**Diabetic Foot: Implosion:**
The Tragedy of Diabetic Charcot Arthropathy

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**Diabetic Epidemiology**

- Foot Infection:
  - Leading cause for hospitalization
  - Leading cause for ↑ length of stay
    * 25% of all DM admissions in US
    * 50% of all DM admissions in Europe and UK
- Survival rate following one major LEA:
  50% @ 3 yrs.; 40% @ 5 yrs. (1)

(1) Lee JS, Diabetes, 42: 876-82, 1993
The relative 5-year mortality rate after limb amp is at least 50%. When compared with cancer, it is second only to lung cancer (86%). Colorectal cancer (39%), Breast cancer (8%).

Diabetic Epidemiology

- Toe amputations are most common LEA (2.6/1000)
- Below the knee (1.6/1000), AKA (0.8/1000)
- 15-20 times higher in DM than non-DM
- Costs for diabetic foot disease can exceed $6 billion/year
- $20,000-60,000 per event, greater cost with higher amp.


Diabetic Epidemiology

- Diabetes: 8 of 10 non-traumatic amputations
- 85% of diabetic amputations follow a foot ulcer
DISEASES THAT MAY CAUSE NEUROPATHIC ARTHROPATHY

- Diabetes mellitus
- Tertiary syphilis
- Leprosy
- Syringomyelia
- Pernicious anemia
- Multiple sclerosis
- Poliomyelitis
- Congenital insensitivity
- Hysterical insensitivity
- Paraplegia
- Peripheral nerve lesions
- Spinal cord lesions
- Riley-Day syndrome
- Intra-articular injection of corticosteroid

SEMMES - WEINSTEIN MONOFILAMENTS

- Simple application, inexpensive
- Accurate assessment neuropathy
  (Sosenko, Diabetes Care ‘90)
- Risk categorization (5.07/10 gram filament)
  (Rith, Najarian, Diabetes Care, ‘92)
Sensory Assessment Tools

- Semmes-Weinstein Monofilaments
- Tuning Fork
- Biothesiometer
- 2-Point Discriminator
- PSSD
- Neurometer
- EMG/NCV

Monofilament Test

- Test with the 3.61(0.2 gram) monofilament first to determine if the patient has normal plantar sensation
- Sensory loss can be documented by testing with the 4.31(2.0 gram), 4.56(4 gram), and finally the 5.16(10 gram) filaments respectively
- The standard 5.07 monofilament is generally accepted to represent 10 grams of force and has been used extensively to screen for loss of protective sensation
BIO - THESIOMETER

- VPT: Vibratory Perception Threshold
- Patients VPT < 15V cumulative incidence of foot ulceration = 2.9%
- Patients VPT > 25V incidence of foot ulceration = 19.8%

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2 Point Discrimination

 Dermatomes:
- L4: ant. & med. leg, dorsal 1st ray & hallux plantar hallux and 1st MPJ
- L5: ant. & lat. leg, central dorsal & plant. foot inc. rays & toes 2, 3 & 4
- S1: post. & lat. leg, lat. dorsal & plant. foot inc. 5th ray and toe
- S2: post. leg, post. & plant. med. heel

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Radiculopathy
I. Mononeuropathy simplex
* Etiology: vascular; esp. 3rd cranial nerve → diplogia
  compression & entrapment; susceptibility
* Femoral, sciatic, peroneal nerve → drop foot
* Exacerbated at night

II. Mononeuropathy multiplex
(proximal amyotrophic neuropathy)
* Males, Type II (NIDDM), Etoh abuse, age: 50 - 60
* Iliopsoas & quads → girdle weakness, diff rise from sitting

III. Distal Symmetrical Polyneuropathy (DSP)
* Most common
* 50% all DM patients → sensory DSP
* 70% of pts. with DSP → sensory & motor
* Stocking-and-glove distribution, FEET
* Acute phase may → weight loss, impotence, depression

III. DISTAL SYMMETRICAL POLYNEUROPATHY
* Most common (50% DM pts. → sensation)
* Small fiber dis.: Dysesthesias (tingling, burning, numbness), pruritis, temperature, PAIN
* Large fiber: vib., proprioception, DTRs
* Stocking-and-glove distribution, FEET
* Motor deficit: digital def. → arthropathy, ulcers
* BIOMECHANICS: Large fiber disease & motor deficit
Diabetic Neurotrophic Foot Ulcers

- Diabetic Neuropathy
  - Atrophy / weakness of the intrinsic muscles
  - Destabilize the MP joints and toes
    - hammertoes, dorsal IPJ prominence
    - prominent plantar metatarsal heads
    - plantar MPJ fat pad atrophy
    - high peak pressures, calluses, ulcers

