Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) and Guillain-Barré Syndrome (GBS)

James J. Sejvar, MD, FAAN

Centers for Disease Control and Prevention

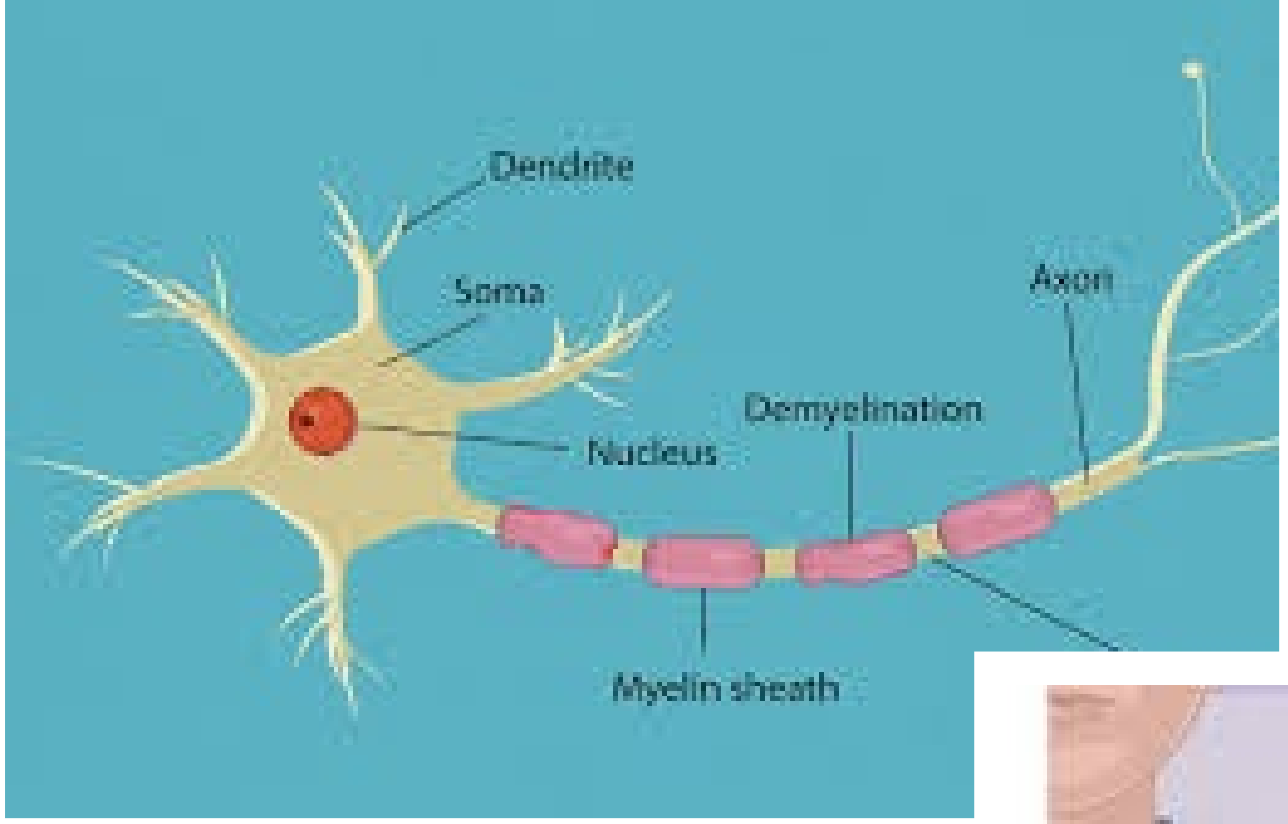
The findings and conclusions contained in this presentation are those of the author and do not represent the official position of the Centers for Disease Control and Prevention

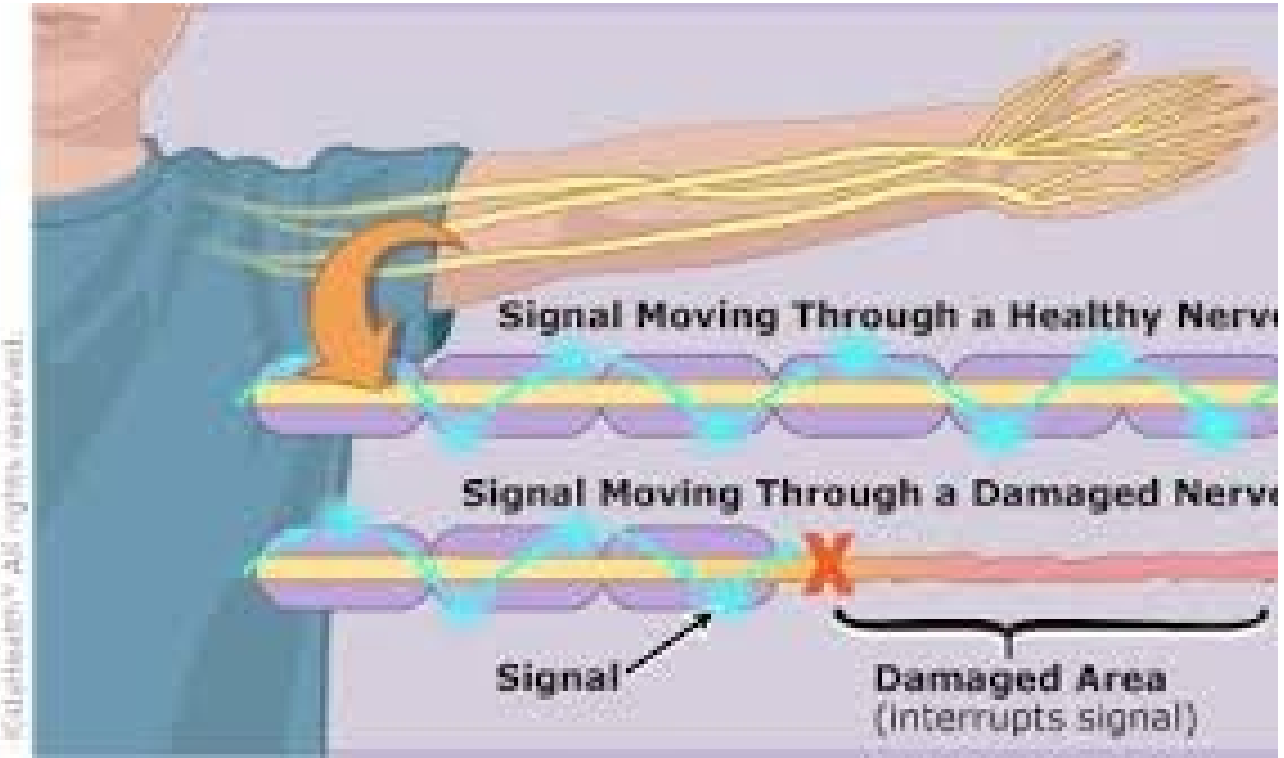
Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)

