, physiological, and embryological characteristics to cerebral vessels
3. Ocular signs as clues to cerebrovascular disease

II. Patient Ocular Symptoms

A. Transient Monocular Vision Loss (TMVL)
1. Causes
   - Embolic, thrombotic, hematological, vasospastic
2. Transient Ischemic Attack (TIA)
   - Can result in painless monocular vision loss or no ocular involvement at all
   - Symptoms similar to stroke, but typically lasting a few minutes and causing no permanent damage
   - Signs/symptoms lasting longer than 24 hours or causing lasting brain damage constitutes a stroke
   - TIA strong predictor of subsequent stroke, 90 day risk 9.5% to 14.6%
3. Amaurosis Fugax
   - Transient monocular blindness
     a. Painless, monocular vision loss, total or sectoral
     b. Last few seconds to few hours, always resolves completely
4. ABCD and $ABCD^2$ score
   - Age > 60 = 1 point
   - Blood pressure: Systolic > 140 and/or diastolic > 90 = 1 point
   - Clinical symptoms
     a. Unilateral weakness/numbness = 2 points
     b. Speech disturbance = 1 point
   - Duration
     a. $0 < x < 10$ minutes = 0 points
     b. $10 < x < 59$ minutes = 1 point
     c. > 60 minutes = 2 points
   - Score of 5 or 6 = 7-8 fold greater 30 day risk of stroke
     a. Early initiation of treatment after TIA or minor stroke associated with 80% reduction in risk of early recurrent stroke (EXPRESS study)

- $ABCD^2$
    a. Diabetes = 1 point
    b. Score can predict individuals with high risk of early and very early stroke (2, 7, 30 days) after TIA or minor stroke
    c. Also useful in predicting long term stroke risk after TIA up to 54 months
    d. 90 day risk of stroke 7 fold higher in patients > 3 points

5. Management
    - Based on $ABCD^2$ score, consider emergent referral to local emergency room for SAME DAY observation/treatment
    - Patients with low score, inform primary care provider of suspected TIA and refer for proper evaluation and work-up within 1 week
        a. Consider daily aspirin until primary care exam

III. Patient Ocular Signs

A. Terson's Syndrome

1. Characteristics
    - Vitreous hemorrhage associated with subarachnoid hemorrhage (SAH)
    - 13% with SAH had evidence of vitreous hemorrhage
    - Vitreous hemorrhage associated with worse outcome than patients with SAH without vitreous hemorrhage
    - Consequence of ruptured cerebral aneurysm
    - Mild retinal hemorrhages associated with better prognosis than large preretinal hemorrhages and/or vitreous hemorrhage
    - Average age 51.7 years
    - 2 times more common in women than men
    - In patients with SAH, death more common in patients with Terson's syndrome than in those without (43% vs. 9%)
2. Management
    - Suspect aneurysmal rupture in patient with any retinal hemorrhage who has temporarily lost consciousness
        a. 1/3 of SAH cases
        b. Internal carotid artery and anterior communicating artery aneurysms resulted in most severe hemorrhage in 1 study
    - Conscious patients almost always report sudden onset of severe headache due to rapid rise in intracranial pressure
        a. Case report of Terson's syndrome who suffered SAH w/o headache
        b. Patient may also forget to report transient severe headache due to persistent loss of vision
    - Emergent CT scan in patients that have no explanation for vitreous hemorrhage and report recent severe headache and/or loss of consciousness

B. Cholesterol Deposits

1. Xanthelasma

- Flat or minimally elevated yellow lesions, most common medial eyelid
- Bilateral and symmetric
- 50% have essential hyperlipidemia

2. Corneal Arcus
    - Arcus Senilis vs. Arcus Lipoides
    - Bilateral gray, white circumferential deposit in peripheral cornea
    - Younger patients (< 40 years old) greater risk of death from cardiovascular disease
3. Schnyder's Crystalline Dystrophy
4. Management
    - Evaluate for systemic lipid abnormalities
    - Surgical excision of Xanthelasma with larger or cosmetically unacceptable lesions

