

Peripheral Neuropathy

Acuity / Time course	Likely causes of polyneuropathy	Clinical clues / phenotype	
Hyperacute (hours–days)	Guillain–Barré syndrome (AIDP/AMAN/AMSAN)	Rapid ascending weakness, areflexia, autonomic dysfunction, cranial nerve involvement, post-infectious history	
	Acute intermittent porphyria	Abdominal pain, psychiatric symptoms, autonomic instability, motor-predominant neuropathy	
	Vasculitic neuropathy (fulminant)	Painful, asymmetric onset (may present as mononeuritis multiplex evolving to confluent polyneuropathy), systemic inflammatory features	
	Toxic neuropathy (arsenic, thallium, organophosphates, hexacarbon solvents)	Exposure history, GI symptoms, encephalopathy, painful neuropathy	
	Critical illness polyneuropathy	ICU/sepsis context, difficulty weaning ventilation, diffuse weakness	
	Diphtheritic neuropathy (less common)	Recent pharyngitis, bulbar symptoms	
	Subacute (days–8 weeks)	CIDP (acute-onset variant / evolving CIDP)	Similar to GBS initially but progression >8 weeks or relapsing course
Nutritional deficiency (B12, copper, thiamine, vitamin E)		Myeloneuropathy, sensory ataxia, malnutrition/bariatric history	
Toxic/drug-induced (chemotherapy—vincristine, platinum, taxanes; metronidazole; nitrofurantoin; linezolid; amiodarone)		Medication temporal association	
Paraneoplastic neuropathy		Weight loss, cancer risk factors, painful sensory neuronopathy	
Infectious (HIV seroconversion, Lyme, CMV, hepatitis)		Constitutional symptoms, exposure history	
Amyloid neuropathy		Painful small-fiber/autonomic neuropathy, weight loss, cardiomyopathy	
Sjögren syndrome / connective tissue disease		Sicca symptoms, sensory ganglionopathy	
POEMS syndrome		Organomegaly, endocrinopathy, skin changes, monoclonal protein	
Uremic neuropathy (accelerated)		Renal failure context	
Chronic (>8 weeks to months/years)		Diabetic distal symmetric polyneuropathy	Length-dependent sensory symptoms, diabetes/metabolic syndrome
		Alcohol-related neuropathy	Chronic alcohol use ± nutritional deficiency
	Chronic kidney disease (uremic)	Distal sensorimotor neuropathy	
	Hypothyroidism	Fatigue, weight gain, entrapment neuropathies	
	Monoclonal gammopathy-associated neuropathy (MGUS, anti-MAG)	Slowly progressive sensory > motor neuropathy	

Acuity / Time course	Likely causes of polyneuropathy	Clinical clues / phenotype
	CIDP	Progressive/relapsing weakness, proximal + distal involvement, areflexia
	Hereditary neuropathies (CMT, HNPP, hereditary amyloid)	Family history, pes cavus, long-standing course
	Chronic toxic exposure (alcohol, heavy metals, solvents)	Occupational/exposure history
	HIV-associated DSP	Chronic painful distal neuropathy
	Leprosy (geographic context)	Skin lesions, palpable nerves
	Sarcoidosis	Multisystem granulomatous disease
Relapsing / episodic	CIDP	Stepwise deterioration/improvement
	Hereditary neuropathy with liability to pressure palsies (HNPP)	Recurrent focal deficits
	Porphyria	Episodic neurovisceral attacks
	Autoimmune / vasculitic neuropathy	Recurrent painful asymmetric deficits

Useful “pattern recognition” overlays

Phenotype	Think of
Predominantly motor, acute	GBS, porphyria, lead toxicity
Painful asymmetric neuropathy	Vasculitis, diabetes (mononeuritis multiplex), amyloid
Sensory ataxic neuropathy	Sjögren, paraneoplastic, cisplatin
Small-fiber + autonomic	Diabetes, amyloid, Fabry, HIV
Demyelinating	GBS, CIDP, anti-MAG, hereditary demyelinating neuropathy
Axonal	Diabetes, toxins, nutritional deficiency, vasculitis

Practical diagnostic heuristic

Acute + weakness + areflexia = GBS until proven otherwise.

Chronic length-dependent sensory symptoms = diabetes/metabolic/toxic until proven otherwise.

Painful asymmetric neuropathy = vasculitis/amyloid/diabetic multifocal process until proven otherwise.