- Chronic, ongoing, progressive and/or relapsing disease of the peripheral nerves
- Results in weakness, pain, and numbness in the limbs
- “Typical” and “Atypical” presentations of CIDP
  - Typical – chronically progressive, stepwise or recurrent weakness prox. and dist. weakness and sensory dysfunction in all extremities over 2 months; decreased or absent reflexes
  - Atypical – as above, but:
    - Predominantly distal
    - Asymmetric
    - Pure motor, pure sensory

Guillain-Barré Syndrome (GBS)

- Acute, monophasic illness affecting the peripheral nerves
- Results in weakness, pain, and numbness in limbs and /or cranial nerve innervated muscles, as well as decreased/absent reflexes
- Several variants
  - Acute inflammatory demyelinating polyneuropathy (AIDP)
  - Acute motor axonal neuropathy (AMAN)
  - Fisher Syndrome


Dendrite
Soma
Axon
Demyelination
Nucleus
Myelin sheath


Signal Moving Through a Healthy Nerve
Signal Moving Through a Damaged Nerve
Signal
Damaged Area
(interrupts signal)

Case Definitions

GBS

Brighton Collaboration Criteria for GBS

Level 3
- Acute onset of bilateral, symmetric weakness of the limbs
- Decreased/absent deep tendon reflexes in affected limbs

Level 2
- Monophasic illness pattern with maximal weakness 12 hours - 28 days, followed by clinical plateau
- CSF total white count < 50 cells/mm^3 (elevated protein, absence of pleocytosis)

Level 1
- Electrophysiologic findings consistent with GBS

Level 4 – Lacking documentation to fulfill case definition, "reported case of GBS"

Level 5 – Not a case

Seivar J, et al. Guillain-Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data. Vaccine. 2011;29:599-612.

CIDP

- Clinical inclusion criteria for typical CIDP require both of the following:
  - Chronically progressive, stepwise, or recurrent symmetric proximal and distal weakness and sensory dysfunction of at least two limbs, developing over two months or longer; cranial nerves may be affected .
  - Absent or reduced tendon reflexes in all extremities
- Clinical inclusion criteria for CIDP variants require one of the following, but otherwise as in typical CIDP. However, tendon reflexes may be normal in unaffected limbs:
  - Predominantly distal (distal acquired demyelinating symmetric neuropathy) or
  - Asymmetric (multifocal acquired demyelinating sensory and motor neuropathy) o
  - Focal
  - Pure motor
  - Pure sensory
- Clinical exclusion criteria:
  - Neuropathy by Borrelia burgdorferi infection (Lyme disease), diphtheria, drug or toxin exposure
  - Hereditary demyelinating neuropathy
  - Prominent sphincter disturbance
  - Diagnosis of multifocal motor neuropathy
  - gM monoclonal gammopathy with high titer antibodies to myelin-associated glycoprotein
  - Other causes of demyelinating neuropathy include POEMS syndrome, a osteosclerotic myeloma, diabetic and non-diabetic lumbosacral radiculoplexus neuropathy, peripheral nervous system lymphoma and amyloidosis
- Electrodiagnostic criteria for CIDP include:
  - Parameters to identify motor and sensory conduction abnormalities that suggest demyelination .
- Supportive criteria for CIDP:
  - Elevated CSF protein with leukocyte count < 10/mm3 (albuminocytological dissociation)
  - MRI showing gadolinium enhancement and/or hypertrophy of the cauda equina, lumbosacral or cervical nerve roots, or the brachial or lumbosacral plexuses .
  - Abnormal sensory electrophysiology in at least one nerve
  - Objective clinical improvement following immunomodulatory treatment .
  - Nerve biopsy showing unequivocal evidence of demyelination and/or remyelination by electron microscopy or teased fiber analysis .

