Overview of Myopathies, and Neuropathies in the Hospital

Myopathies, neuropathies, and polyneuropathies are neuromuscular disorders frequently encountered in the hospital setting, often presenting as weakness, sensory deficits, or autonomic dysfunction. Myopathies involve primary muscle dysfunction (e.g., statin-induced myopathy, critical illness myopathy, dermatomyositis), neuropathies affect peripheral nerves (e.g., Guillain-Barré syndrome (GBS), acute motor axonal neuropathy (AMAN), chronic inflammatory demyelinating polyneuropathy (CIDP)), and polyneuropathies indicate widespread peripheral nerve involvement (e.g., diabetic polyneuropathy, chemotherapy-induced polyneuropathy, critical illness polyneuropathy (CIP)). These conditions can be acute or chronic, with prevalence varying by cause—diabetic polyneuropathy affects ~30% of diabetic patients, while critical illness polyneuropathy occurs in 25-50% of ICU patients (StatPearls, 2025). Hospitalists play a critical role in early recognition, diagnostic workup, and management to prevent complications like respiratory failure or permanent disability. This guide provides a comprehensive overview of myopathies, neuropathies, and polyneuropathies in the hospital, including clinical presentation, pathophysiology, diagnostic studies, complications, causes, treatment strategies, hospitalist implications, and includes tables and clinical scenarios for practical application.

Pathophysiology

Myopathies:

- Statin-Induced Myopathy:
  - Statins impair mitochondrial function and increase oxidative stress, leading to muscle fiber necrosis and myotoxicity.
- Critical Illness Myopathy (CIM):
  - Sepsis and prolonged corticosteroid use cause muscle membrane inexcitability and reduced protein synthesis, resulting in muscle fiber atrophy.
- Dermatomyositis:
  - Autoimmune-mediated inflammation with CD4+ T-cell infiltration and complement activation causes perifascicular atrophy and muscle fiber damage.

Neuropathies:

- Guillain-Barré Syndrome (GBS):
  - Post-infectious autoimmune demyelination (often Campylobacter jejuni) with anti-ganglioside antibodies (e.g., anti-GM1) targeting Schwann cells, causing conduction block and weakness.
- Acute Motor Axonal Neuropathy (AMAN):
  - A GBS variant with primary axonal degeneration (not demyelination), often associated with anti-GD1a antibodies, leading to motor-predominant weakness.
- Chronic Inflammatory Demyelinating Polyneuropathy (CIDP):
  - Chronic autoimmune demyelination with macrophage-mediated stripping of myelin, causing progressive or relapsing weakness and sensory loss.

Polyneuropathies:

- Diabetic Polyneuropathy:
  - Hyperglycemia causes oxidative stress, advanced glycation end-products (AGEs), and microvascular damage, leading to axonal degeneration and sensory loss.
- Chemotherapy-Induced Polyneuropathy (CIPN):
  - Neurotoxic agents (e.g., cisplatin, paclitaxel) cause axonal damage via microtubule disruption or mitochondrial toxicity, resulting in sensory neuropathy.
- Critical Illness Polyneuropathy (CIP):
  - Sepsis and multi-organ failure lead to axonal degeneration via microcirculatory dysfunction, oxidative stress, and cytokine-mediated nerve injury.

Causes

Myopathies:

- Statin-Induced Myopathy:
  - Statins (e.g., atorvastatin), often dose-dependent, risk increased with fibrates.
- Critical Illness Myopathy (CIM):
  - Sepsis, prolonged corticosteroid use, neuromuscular blockers in ICU.
- Dermatomyositis:
  - Autoimmune, often paraneoplastic (e.g., lung, breast cancer), associated with anti-Jo-1 antibodies.

- Other:
  - Hypokalemia, hypophosphatemia, viral infections (e.g., influenza), muscular dystrophies.

Neuropathies:

- Guillain-Barré Syndrome (GBS):
- Post-infectious (Campylobacter jejuni, CMV, EBV), vaccines (rare).
- Acute Motor Axonal Neuropathy (AMAN):
- Variant of GBS, often post-Campylobacter, more common in Asia.
- Chronic Inflammatory Demyelinating Polyneuropathy (CIDP):
- Autoimmune, often idiopathic, associated with HIV, diabetes.
- Other: Traumatic nerve injury, vitamin B12 deficiency, toxic (e.g., lead, arsenic).

