Evaluation & Treatment of Diabetic Neuroarthropathy

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Evaluation & Treatment of Diabetic Neuroarthropathy

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I have no professional conflicts associated with this lecture

consultant: Biomet, Tornier
Objectives

• discuss the musculoskeletal care of patients with diabetes mellitus
• foot and ankle issues
  neuropathy
  ulceration
  Charcot neuroarthropathy
  amputation
Objectives

• epidemiology
• etiology
• diagnosis
• treatment
  medical
  surgical
Diabetes; the “giving” disease

- endocrinology
- nephrology
- cardiology
- neurology
- ophthalmology
- P M & R
- physical therapists
- orthopaedic surgery
- vascular surgery
- plastic surgery
- transplant surgery
- wound care
- dieticians
- orthotists, prosthetists
Diabetes mellitus

**Epidemiology**

25,800,000 people (8.3% population)
- diagnosed: 18,800,000
- undiagnosed: 7,000,000

“it sees you, but you may not see it…”

National Diabetes Fact Sheet

http://www.cdc.gov/diabetes
Diabetes mellitus

**Epidemiology**

>65 y.o.: 10,900,000 (26.9%) have D.M.

‘05–’08: fasting glucose/HbA1c levels

35% > 20 y.o. have prediabetes

50% > 65 y.o. have prediabetes

National Diabetes Fact Sheet

http://www.cdc.gov/diabetes
Neuropathy

**Epidemiology**
60 – 70% of diabetics

National Diabetes Fact Sheet
http://www.cdc.gov/diabetes

differential:
- alcohol
- cord trauma/tumor
- idiopathic
- syringomyelia
- myelomeningocele
- cerebral palsy
- leprosy
- syphilis
Neuropathy

Epidemiology
associated with:
↑ age, duration
poor glucose control
no correlation with type I/II, sex

Diabetes control research grp
Ann Int Med 122: 1995
Neuropathy

**Etiology**
symmetrical > asymmetrical
sensory fibers > autonomies
distally accentuated loss of myelinated and unmyelinated fibers
electrophys. studies indicate axonal loss
sensory and/or motor

Dyck, et al:
Neurology 43: 1993
Neuropathy

**metabolic contribution:**
hyperglycemia results in: accumulation of sorbitol; ↓ myo-inositol; conduction block of myelinated axons

**vascular contribution:**
eextraneural A-V shunting; hypoxia
narrowed capillary/arteriolar lumens
poor regulation of microcirculation
Neuropathy

**Sensory dysfunction:**
distal → proximal; stocking-glove pattern
large fiber: ↓ light touch, proprioception
small fiber: ↓ pain, temp. perception
paresthesias; possibly due to spontaneous depolarization of regenerating nerve fibers
Neuropathy

**Autonomic dysfunction:**
- arteriovenous shunting
- loss of skin temperature regulation
- perspiration loss
  - nonpliable skin
- hypertrophic callosities
Neuropathy

**Motor dysfunction:**

demyelination causes conduction deficits
more commonly distal
results in intrinsic musculature dysfunction
clawing of toes
metatarsal head depression
plantar pressure concentration
Neuropathy

**Immune dysfunction:**
malnutrition, hyperglycemia, hypoxia results in inefficient anaerobic metabolism

↑ acidity, hypertonicity, edema

altered PMN chemotaxis
Neuropathy

**Diagnosis:**

Semmes-Weinstein monofilaments
- 5.07 (10 gm.) size
- 100% sensitive, 75% specific

Biothesiometer (vibratory stimulus)
- 79% sensitive, 93% specific

Kumar, et al
Neuropathy treatment

- maximize glucose control
  intensive tx. ↓ neuropathy 64%
- oral medication for painful neuropathy*
  gabapentin, cymbalta, lyrica
- compounded nerve creams

Diabetes control research grp
Ann Int Med 122: 1995
Neuropathy treatment

Guidelines for diabetic foot care
foot-specific patient education
skin and nail care
therapeutic footwear

Medicare therapeutic foot bill; 1993

FAI 20: 1999
Diabetic foot ulcer

Epidemiology
15% of diabetics (3-4% of pop.)
precede 85% of L.E.A. in diabetics

National Diabetes Fact Sheet
http://www.cdc.gov/diabetes
Diabetic foot ulcer

Epidemiology

35% Charcot have ulcer @ 1st presentation

Game, et al:
Diabetologia 55: 2012

ulcer @ 1st presentation → 28% amputation

Dalla Paola, Fagia:
Curr Diab Rev 2: 2006
Ulcer formation

pressure + neuropathy
DIABETIC NEUROPATHY

SENSORY
- Impaired sensory perception of:
  - Pain
  - Temperature
  - Vibration
  - Touch
  - Injury:
    - Mechanical
    - Thermal
    - Chemical

MOTOR
- Small muscle atrophy
  - Imbalance in flexor & extensor muscles
    - Clawed toes
      - Prominent metatarsal heads
      - Altered gait
      - Callus