Case Definitions

GBS

Brighton Collaboration Criteria for GBS

Level 3
- Acute onset of bilateral, symmetric weakness of the limbs
- Decreased/absent deep tendon reflexes in affected limbs

Level 2
- Monophasic illness pattern with maximal weakness 12 hours - 28 days, followed by clinical plateau
- CSF total white count < 50 cells/mm^3 (elevated protein, absence of pleocytosis)

Level 1
- Electrophysiologic findings consistent with GBS

Level 4 – Lacking documentation to fulfill case definition, "reported case of GBS"

Level 5 – Not a case

Seivar J, et al. Guillain-Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data. Vaccine. 2011;29:599-612.

CIDP

- Clinical inclusion criteria for typical CIDP require both of the following:
  - Chronically progressive, stepwise, or recurrent symmetric proximal and distal weakness and sensory dysfunction of at least two limbs, developing over two months or longer; cranial nerves may be affected .
  - Absent or reduced tendon reflexes in all extremities
- Clinical inclusion criteria for CIDP variants require one of the following, but otherwise as in typical CIDP. However, tendon reflexes may be normal in unaffected limbs:
  - Predominantly distal (distal acquired demyelinating symmetric neuropathy) or
  - Asymmetric (multifocal acquired demyelinating sensory and motor neuropathy) o
  - Focal
  - Pure motor
  - Pure sensory
- Clinical exclusion criteria:
  - Neuropathy by Borrelia burgdorferi infection (Lyme disease), diphtheria, drug or toxin exposure
  - Hereditary demyelinating neuropathy
  - Prominent sphincter disturbance
  - Diagnosis of multifocal motor neuropathy
  - gM monoclonal gammopathy with high titer antibodies to myelin-associated glycoprotein
  - Other causes of demyelinating neuropathy include POEMS syndrome, a osteosclerotic myeloma, diabetic and non-diabetic lumbosacral radiculoplexus neuropathy, peripheral nervous system lymphoma and amyloidosis
- Electrodiagnostic criteria for CIDP include:
  - Parameters to identify motor and sensory conduction abnormalities that suggest demyelination .
- Supportive criteria for CIDP:
  - Elevated CSF protein with leukocyte count < 10/mm3 (albuminocytological dissociation)
  - MRI showing gadolinium enhancement and/or hypertrophy of the cauda equina, lumbosacral or cervical nerve roots, or the brachial or lumbosacral plexuses .
  - Abnormal sensory electrophysiology in at least one nerve
  - Objective clinical improvement following immunomodulatory treatment .
  - Nerve biopsy showing unequivocal evidence of demyelination and/or remyelination by electron microscopy or teased fiber analysis .

Brighton Collaboration Criteria for GBS

- Acute onset of bilateral, symmetric weakness of the limbs
- Decreased/absent deep tendon reflexes in affected limbs
- Monophasic illness pattern with maximal weakness 12 hours - 28 days, followed by clinical plateau
- CSF total white count < 50 cells/mm^3 (elevated protein, absence of pleocytosis)
- Electrophysiologic findings consistent with GBS

Level 4 – Lacking documentation to fulfill case definition, "reported case of GBS"