Polyneuropathies:

- Diabetic Polyneuropathy:
  - Chronic hyperglycemia, poor glycemic control (HbA1c >7%).
- Chemotherapy-Induced Polyneuropathy (CIPN):
  - Platinum agents (cisplatin), taxanes (paclitaxel), vinca alkaloids (vincristine).
- Critical Illness Polyneuropathy (CIP):
  - Sepsis, multi-organ failure, prolonged ICU stay, mechanical ventilation.
- Other:
  - Alcohol, uremia, hypothyroidism, vitamin deficiencies (B12, thiamine).

Clinical Presentation

Myopathies:

- Statin-Induced Myopathy:
  - Proximal weakness (e.g., difficulty rising from chair), myalgias, fatigue, onset weeks to months after starting statin.
  - Normal reflexes, no sensory loss, possible muscle tenderness.
- Critical Illness Myopathy (CIM):
  - Generalized weakness, often after 7-10 days in ICU, difficulty weaning from ventilator.
  - Hyporeflexia, flaccid paralysis, normal sensation, muscle atrophy.
- Dermatomyositis:
  - Proximal weakness, heliotrope rash (periorbital edema), Gottron’s papules (over knuckles), myalgias.

- Normal reflexes, no sensory loss, possible dysphagia (esophageal involvement).

Neuropathies:

- Guillain-Barré Syndrome (GBS):
  - Ascending weakness (legs to arms), areflexia, sensory loss (paresthesia), onset over days to weeks.
  - Autonomic dysfunction (e.g., orthostatic hypotension, arrhythmias), respiratory involvement (dyspnea).
- Acute Motor Axonal Neuropathy (AMAN):
  - Rapid motor weakness (more prominent than sensory), areflexia, minimal sensory loss.
  - Often post-Campylobacter, faster progression than GBS, less autonomic involvement.
- Chronic Inflammatory Demyelinating Polyneuropathy (CIDP):
  - Progressive or relapsing weakness (proximal and distal), sensory loss, hyporeflexia, onset over >8 weeks.
  - May mimic GBS but chronic course, less autonomic dysfunction.

Polyneuropathies:

- Diabetic Polyneuropathy:
  - Symmetrical distal sensory loss ("stocking-glove"), burning pain, numbness, hyporeflexia (ankle jerks absent).
  - Chronic, often with foot ulcers, gait instability.
- Chemotherapy-Induced Polyneuropathy (CIPN):
  - Distal sensory loss (paresthesia, numbness), burning pain, hyporeflexia, onset during/after chemotherapy.
  - Motor weakness rare, sensory-predominant, dose-dependent.
- Critical Illness Polyneuropathy (CIP):
  - Distal weakness, sensory loss, hyporeflexia, onset after 7-14 days in ICU.
  - Difficulty weaning from ventilator, flaccid paralysis, normal CK (distinguishes from CIM).

Diagnostic Studies

Labs:

- Myopathies:
  - **CK:** Elevated (e.g., >5,000 U/L in statin-induced myopathy, CIM; 1,000-5,000 U/L in dermatomyositis).
  - **Aldolase:** Elevated in muscle damage.
  - **Autoantibodies:** Anti-Jo-1, anti-Mi-2 (dermatomyositis).
  - **Electrolytes:** Hypokalemia, hypophosphatemia (metabolic myopathy).
- Neuropathies/Polyneuropathies:
  - **HbA1c:** Elevated in diabetic polyneuropathy (>7%).
  - **Vitamin B12/Folate:** Deficiency (<200 pg/mL for B12, CIPN differential).
  - **Serologies:** HIV, Lyme, Campylobacter (GBS, AMAN).
  - **Autoantibodies:** Anti-GM1 (GBS), anti-MAG (CIDP).
  - **Chemotherapy History:** Review for cisplatin, paclitaxel exposure (CIPN).
  - **Electrolytes:** Hypokalemia, hypophosphatemia (metabolic myopathy).

Electrophysiology:

- EMG/NCS:
  - **Myopathies:** Myopathic pattern (small, polyphasic motor units, early recruitment in statin-induced myopathy, CIM, dermatomyositis).
- Neuropathies:
  - **GBS:** Demyelination (slowed conduction velocity, prolonged distal latency).
  - **AMAN:** Axonal loss (reduced CMAP amplitude, normal conduction velocity).
  - **CIDP:** Demyelination (conduction block, temporal dispersion).
- Polyneuropathies:
  - **Diabetic Polyneuropathy:** Axonal loss (reduced amplitudes, distal slowing).
  - **CIPN:** Axonal sensory neuropathy (reduced sensory nerve action potentials).
  - **CIP:** Axonal motor and sensory loss, normal nerve conduction velocity.

