GB Syndrome

Dr. Jasodhara Chaudhuri
Assistant Professor, Dept. of Neuromedicine
NRS MCH, Kolkata

Panelists

- Prof Dr Swapan Ray
- Dr Arijit Chatterjee
- Prof BK Ray
- Prof Dr Subhasis Bhattacharya

GB Syndrome (Guillain-Barré syndrome)

- Acute inflammatory polyneuropathy.
- Most common cause of acute/subacute generalized paralysis in clinical practice.
- Rapidly progressive, symmetric, ascending weakness (in majority) with areflexia.
- Usually preceded by a mild RTI, GI infection, or immunization 1-3 weeks before the neurologic symptoms.

Infections Preceding GBS

Preceding Infections

✓ 90%+ of GBS cases follow a respiratory or gastrointestinal infection

Campylobacter jejuni (C. jejuni)

✓ Most common bacterial trigger of GBS

✓ Up to 30% of GBS cases linked to C. jejuni

GBS Risk After C. jejuni

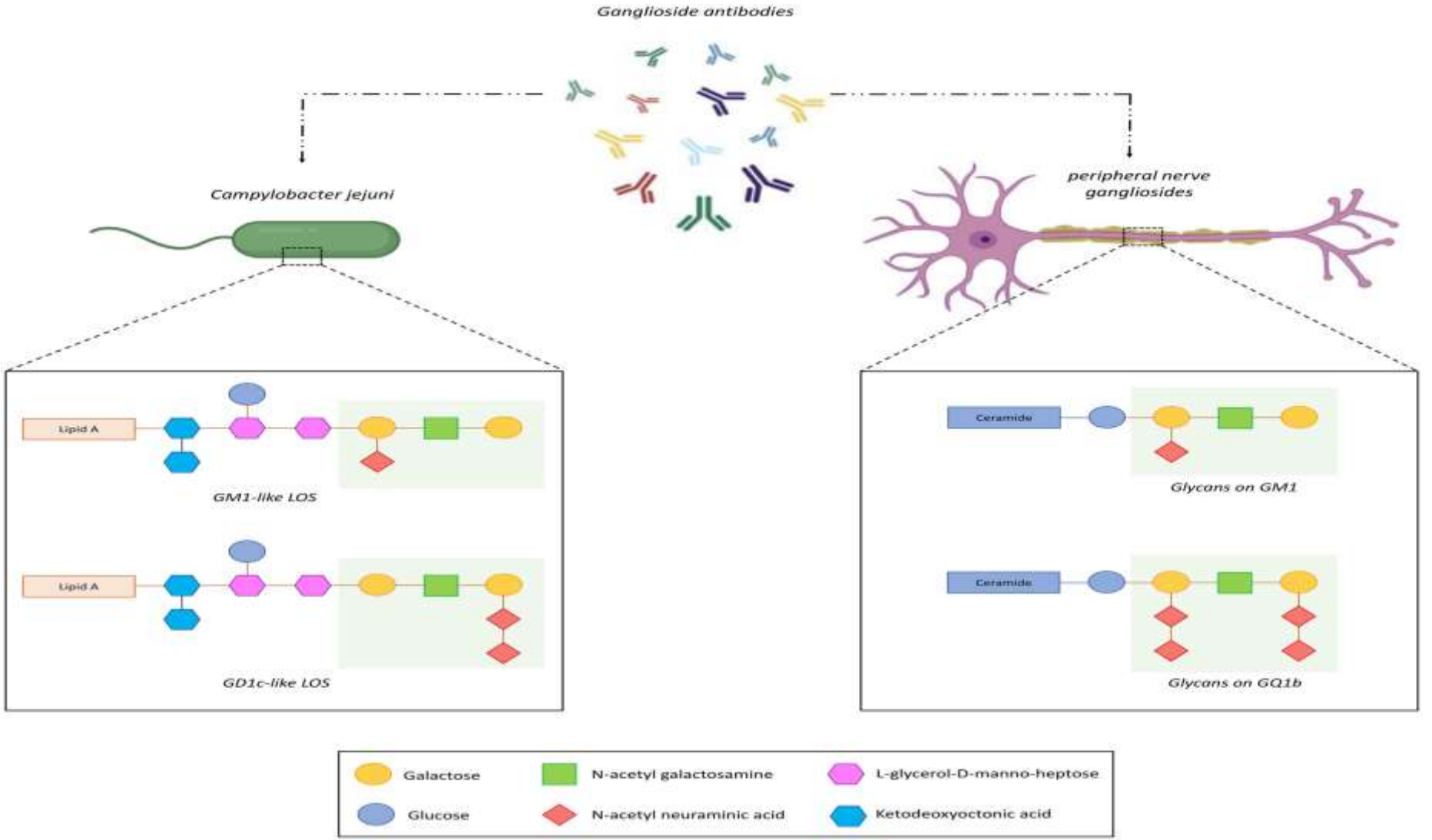
✓ 1 in 1000 infected individuals develop GBS

Symptom Onset

✓ Median: 10 days post-diarrhea (Range: 3 days – 6 weeks)

SR

Guillain-Barré syndrome and campylobacter jejuni



Lipo-oligosaccharide (LOS) on the outer membrane of Campylobacter jejuni induces cross-reactive antibodies which, through molecular mimicry, bind to the structurally identical glycans (areas in green) present on peripheral nerve gangliosides (GM1 and GQ1b in the example above), resulting in damage to axons and Schwann cells