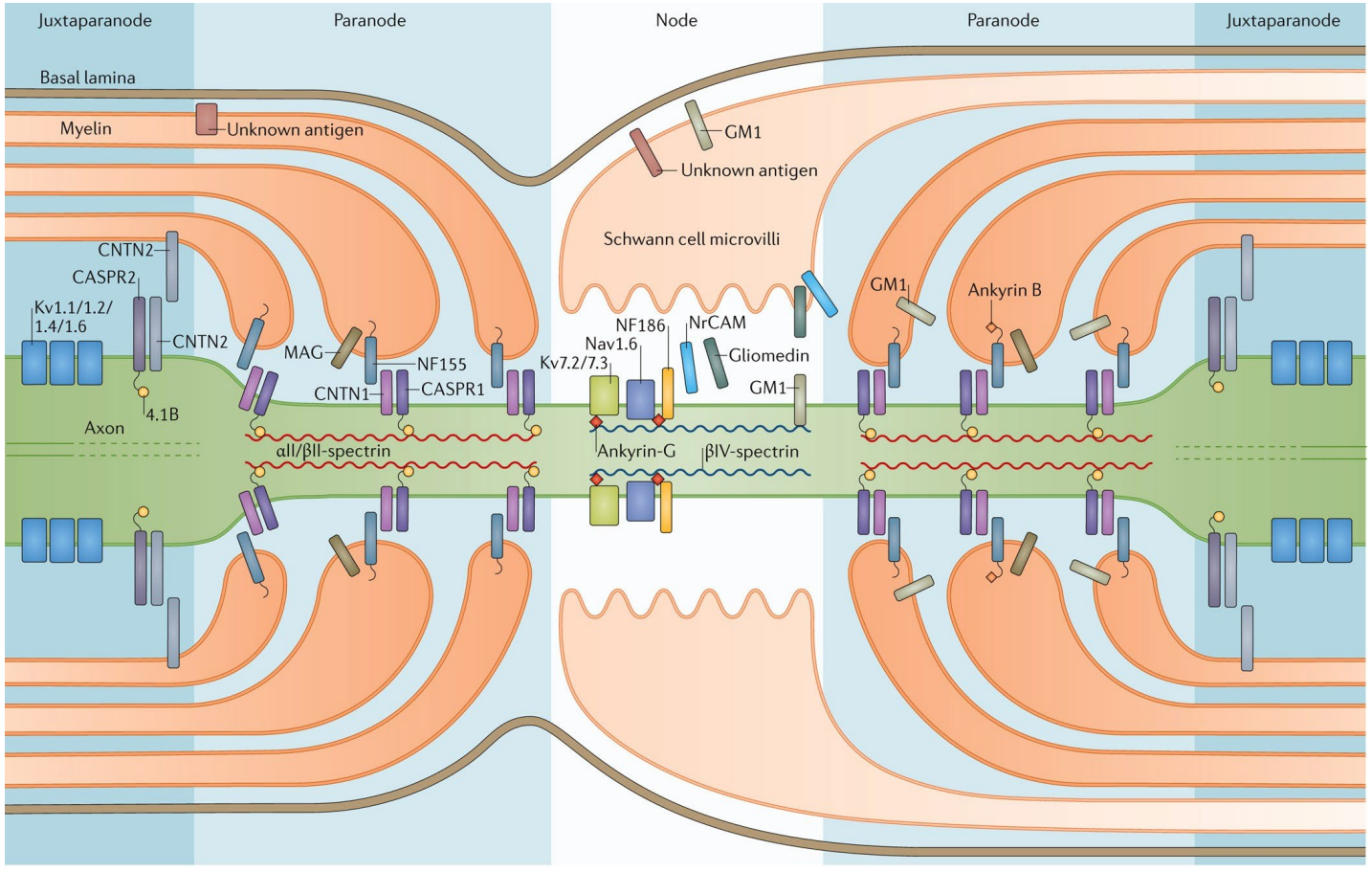
Level 5 – Not a case

Sejvar J, et al. Guillain-Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data. Vaccine. 2011;29:599-612.

CIDP Pathogenesis

- CIDP is an immune-mediated, demyelinating disease
  - Immune-mediated: body's immune system attacks itself, in this case, peripheral nerves
  - Demyelinating: Attack on the 'insulation' of the peripheral nerve, interrupting the flow of electrical information in the body
- Principal antibodies involved in CIDP include antibodies against Neurofascin (NF155, NF186), Contactin-1 (CNTN-1)
  - Antibodies involved in 2- 18% of CIDP
- Antibodies disrupt and destroy nodal structures on nerve

