Casual Friday Series

Interrupting Neuropathy Pt II

A Biogenetix Clinical Presentation BIOGENETIX.COM



Disclaimer

- Information in this presentation is not intended to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.
- The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.







CLINICAL FEATURES





Signs

Signs of peripheral neuropathy also include sensory, motor and autonomic components.

• The most prominent autonomic sign of neuropathy is orthostatic hypotension



CLASSIFICATION

There are many ways to classify peripheral neuropathy. One helpful method is to consider four categories, namely <u>etiology, distribution, pathology and</u> <u>modality</u>.



https://www.urmc.rochester.edu/MediaLibraries/URMCMedia/center-experiential-learning/cme/types-of-activities/documents/PERIPHERAL-NEUROPATHY-HANDOUT.pdf The mnemonic **DANG THERAPIST** is helpful in recalling the more common causes of peripheral neuropathy:

Diabetes Mellitus Alcohol Nutritional (B₁₂ deficiency) Guillain-Barre Syndrome Toxins (Pb, As, Zn, Hg) Hematologic (cancers, etc.) Endocrine (hypothyroid) Rheumatologic (SLE, rheumatoid arthritis, vasculitis) **A**myloid Porphyria Infectious (syphilis, HIV) **S**arcoid **T**umor (paraneoplastic neuropathy)





PROLONGED OVERWHELMING IMBALANCE

OXIDATIVE AGENTS: A chemical species that steals electrons from another substance, leading to the oxidation. These are reactive oxygen species (ROS), referring to a group of highly reactive molecules and free radicals containing oxygen. ANTIOXIDANTS: Molecules that help protect the body from the damage caused by ROS and free radicals. Antioxidants neutralize free radicals by donating an electron without becoming destabilized themselves.

LABORATORY INVESTIGATION

Laboratory studies play an important role in diagnosing and categorizing the peripheral neuropathies. Electrodiagnostic studies are helpful in quantitating the neuropathy, while blood and urine studies are helpful in identifying an etiology.

Electrodiagnostic Studies:

- Nerve Conduction Study
- Electromyography (EMG)



Nerve Conduction Study:

The recording and measurement of the compound nerve and muscle action potential elicited in response to a single supramaximal electrical stimulus, to measure the terminal latency, amplitude and duration of the evoked potential, as well as the conduction velocity.

Nerve conduction studies are helpful in documenting that a neuropathy exists, quantitating the severity, and noting the distribution of the neuropathy, i.e. whether it is distal, proximal or diffuse. In addition, nerve conduction studies can provide information on the modality involved, i.e. motor versus sensory, and can also give clues as to the underlying pathology, whether axonal or demyelinating. **Demyelinating neuropathies** (neuropathies due to loss or destruction of myelin) result in slowed conduction velocities and prolonged distal latencies, because conduction velocity is proportional to the velocity of the largest-diameter myelinated fibers. Dispersion of evoked compound action potentials (CAP) can also be seen in demyelinating neuropathies, because all of the action potentials elicited in response to a single electrical stimulus will not reach the recording potential at the same time. Severe demyelinating neuropathies can also produce conduction block, which is a major decrease in amplitude of the muscle CAP upon proximal stimulation of its nerve as compared to distal stimulation.

Axonal neuropathies (neuropathies due to loss of axons or their cell bodies) generally result in a reduced amplitude of the compound motor or sensory nerve action potentials.

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Electromyography (EMG):

The recording and study of insertional, spontaneous, and voluntary electrical activity of muscle.

This test allows one to physiologically evaluate the motor unit, including the anterior horn cell, peripheral nerve, and muscle. EMG is helpful when evaluating patients with weakness, in that it can help one determine whether weakness is due to anterior horn cell disease, nerve root compression, peripheral neuropathy, or an intrinsic disease of muscle itself (myopathy).

EMG can differentiate acute denervation from chronic denervation, and may thus give an indication as to the time course of the lesion causing the neuropathy.

https://www.urmc.rochester.edu/MediaLibraries/URMCMedia/center-experiential-learning/cme/types-of-activities/documents/PERIPHERAI NEUROPATHY-HANDOUT.pdf •Acute Denervation: Fibrillations and positive waves are present indicating spontaneous discharge of individual muscle fibers.

•Chronic Denervation: Voluntary motor unit potentials are of large amplitude and long duration, and are frequently polyphasic, because the motor units are enlarged as a result of reinnervation of adjacent previously denervated muscle fibers. Recruitment of additional motor units in response to increasing the force of muscular contraction is reduced for the same reason.

•Demyelinating Neuropathy: A decreased recruitment pattern is seen, since demyelination interferes with conduction of individual action potentials along the course of a peripheral nerve. Because denervation and reinnervation of muscle fibers are not features of demyelinating neuropathies, the configuration of the voluntary motor unit potentials is usually normal, and fibrillation potentials are not seen.

Blood Studies

Routine blood studies should be obtained in all patients with peripheral neuropathy in order to screen for reversible causes. The following blood tests are recommended:

Complete blood count
Chemistry profile
Sedimentation rate
Thyroid studies
Vitamin B₁₂ level
ANA, rheumatoid factor
Serum protein electrophoresis, serum immunoelectrophoresis
RPR and HIV (if the clinical situation warrants)