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Other Infections Linked to GBS

- **Bacterial & Viral Triggers**
  - ✓ **Bacteria:** *Haemophilus influenzae, Mycoplasma pneumoniae*
  - ✓ **Viruses:** *Cytomegalovirus, Epstein–Barr virus, Varicella zoster virus*

🔍 **Asymptomatic Infections**

- ✓ Some GBS patients **show no prior symptoms**
- ✓ **50% of C. jejuni infections** are **asymptomatic**

📌 **Key Takeaway:**

- ✓ GBS may follow **various infections**, even without visible symptoms

Non-Infective Associations with GBS

💉 **Medical Triggers**

- ✓ **Parenteral gangliosides** (used for peripheral neuropathy)
- ✓ **Vaccines** → *Potential molecular mimicry*

Other Associations

- ✓ **Autoimmune diseases** (e.g., lupus)
- ✓ **Immunosuppressive drugs** (e.g., anti-TNFα therapy)
- ✓ **Surgery** → *May increase susceptibility to infections*

- SB

Vaccines & GBS

💉 **Possible Links**

- ✓ **H1N1 & seasonal flu vaccines** → Slight risk (~1 case per million)
- ✓ **Adenovirus-based COVID-19 vaccines** → Rare association

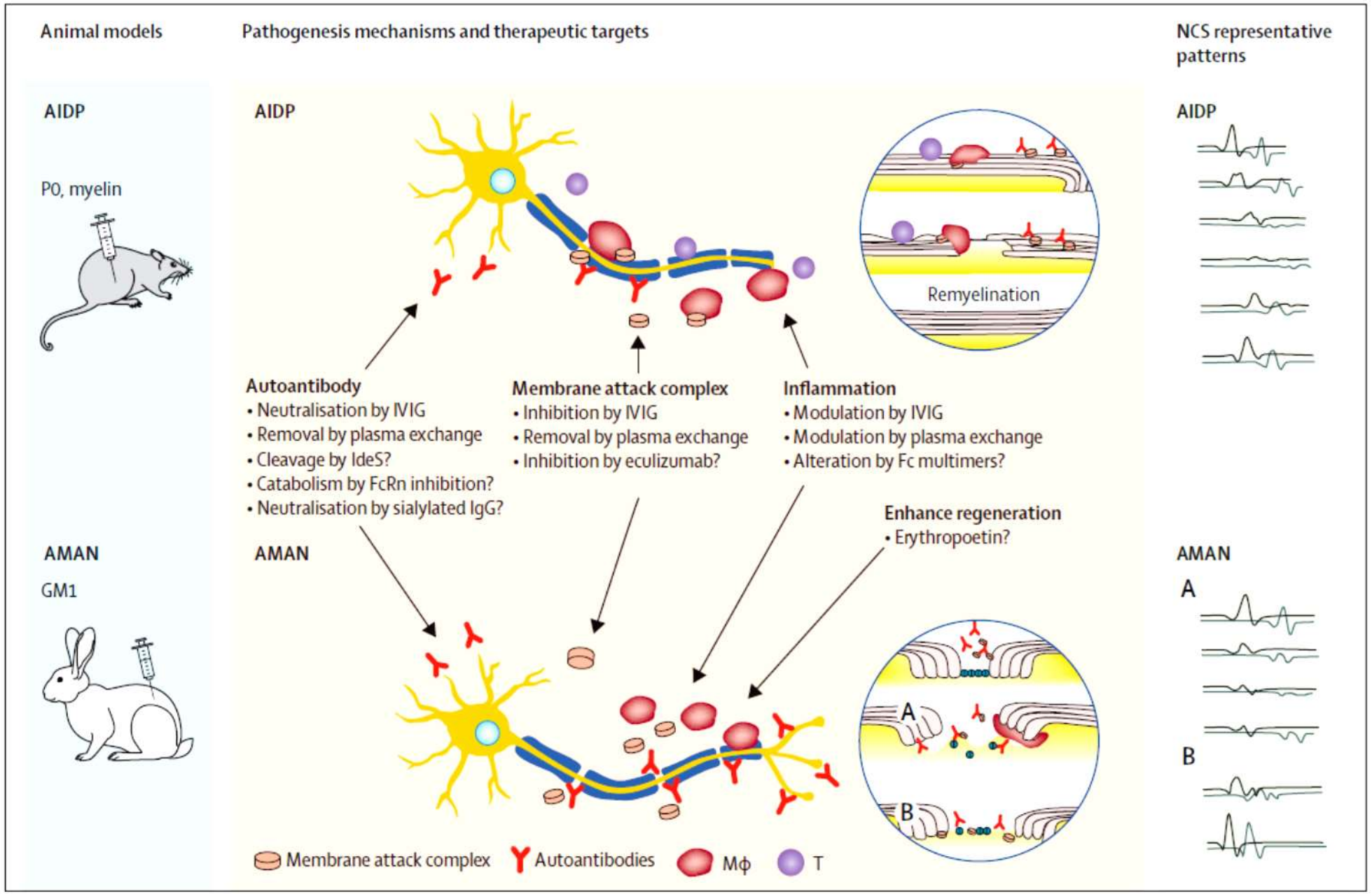
🛡 **Low or No Risk**

- ✓ **mRNA COVID-19, Hepatitis B, MMR, HPV, tetanus** → No strong evidence

📌 **Key Takeaway:**

- ✓ Vaccine-related GBS is **extremely rare**, and infections pose a **higher risk.**