Diabetic Neurotrophic Foot Ulcers

DIABETIC NEUROPATHY

- (1) Anterior crural weakness
- Absolute or relatively high posterior strength

"Diabetic Neuropathy (not other neuropathy) is statistically correlated with the development of equinus"

(2) Kravitz, et al, 500 patients in study, randomized

Diabetic Neurotrophic Foot Ulcers

DIABETIC NEUROPATHY

- Equinus: Lack of 10 Degrees Dorsiflexion
  - Lack of dorsiflexion with knee extended and flexed
    - Gastrocnemius-soleus equinus
    - Bone blockage (Ankle Equinus) Rare, imaging
  - Lack of dorsiflexion with knee extended ONLY Normal dorsiflexion with knee flexed
    - Gastrocnemius equinus
Diabetic Foot Ulcers / Autonomic Neuropathy

- Sweat gland denervation leads to dry, cracked, atrophic skin, with increased callus formation
- Increased arteriovenous shunting
- Neurogenic edema secondary to impaired posturally induced vasoconstriction
- Increased blood flow with distended dorsal veins & increased capillary pressure leading to microvascular sclerosis
- Tissue ischemia due to impaired microvascular vasodilation, the normal hyperemic response to injury

Diabetic Autonomic Neuropathy (DAN)

Present in 70% Diabetic patients

- Lower extremity: anhydrosis
- Cardiovascular: tachycardia, postural hypotension
- GI: diarrhea or constipation, esophageal dysfunction (heartburn, dysphagia)
- GU: bladder dysfunction, impotence
  Symptomatic DAN poorest prognosis → renal failure

Clinical Features of Acute Charcot Joint Neuropathy

Vascular
- Bounding pulses
- Erythema
- Swelling
- Warmth

Sensorium
  * ABSENT OR
  * DIMINISHED
  * Pain
  * Proprioception
  * Vibration
  * Deep tendon reflexes
  Hypo or Hyperhidrosis
CHARCOT JOINT
DIABETIC NEUROPATHIC OSTEARTROPATHY (DNOA)

- 1868, Jean-Martin Charcot, Tabes dorsalis
- Diabetes most common cause today
- 1966, Eichenholtz, 3 radiographic stages
- stage of DEVELOPMENT
- stage of COALESCENCE
- stage of RECONSTRUCTION

DNOA CLINICAL FEATURES OF ACUTE CHARCOT JOINT

SKELETAL
- Rocker bottom deformity
- Medial tarsal subluxation
- Digital subluxation
- Crepitus
- Hypermobility

CUTANEOUS
- Hyperkeratoses
- Neuropathic ulcer
- Infection

DIABETIC NEUROPATHIC OSTEOARTHRITIS (DNOA)

- **Acute** or early stage, also termed: atrophic, destructive, hyperemic
- **Chronic** or late stage, also termed: hypertrophic, sclerotic, and quiescent
STAGE OF DEVELOPMENT

- Hyperemia
- Swelling/Jt. effusion
- Skin temperature
- Osteolysis/Osteopenia

BONE RESORPTION
- Jt. mobility/subluxation
- Bone/cartilage erosion
- Bone fragmentation
- **Increased instability**

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STAGE OF COALESCEENCE

- Absorption fine debris
- Decreased edema
- Decreased erythema
- Fracture healing
- Fusion / rounding large fragments
- Decreased joint mobility
- Increased bone density
- **Increased stability**

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PATTERNS OF DIABETIC NEUROPATHIC OSTEOARTHRITIS

- Pattern I: IPJ’s & phalanges
  (* = ulcer prone)
- Pattern II: Lisfranc’s Jt. (TMT)
- Pattern III: MTJ (TN, CC) and naviculocuneiform
- Pattern IV: Ankle
- Pattern V: Calcaneus

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DNOA RADIOGRAPHIC FINDINGS
DIABETIC OSTEOARTHROPATHY

STAGE  ATROPHIC  HYPERTROPHIC
Development  *phalangeal  osteochondral fragments
           “bourglassing”  w/ joint mice
           *met.resorption  jt. subluxation &
           “pencil pointing”  jt. effusion

Coalescence
periosteal new bone &
osteophyte formation
resorption debris
subchondral sclerosis
w/ AVN
Reconstruction
anklyosis or rounding
bone ends, swelling,
decreased sclerosis

DNOA CONDITIONS CAUSING
JOINT CHANGES SIMILAR TO
CHARCOT JOINT
Gout  Septic arthritis
Osteoarthritis  Tuberculous arth.
Rheumatoid arth.  Paraplegia
Psoriatic arthritis  Neoplasm

Korak et al, Management of Diabetic Foot Problems, Saunders, 2nd ed, 1995
CHARCOT JOINT DISEASE: TREATMENT: long term

- **Conservative** management: most often
- **Surgery**: never indiscriminate
  * Assess: metabolic, vascular & neuropathic
  rectus vs. deformity and stable vs. unstable
  * Multidisciplinary approach: risk, outcome
  * Pretrophic and trophic sites: imminent risk,
  ulceration, infection, exostectomy, reconstruction, amputation
CHARCOT JOINT DISEASE: TREATMENT: acute phase

- Weight bearing: **completely avoided**
- Immobilization: severe joint destruction,
  - Fractures: eg. Lisfranc’s jt. fracture/subluxation
  - Casts avoided until edema resolved,
    condition stabilized, (1 - 2 weeks); Post. splint, Jones dressing, etc, temporary immobilization
    Immobilize until osseous repair complete,
    min. 12 wks., ** 16-20 wks

CHARCOT JOINT DISEASE: TREATMENT: long term

- Healing sandal: Plastazote molded impression on surgical shoe, short term following cast
- Minimal destruction: accommodative inlay,
  custom orthosis (Plastazote), extra depth shoe,
- Significant destruction: molded shoe
- Impression casting for orthosis or molded shoe, MAFO: prior to cast immobilization removal

CHARCOT TREATMENT ALGORITHM

<table>
<thead>
<tr>
<th>Stage</th>
<th>ACUTE</th>
<th>CHRONIC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Degree</td>
<td>Degree</td>
</tr>
<tr>
<td>I.</td>
<td>Deformity (Min/none)</td>
<td>I. Stable Foot, Deformity Min/none</td>
</tr>
<tr>
<td>II.</td>
<td>Deformity (moderate)</td>
<td>II. Foot deformity (mod) foot/ankle instability</td>
</tr>
<tr>
<td>III.</td>
<td>Deformity nonreducible rocker bottom</td>
<td>III. Foot deformity, recurrent ulceration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV. Foot/ankle instability and/or not amenable to bracing or exostectomy</td>
</tr>
</tbody>
</table>
CHARCOT TREATMENT ALGORITHM

Stage: **ACUTE**

### Degree I. Minimal or no deformity
Tx. Non weight bearing x 16 wks
a. Posterior splint or Jones compression dressing initial 2 weeks or until edema resolves
b. Cast immobilization follows (a) above for additional 14 wks or until stage 3

Consider electrical bone stim, phospholipids to augment therapy anecdotal, no clear evidence, some positive reports for Acute Degrees II and III

### Degree II. Foot Deformity - minor or may be reducible
Tx. Closed or Open Reduction with fixation (int./ext.)
Non weight bearing x 16 wks
a. Posterior splint or Jones compression dressing initial 2 wks followed by
b. Cast immobilization x additional 14 wks or until quiescent

Long term therapy: Padded orthopedic or custom molded footwear or MAFO (molded ankle foot orthosis)

### Degree III. Deformity nonreducible rocker-bottom
Tx. (A) 1. Treatment protocol under degree 1
2. Followed by reconstructive surgery ORIF (open reduction w internal fixation) followed by additional 16 wks NWB
(B) Consider immediate reduction with external fixation (Ilizarov), elect. bone stim. Non weight bearing to partial weightbearing X 16 to 20 weeks
CHARCOT TREATMENT ALGORITHM

Stage: CHRONIC

Grade I. Stable Foot, Minimal or No Deformity
Tx. Properly padded and supportive orthopedic or custom footwear

Grade II. Foot/ankle deformity and/or instability
Tx. Properly padded custom molded shoes
Consider above ankle laced shoes
Molded ankle/foot orthosis bracing

Grade III. Foot Deformity, recurrent ulceration not amenable to off-loading techniques
Tx. Exostectomy followed by proper shoeing and/or bracing

Grade IV. Foot/ankle instability and/or significant deformity not amenable to bracing or exostectomy
Tx. (A) ORIF or ext. fixation eg. Ilizarov, bone stim.
NWB X 16 – 20 weeks
(B) Partial foot/BK amputation

CHARCOT JOINT DISEASE: TREATMENT: long term

* Salvage surgery: tissue morbidity, osteo., ulcers
* Amputation: unresponsive ulceration/infection
CHARCOT

Some Newer Concepts.

