QUESTION 26 Trigeminal neuralgia
Which one of the following best explains the mechanism of trigeminal neuralgia?
A. Herpes virus ganglionitis.
B. Microvascular compression.
C. Central demyelination.
D. Vascular steal phenomena.
E. Trigeminal neuroma.

Trigeminal nerve: sensory & motor supply to the muscles of mastication
3 major divisions: ophthalmic, maxillary & mandibular

Differential diagnosis
TN is most common cause of facial pain
Migraine pain is deep seated and steady
Cluster headache is associated with TN
Temporal arteritis: superficial facial pain is present but not shock like and ESR is high
If TN develops in young adults, exclude MS
- cause is a demyelinating plaque at the root entry zone of the 5th nerve in the pons
- evidence of facial sensory loss

Clinical features
- paroxysms and is maximal at or near onset
- Described as “electric shock” or “stabbing”
- V2/V3 are more frequently involved than V1
- Pain usually last from one to several seconds but may occur repetitively
- Unilateral + deep continuous dull pain
- Unilateral, severe,brief, stabbing recurrent episodes of pain in the distribution of one or more branches of the 5th cranial (trigeminal) nerve
- Trigger zones: lightly touching area triggers attack, chewing, talking, brushing teeth, cold air, smiling and grimacing
- Course: last weeks or months, recurrence is common. Some patients have continuous pain

Pathogenesis
Caused by compression of nerve root → demyelination of large fibers
1. aberrant loop of artery or vein (80-90%) often superior cerebellar artery
2. vestibular schwannoma (acoustic neuroma)
3. meningioma
4. epidermoid cyst
5. AV malformation

Diagnosis
Based upon characteristic features
Trigger zones demonstrated on physical examination (located near midline)
Search for ipsilateral dental pathology

Imaging: MRI – rule out mass lesion or MS
Pick up:
demyelinating lesions
mass lesion in cerebellopontine angle
ectatic blood vessel
serious structural lesions (tumour, MS)
Medical Therapy
1st line:
Carbamazepine (most effective)

Should be done in
Young patients (<40)
Sensory loss
Not responding to conservative therapy
Bilateral symptoms
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- Responding to carbamazepine is diagnostic for TN
- Blocks Na and K conductance
- Dose is 100 – 200mg bd
- Dose can be gradually increased by 200mg until pain relief occurs
- Maintenance dose is 600 – 1200mg
- S.E: drowsiness, dizziness, nausea and vomiting. Carbamazepine induced leukopenia (uncommon)

If refractory to CBZ monotherapy, try combination therapy:

1. Baclofen
   - Depress excitory synaptoc transmission in spinal trigeminal nucleus
   - Normally used in combination with carbamazepine

2. Phenytoin
   - Blocks Na channels and inhibits presynaptic glutamate release
   - IV therapy was effective in reducing acute exacerbations of neuropathic pain – useful tx in pts with severe symptoms

3. Valproate
   - 50-80% have relief of Sx
   - increase amount of GABA, binds GABA receptors and prolongs repolarization of voltage gated Na channels

4. Lamotrigine
5. Gabapentin
6. Clonazepam

Surgical therapy
Reserved for Sx in pt refractory to drug therapy

1. Microvascular decompression  (Best long term outcome)
   - 60 – 70% have good outcomes

2. Radiofrequency rhizotomy
   - Creates lesion in gaseerian ganglion of TN by application of heat
   - Less invasive than microvascular decompression
   - Higher initial success rate but recurrence rate of 25 – 50% within 2 – 3 years

3. Glycerol rhizolysis
   - Injection of 0.1 – 0.4mL of glycerol into trigeminal cistern
   - Recurrence rates by 6 years is as high as 92%

Answer: B  Microvascular compression