C. Hypertensive Retinopathy
1. Characteristics
    - Related to small vessel arteriosclerosis, retinal ischemia, breakdown of blood retina barrier
    - Microaneurysms, flame shaped or dot blot hemes, cotton wool spots, hard exudates, AV nicking, arteriolar narrowing, copper/silver wire, disk edema
    - Other signs of uncontrolled hypertension – increase stroke 4-6 fold
        a. Malignant hypertension
        b. Retinal arterial macroaneurysm
2. Risk of Stroke
    - Most important modifiable risk factor for stroke
    - Atherosclerosis Risk on Communities Study (ARIC) - AV nicking, focal retinal arteriolar narrowing, microaneurysms, CWS, associated with increased risk of MRI-detected silent cerebral infarcts (2-3 times increased risk)
    - Blue Mountain Eye Study (BMES) and Beaver Dam Eye Study (BDES)- both focal retinal arteriolar narrowing and AV nicking at baseline associated with increased risk of incident stroke or stroke mortality at 7 and 10 years
3. Management
    - Measure blood pressure in office, co-manage with primary care provider
    - Patient education

D. Diabetic Retinopathy
1. Risk of stroke in Non Proliferative Diabetic Retinopathy (NPDR) and PDR
    - Type 2 Diabetes and NPDR – incident ischemic stroke higher in mild NPDR vs. no DR in ARIC Study
    - Type 2 Diabetes and PDR – 6 fold higher risk incidence of stroke, risk of stroke mortality was 2 fold higher in Wisconsin Epidemiological Study of Diabetic Retinopathy (WESDR)

2. Asymmetric Proliferative Diabetic Retinopathy and Carotid Artery Disease
    - PDR in 1 eye, NPDR in fellow eye OR 2 stage/grade difference in retinopathy (ie: mild retinopathy OD vs severe retinopathy OS)
    - Found in approximately 5 to 10% of diabetic patients
    - Risk Factors
        a. Carotid artery disease, BRVO, cataract surgery, vitreous loss, trauma, radiation, uveitis, tumor
    - Protective Factors – reduction in metabolic requirements reduces ischemic stimulus for neovascularization
        a. Chorioretinal scarring, optic atrophy, myopia, RPE dystrophy, complete PVD, amblyopia
    - Worse retinopathy on side with more patent flow (Gay and Rosenbaum, Arch Oph 1966)
        a. Ophthalmodynamometry: retinal diastolic difference of 15% or more between the 2 eyes in 8 of 10 patients
        b. Eye with less advanced or minimal DR always on same side as carotid insufficiency
        c. Carotid disease retarding progression of DR in ipsilateral eye or accelerating it in the contralateral eye
    - Worse retinopathy on side with worse carotid stenosis vs. chance
        a. Only 1 patient in study documented with unilateral carotid vascular disease and showed only background DR in involved side, not PDR (Browning et al. Asymmetric Retinopathy in Patients with Diabetes Mellitus AJO 1998)
        b. 4 of 20 patients with asymmetry diagnosed with carotid artery stenosis. 2 of 4 patients with PDR ipsilateral to severe carotid stenosis. (Duker et al. Asymmetric Proliferative Diabetic Retinopathy and Carotid Artery Disease. Ophthalmology 1990)
        c. Ocular Ischemic Syndrome (OIS) and DR additive in patients with carotid stenosis ipsilateral to the proliferative retinopathy
3. Management
    - Emphasize glycemic control
    - Patient education about signs of stroke
    - With 2 level difference in retinopathy, consider carotid ultrasound to rule out hemodynamically significant stenosis

E. Ocular Ischemic Syndrome (OIS)
1. Venous Stasis Retinopathy OU
    - Early stage of ocular ischemia, often asymptomatic
    - Chronic low perfusion pressures, diffuse retinal ischemia
    - Dilation, irregularity of caliber, tortuosity of BV
    - Mid-peripheral hemorrhages and microaneurysms
    - ~9% develop OIS

2. Chronic Ocular Ischemia
    - Progressive visual loss
    - Mid peripheral hemorrhages and microaneurysms in over 80% cases
    - Macular edema, disk edema, retinal artery occlusion
    - NVE, NVD, NVI, NVA, neovascular glaucoma (High Risk)
    - Corneal edema, mild a/c reaction
    - Asymmetric IOP due to low perfusion of ciliary body
    - Hypotony: cataracts, K edema, maculopathy
    - Dull periocular pain
3. Management
    - Patient education about signs/symptoms of stroke
    - Monitor regularly for development of neovascularization
        a. Refer to retinal specialist for panretinal photocoagulation when appropriate
        b. Refer to glaucoma specialist if neovascular glaucoma develops
    - Carotid ultrasound
        a. Refer to vascular surgeon for consultation when appropriate
        b. Carotid Endarterectomy, Stent