AUTONOMIC
- Decreased sweating
- Dry scaly skin
  - Fissures
  - A-V shunt
    - Cell nutrition
      - Capillary pressure
        - Edema
        - Poor healing

FOOT ULCER

DEMYELINIZATION
Ulcer formation

intrinsic imbalance → clawtoes; (+) neuropathy ...... → ulcers
Ulcer classification

**depth**
0: “at risk”, intact skin
1: superficial
2: deep; tendon/joint exposed
3: extensive; bone exposed *

**ischemic**
 a: nml
 b: ischemia
 c: partial gangrene
 d. nasty gangrene
Ulcer treatment

- structural factors: WB x-rays
- sensation: monofilaments
- circulation: ABIs with toe pressures
- infection: labs, imaging
- healing potential: labs
Mechanical, structural factors

- clawing of toes
  - plantar pressure, dorsal impingement
- unstable Charcot foot
- stable foot with bony prominence
- requires *weight-bearing* films !!
Ulcer treatment

• neurogenic vs. vasculogenic
  neurogenic ulcers might heal
  vasculogenic ulcers won’t heal
• must distinguish between the 2
  (or combination)
Sensation

Monofilament testing

5.07 (10 gm) threshold

protective sensation
ABIs with toe pressures

Vessel compliance affected by small vessel calcification; toe pressures necessary

ABI > .45 required for amp. healing*

T.p. > 45 mm Hg for amputation

T.p. > 60 mm Hg for ulcer healing

• If not, → Vascular consult
Circulation

Waveform characteristics
Circulation

• **TcPO$_2$**
• skin heated to 44°C, 10 min.s
• tests autonomic response
• adversely affected by edema, cellulitis

<table>
<thead>
<tr>
<th>Pressure Range</th>
<th>Percentage</th>
</tr>
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<tbody>
<tr>
<td>1-19 mm Hg</td>
<td>50%</td>
</tr>
<tr>
<td>20-29 mm Hg</td>
<td>75%</td>
</tr>
<tr>
<td>&gt;30 mm Hg</td>
<td>92%</td>
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Infection

- screening labs
  CBC, ESR, CRP
- advanced imaging
- if concern for osteomyelitis, abscess
  triple phase (+) tagged WBC (Indium)
  MRI (+) gadolinium, fat suppression
Infection
Infection
Infection

Probe to bone = osteo.
Healing potential

**Screening labs**
- total protein > 6.2 g/dL
- albumin > 3.5 g/dL
- total lymphocyte count > 1,500/mm$^3$
Ulcer treatment

• off-loading
  crutches, walker, wheelchair
  relief shoe, boot, TCC

• protection
  orthotic, brace, CROW

• correction
  exostectomy, reconstruction
Off-loading

• pressure prevents ulcer healing !!
• location of ulcer determines type of immobilization
  relief shoe with plastazote lining
  boot brace with plastazote lining
  total contact cast
• assist device to help unweight
Total contact cast

- edema control device
- vertical forces distributed over sole
- shear forces reduced
- labor-intensive
- prevents local wound care

Total contact cast
TCC results

- 28/36 (78%) heal in 6.3 weeks
- 16/28 (57%) re-ulcerated
- 16/22 (73%) heal in 5.8 weeks
  - Helm; Arch PMR 65: 1984
- 49% ulcers recur
  - Saltzman, et al; CORR 435: 2005
Protection
Correction

**exostectomy**
stable deformity
over-resection can create collapse

**reconstruction**
unstable deformity
chronic Charcot
Exostectomy
Exostectomy
Exostectomy

- #41 Charcot pt.s followed > 8 yr.
  2/3 ulcerated → 50% required surgery
  exostectomy successful: 62%

- exostectomy: 74 – 91% healing
  lateral exostectomy → 2/3 recur
  Lowery, et al; FAI 33: 2012
Reconstruction
Neuroarthropathy

- Jean-Martin Charcot (1868)
  1st described neuropathic changes in a syphilitic patient; disease of “trophic centers” of anterior horn cells
- Jordan (1936)
  1st described diabetic neuropathy
- Bailey (1942)
  tarsal destruction in 14 uncontrolled DM
Neuroarthropathy

- bony destruction in neuropathic patients
- associated instability
- ulceration 2° to bony prominence
- confused with:
  - cellulitis
  - osteomyelitis
  - acute fracture
Neuroarthropathy

Typical scenario
initial dx. of cellulitis/osteomyelitis
“confirmatory” bone scan
intravenousous antibiotics
bone biopsy request
no x-rays obtained
Neuroarthropathy

40 y.o.; IDDM
3 wk. h/o red, swelling
no constitutional sx.
no ulcer
abx. not improving
no immobilization
Neuroarthropathy

- “no ulcer – no osteo”
- neuropathic swelling ↓↓ with elevation
Neuroarthropathy

**Epidemiology**

1.4 – 2.5% of all diabetics

25% delay in diagnosis

35% Charcot with ulcer at presentation

30% contralateral within 2 yr.