CBC With Differential/Platelet

Test	Current Result and Flag	Previous Result and Date	Units	Reference Interval
WBC 01	8.1		x10E3/uL	3.4-10.8
RBC ⁰¹	4.62		x10E6/uL	3.77-5.28
Hemoglobin ⁰¹	13.6		g/dL	11.1-15.9
Hematocrit ⁰¹	41.8		%	34.0-46.6
MCV ⁰¹	91		fL	79-97
MCH 01	29.4		pg	26.6-33.0
MCHC ⁰¹	32.5		g/dL	31.5-35.7
RDW ⁰¹	12.8		%	11.7-15.4
Platelets 01	360		x10E3/uL	150-450
Neutrophils ⁰¹	49		%	Not Estab.
Lymphs ⁰¹	38		%	Not Estab.
Monocytes ⁰¹	8		%	Not Estab.
Eos 01	3		%	Not Estab.
Basos ⁰¹	1		%	Not Estab.
Neutrophils (Absolute) ⁰¹	4.1		x10E3/uL	1.4-7.0
Lymphs (Absolute) ⁰¹	3.1		x10E3/uL	0.7-3.1
Monocytes(Absolute) ⁰¹	0.6		x10E3/uL	0.1-0.9
Eos (Absolute) 01	0.2		x10E3/uL	0.0-0.4
Baso (Absolute) 01	0.0		x10E3/uL	0.0-0.2
Immature Granulocytes 01	1		%	Not Estab.
Immature Grans (Abs) 01	0.1		x10E3/uL	0.0-0.1



Comp. Metabolic Panel (14)

	Test	Current Resu	lt and Flag	Previous Result and Date	Units	Reference Interval
	Glucose 01	201	High		mg/dL	70-99
	BUN ⁰¹	17			mg/dL	6-24
	Creatinine ⁰¹	0.97			mg/dL	0.57-1.00
	eGFR	69			mL/min/1.73	>59
	BUN/Creatinine Ratio	18				9-23
	Sodium ⁰¹	137			mmol/L	134-144
	Potassium ⁰¹	4.1			mmol/L	3.5-5.2
	Chloride ⁰¹	100			mmol/L	96-106
•	Carbon Dioxide, Total ⁰¹	19	Low		mmol/L	20-29
	Calcium 01	9.9			mg/dL	8.7-10.2
	Protein, Total ⁰¹	7.3			g/dL	6.0-8.5
_	Albumin	4.8			g/dL	3.8-4.9
Co	mp. Metabolic Panel (14	l) (Cont.)				
	Globulin, Total	2.5			g/dL	1.5-4.5
0	A/G Ratio	1.9				1.2-2.2
	Bilirubin, Total ⁰¹	<0.2			mg/dL	0.0-1.2
	Alkaline Phosphatase ⁰¹	110			IU/L	44-121
	AST (SGOT) 01	34			IU/L	0-40
	ALT (SGPT) ⁰¹	46	High		IU/L	0-32



Hgb A1c with eAG Estimation

Test		Current Result and Flag		Previous Result and Date	Units	Reference Interval	
A Hemoglobin A1c 01		10.6	10.6 High		%	4.8-5.6	
Please	Note: 01						
		Prediabetes: 5.7 - 6.4 Diabetes: >6.4 Glycemic control for adults with diabetes: <7.0					
Estim. A	Avg Glu (eAG)	258		mg/dL			
Thyroxin	e (T4) Free, Direct						
Test		Current Result and	Flag	Previous Result and Date	Units	Reference Interval	

C-Peptide, Serum

T4,Free(Direct) 01

 Test	st Current Result and Flag		Previous Result and Date	Units	Reference Interval
C-Peptide, Serum ⁰¹	8.5	High		ng/mL	1.1-4.4
	C-Peptide reference	interval	is for fasting patients.		

ng/dL

0.82-1.77

1.42

Vitamin D, 25-Hydroxy

Tes	st	Current Result and Flag		Previous Result and Date	Units	Reference Interval	
Vit	tamin D, 25-Hydroxy ⁰¹	28.5	Low		ng/mL	30.0-100.0	

C-Reactive Protein, Cardiac

Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
▲ C-Reactive Protein, Cardiac ⁰¹	8.69	High		mg/L	0.00-3.00
		Relat	ovascular Event		
			Low	<1.00	
			Average	1.00 - 3.00	
			High	>3.00	
Homocyst(e)ine					
Test	Current Resu	lt and Flag	Previous Result and Date	Units	Reference Interval
Homocyst(e)ine ⁰¹	14.9	High		umol/L	0.0-14.5



Urine Studies

1. The following studies are recommended to screen for reversible causes of neuropathy:

- Heavy metal screen (Hg, Pb, Zn, As)
- Urine protein electrophoresis, urine immunoelectrophoresis

***Chest x-ray**, helpful to screen for asymptomatic lung cancer that can sometimes cause a purely sensory neuropathy.



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Moderate (75th-95th perc	entile)				_e © He	eavy Metals	🎼 Envi	onmental Toxins
TEST NAME	CURRENT RESULT	PREVIOUS RESULT		CURRENT	RESULT	PREVIOUS R	ESULT	REFERENCE
₀ [©] Arsenic*	40.39		0	11.9	52			≤52 ug/g
_e [©] Lead*	0.64		0	0.52	1.16			≤1.16 ug/g
₀ [©] Mercury*	0.73		0	0.57	1.61			≤1.61 ug/g
a [©] Nickel	6.83		0	6.37	12.1			≤12.13 ug/g
🖗 Thallium*	0.29		0	0.24	0.43			≤0.43 ug/g
_ø © Tin*	1.06		0	1	3.72			≤3.72 ug/g
Tazine mercapturate*	0.05		0	0.02	0.05			≤0.05 ug/g
👫 Glyphosate	7.19		0	1.65	7.6			≤7.6 ug/g
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)*	12.24		0	8.99	23.4			≤23.4 ug/g

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59-year-old, female, dx c/ diabetes



Rescue Stage

Repair Stage

Recovery Stage









21-Day Metabolic Clearing Program Neuropathy Support Kit or Regen Support Bundle Monthly Wellness Bundle + Nexugen





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