“Doc, DO I Have Neuropathy?”

Stanley Jones P. Iyadurai, MSc, PhD, MD
Assistant Professor of Neurology, Neuromuscular Division, Department of Neurology, The Ohio State University Wexner Medical Center

Case Vignettes

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<th>Duration</th>
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“Neuropathy” - Definition

- “Neuron” and “Pathos” (Greek)
- Disease of the Peripheral Nerve
- Dysfunction of the nerves outside of the central nervous system

Neuropathy - General Theme

- Symmetric
- Insidious
- More prominent distally and starts in legs
- Involves both motor and sensory components
- May cause pain
- May cause loss of balance
- Progressive, but not debilitating
Not all that tingles is neuropathy

‘numbness and tingling’
- not all numbness and tingling is peripheral nerve
  - RLS
  - edema
  - deconditioning
  - central nervous system
  - nerve root ['sciatica']

Classification - Functional

- Motor – affects the motor nerves ➔ Weakness
- Sensory
  - Pain
  - Small Fiber
  - Large Fiber
  - Large and Small Fiber
  - Neuronopathy (ganglionopathy)
- Autonomic
  - Sweating Changes, Blood Pressure Changes

Classification – Time-course

- Acute
  - Immune-mediated
- Infantile Weakness
- Childhood-onset
- Relapsing
- Hereditary
  - Motor-Sensory
  - Motor Syndromes
  - Sensory Syndromes

Classification – Time-course

- Congenital/Hereditary

- Acquired
  - Reversible?
  - Demyelinating?
  - Immune-mediated?
  - Systemic diseases?
Neuropathy - Evaluation

• Detailed History
• Family History
• Clinical Examination
• Blood Work
• Special Tests – EMG/NCS
• Special Tests – Imaging
• Special Tests – Nerve/Muscle/Skin Biopsy

Examination

• Vibration sense – the most sensitive test
• Quantitative Tuning Fork (Rydel-Seiffer)
• Pin scratch test (gradient)
• Proprioception
• Temperature sense – comparisons
• Reflexes
• Distal strength testing
  – “Bring your toes up”
  – “Curl your toes down”
  – “Spread your toes”

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Work-up

• CBC, CMP, LFTs
• ESR, CRP, RF, ANA, ENA, ANCAs, ACE
• Hgb A1c, fasting glucose, 2h glucose tolerance, TSH, T3, T4, B12, MMA, B6, B1, D
• Lyme titer, RPR, HIV, Hepatitis panel,
• Quantitative immunoglobulins, Special antibodies
• immunofixation electrophoresis
• CSF
• EMG/NCS, consider MRI Brain/Spine, CT-C,A,P
• Nerve/muscle biopsy, Skin biopsy*, Autonomic testing*

*consider if EMG/NCS normal
‘The Big 4’ – The Most Common

- Diabetes AND impaired glucose tolerance
  - A1c, 2h glucose tolerance test, fasting glucose
- B12 deficiency
  - B12 + MMA
- Alcohol
  - CAGE questionnaire
- Plasma cell dyscrasias (paraproteinemias)
  - immunofixation electrophoresis

Nerve Conduction Studies (NCS)

Electromyography (EMG)

Autonomic testing – sweat output as measure of small fiber neuropathy = QSART

LENGTH DEPENDENT
SWEAT LOSS – DISTAL IS < 1/3 PROXIMAL SWEAT VOLUME
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Why Do We Care About Details?