Imaging:

- MRI Muscle:
  - Edema, fatty infiltration (dermatomyositis, statin-induced myopathy).

- MRI Spine:
  - Rule out cord compression (if sensory/motor deficits overlap, e.g., GBS differential).
- CT/MRI Brain:
  - If CNS involvement suspected (e.g., stroke mimic in neuropathy).

Other Tests:

- Muscle Biopsy:
  - **Statin-Induced Myopathy:** Necrotic fibers, no inflammation.
  - **CIM:** Muscle fiber atrophy, loss of thick filaments.
  - **Dermatomyositis:** Perifascicular atrophy, inflammatory infiltrates.
- Nerve Biopsy:
  - CIDP (demyelination, onion-bulb formation), rarely needed.
- CSF Analysis:
  - **GBS/AMAN:** Albuminocytologic dissociation (protein 100-200 mg/dL, WBC <10/µL).
  - **CIDP:** Similar to GBS but chronic course.
- Genetic Testing:
  - If hereditary neuropathy suspected (e.g., Charcot-Marie-Tooth, CMT).

Complications

Myopathies:

- Rhabdomyolysis:
  - 10-20% in statin-induced myopathy, AKI (Cr rise), hyperkalemia (K+ >5.5 mEq/L).
- Respiratory Failure:
  - 5-10% in CIM, FVC <15 mL/kg, often with CIP overlap.
- Cardiomyopathy:
  - 20-30% in dermatomyositis, EF <40%, heart failure.

Neuropathies:

- Respiratory Failure:
  - 20-30% in GBS/AMAN, FVC <15 mL/kg, intubation required.
- Autonomic Dysfunction:
  - 10-20% in GBS/AMAN, orthostatic hypotension, arrhythmias.
- Chronic Disability:
  - 30-50% in CIDP, persistent weakness, sensory deficits.

Polyneuropathies:

- Falls/Injuries:
  - 30-50% in diabetic polyneuropathy/CIPN, due to sensory loss, weakness.
- Foot Ulcers/Amputations:
  - 5-10% in diabetic polyneuropathy, due to sensory loss, infection.
- Prolonged ICU Recovery:
  - 50-70% in CIP, delayed ventilator weaning, 3-6 month recovery.

Treatment Strategies

Myopathies:

- Statin-Induced Myopathy:
  - **Discontinue Statin:** Stop immediately, monitor CK.
  - **Supportive:** Fluids (NS 1-2 L/h for rhabdomyolysis, target urine output 200-300 mL/h).
- Critical Illness Myopathy (CIM):
  - **Supportive:** Minimize corticosteroids, physical therapy, optimize nutrition.
  - **Respiratory:** Monitor FVC q4-6h, NIV/intubation if FVC <15 mL/kg.
- Dermatomyositis:
  - **Steroids:** Prednisone 1 mg/kg/day PO x 4-6 weeks, taper slowly.
  - **Immunosuppressants:** Methotrexate 15 mg PO weekly or azathioprine 2 mg/kg/day.
  - **IVIG:** 2 g/kg IV over 2-5 days (refractory cases).
  - Duration: Supportive until resolution (statin-induced, CIM), steroids 6-12 months (dermatomyositis).

Neuropathies:

- Guillain-Barré Syndrome (GBS) and Acute Motor Axonal Neuropathy (AMAN):
  - **IVIG:** 0.4 g/kg/day IV x 5 days (first-line for both).
  - **Plasmapheresis:** 5 sessions over 7-10 days (alternative, equally effective).
  - **Supportive:** Monitor FVC q4-6h, intubate if FVC <15 mL/kg, DVT prophylaxis (enoxaparin 40 mg SC daily).
- Chronic Inflammatory Demyelinating Polyneuropathy (CIDP):
  - **IVIG:** 0.4 g/kg/day IV x 5 days, then 1 g/kg q3 weeks maintenance.
  - **Steroids:** Prednisone 1 mg/kg/day PO, taper over months.
  - **Immunosuppressants:** Azathioprine 2 mg/kg/day (steroid-sparing).

    - **Pain Management:** Gabapentin 300 mg PO TID, titrate to 1,800-3,600 mg/day; amitriptyline 25-100 mg PO qHS.
- Duration: IVIG/plasmapheresis for GBS/AMAN (5-10 days), CIDP maintenance (months to years), pain management indefinite.