Pathophysiology in GBS



💪 Key GBS Features

- ✓ **Symmetric limb weakness** (sometimes asymmetric/unilateral)
- ✓ **Classic GBS:** Bilateral limb weakness, often with bi**facial weakness**
- ✓ **Ascending weakness**, possible **respiratory & cranial nerve involvement**
- ✓ **10% may have normal/exaggerated reflexes**

◌◌ **MFS (**Miller Fisher syndrome) **Features**

- ✓ **Ophthalmoplegia, ataxia, areflexia** (no limb weakness)

  ✓ **Bickerstaff's Brainstem Encephalitis (BBE):** MFS + **hypersomnolence**

  ✓ **Incomplete MFS:** May lack ophthalmoplegia or ataxia

- AC

🔄 Subtypes & Overlaps

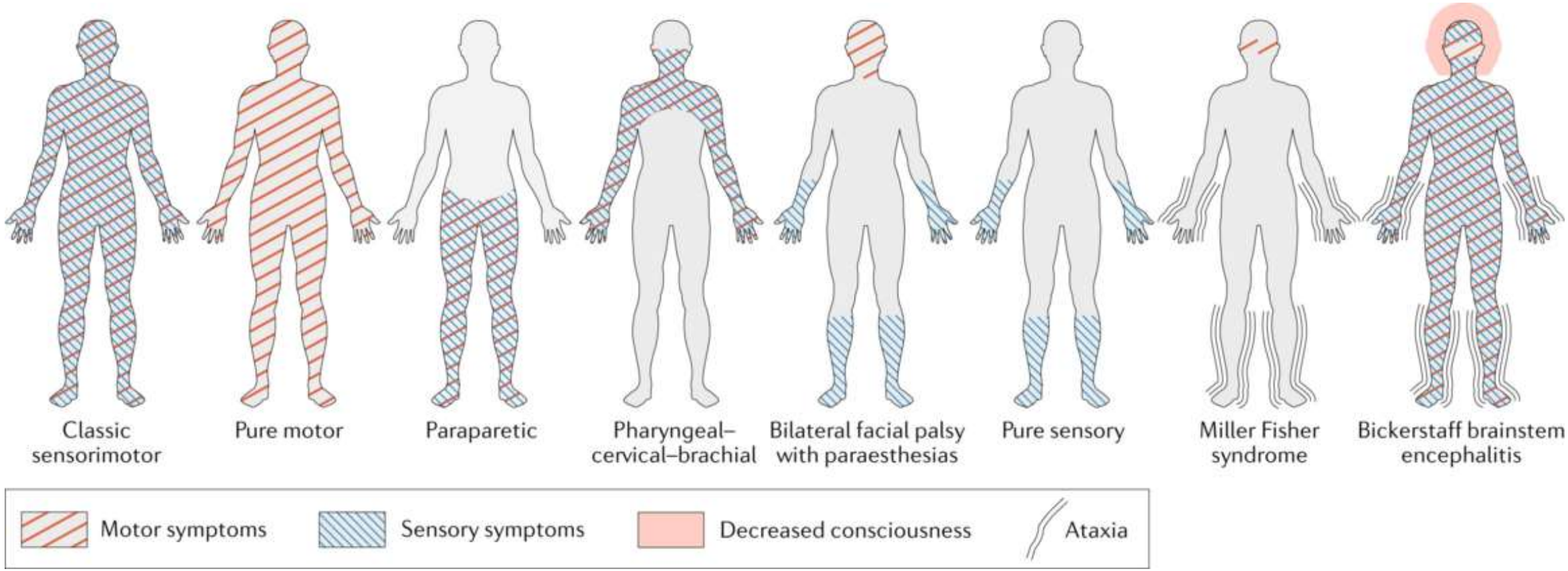
✓ **MFS/BBE + limb weakness** → Overlap with **GBS**

✓ **GBS with ophthalmoplegia/ataxia** → Overlap with **MFS**

✓ **Pharyngeal-cervical-brachial (PCB) weakness + ataxia** → Overlap with **GBS/MFS subtypes**

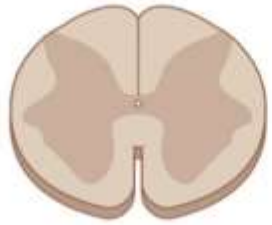
BKR

Types :


Classic sensorimotor
Pure motor
Paraparetic
Pharyngeal–cervical–brachial
Bilateral facial palsy with paraesthesias
Pure sensory
Miller Fisher syndrome
Bickerstaff brainstem encephalitis
Motor symptoms
Sensory symptoms
Decreased consciousness
Ataxia

Guillain-Barré syndrome: Differentials

BKR

Nerve / nerve root	Muscle	Neuromuscular junction	Spinal cord	Higher CNS diseases
				
Acute onset CIDP	Hypokalaemic Periodic Paralysis	Myasthenia Gravis	Acute transverse myelitis	Brainstem stroke
Autoimmune nodopathy	Acute viral myositis	LEMS	MOGAD-related conus medullaris syndrome	Rhombencephalitis
Vasculitic neuropathy	Acute colchicine myopathy	Botulism		Wernicke's encephalopathy
Haematological malignancy / carcinomatosis with nerve root infiltration		Organophosphate intoxication		
Acute infection / HIV seroconversion				
Acute intermittent porphyria				
Nutritional (thiamine deficiency)				
Heavy metals poisoning				

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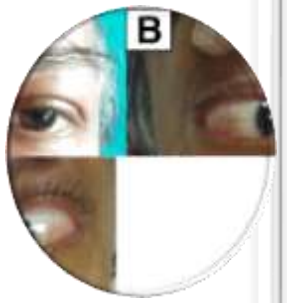
The 10 steps

Step 1 : When to Suspect? SR

Rapidly progressive bilateral limb weakness and /or sensory deficits.