median time resolution: 9 months
Neuroarthropathy

“at-risk” factors
retinopathy
nephropathy
previous ulceration
↓ 5.07 monofilament

Foltz, et al:
J Foot Ankle Surg 43: 2004
Etiology

**Neurotraumatic theory**

↓ proprioceptive reflexes via post. roots

repetitive trauma to insensitive joints

results in fracture and/or dislocation

Volkman, Virchow

Johnson; JBJS 49a: 1967
Etiology

Neurovascular theory
neurally initiated vascular reflex
“autosympathectomy”
↑ blood flow, osteoclastic bone resorption
initial atrophic, destructive changes
secondary reparative phase

Charcot; 1868
Brower, Allman; Radiology 139: 1981
Classification

Eichenholtz

stage I: acute, inflammatory
stage II: reparative, unstable
stage III: consolidated, stable

stage “0”: neuropathic foot “at risk”

Schon, Marks;
Orth Clinics N.A. 26:1995
stage I Charcot

- x-ray: fragmentation, subluxation
- clinical: red, warm, swollen foot
- no ulcer, no osteo
- elevation improves clinical signs
stage II Charcot

- x-ray: coalescence of bone
- clinical: decreased warmth, swelling
- fluctuation of clinical signs with increased activity
- joints not stable
stage III Charcot

- x-ray: reconstitution of bone
- clinical: resolution of redness, warmth, swelling
- joints stable on exam
stage “0” Charcot

- x-ray: normal
- clinical: history of trauma
- +/- swelling
- any diabetic, neuropathic patient is considered “at-risk” with any history of trauma, even in light of a normal xray and exam
Neuroarthropathy

Treatment goals
create a mechanically stable foot
plantigrade; can accommodate a shoe
and/or a brace
prevent bony prominence
that will ulcerate
Treatment considerations

- stage of Charcot
- deformity +/- instability
- ulceration +/- deep infection
- healing potential
  - vascular status, nutrition, tobacco
Charcot acute treatment

- strict immobilization
  TCC
  boot brace (no deformity)
- strict off-weighting
- elevation
- patient education
Charcot acute treatment

- change initial cast @ 1 week
- successive casts based on swelling
- serial radiographs
- months to resolve
- role of bisphosphonates?
  - inhibit osteoclasts, ↓ skin temp
- intranasal calcitonin + Ca.?
Stage II treatment

- protected weightbearing
  TCC, boot
- slow, gradual increase
  10-25% over 4 weeks, → x-ray
  25-50% over 4 weeks, → x-ray
  50-100% over 6-8 weeks, → x-ray
Stage III treatment

- wean into protective orthotic/brace, shoewear
- foot must be able to accommodate shoes, orthotics, braces
- evaluate any bony prominence
Surgical indications

- gross instability
- cannot maintain a plantigrade position with a shoe/brace
- irreducible fracture-dislocation
- recurrent ulceration
- reconstruction in stage I (acute) only for dislocation or threatened skin
Amputation

**Epidemiology**

#1 cause nontraumatic amputations

> 60% in diabetics

2006: 65,700 L.E.A. in diabetics

2 yr. mortality

BKA: 36%

AKA: > 50%

National Diabetes Fact Sheet

http://www.cdc.gov/diabetes
Amputation

Epidemiology

20X higher amputation rate vs. general population.

Moss, et al:
Diabetes Care 22: 1999

26% require an additional amputation

1/3 die within 1 yr.

Dillingham, et al:
Arch PMR 86: 2005
Diabetic salvage failure

- poor control
- poor compliance
- poor physician recognition
- lack of coordinated approach
Limb salvage vs. amputation

- will salvage outperform a prosthesis?
- reasonable outcome expectation?
- what are the costs?
- what are the risks?
Conclusions

Neuropathy treatment
monofilament testing
maximize glucose control
protective orthotics
protective shoewear
education
Conclusions

**Ulcer treatment**

pressure (+) neuropathy = ulcer
neurogenic vs. vasculogenic

ABIs + toe pressures
vascular consult for suspected insuff.
nutritional status ?
Conclusions

**Ulcer treatment**

infectious evaluation

  triple phase (+) tagged WBC

MRI for suspected abscess

protection !!

  offweight, followed by orthotics

exostectomy vs. reconstruction
Conclusions

**Charcot treatment**

redness, warmth, swelling = Charcot
no ulcer, no osteo.
weightbearing x-rays
offweight
elevate
evaluate
Conclusions

Charcot treatment
recognize stages
recognize at-risk patients
acute surgery for dislocation, skin at risk
reconstruction for chronic instability, nonbraceable deformity
protective orthotics, braces shoewear
education
Limb preservation in the diabetic, neuropathic patient
Primary Care Foot Program

Jessica Altis, PA-C
Amanda Fuchs, PA-C

- diabetic foot care
- ulcer, nail care
- orthotics, shoes
- fractures, sprains
- nonoperative tendinitis, arthritis

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