Polyneuropathies:

- Diabetic Polyneuropathy:
    - **Glycemic Control:** Target HbA1c <7% (insulin, metformin).
    - **Pain Management:** Pregabalin 150-300 mg PO BID, duloxetine 60 mg PO daily.
- Chemotherapy-Induced Polyneuropathy (CIPN):
    - **Prevention:** Reduce dose (e.g., cisplatin), neuroprotective agents (e.g., duloxetine 60 mg PO daily).
    - **Pain Management:** Gabapentin 300-1,800 mg/day, duloxetine 60 mg PO daily.
- Critical Illness Polyneuropathy (CIP):
    - **Supportive:** Physical therapy, minimize sedatives (e.g., propofol), optimize nutrition.
    - **Recovery:** Gradual, 3-6 months post-ICU, focus on rehabilitation.
- Duration: Chronic management (diabetic neuropathy, CIPN), supportive until recovery (CIP).

Hospital Medicine Implications

Early Recognition:

- Myopathies:
    - Proximal weakness, elevated CK (e.g., statin-induced, CIM, dermatomyositis).
- Neuropathies:
    - Rapid weakness (GBS, AMAN), chronic sensory loss (CIDP).
- Polyneuropathies:
    - Symmetrical sensory loss (diabetic, CIPN), ICU setting (CIP).

Consultations:

- Neurology:
    - For EMG/NCS, IVIG/plasmapheresis, biopsy.
- Rheumatology:
    - For dermatomyositis, inflammatory myopathies.

- Endocrinology:
  - For diabetic neuropathy, metabolic causes.
- Physical Therapy:
  - For mobility, rehabilitation.

Monitoring:

- FVC q4-6h (GBS, AMAN, CIM with respiratory risk).
- CK, Cr q12h (rhabdomyolysis in myopathies).
- EMG/NCS results, monitor for recovery (CIDP, CIP).

Discharge Planning:

Medications: Pain control (gabapentin), steroids/IVIG (inflammatory).

Follow-Up: Neurology, endocrinology within 1-2 weeks.

Education: Fall prevention, foot care (diabetic neuropathy), medication adherence.

Table: Myopathies, Neuropathies, and Polyneuropathies - Key Features

Condition	Presentation	Pathophysiology	Labs/Tests	Complications	Treatment
Statin-Induced Myopathy	Proximal weakness, myalgias	Mitochondrial toxicity, oxidative stress	CK >5,000 U/L, EMG: Myopathic	Rhabdomyolysis, AKI	Discontinue statin, fluids
Critical Illness Myopathy (CIM)	Generalized weakness, ICU	Muscle membrane inexcitability, atrophy	CK 1,000-5,000 U/L, EMG: Myopathic	Respiratory failure, prolonged recovery	Supportive, PT, minimize steroids
Dermatomyositis	Proximal weakness, heliotrope rash	Autoimmune, perifascicular atrophy	CK 1,000-5,000 U/L, anti-Jo-1	Cardiomyopathy, respiratory failure	Prednisone 1 mg/kg/day, IVIG
Guillain-Barré Syndrome (GBS)	Ascending weakness, areflexia	Demyelination, anti-GM1 antibodies	CSF: Elevated protein, EMG: Slowed conduction	Respiratory failure, dysautonomia	IVIG 0.4 g/kg/day x 5 days
Acute Motor Axonal Neuropathy (AMAN)	Motor weakness, minimal sensory loss	Axonal degeneration, anti-GD1a antibodies	EMG: Reduced CMAP, CSF: Protein rise	Respiratory failure, disability	IVIG, plasmapheresis

Condition	Presentation	Pathophysiology	Labs/Tests	Complications	Treatment
Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)	Progressive weakness, sensory loss	Chronic demyelination, macrophage-mediated	EMG: Conduction block, CSF: Protein rise	Chronic disability, falls	IVIG, steroids, azathioprine
Diabetic Polyneuropathy	Stocking-glove sensory loss, pain	Axonal degeneration, hyperglycemia	HbA1c >7%, EMG: Reduced amplitudes	Foot ulcers, amputations	Pregabalin 150-300 mg BID, glycemic control
Chemotherapy-Induced Polyneuropathy (CIPN)	Distal sensory loss, burning pain	Axonal damage, microtubule disruption	EMG: Sensory axonal loss, normal CK	Chronic pain, falls	Gabapentin, duloxetine
Critical Illness Polyneuropathy (CIP)	Distal weakness, ICU patient	Axonal injury, sepsis-related	EMG: Reduced amplitudes, normal CSF	Prolonged ICU recovery, disability	Supportive, PT, minimize sedatives