Hyporeflexia /areflexia

Facial /cranial nerve palsy



Ophthalmoplegia

ataxia.

Dysautonomia

- acute to subacute onset

- maximum disability is within 2 weeks.

Alternate diagnosis : When maximum disability is within 24 hrs or after 4weeks of disease onset

,

2/3 patients have symptoms of infections in the preceding 6 weeks.

Atypical clinical presentation :

- Weakness and sensory signs could be asymmetrical or predominantly proximal or distal.
- Severe and diffuse pain or isolated cranial nerve dysfunction can precede the onset of weakness.
- In minority of patient particularly those with pure motor variant and an AMAN subtype might have normal or even exaggerated reflexes.

GBS variants and Clinical manifestations

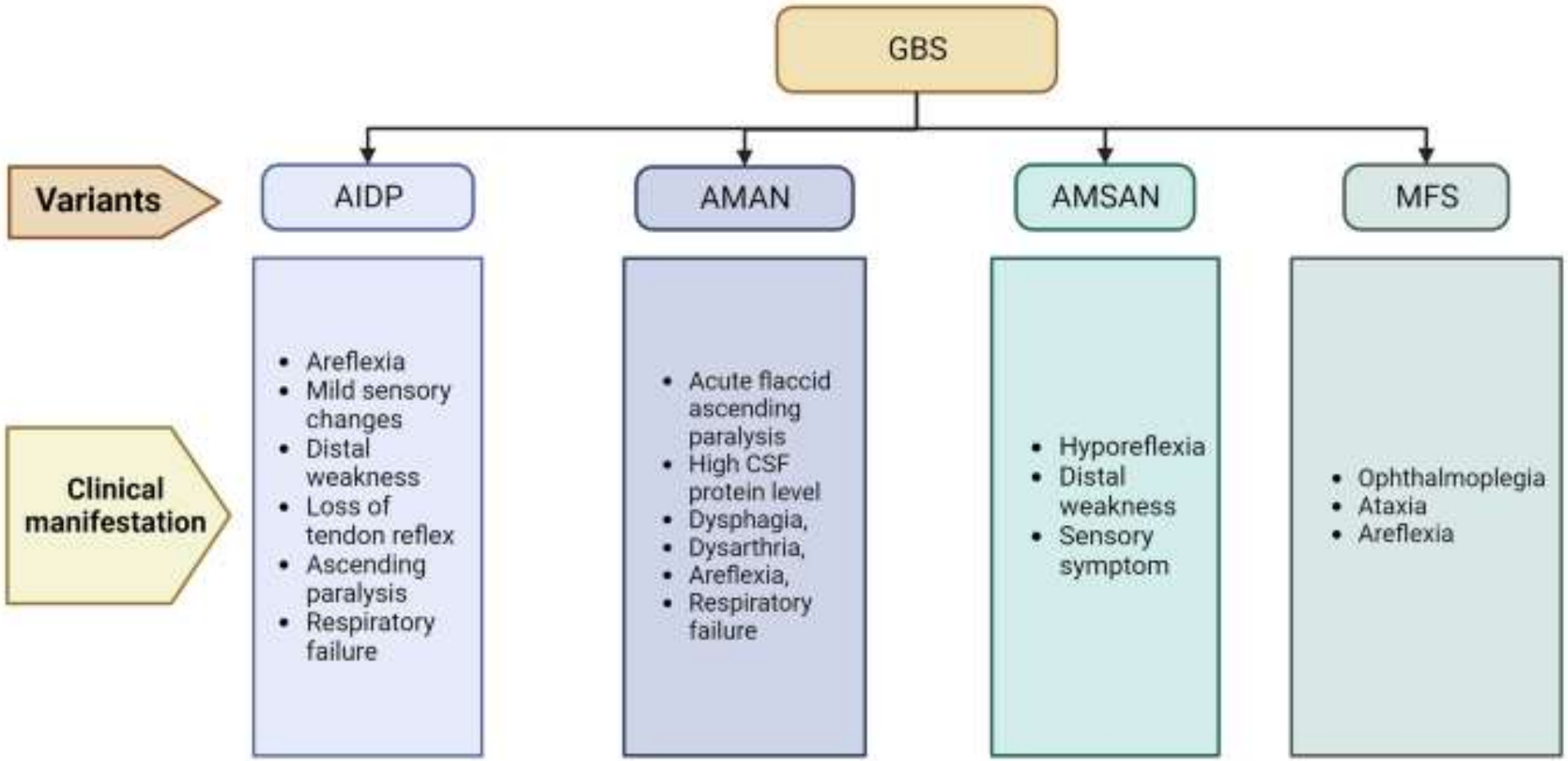


Table 1 | **Variants of Guillain–Barré syndrome**

Variant	Frequency (% of GBS cases)[a]	Clinical features
Classic sensorimotor GBS[b]	30–85	Rapidly progressive symmetrical weakness and sensory signs with absent or reduced tendon reflexes, usually reaching nadir within 2 weeks
Pure motor[c]	5–70	Motor weakness without sensory signs
Paraparetic	5–10	Paresis restricted to the legs
Pharyngeal–cervical–brachial	<5	Weakness of pharyngeal, cervical and brachial muscles without lower limb weakness
Bilateral facial palsy with paraesthesias[d]	<5	Bilateral facial weakness, paraesthesias and reduced reflexes
Pure sensory[d]	<1	Acute or subacute sensory neuropathy without other deficits
Miller Fisher syndrome	5–25	Ophthalmoplegia, ataxia and areflexia. Incomplete forms with isolated ataxia (acute ataxic neuropathy) or ophthalmoplegia (acute ophthalmoplegia) can occur[31]. Overlaps with classical sensorimotor GBS in an estimated 15% of patients
Bickerstaff brainstem encephalitis[d]	<5	Ophthalmoplegia, ataxia, areflexia, pyramidal tract signs and impaired consciousness, often overlapping with sensorimotor GBS

Step 2 : How to diagnose?

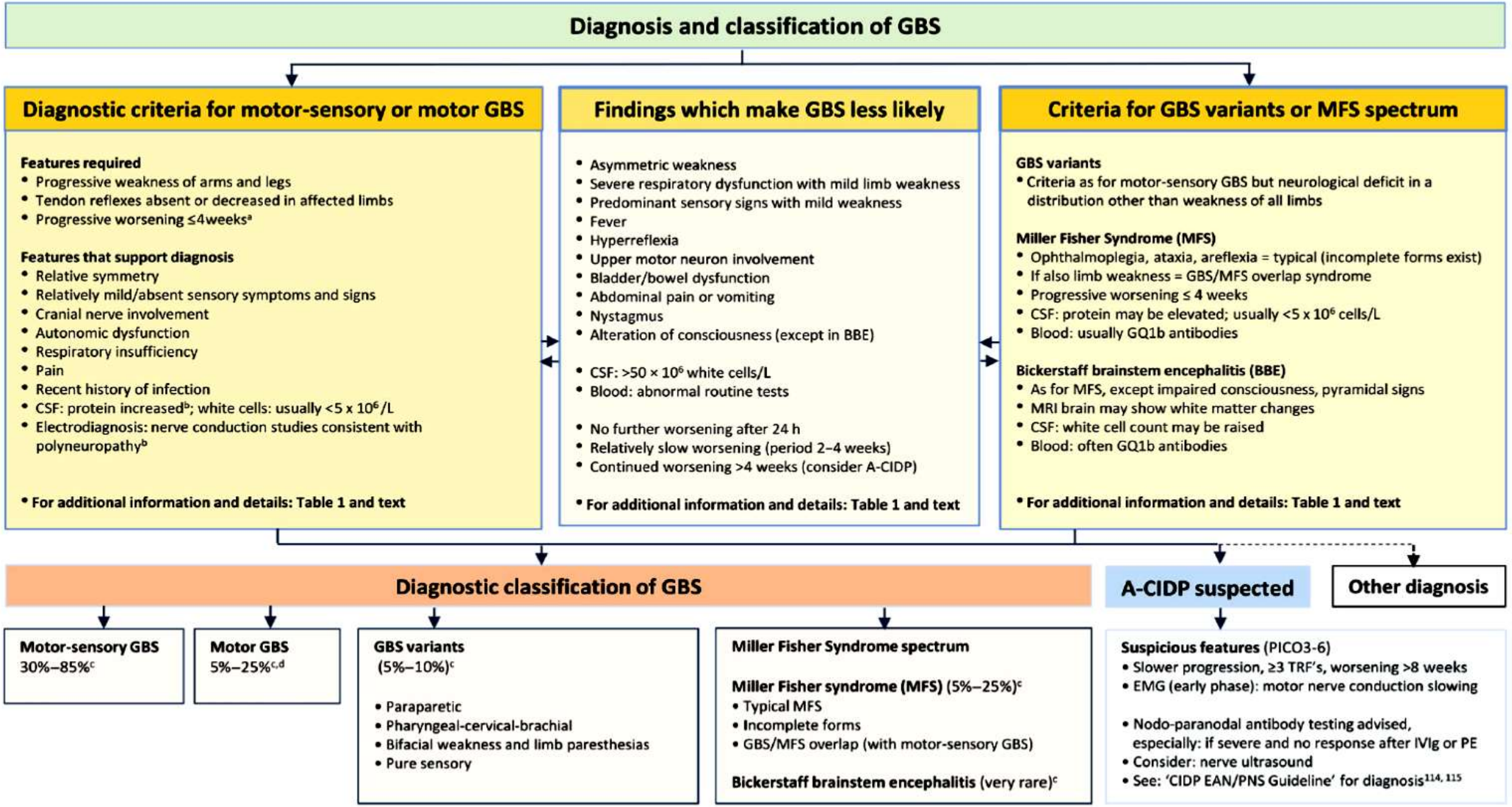
- There are several published sets of criteria for the diagnosis of GBS, of which the most frequently used are
- The NINDS criteria revised by Asbury and Cornblath (1990)
- The Brighton Collaboration Consensus Criteria (2011)
- The Wakerley and Yuki (2015) criteria
- The consensus guideline by Leonhard et al. (2019)

- Clinical
- Investigations
- AC +BKR

National Institute of Neurological Disorders and Stroke (NINDS) GBS diagnostic criteria

Required Features	Supportive Features
• Progressive weakening of the trunk, bulbar, and face muscles as well as external ophthalmoplegia, ranging from mild weakness of the legs to complete paralysis of all four limbs • Weak limbs with areflexia or hyporeflexia	• From days to four weeks, symptoms progress • Symptoms are bilateral and symmetrical in nature. • Pain in the trunk or limbs • Cranial nerve symptoms or signs • failure of the autonomic system • A mild form of sensory dysfunction • No fever at symptom onset • CSF with normal to mildly raised leukocyte count (typically 5 cells/mm3) and high protein. • Anomalies on the electrodiagnostic screen that are GBS-relatedRecovery starting two to four weeks after progression halts

European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of GBS (2023)



Lab investigations:

- Help to exclude other causes of acute flaccid paralysis such as infections or metabolic or electrolyte dysfunctions (Hypokalemia, Hypomagnesemia, Hypophosphatemia etc.).

CSF examinations :

- Done when the diagnosis is uncertain or when an alternative diagnosis needs to be excluded.
- Classic finding is albumino-cytological dissociation.
- However it could be normal in about 30-50%of patients in the first week and 10-30% in the second week.
- Usually <5 cells/µL, rarely 5–50 cells/µL
- CSF cells >50 cells/µL : May be alternative diagnosis

Electrodiagnostics:

- **Early Electrodiagnostic Features of GBS**
- 🔻 **High Sensitivity, Low Specificity** – Supportive but not definitive
- ✅ **Findings:**
- **Polyneuropathy** – Sensory and/or motor conduction abnormalities
- **Absent H-reflexes** – Common early sign
- **Facial Nerve Involvement** – ↑ Distal motor latency / ↓ CMAP amplitude
- **Blink Reflex Abnormalities** – Absent or prolonged R1, R2 responses

Electrodiagnostic Features Supporting GBS Diagnosis

🔹 **Low to Moderate Sensitivity, High Specificity**

✅ **Key Findings:**

- **Sural Sparing Pattern** – Abnormal median/ulnar SNAP with normal sural SNAP
- **Indirect Discharges** – Multiple A-wave-like responses
- **Prolonged Distal CMAP Duration** – >8.5 ms

🚫 **Findings Against GBS:**

Presence of **H-reflexes**

📌 **Additional Notes:**

- Normal NCS early on **does not exclude GBS** – Repeat testing may be needed
- **Miller Fisher Syndrome (MFS) Clues:**
  - Sural sparing
  - Polyneuropathy-like conduction abnormalities

Role of MRI & Ultrasound in GBS Diagnosis

🔻 **Not Recommended for Routine Use**

✅ **When to Consider:**

Atypical cases – May help differentiate GBS from mimics

📌 **Key Findings:**

MRI Nerve Root Enhancement – Supports GBS but not exclusive

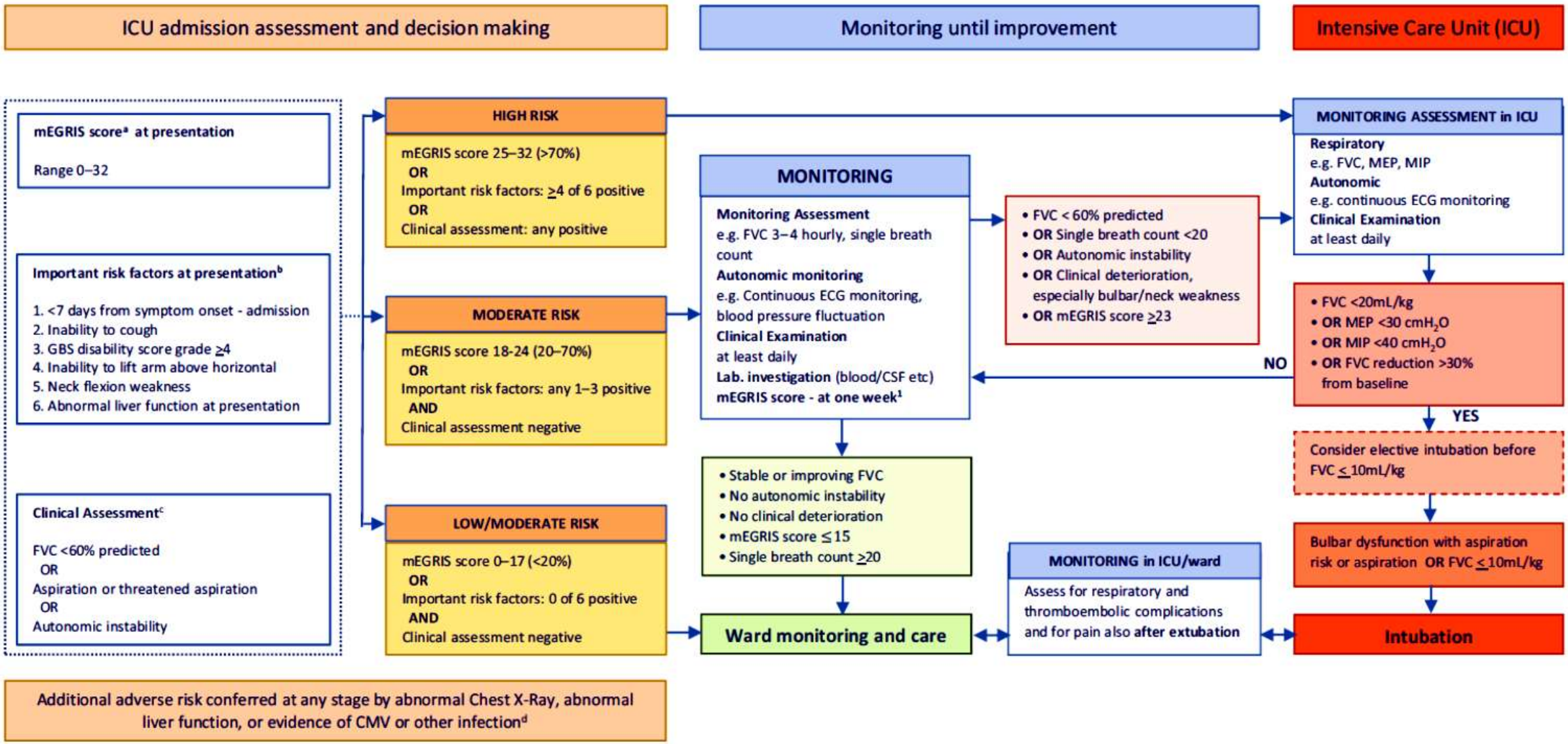
Widespread Nerve Enlargement (US/MRI) – Suggests A-CIDP but not definitive

