Posterior Circulation Cerebrovascular Accident

CLINICAL

- Often complex symptoms, can vary from mild to extreme
- Unilateral or bilateral sensory loss
- Visual field deficits
- Vertigo, diplopia, dysarthria, dysphagia, loss of balance
- Usually two of the following: dizziness, slurred speech, double vision, dysphagia, and unilateral or bilateral weakness

ETIOLOGY

- Vertebrobasilar circulation occlusive disease
- Vertebral dissection
- Atherothrombotic, embolic, inflammatory, mechanical, or hypercoaguable
- Atrial fibrillation- 20 % of cardiac emboli lodge in posterior circulation

WORK UP

- MRI Brain
- MRA of both intracranial and extracranial circulation
- Emergent CT head if bleed is suspected
- Consider 4-vessel catheter angiography (gold standard)
- Color Duplex Ultrasound
- Transesophageal echocardiogram

TREATMENT

- Antiplatelets- Aspirin, Aggrenox, or Plavix
- If cardio embolic source or dissection, then Coumadin for six months
- Vertebrobasilar stenting
- Modification of risk factors

Benign Paroxysmal Positional Vertigo

CLINICAL

- Most common vestibular disorder (20%)
- Involves the semicircular canals
- Brief attacks of vertigo precipitated by rapid extension/flexion of head or head tilt
- Typically experience symptoms by rolling over in bed, lying/sitting, or bending over
- Illusion of movement is usually rotatory
- Associated nausea, diaphoresis, tachycardia
- 10-20 seconds long, never longer than 1 minute, but flurry of attacks can occur
- Episodic, can last days to several months

ETIOLOGY

- Dislodged otolith moving freely within endolymph of semicircular canals
- Advanced age
- Head trauma
- Other ear disease
- Migraine
- Bed rest
- Idiopathic
- Peripheral disease

WORK UP

- Dix Hall pike maneuver
- Nystagmus must torsional, latent, less than 60 seconds, reversible, and fatigues
- If Nystagmus occurs during positional testing, no further work up is indicated
- If central cause cannot be ruled out, then MRI of the posterior fossa is indicated

TREATMENT

- Self-limiting
- Epley maneuver
- Vestibular rehab/exercises
- Vestibular sedatives, antiemetics

Multiple Sclerosis

CLINICAL

- Lesions in the brain and spinal cord damage every function of the central nervous system
- Fatigue, disturbed sensation, motor function, bladder function, dowel function, sexual function, cerebellar, brainstem, optic nerve, cognition
- Lesions primarily in periventricular white matter
- Intranuclear ophthalmoplegia present
- Symptoms arise over hours to days, can last from 2-6 weeks, and can resolve completely
- 40% of attacks cause long lasting deficits

ETIOLOGY

- Unclear
- Attack against myelin and myelin forming cells in brain and spinal cord
- Three phases- initiating event, recovery from relapse, chronic progression

WORK UP

- Contrast enhanced MRI of Brain and Cervical Spine
- Lumbar puncture with CSF for oligoclonal bands
- VEP

TREATMENT

- Management of symptoms
- Immunosuppression, immunomodulation
- Steroid therapy for acute exacerbation
- Plasmaphoresis
- Intravenous immunoglobulin

Guillain-Barre Syndrome

CLINICAL

- Major complaint is ascending weakness, although can begin in arms and face
- Progressive over 2-4 weeks
- Facial diplegia develops in >50% of cases
- Severity varies from gait difficulty to respiratory failure
- Transient urinary retention, autonomic dysfunction
- Late in course is objective sensory loss

ETIOLOGY

- Cell mediated autoimmune disease of peripheral nerves
- 2/3 of cases follow viral illness- CMV, EBV, Smallpox, Hepatitis B and C, HIV, C. Jejuni
- Can also occur after vaccinations, surgery, epidural anesthesia, thrombolytic agents, and heroin use

WORK UP

- Abnormal Electromyelogram and Nerve conduction study
- Increased protein in CSF after one week

TREATMENT

- Steroids of little benefit
- Plasmaphoresis
- Intravenous Immunoglobulin
- Prevention and monitoring of complications (respiratory and vascular collapse)
- Note: Patients will be sensitive to succinylcholine, muscle relaxants, and local anesthesia

Transverse Myelitis

CLINICAL

- Symptoms evolve over one week
- Nonspecific fever and myalgias
- Signs are bilateral, but usually some longitudinal, expanding spinal cord pathology
- Acute partial cord lesions cause unilateral or markedly asymmetric bilateral sensory and motor dysfunction
- First symptoms are ascending paresthesias and/or back pain at level of myelitis, plus leg weakness and sphincter dysfunction
- Tingling or paresthesias progress to loss of pain, temperature, vibration that halts at sensory level (usually thoracic- 80%)
- Weakness is severe, in 2/3 of cases, often leads to total leg paralysis and spasticity
- 5% of patients have respiratory symptoms
- Bladder and bowel function frequently lost
- Maximal improvement at six months

ETIOLOGY

- 2/3 of cases are idiopathic
- 1/3 of cases thought to be triggered by a virus
- Typically develops in 3-15 days after upper respiratory infection
- Lesions restricted to the spinal cord