CIPD Pathogenesis

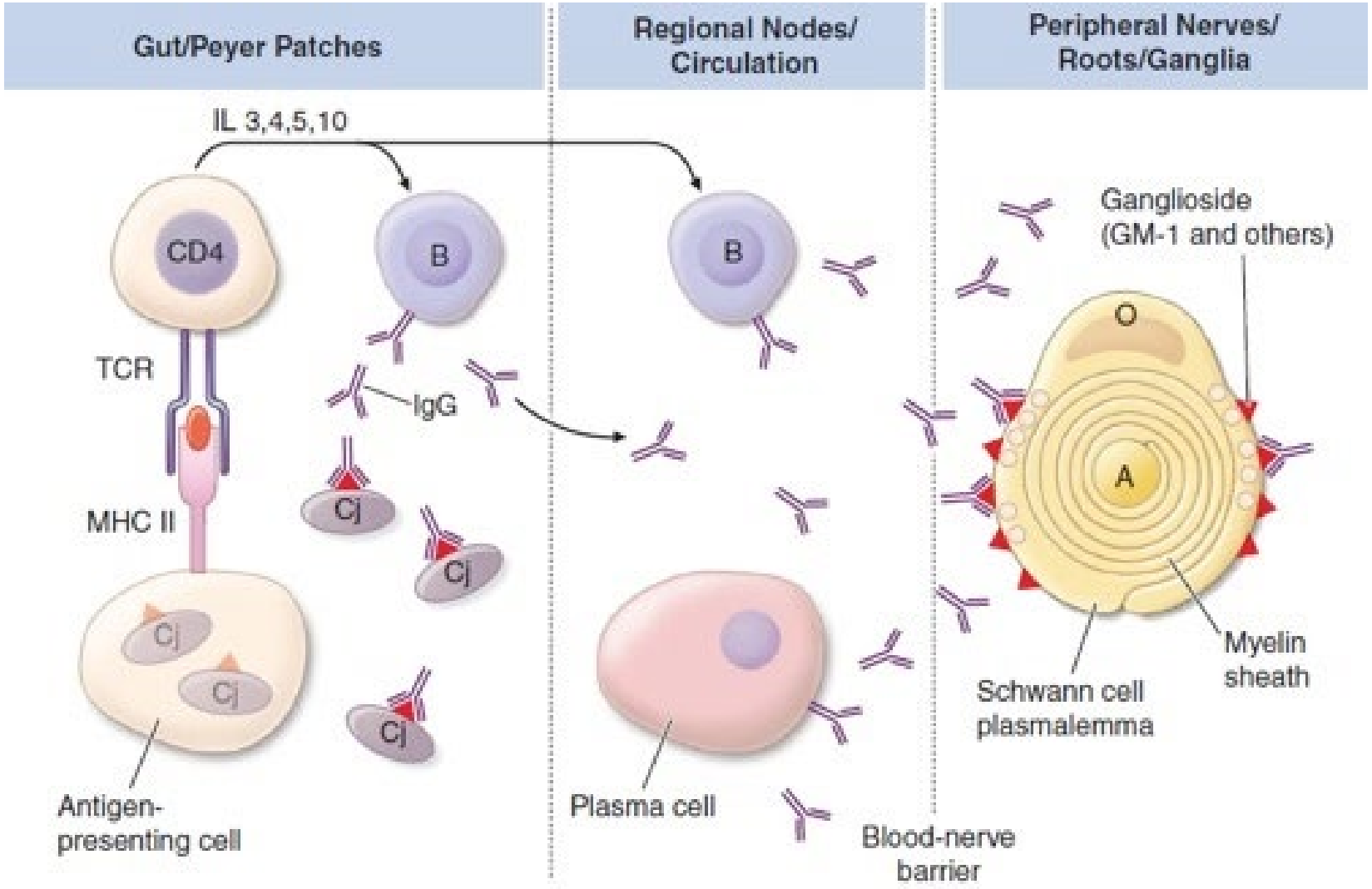


Nature Reviews | Neurology

GBS Pathogenesis

- Predominantly a post-infectious autoimmune inflammatory disease
- Antecedent antigenic stimulus (infection, vaccine, rarely other stimuli) result in cross-reactive antibodies, leading to myelin / axonal damage
- Autoantibodies and/or autoreactive T cells mediate cellular damage
- Frequently associated with anti-ganglioside antibodies
  - Anti-GM1
  - Anti-GD1b
  - Others…
- *Campylobacter jejuni* – has demonstrated ‘molecular mimicry’ with anti-ganglioside antibodies

GBS Pathogenesis



Source: Eric Shamus: *The Color Atlas of Physical Therapy*: www.accessphysiotherapy.com

Acute Onset CIDP (A-CIDP)

- “Rapid”, acute onset
- Particularly difficult to distinguish from GBS in the early phases
- About 16% of cases of CIDP
- Several features help to distinguish A-CIDP from AIDP:
  - Slightly older age
  - More frequently with concurrent diabetes
  - Greater incidence of proprioceptive abnormalities
- That being said, the only thing that is reliably going to differentiate A-CIDP from AIDP is time
  - A-CIDP – by definition, evolves over 8 weeks or more
  - AIDP – reaches nadir by 4 weeks, then plateaus or improves

Age & Sex

GBS	CIDP
• The incidence increases with age and reaches its peak between 50 and 70 years • Men are about 1.5 times more likely to be affected than are women.	• Typical CIDP can occur at any age, but most commonly between 40 and 60 year • Predominantly affects males more than females, with a ratio of 2:1

Incidence & Prevalence

GBS	CIDP
• Worldwide incidence rate of 1–2 cases per 100,000 people per year	• Incidence of 0.2 – 1.6 / 100,000 population per year • Prevalence of 0.8–8.9 per 100,000, • Depends heavily on diagnostic criteria used

Geographic Distribution

GBS

High-income Asia Pacific [1.9 (95% UI: 1.5 to 2.4)] and East Asia [0.8 (95% UI: 0.6 to 1.0)] had the highest and lowest age-standardized prevalence rates (per 100,000), respectively, in 2019.

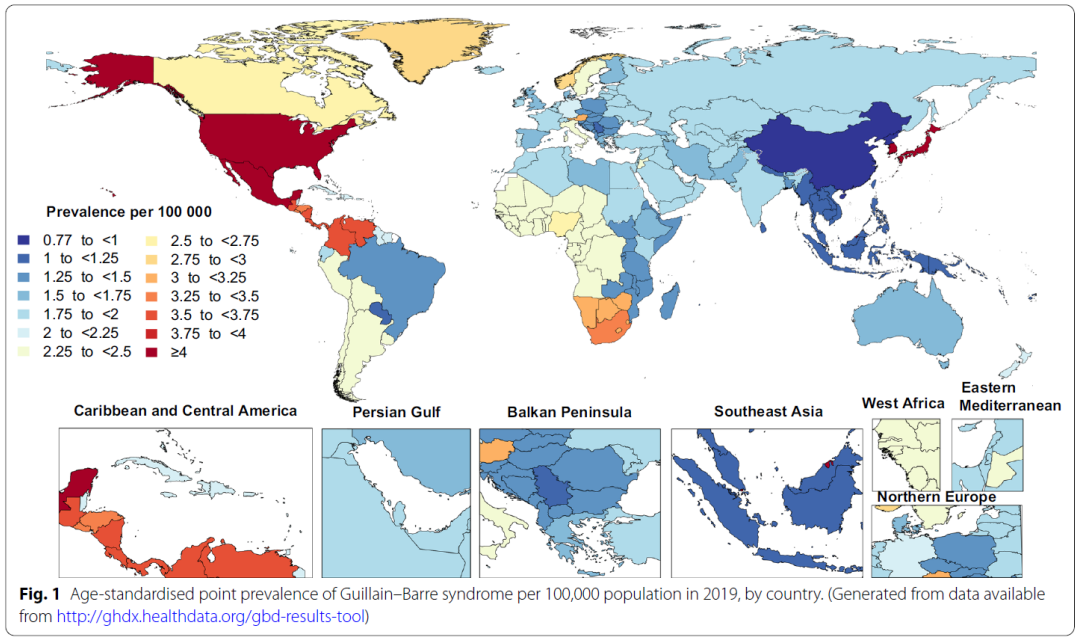


Fig. 1 Age-standardised point prevalence of Guillain–Barre syndrome per 100,000 population in 2019, by country. (Generated from data available from http://ghdx.healthdata.org/gbd-results-tool)