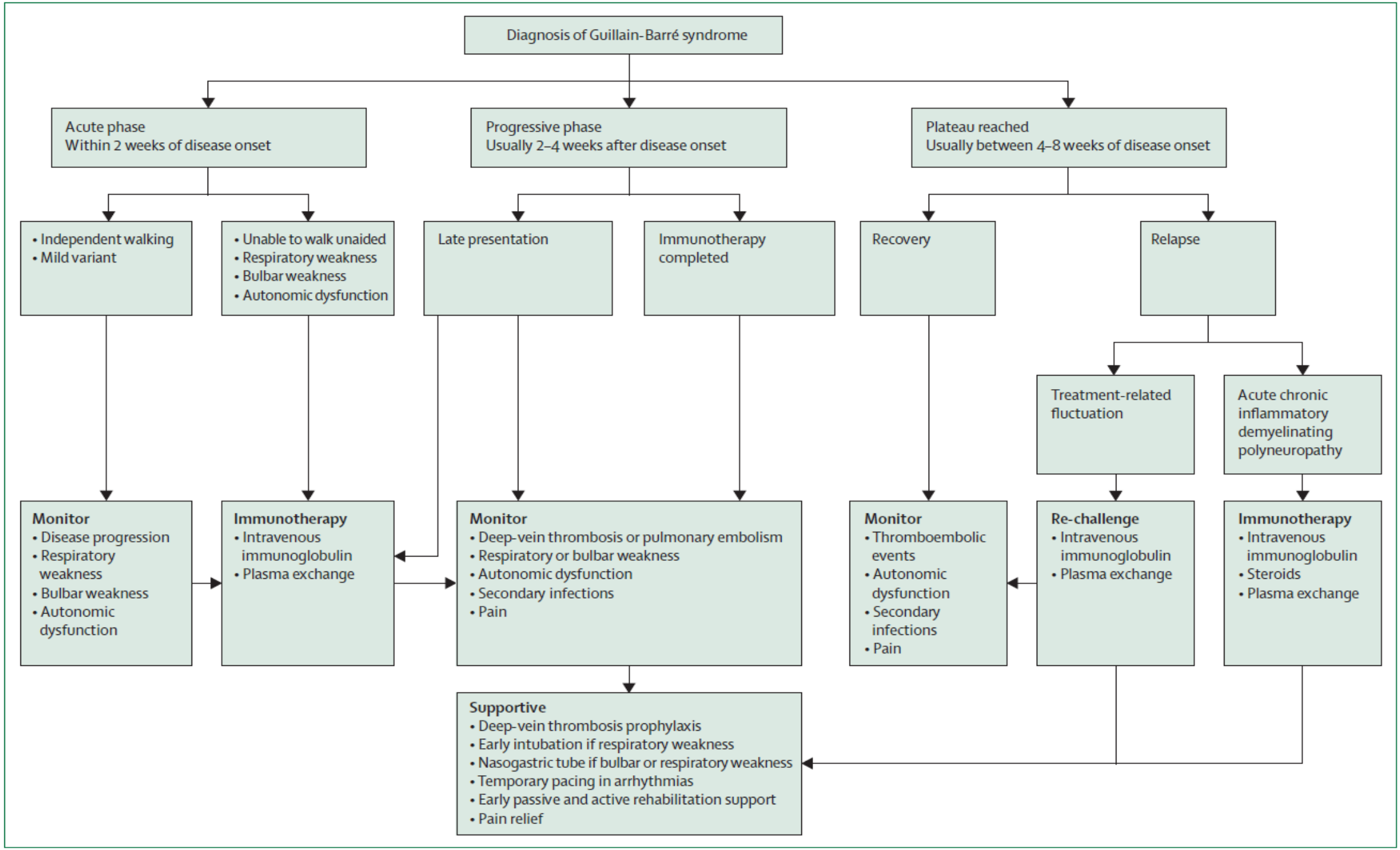
Whole Spine MRI with Contrast – Helps rule out **spinal cord compression, transverse myelitis, tumors**

Step 3 : When to refer and admit in ICU?

