Surgical Decompression for Painful Diabetic Neuropathy

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Background-Diabetic Neuropathy

- Most common complication of DM
  - 10-90% of patients w/ DM
- Most common neuropathy (in developed countries)
  - 50-75% nontraumatic amputations
- Symmetrical, length-dependent sensorimotor polyneuropathy
  - Insidious, progressive, irreversible

Pathophysiology

- Excess glucose conversion → intracellular sorbitol
  - Sorbitol blocks uptake of myoinositol
    - Reduced sodium and potassium activity (affects conduction)
    - Increased intracellular sodium → demyelination
  - Attracts water molecules → swelling
  - Decreased axoplasmic flow → axonal degeneration
- End products interfere with nerve physiology on many levels
- Vascular
  - Less accepted than it used to be
    - Not from small or large vessel disease
    - Axonal swelling may cause compression
      - Analogous to nerve ischemia seen in other compressive neuropathies
      - Microvascular perfusion impaired from dyslipidemia, oxidative stress, premature atherosclerosis
  - Compression from nerve swelling
    - Diabetic neuropathy w/ superimposed compression neuropathy
- Double-crush hypothesis
  - Serial constraints on axoplasmic flow additive effect
  - Axoplasmic flow decreased in diabetic rats
    - Increased susceptibility to nerve compression (in a rat model)
  - Doesn’t behave like compression neuropathy
- Loss of small-fiber-mediated sensation → pain and temp
- Large-fiber → touch and vibration
  - Replaced with pain/ paresthesias
  - Neuropathic pain noted even if impaired glucose tolerance

*Pain reported in 30-40% of both Type I’s and II’s

Surgical Release Rationale

- Carpal Tunnel Syndrome → abnormally tight space damages normal median nerve
- Diabetic Neuropathy → normally tight spaces irritates damaged nerves

Natural “narrowing”
Known sites:
- Fibular neck
- Tarsal tunnel (distal medial plantar, lateral plantar, and calcaneal tunnels)
- Deep peroneal nerve on dorsum of foot

Increased water content $\rightarrow$ swelling
Loss of tissue elasticity (from binding of glucose to collagen in epineurium)$^{15}$
- Loss of gliding $\rightarrow$ increased tension (dec in blood flow)

Animal Data
- Releasing tarsal tunnel in “streptozotocin-induced diabetic” rats blocked development of “abnormal” walking pattern$^{16}$
- Diabetic Zucker rats demonstrated therapeutic benefit w/ decompression early in disease process$^{17}$

Surgical Procedure$^{18}$ - Release at fibular neck, tarsal tunnel, dorsum of foot

Clinical Experience
- Dellon et al.$^{19}$
  - 57 lower ext diab nerve releases
  - Subjective improvement: good or excellent
  - Tibial: 85%
  - Common Peroneal: 72%
  - Deep Peroneal: 62%
  - Best predictor: strong Tinel’s at point of “narrowing”

- 26 patients$^{20}$
  - Tarsal tunnel on 33 legs $\rightarrow$ pain better in 92%; sensation better 76%
  - Two patients w/ neg Tinel Test with no improvement

- 36 patients$^{21}$
  - 58 tibial nerve releases $\rightarrow$ pain better in 86%; sensation better 60%

- 18 patients (15 w/ diab neur and 3 w/ idopathic neuropathy)$^{15}$
  - Triple releases in 25 legs $\rightarrow$ 88% pain relief

- 39 patients$^{22}$
  - 49 limbs $\rightarrow$ all with improvement in pain and sensation

- 100 patients (60 w/ diab neuropathy and 40 idiopathic)$^{23}$
  - At 1 year f/u, 87% and 86% of patients with numbness and severe pain reported improvement

- 33 patients$^{24}$
  - Triple release
  - Pain and sensory testing
  - 90% good to excellent pain relief (VAS improvement 6-10 points)
  - 67% good/exc sensation improvement
  - 40% return to normal sensation

Meta-analysis$^{25}$
- 90% predictive value of positive Tinels
- Pain relief in 80% of patients VAS mean of 8.5 $\rightarrow$ 2
- 80% gained $>$ protective sensation
References:


