Trigeminal Neuralgia

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Trigeminal Neuralgia- Typical (TN)

- Severe, paroxysmal, intermittent, lancinating pain on one side of the face
- Sudden onset (electrical shock like)
- Triggered by touching the face, brushing teeth, chewing, cool wind blowing on face, air conditioning, talking
- May be triggered by changing head position
- Patients can usually remember exactly when the first event occurred because the pain is so memorable
- May resolve spontaneously and then return
- No neurologic deficits except for possible decrease in sensation to face and/or cornea

Trigeminal Neuralgia- Atypical

- Unilateral face pain similar to typical TN but having some different characteristics such as persistent, no triggers, etc.
- May develop in an individual who formerly suffered from typical TN

Facial Pain

- May be unilateral or bilateral
- May be burning or aching
- May have numbness associated
- No typical triggers
- Longer lasting or continuous

Differential Diagnosis

- Typical TN
- Atypical TN
- Facial pain syndrome
- TN secondary to multiple sclerosis and TN demyelinating plaque in pons (2% of patients with MS have TN; 18% of patients with bilateral TN have MS)
- Herpes simplex infection with shingles
- TMJ
- Dental disease
- Orbital disease
- Giant cell arteritis
- Tumor(carcinoma, meningioma, acoustic neuroma)
- Arteriovenous malformation
- Traumatic nerve injury

What Causes Trigeminal Neuralgia?

- Compression of the trigeminal nerve as it exits the pons by arteries and/or veins
  - Typical offending vessels are superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), pontine veins
  - **DO NOT** underestimate venous pathology especially in young females who have some atypical pain components to their history
- Compression may lead to demyelination of different types of pain fibers within the TN.
- Demyelination may allow electrical impulses to travel between these different types of nerve fibers so that one type of sensation such as touch is interpreted by the brain as severe pain
  - Ultimately the underlying neurologic etiology is still unknown

Medical Therapy

- Anti-seizure medications may work by raising the threshold for demyelinated nerve fibers to trigger one another with aberrant signals
  - Carbamezapine (Tegretol)
  - Phenytoin (Dilantin)
  - Gabapentin (Neurontin)
- Baclofen (Gabapentin)
  - Stimulates GABA receptors to control pain gating system

Typical TN

- In terms of medical therapy most cases of typical TN should improve with the use of Tegretol
- When TN controlled by Tegretol recurs in a severe form it can often be controlled temporarily using IV Dilantin

Surgical Treatments

- Microvascular decompression (see **video** in video section of this web site for surgical procedure and information)
- Glycerol Rhizotomy
- Radiosurgery
- Percutaneous Balloon Compression
- Percutanous radiofrequency rhizotomy

Benefits of MVD Over Percutaneous Procedures

- >85 - 90%% initial cure rate
- 15% recurrence rate at 5 years (lowest of all procedures)
- Long term recurrence rate lowest of all procedures
- Lowest incidence of facial numbness of all procedures

Risks of MVD (not all inclusive)

- Stroke (<5%; may be higher if there is extensive venous pathology secondary to venous infarction if veins need to be sacrificed)
- Death (<1%)
- Hearing loss/reduction (<1%)
- Facial weakness (<1%)
- Facial numbness (5%; may be higher if thre is extensive venous compression)
- Double vision (<3%)
- Infection (5%)
- Spinal fluid leak (5%)