- Not to Miss a Treatable Cause
- Not to Miss an Immune-mediated Neuropathy – since it is often treatable
- Prevent Further Worsening of Neuropathy
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<td>• Vitamin deficiencies</td>
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<td>• Inflammatory conditions</td>
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<tr>
<td>• Cancer</td>
<td></td>
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<tr>
<td>• Drugs, Toxins</td>
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<tr>
<td>• Nerve injury at specific locations</td>
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<tr>
<td>• Immune Mediated Neuropathies</td>
<td></td>
</tr>
<tr>
<td>• Genetic</td>
<td></td>
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<tr>
<td>• Diabetes</td>
<td></td>
</tr>
<tr>
<td>• Thyroid</td>
<td></td>
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<td>• Uremic (usually on hemodialysis)</td>
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<td>• Vitamin B12</td>
<td>• Vasculitides – often are very painful, multifocal, and require nerve and muscle biopsy. Treated with steroids and immunosuppressants</td>
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<td>• Vitamin B6</td>
<td>EVALUATE FOR SECONDARY CAUSES</td>
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<tr>
<td>• Vitamin B1</td>
<td>• Systemic Lupus Erythematosus</td>
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<tr>
<td>• Vitamin E</td>
<td>• Celiac Sprue-related Neuropathy</td>
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<td>• Connective Tissue Disorders</td>
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<td>• Sarcoidosis</td>
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## Infections

- HIV
- Hepatitis
- Lyme disease
- Leprosy

## Cancer

- Due to any cancer in general
- Lymphoma
- Multiple Myeloma
- Result of Cancer Treatments
- Paraneoplastic – Specifically anti-Hu

## Paraproteinemias

- MGUS – often mimics CIDP (demyelinating)
- Waldenstrom – more axonal
- Amyloidosis – get significant autonomic disturbance
- Multiple Myeloma – neuropathy usually results more from chemotherapy (bortezomib)
- POEMS – Polyneuropathy, Organomegaly, Endocrinopathy, M-protein Spike, Skin Changes.

## Drugs, Toxins

- Chemotherapy drugs
- Amiodarone
- Dilantin
- Disulfiram
- Dapsone, Ethambutol
- Leflunomide
- Alcohol
- “Huffing”
- Agent Orange
### Nerve Injuries
- Carpal Tunnel
- Ulnar Neuropathy
- Common Peroneal Neuropathy

### Immune-Mediated Neuropathies
- Evaluation based on specific antibodies in the serum
- Common ones: anti-GM-1, anti-GQ1b, anti-tubulin and anti-Hu, anti-TS-HDS
- Treatment is related to immune therapies and immunomodulation
- IVIG, Plasmapheresis, Steroids, Rituximab

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### Autoimmune Neuropathy Spectrum
- Acquired, Immune Mediated Neuropathies
  - GBS – reaches nadir in < 4 weeks
  - CIDP – reaches nadir in > 8 weeks
Acquired Demyelinating Features

Typical GBS (AIDP)
- 90% + of GBS have AIDP presentation
- Sensory phase of 3 - 10 days
  - Numbness, tingling, tightness
  - Pain in 75-90%: often in the lower back or proximal legs
- Weakness spreads from legs to arms
  - Facial involvement in about 50% of otherwise typical cases
  - DTRs decreased to absent –
    - not always global
- Autonomic involvement in ~ 70% (may be severe)
  - Requires ICU stay
- 25-40% require ventilator assistance (may be very rapid)
  - 1/3 require intubation
  - Predictors of impending respiratory failure may prompt PLEX instead of IVIG to allow for faster treatment response

GBS Diagnostic Criteria

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<th>Necessary Criteria</th>
<th>Supportive Criteria</th>
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<td>Symmetric weakness</td>
<td>Sensory sx./signs</td>
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<tr>
<td>Areflexia</td>
<td>CN weakness (VII)</td>
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<tr>
<td>Progression &lt; 4 weeks</td>
<td>ANS dysfunction</td>
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<td>High CSF protein</td>
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<td>Demyelinating EPS</td>
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GBS Overview

Typical
- Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP)

Atypical
- Acute Motor Axonal Neuropathy (AMAN)
- Acute Motor Sensory Axonal Neuropathy (AMSAN)