Clinical Scenarios

Scenario 1: Middle-Aged Female with Statin-Induced Myopathy

- Presentation: A 55-year-old female on atorvastatin 80 mg daily for 2 months presents with 1 week of proximal muscle weakness and myalgias. Exam shows T 37°C, BP 130/80 mmHg, HR 80 bpm, RR 16/min, proximal weakness (4/5), normal reflexes, no sensory loss.
- Diagnostic Workup: **Labs:** CK 6,000 U/L, Cr 1.8 mg/dL (baseline 1.0), K+ 5.8 mEq/L, EMG: Myopathic pattern, muscle biopsy: Necrotic fibers, no inflammation.
- Diagnosis: Statin-induced myopathy → Proximal weakness, elevated CK, recent statin use.
- Management: Admit to medicine (rhabdomyolysis risk). Discontinue atorvastatin. Start fluids (NS 2 L/h, target urine output 200-300 mL/h). Correct K+ (insulin/glucose, calcium gluconate for hyperkalemia). Monitor CK, Cr q12h (CK decreases to 2,000 U/L by day 3). Day 4: Strength improves (5/5), Cr 1.2 mg/dL, discharged with cardiology follow-up for alternative lipid management.

Scenario 2: Young Male with Acute Motor Axonal Neuropathy (AMAN)

- Presentation: A 28-year-old male presents with 4 days of rapidly progressive weakness and difficulty walking after a recent Campylobacter infection. Exam shows T 37°C, BP 110/70 mmHg, HR 90 bpm, RR 20/min, distal weakness (3/5), areflexia, minimal sensory loss.
- Diagnostic Workup: **FVC 18 mL/kg, CSF:** Protein 120 mg/dL, WBC 3/μL, EMG/NCS: Reduced CMAP amplitude (axonal pattern), anti-GD1a antibodies positive, labs: WBC 9,000/μL, Cr 0.8 mg/dL.
- Diagnosis: Acute motor axonal neuropathy (AMAN) → Rapid motor weakness, areflexia, post-infectious.
- Management: Admit to ICU (respiratory risk). Start IVIG 0.4 g/kg/day IV x 5 days. Monitor FVC q4h (drops to 14 mL/kg, intubated day 2). DVT prophylaxis (enoxaparin 40 mg SC daily). Consult neurology: Plasmapheresis deferred (IVIG started). Day 7: Extubate (FVC 20 mL/kg), discharged to rehab with neurology follow-up.

Scenario 3: Elderly Female with Chemotherapy-Induced Polyneuropathy (CIPN)

- Presentation: A 60-year-old female with breast cancer, on paclitaxel for 3 months, presents with 2 weeks of numbness, burning pain, and tingling in her hands and feet. Exam shows T 37°C, BP 120/80 mmHg, HR 80 bpm, RR 16/min, distal sensory loss (pinprick reduced), hyporeflexia, no weakness.
- Diagnostic Workup: **Labs:** HbA1c 5.5%, B12 400 pg/mL, EMG/NCS: Sensory axonal neuropathy (reduced SNAP amplitudes), normal CK, recent chemo history confirmed.
- Diagnosis: Chemotherapy-induced polyneuropathy (CIPN) → Distal sensory loss, burning pain, paclitaxel exposure.
- Management: Admit to medicine (symptomatic neuropathy). Start gabapentin 300 mg PO TID, titrate to 1,800 mg/day. Consult oncology: Paclitaxel dose reduction planned. Monitor sensory exams qshift, pain scores q4h (improves to 2/10 by day 3). Day 4: Pain controlled, discharged with oncology follow-up, gabapentin continued.

Table: Hospitalist Management Checklist

Task	Myopathies (Dermatomyositis)	Neuropathies (GBS/AMAN)	Polyneuropathies (CIP)	Monitoring	Consults
Initial Diagnosis	CK, EMG, muscle biopsy	FVC, EMG/NCS, CSF analysis	EMG, HbA1c, B12 levels	FVC q4-6h, CK q12h	Neurology, rheumatology

Task	Myopathies (Dermatomyositis)	Neuropathies (GBS/AMAN)	Polyneuropathies (CIP)	Monitoring	Consults
Treatment	Prednisone, IVIG	IVIG, plasmapheresis	Gabapentin, supportive	EMG/NCS, Cr q24h	Endocrinology, PT
Supportive Care	Fluids (rhabdomyolysis), PT	DVT prophylaxis, intubation	Foot care, fall prevention	Vitals q4h, sensory exams	Neurology, PT
Follow-Up	Rheumatology, monitor CK	Neurology, monitor recovery	Endocrinology, foot exams	PFTs post-discharge	Primary care