CIDP

Study region	Diagnostic criteria	Population (year of prevalence)	Prevalence per 100.000 population	Male prevalence per 100. 000 population	Female prevalence per 100.000 population	Incidence per 100 000 population	Mean age at onset (years)
Tottori, Japan [2	AAN	614 725 (1992)	0.8 (0.3–1.9)	1.4 (0.4–3.6)	0.3 (0.01–1.7)	Not reported	Not reported
Southeast England [3]	AAN	3 717 638 (1995)	1.2 (0.9–1.7)	Not reported	Not reported	Not reported	54.4
New South Wales, Australia [4]	AAN	5 999 544 (1996)	1.9 (1.5–2.2)	2.2 (1.7–2.8)	1.6 (1.2–2.1)	0.2 (CI not given)	53.5
Vest-Agder, Norway [5]	Albers and Kelly	155 464 (1999)	7.7 (3.2–12.2)	14.7 (7.3–26.4)	5.0 (1.4–12.8)	Not reported	48
Olmsted county, USA [8]	Dyck & Mayo EMG Lab	Not available (2000)	8.9 (CI not given)	Not available	Not available	1.6 (CI not given)	Not available
Piemonte, Italy [1]	AAN	4 334 225 (2001)	3.6 (3.0–4.2)	5.0 (4.1–6.1)	2.2 (1.7–2.9)	0.4 (0.2–0.5)	59.6
Japan [6]	AAN INCAT	127 655 000 (2005)	1.6 (CI not given)	2.0 (CI not given)	1.2 (CI not given)	0.6 (CI not given)	Not reported
Leicestershire / Rutland, UK [7]	EFNS/ PNS AAN	963 600 (2008)	4.8 (3.5–6.3) 2.0 (1.2–3.1)	6.7 (4.6–9.5) 2.9 (1.6–4.9)	2.9 (1.6–4.8) 1.0 (0.3–2.4)	0.7 (0.4 –1.1) 0.4 (0.2–0.6)	52.9
Mahdi-Rodgers (This study)	EFNS/PNS	3 557 352 (2008)	2.8 (2.3–3.5)	3.8 (3.0–4.9)	1.9 (1.3–2.7)	-	57.7

Geographic Distribution

GBS

High-income Asia Pacific [1.9 (95% UI: 1.5 to 2.4)] and East Asia [0.8 (95% UI: 0.6 to 1.0)] had the highest and lowest age-standardized prevalence rates (per 100,000), respectively, in 2019.

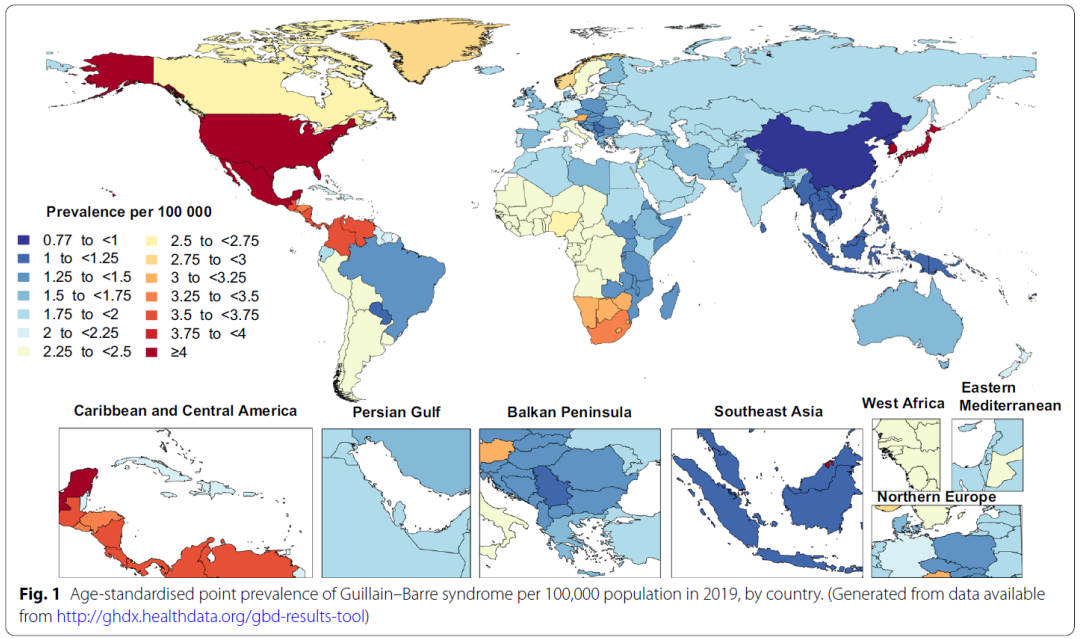


Fig. 1 Age-standardised point prevalence of Guillain–Barre syndrome per 100,000 population in 2019, by country. (Generated from data available from http://ghdx.healthdata.org/gbd-results-tool)

CIDP

Study region	Diagnostic criteria	Population (year of prevalence)	Prevalence per 100.000 population	Male prevalence per 100. 000 population	Female prevalence per 100.000 population	Incidence per 100 000 population	Mean age at onset (years)
Tottori, Japan [2	AAN	614 725 (1992)	0.8 (0.3–1.9)	1.4 (0.4–3.6)	0.3 (0.01–1.7)	Not reported	Not reported
Southeast England [3]	AAN	3 717 638 (1995)	1.2 (0.9–1.7)	Not reported	Not reported	Not reported	54.4
New South Wales, Australia [4]	AAN	5 999 544 (1996)	1.9 (1.5–2.2)	2.2 (1.7–2.8)	1.6 (1.2–2.1)	0.2 (CI not given)	53.5
Vest-Agder, Norway [5]	Albers and Kelly	155 464 (1999)	7.7 (3.2–12.2)	14.7 (7.3–26.4)	5.0 (1.4–12.8)	Not reported	48
Olmsted county, USA [8]	Dyck & Mayo EMG Lab	Not available (2000)	8.9 (CI not given)	Not available	Not available	1.6 (CI not given)	Not available
Piemonte, Italy [1]	AAN	4 334 225 (2001)	3.6 (3.0–4.2)	5.0 (4.1–6.1)	2.2 (1.7–2.9)	0.4 (0.2–0.5)	59.6
Japan [6]	AAN INCAT	127 655 000 (2005)	1.6 (CI not given)	2.0 (CI not given)	1.2 (CI not given)	0.6 (CI not given)	Not reported
Leicestershire / Rutland, UK [7]	EFNS/ PNS AAN	963 600 (2008)	4.8 (3.5–6.3) 2.0 (1.2–3.1)	6.7 (4.6–9.5) 2.9 (1.6–4.9)	2.9 (1.6–4.8) 1.0 (0.3–2.4)	0.7 (0.4 –1.1) 0.4 (0.2–0.6)	52.9
Mahdi-Rodgers (This study)	EFNS/PNS	3 557 352 (2008)	2.3 (2.3–3.5)	3.8 (3.0–4.9)	1.9 (1.3–2.7)	-	57.7