Where is diagnosis going and how might that effect management

Definitions:

Proinflammatory cytokines - promote inflammation

Prostaglandin-E2 (PGE2)
- A prostaglandin class derived from arachidonic acid via the cyclooxygenase pathway
- Prostaglandins induce fever, pain, vasodilation, smooth muscle relaxation, effects nerve transduction & renal function
- Prostaglandins affect cytokine gene expression


Tumor Necrosis Factor-α (TNF-α): a proinflammatory cytokine with multiple effects on vascular endothelium, immune system and mediator in rheumatoid arthritis and other diseases

Lipopolysaccharide Promotes the Survival of Osteoclasts

Receptor Activator (RANKL) is a membrane-bound or soluble cytokine essential for osteoclast activation
Definitions

**RANKL:** Receptor Activator of NF-κB Ligand [nuclear factor kappa B ligand (RANKL)] is a membrane bound or soluble cytokine essential for osteoclast activation

**Osteoprotegerin ligand (OPGL)** - a TNF cytokine
- Stimulates osteoclasts, increases bone resorption

**Osteoprotegerin (OPG)**
- Member of the TNF receptor superfamily
- Binds receptor activator of NF-κB (RANK)
- Inhibits osteoclast activation and decreasing bone resorption
- Also known as osteoclastogenesis inhibitory factor


Theorized Charcot Arthropathy is due to increased proinflammatory cytokines, including nuclear factor kappa B ligand (RANKL) causing increased osteoclastic activity resulting in osteopenia.

*If the above is true then new therapies can be used to reduce devastation in bone disease by decreasing the expression of these cytokines*

**Biomechanics of the Diabetic Foot**

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DYNAMIC LOADING & PEDAL ULCERS

- Friction: resistance between one body moving over another body
- Pressure: force exerted by one body on another inversely proportional to area
- Shear: combination friction & compression "Sliding" of contiguous body parts relative to each other→ callus and ulcer

DIABETIC PEDAL BIOMECHANICS

Multifaceted
- Foot type
- Neuropathy
- Glycosylation
- Nephropathy

- Vertical pressure met heads
  - hammertoes
  - Soft tissue, fat pad atrophy
  - Calluses
  - Ulcers
Biomechanics: Diabetic Foot

- **Diabetic Neuropathy**
  - Atrophy / weakness of the intrinsic muscles
  - Destabilize the MP joints and toes leads to:
    - hammertoes, dorsal IPJ prominence
    - prominent plantar metatarsal heads
    - plantar MPJ fat pad atrophy
    - high peak pressures, calluses, ulcers

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DIABETIC NEPHROPATHY

- Decreased *renal function* → vitamin D conversion to active form → serum calcium levels
- Demineralized bone: osteomalacia
- Stress and microfracture common may exacerbate charcot changes
  * Conversion of 25-hydroxycholecalciferol (25-HCC) to 1, 25-dihydroxycholecalciferol & 24, 25-DHCC

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Limited Joint Mobility

- (1) Hyperglycemia → glycosylation collagen
  - increased cross linking collagen
  - collagen and keratin increased rigidity
  - less resistance to collagenase
  - decreased joint mobility, stiff foot

Biomechanics: Diabetic Foot

AMPUTATION: Weight Redistribution

- Normal Foot Propulsive Phase
  - Loads forefoot lateral to medial
  - Hallux last segment loaded

### AMPUTATED WEIGHT TRANSFER RESULT

<table>
<thead>
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<th>SEGMENT</th>
<th>WEIGHT</th>
<th>TRANSFER</th>
<th>RESULT</th>
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</thead>
<tbody>
<tr>
<td>Hallux</td>
<td>2nd MPJ &amp; Toe</td>
<td>Contracture</td>
<td>Loss of EHL ankle D/F</td>
</tr>
<tr>
<td>Lesser toe</td>
<td>minimal if any</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>First ray</td>
<td>2nd met head</td>
<td>Contracted 2nd MPJ</td>
<td>excessive pronation</td>
</tr>
<tr>
<td>Fifth ray</td>
<td>4th met head</td>
<td>Contracted 4th MPJ</td>
<td></td>
</tr>
</tbody>
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1st and 5th ray resections better tolerated than central ray

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<table>
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<th>WEIGHT</th>
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<th>RESULT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central ray</td>
<td>adjacent met heads</td>
<td>high rate transfer lesions</td>
<td></td>
</tr>
</tbody>
</table>

3rd met (ray) amp better than 2nd & 4th. All central ray resection have poor prognosis often transmitted amp

- Transmet: distal plantar medial stump lesions
  - Well tolerated augment: TAL long supinators intact invert plant (equinovarus) loss extensors, flexors dominant, short stride gait:
Biomechanics: Diabetic Foot

DYNAMICS: FOOT ULCER

- Rate of load rather than magnitude
- Orthoses: should they off-load or rate of dynamic load?
- MAFO: high velocity impact: definitive

Biomechanics: Diabetic Foot; Off-Loading Techniques

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Biomechanics: Diabetic Foot; Off-Loading Techniques

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Biomechanics: Diabetic Foot; Off-Loading Techniques

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MERCI
DANKE
GRACIAS
EUCHARISTO
THANK YOU!