Both AMAN and AMSAN have association with GD1a and GM1
- Miller Fisher Variant (MFS) – GQ1b
- Pharyngeal Cervical Variant – GT1a
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<th>Chronic Inflammatory Demyelinating Polyradiculoneuropathy</th>
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<td>• Responds to IVIG or Plasmapheresis</td>
<td>• Chronic progressive, stepwise, or recurrent symmetric proximal and distal weakness and sensory dysfunction of all extremities, developing over at least 2 months and — Absent or reduced reflexes in all extremities</td>
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<td>1. Elevated CSF protein with leukocyte count &lt; 10/mm (level A)</td>
<td>• Responds to IVIG, Steroids, or Plasmapheresis</td>
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<td>2. MRI showing GAD enhancement and/or hypertrophy of cauda, lumbosacral, or cervical nerve roots, or brachial or lumbosacral plexus (level C)</td>
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<td>3. Abnormal sensory electrophysiology in at least one nerve (good practice point) 1. Normal sural with abnormal median (excluding CTS) or radial SNAP 2. CV &lt; 80% of LLN (&lt;70% if SNAP amplitude &lt;80% of LLN) 3. Delayed SSEPs without CNS disease</td>
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**Multifocal Motor Neuropathy**

- Associated with anti-GM1 IgM antibodies
- Antibodies bind to node of Ranvier structures of motor axons (GM1 enriched) and fix complement.
- Incidence: 0.6/100K
- Clinically:
  - non-contiguous motor nerves affected
  - weakness far in excess of atrophy noted (in distinction to MND)
  - asymmetric upper limb onset without sensory complaints
  - 20-30% can have brisk tendon reflexes
  - Combining galatocerbroside with GM1 increases sensitivity to 75%

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**Neuropathy – Treatment Principles**

- Address Reversible Causes of Neuropathy
  - B12 deficiency - 2000mcg daily by mouth or IM
  - B6 toxicity – reduce amount
  - Alcohol abuse – abstinence, vitamin replenishment
- Pain Management – Try different classes +/- Tramadol before going to Opiates
  - Tricyclics
  - SNRIs
  - Sodium Channel Blockers
  - Calcium Channel Blockers
  - Tramadol
  - Opiates (long-acting)

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**Treatment**

- **Anticonvulsants**
  - Gabapentin
  - Pregabalin
  - Topiramate
  - Lamotrigine
  - Carbamazepine
  - Oxcarbazepine

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**Treatment**

- **Antidepressants**
  - Amitriptyline (TCA)
  - Nortriptyline (TCA)
  - Desipramine (TCA)
  - Duloxetine (SNRI)
  - SSRIs
Treatment

- Topical Anesthetics
  - 5% Lidocaine patch
  - 0.075% Capsaicin patch

- Opioids
  - Tramadol
  - Oxycodone

Common Mimics

- RLS
  - Requip
  - Check Ferritin

- PLMS
  - Sleep study

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Case Presentation

- 54-year old man presents to the ER with recent onset of numbness and tingling in bilateral hands and feet (“stocking-glove distribution”)

- Examination – Reportedly Normal
- Blood sugar – 127 (Elevated)
- Diagnosis?
### Case presentation (Cont’d)

- **Diagnosis:** “Diabetic neuropathy”
  - Start metformin
- 2 days later, “increased numbness, worse tingling, and with pain”
- Examination with diminished reflexes
  - Diagnosis: ?

### Case Follow-up

- CSF: Protein elevated at 124
- Diagnosis: Guillain-Barre Syndrome
  - IVlg treatment with full remission

### Genetic causes

- Suggested by a very long course
- No treatable reason found despite repetitive evaluation
- Loss of strength (motor nerves damaged) without much reported sensory loss (usually)
- Pain < dysfunction
- Family history

### Genetic causes

- Charcot-Marie-Tooth disease
  - Many forms, many genes
- HNPP (PMP-22)
  - “Tool-belt” pressure causing pain and weakness
  - “Foot drop”
- Porphyria
- Amyloidosis
### Rare genetic causes

- Fabry’s disease (alpha-galactosidase)
- Metachromatic leukodystrophy (aryl sulfatase)
- Adrenoleukodystrophy
- Refsum's disease (phytanic acid)

### Hereditary Neuropathies

#### Neuropathic Gait

- Chronic sensory neuropathy
- High arches & Hammer toes
- Preserved gastrocnemius size

#### Hereditary Neuropathies

- CMT 1A
- PMP-22 duplication
- CMT 1X
- CMT 2A2

Pictures from neuromuscular.wustl.edu, courtesy of Dr. Alan Pestronk
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<td>Normal Strength</td>
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