Seasonality

GBS	CIDP
• Most studies have suggested a seasonality to GBS, with some showing a winter peak, and others showing a peak in late summer and autumn	• No data

Risk factors: Pre-existing/concurrent illnesses

GBS	CIDP
• Some studies have suggested that persons with underlying immunosuppression status are at higher risk of GBS, but this is inconsistent • Several surgeries, including GI and orthopedic surgeries, have been suggested as being risk factors for GBS	• CIDP has been found in some studies to be more frequent in people with diabetes mellitus; however, diabetes as a risk factor for CIDP has not been established.

Risk factors: Vaccines

GBS	CIDP
• Strong association between 1976 formulation of A/H1N1 influenza vaccine and subsequent GBS in the 6 weeks following vaccination – 1 excess case of GBS per 100,000 vaccinations • Same risk has not been demonstrated for subsequent formulations of influenza vaccine, despite intensive investigation • IOM has not found a causal association between GBS and any other single vaccine (DTAP, MMR, shingles, HBV, meningococcal) • That said, some patients do report receipt of a vaccine in the 6 weeks prior to onset of their GBS illness	• Based upon 2 studies, approximately 1% of CIDP patients report receipt of vaccine in the month prior to onset of illness

Risk factors: Infections

GBS	CIDP

• Strong (causal) association between infection with *Campylobacter jejuni* and AMAN, due to molecular mimicry
  • 8-12% of GBS cases in USA attributable to *Campylobacter*
• Other infectious illnesses have been associated with GBS in a temporal fashion
  • VZV, influenza, Hepatitis E, CMV, Mycoplasma, Salmonella
• 70% of GBS patients report antecedent respiratory or gastrointestinal illness in 6 weeks prior to onset
	• Antecedent infectious illness reported in 10% of cases of CIDP
	
	• Not the same association with *Campylobacter*

Antecedent Vaccines / Infections and CIDP / GBS

- There appear to be differences in the rate and in the strength of evidence in antecedent vaccines and infectious illnesses preceding CIDP and GBS
- GBS: Evidence to suggest
  - Causal association between *Campylobacter jejuni* infection and AMAN (motor variant of GBS)
  - Temporal association between 1976 influenza A/H1N1 vaccine and GBS (variants not specified)
  - Loose temporal association between GBS and several other vaccines and infections (shingles vaccine, some influenza vaccines; Mycoplasma and hepatitis E infections)
  - Overall, antecedent infection or vaccination reported in 70% of GBS cases
- CIDP: The same data are not present
  - Overall, about 9% of CIDP cases report antecedent infection, and 1% report antecedent vaccination

Progression

GBS	CIDP

• Presents acutely; evolution over hours - days
• Symptoms and signs typically progress within 1 to 2 weeks
• Nadir reached within 4 weeks in 90% to 97% of patients
• Plateau phase is variable with duration lasting days to weeks; in some cases, it can last months
• Gradual improvement
	• Develops over several weeks, months, or years; chronic autoimmune neuropathy
	• By definition, it progresses over 8 weeks or greater
	• 3 temporal patterns to illness
	  • Progressive
	  • Stepwise
	  • Relapsing
	• May have periods of stabilization

Electrodiagnostic (EDx)

GBS	CIDP

• Goals of EDx:
  • To diagnosis, subtype, and predict outcome of GBS.
  • To demonstrate features supportive of demyelination
• Useful in the differential diagnosis of disorders, which may mimic GBS.
• 60–70% of GBS patients have abnormal sensory nerve conduction studies.
• The finding of multiple A-waves, the electrophysiological sural sparing next to ultrasonic sensory sparing pattern, and swelling of the roots and vagus nerve are diagnostic hallmarks
• May indicate normal measures in the early stages of the disease (within 1 week of symptom onset).
  • Repeated study 2–3 weeks later provides valuable insight.
• After 6 months, nerve enlargement disappears or reduces markedly
	• Goal of Edx:
	  • Confirm diagnostic
	  • Characterize peripheral nerve demyelination
	• Electrophysiological criteria:
	  • Definite diagnosis: at least 2 demyelinating features in 2 different nerves.
	  • Probable diagnosis: 2 probable blocks or a probable block and 1 other demyelinating feature in a different nerve
	  • Possible diagnosis: 1 demyelinating feature in 1 nerve.
	• Enlarged sensory nerves and/or multifocally enlarged peripheral nerves and heightened echointensity are distinctive marker of CIDP
	• After 6 months, nerve enlargement remains