F. Retinal Arterial Emboli
1. Types of Emboli
    - Cholesterol
    - Calcific
    - Fibrino-platelet
2. Background
    - 1% of patients over age of 40
    - Distal portion of occluded artery may be ischemic due to occlusion
        a. Branch Artery Occlusion
        b. Central Artery Occlusion
        c. Cilioretinal Artery Occlusion
    - With or without occlusion, presence of emboli puts individual at higher risk of stroke and mortality from cardiovascular disease
3. Risk of Stroke
    - Associated with high risk of stroke-related death, 3 fold greater in individuals with retinal emboli over 8 year period in BDES
    - 10-fold increase in annual rate of stroke: 8.5% patients with emboli vs. 0.8% of patients w/o emboli. 71% of stroke cases involved carotid artery on same side of embolus (Bruno et al. Vascular outcome in men with asymptomatic retinal cholesterol emboli. A cohort study. Ann Intern Med.1995)
    - Asymptomatic patients with emboli
        a. 18% had internal or common carotid artery stenosis > 75%
    - Hollenhorst Plaque

a. Plaques associated with positive scan(60% if greater stenosis) in 18.2% of carotid arteries (McCullough et al. Journ Vasc Surg 2004)
b. Presence of plaque or retinal artery occlusion is not associated with high risk hemispheric neurologic event (Dunlap et al. J Vasc Surgery 2007)
c. 80% of people with asymptomatic retinal emboli do not have significant carotid stenosis (Bruno et al. Stroke 1992)

4. Management
   - Patient education about signs/symptoms of stroke
   - Work-up to find origin of plaque (Carotid artery vs heart valve)
     a. Carotid Ultrasound and Echocardiogram
     b. Hollenhorst, Calcific, Fibrinoplatelet plaques
   - Monitor for neovascularization with retinal artery occlusion
   - Baseline visual field

IV. Management of Stroke Patients

A. Homonymous Visual Field Defects

1. Visual Field Testing
   - Binocular Confrontation Visual Fields
     a. Detect homonymous defect
     b. Test superior and inferior quadrant with both hands
   - Monocular Confrontation Visual Fields
   - Degree of field loss
     a. Monocular test, have patient fixate on nose and bring red cap from periphery towards noses from 8 directions
     b. Formal visual field testing still standard
2. Complete Homonymous Hemianopia
   - 8% of all stroke patients
   - Middle cerebral or posterior cerebral artery stroke affecting either optic radiation or visual cortex of occipital lobe
   - Tip of occipital lobe may receive dual blood from posterior cerebral artery and middle cerebral artery -- > Macular sparing
   - 54% of homonymous field defects in stroke were occipital lobe lesion
3. Quandrantanopia
   - "Pie in the sky" – optic radiation inferiorly in temporal lobe
   - "Pie in the floor" – optic radiation of parietal lobe
   - P.I.T.S. = Parietal – Inferior; Temporal – Superior
   - 33% of homonymous field defects in stroke were optic radiation lesions
4. Recovery
   - Spontaneous VF improvement can occur in up to 50% of patients usually within first 3-6 months and at a slower rate after 6 months
   - Tender patient expectations

B. Other ocular manifestations of stroke

1. Abnormal eye movements
2. Poor saccadic eye movements
3. Poor maintenance of gaze
4. Reading difficulties
5. Poor central vision in up to 27%
6. Cranial Nerve Palsies in ~18%

V. Patient Management
   A. Patient education of signs/symptoms of stroke
   B. Co-manage with primary care provider
      1. Control hypertension, dyslipidemia, diabetes, smoking
      2. Initiation of anticoagulation with aspirin alone, or aspirin and antiplatelet agent may delay onset of CVA and prolong a patient's life
   C. Order appropriate testing
      1. Carotid Ultrasound
         - TMVL
         - Asymmetric diabetic retinopathy, PDR
         - Retinal arterial emboli
         - Venous stasis retinopathy
         - Ocular Ischemic Syndrome
      2. Echocardiogram
         - Cardiac and thoracic sources of retinal emboli
   D. Moderate and High Risk patients
      1. TIA with $ABCD^2$ score of 3 or higher, Terson's Syndrome
      2. Emergency referral to hospital emergency room for imaging/evaluation

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