WORK UP

- MRI with contrast of the entire Neuroaxis, rule out cord compression, vascular or connective tissue disease
- CBC with differential, C-reactive protein, ANA
- CSF analysis shows increased protein, moderate pleocytosis. CSF pressure and glucose will be normal. CSF lacks oligoclonal bands

TREATMENT

- Symptomatic management
- Treat underlying disease process
- Oral/IV glucocorticoids and steroids
- Plasma exchange

Idiopathic Intracranial Hypertension (Pseudotumor Cerebri)

CLINICAL

- Papilledema present in almost all patients, headache present in 90% of patients
- Most often in obese females
- Visual signs are frequent, transient visual disturbances in ¾ of patients; often describe complete loss of vision from seconds to minutes
- Abducens palsy, optic disc swelling with blurring of margins
- Back and shoulder pain, weakness, numbness, tinnitus, intracranial noises, and pulsatile tinnitus
- Elevated opening pressure >250 mm of water with LP, normal CSF composition
- Absence of hydrocephalus, mass, structural, or vascular lesion
- "Secondary" intracranial hypertension includes medication, dural venous thrombosis, and spinal cord tumors
- Important to investigate for secondary causes before concluding idiopathic

ETIOLOGY

- Cause unknown
- May be attributed to cerebral venous hypertension
- May be due to abnormalities of CSF production and absorption
- Linked to polycystic ovarian syndrome, obstructive sleep apnea and obesity
- Less common after 44 years old

WORK UP

- MRI brain with contrast
- Lumbar puncture and measurement of opening pressure
- Ophthalmologic evaluation of visual acuity, the fundus, visual field plotting

TREATMENT

- Weight loss, serial LP, salt and fluid restriction, diuretic treatment, and carbonic anhydrase inhibitors
- Discontinuation of causative agent
- Corticosteroids reserved for emergent management of impending visual loss
- Surgical optic nerve sheath decompression
- Shunting

Myasthenia Gravis

CLINICAL

- Weakness and premature fatigue affecting ocular, bulbar, and peripheral muscles
- Dysfunction of swallowing muscles and respiration particularly dangerous
- Progressive dysphagia and dysarthria
- Diplopia and ptosis
- Ocular manifestations may be only initial symptom
- Improved symptoms at rest differs from fixed central nervous system dysfunction
- Decreased up gaze at target for 30-60 seconds is a specific provocative test for ocular myasthenia
- Mild photophobia due to fatigability of constrictor pupillae
- Head drop
- Proximal extremity weakness after exertion, often misdiagnosed as myopathy

ETIOLOGY

- Acetylcholine receptor antibodies
- Structure and function of neuromuscular junction is altered
- Associated with thymoma

WORK UP

- Acetylcholine receptor antibody assay
- CT chest for thymoma
- Highly a clinical diagnosis
- Anti-MUSK antibody assay
- Repetitive stimulation testing (EMG)

TREATMENT

- Monitor pulmonary function
- Hospitalization may be required
- Thymetomy of thymoma present
- Corticosteroids
- Azathioprine
- Mestinon
- Cellcept
- Cyclosporin
- Cyclophosphamide
- Plasma exchange

Transient Global Amnesia

CLINICAL

- Abrupt onset
- Anterograde memory is impaired
- Disoriented to time, place, but never person
- Patients often repeat motor actions and questions, and recognize memory deficits
- Retrograde memory disturbances can last hours to years
- Still able to do complex tasks (driving, reading, writing)
- Personality unchanged
- Onset usually > 50 years old
- Male > female
- 1/3 of attacks have identifiable physical or psychological precipitant

ETIOLOGY

- Unknown
- Possibilities include TIA, seizure, venous thrombosis, CNS tumor, Drug intoxication, toxic/metabolic disturbances, and hysteria
- Hippocampus involvement
- Increased risk in patients with migraine

WORK UP

- Identify and precipitating stressors
- Neuroimaging of brain to rule out tumor or lesion
- Electroencephalogram to evaluate for seizure activity, especially if impaired consciousness

TREATMENT

- No preventive treatment
- Consider migraine prophylaxis

Normal Pressure Hydrocephalus

CLINICAL

- Gait apraxia, dementia, and urinary incontinence are classic triad
- Insidious onset, symptoms fluctuate daily
- Fecal incontinence is rare
- Postural instability
- Diminished postural reflexes, babinski signs may be present
- Upper extremities often spared
- Must distinguish lower extremity weakness from gait difficulties
- Cognitive deficits, apathetic, bradyphrenic

ETIOLOGY

- 50% idiopathic
- Secondary causes include subarachnoid hemorrhage, head trauma, meningitis, elevated CSF protein, aqueductal stenosis, arachnoid cysts of 3rd and 4th ventricles, basilar artery ectasia, intracranial and spinal tumors
- Enlargement of ventricles
- 6th and 7th decade

WORK UP

- LP will have normal opening pressure; symptoms may improve significantly post LP
- CT or MRI of brain will show enlargement of ventricles relative to sulci

TREATMENT

- Shunt

References

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