Cerebrospinal Fluid (CSF) Analysis

GBS	CIDP

• CSF protein elevation with normal blood cell count (albuminocytologic dissociation)
  • Approximately 80% of AIDP patients have increased protein levels and elevated CSF/serum albumin ratio by second week of illness
• May not be abnormal in early stages of disease
  • Protein levels normal in 30-50% of patients in first week after disease onset and 10-30% in second week
• Limitations:
  • Not specific to GBS
  • May be elevated in other disease processes (DM, CMT)
	• CSF protein elevation with normal blood cell count (albuminocytologic dissociation)
	• Found in about 90% of patients with all variants of CIDP
	• Limitation:
	  • Not specific to CIDP
	  • Elevated CSF protein also found in DM and CMT patients and may result in misdiagnosis
	  • CSF protein can increase with age and may result in underdiagnosis

Outcomes

GBS	CIDP
• The disease can be severe • Reported mortality ranges from 1.9% to 5% • 30% require intubation at some point during illness • About 20% of patients with Guillain-Barré syndrome cannot walk unaided 6 months after onset.	• Mortality rate ranges between 1% to 11% • Loss of ambulation in cases ranges from 2% to 14% • Considerable ongoing, long-term morbidity due to chronic nature of disease

Treatments

GBS	CIDP

• No known cure, but treatment can alleviate symptoms and reduce duration.
• The most common treatment options include:
  • Intravenous immunoglobulins (IVIG)
  • Plasma exchange (plasmapheresis)
• Both treatments are equally effective and have similar outcomes
• Steroids are not helpful in GBS
• Supportive care (pain management, physical therapy, etc.)
	• CIDP is a chronic condition, but it is treatable and early treatment prevents nerve damage.
	• Treatment options include:
	  • Corticosteroids
	  • IVIG
	  • Plasma exchange
	  • Immunotherapy (rituximab, cyclophosphamide)
	  • Steroid Sparing Agents for Maintenance Therapy
	• Early diagnosis and treatment can prevent axonal damage and permanent disability

Conclusions

- Both CIDP and GBS are immune mediated peripheral neuropathies, resulting in limb weakness, sensory deficits, and decreased or absent reflexes
- They share many similarities in terms of clinical presentation and pathophysiology
- CIDP and GBS have important differences
  - Clinical course and progression
  - Treatment
  - Associated morbidity and mortality
  - Antecedent antigenic stimuli
- While GBS has a strong association with preceding infections and some vaccinations, CIDP does not appear to have the same relationship

Reference

- Al-Zuhairy A, Jakobsen J. Outcome in chronic inflammatory demyelinating polyneuropathy: A systematic review and meta-analysis. Muscle Nerve. 2023 Oct;68(4):388-396. doi: 10.1002/mus.27820. Epub 2023 Apr 11. PMID: 36928889.
- Arends S, Drenthen J, Van den Bergh PYK, Hadden RDM, Shahrizaila N, Dimachkie MM, Gutiérrez Gutiérrez G, Katzberg H, Kiers L, Lehmann HC, Péréon Y, Reisin RC, Uncini A, Verhamme C, Waheed W, Cornblath DR, Jacobs BC; IGOS Electrophysiology Expertise Group. Electrodiagnostic subtyping in Guillain-Barré syndrome: Use of criteria in practice based on a survey study in IGOS. J Peripher Nerv Syst. 2022 Sep;27(3):197-205. doi: 10.1111/jns.12504. Epub 2022 Jun 24. PMID: 35700346; PMCID: PMC9542579.
- Fokke C, van den Berg B, Drenthen J, Walgaard C, van Doorn PA, Jacobs BC. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. Brain. 2014 Jan;137(Pt 1):33-43. doi: 10.1093/brain/awt285. Epub 2013 Oct 26. PMID: 24163275.
- Gogia B, Rocha Cabrero F, Khan Suheb MZ, et al. Chronic Inflammatory Demyelinating Polyradiculoneuropathy. [Updated 2023 Jun 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK563249/
- Illes Z, Blaabjerg M. Cerebrospinal fluid findings in Guillain-Barré syndrome and chronic inflammatory demyelinating polyneuropathies. Handb Clin Neurol. 2017;146:125-138. doi: 10.1016/B978-0-12-804279-3.00009-5. PMID: 29110767.
- "NORD. (2021). Chronic Inflammatory Demyelinating Polyneuropathy. Prevots, D. R., & Sutter, R. W. (1997). Assessment of Guillain-Barre syndrome mortality and morbidity in the United States: implications for acute flaccid paralysis surveillance. J Infect Dis, 175 Suppl 1, S151-155. https://doi.org/10.1093/infdis/175.supplement_1.s151“
- Teener, J. W., & Albers, J. W. (2007). Acquired inflammatory demyelinating and axonal neuropathies. Neurobiology of Disease, 859–869.
- Uncini A, Kuwabara S. Electrodiagnostic criteria for Guillain-Barrè syndrome: a critical revision and the need for an update. Clin Neurophysiol. 2012 Aug;123(8):1487-95. doi: 10.1016/j.clinph.2012.01.025. Epub 2012 Apr 4. PMID: 22480600.
- World Health Organization. (n.d.). *Guillain–barré syndrome*. World Health Organization. https://www.who.int/news-room/fact-sheets/detail/guillain-barr%C3%A9-syndrome#:~:text=There%20is%20no%20known%20cure,the%20blood%20or%20intravenous%20immunoglobulin.

Questions?