SB

Assessment and monitoring of GBS




Diagnosis of Guillain-Barré syndrome
Acute phase
Within 2 weeks of disease onset
Progressive phase
Usually 2–4 weeks after disease onset
Plateau reached
Usually between 4–8 weeks of disease onset
• Independent walking
• Mild variant
• Unable to walk unaided
• Respiratory weakness
• Bulbar weakness
• Autonomic dysfunction
Late presentation
Immunotherapy completed
Recovery
Relapse
Treatment-related fluctuation
Acute chronic inflammatory demyelinating polyneuropathy
Monitor
• Disease progression
• Respiratory weakness
• Bulbar weakness
• Autonomic dysfunction
Immunotherapy
• Intravenous immunoglobulin
• Plasma exchange
Monitor
• Deep-vein thrombosis or pulmonary embolism
• Respiratory or bulbar weakness
• Autonomic dysfunction
• Secondary infections
• Pain
Monitor
• Thromboembolic events
• Autonomic dysfunction
• Secondary infections
• Pain
Re-challenge
• Intravenous immunoglobulin
• Plasma exchange
Immunotherapy
• Intravenous immunoglobulin
• Steroids
• Plasma exchange
Supportive
• Deep-vein thrombosis prophylaxis
• Early intubation if respiratory weakness
• Nasogastric tube if bulbar or respiratory weakness
• Temporary pacing in arrhythmias
• Early passive and active rehabilitation support
• Pain relief

Box 3 | **Erasmus GBS Respiratory Insufficiency Score**

The Erasmus Guillain–Barré syndrome (GBS) Respiratory Insufficiency Score (EGRIS) calculates the probability that a patient with GBS will require mechanical ventilation within 1 week of assessment and is based on three key measures. Each measure is categorized and assigned an individual score; the sum of these scores gives an overall EGRIS for that patient (between 0 and 7). An EGRIS of 0–2 indicates a low risk of mechanical intervention (4%), 3–4 indicates an intermediate risk of mechanical intervention (24%) and ≥5 indicates a high risk of mechanical intervention (65%). This model is based on a Dutch population of patients with GBS (aged >6 years) and has not yet been validated internationally. Therefore, it may not be applicable in other age groups or populations. An online resource that automatically calculates the EGRIS for a patient based on answers to a series of questions has been made available by the International GBS Outcome Study (IGOS) consortium (see Related links). The Medical Research Council (MRC) sum score is the sum of the score on the MRC scale for: muscle weakness of bilateral shoulder abduction; elbow flexion; wrist extension; hip flexion; knee extension; and ankle dorsiflexion. A higher MRC sum score denotes increased disability, up to a maximum score of 60.

Measure	Categories	Score
Days between onset of weakness and hospital admission	>7 days	0
	4–7 days	1
	≤3 days	2
Facial and/or bulbar weakness at hospital admission	Absent	0
	Present	1
MRC sum score at hospital admission	60–51	0
	50–41	1
	40–31	2
	30–21	3
	≤20	4
EGRIS	NA	0–7

NA, not applicable. Adapted with permission from REF.[74], Wiley-VCH.

Indicators for Urgent Intubation in GBS in Children

- **Key Parameters:**

- **Vital Capacity** ≤ 20 mL/kg
- **Max Inspiratory Pressure** > -30 cmH2O
- **Max Expiratory Pressure** ≤ 40 cmH2O
- **Tidal Volume** < 5 mL/kg

Respiratory Monitoring in Young Children

⚠ **Challenges in Children <6 Years** – PFTs may be unreliable

✓ **Warning Signs:**

- $paCO_2 \geq 50$ mmHg
- ↑ Respiratory Rate & Oxygen Need
- Accessory Muscle Use & Paradoxical Breathing
- Sweating, Wide Pulse Pressure, Bounding Pulses

Ventilatory Management Considerations

✓ Early Pediatric Critical Care Involvement

✓ Avoid Sedation & Neuromuscular Blockade unless absolutely necessary – Masks progression

✓ Airway Care & Chest Physiotherapy – Prevents pneumonia

⚠ Tracheostomy may be needed for prolonged ventilation

Step 4 :When to start treatment?

SR

Indications for treatment in children :

IVIG and plasma exchange for children with GBS should be reserved for those with any of the following indications :

- Progressing weakness
- Worsening respiratory status or need for mechanical ventilation
- Significant bulbar weakness
- Inability to walk unaided

when to start treatment in adolescents?

One or more :

1. Inability to walk >10 m independently.
2. Rapid progression of weakness
3. Severe autonomic or swallowing dysfunction.
4. Respiratory insufficiency.

Table 2: Treatment dilemmas in GBS and recommendations

Dilemma		Current personal view
Start of treatment	Time window	Treatment should be initiated as soon as possible after diagnosis to prevent further nerve damage (LOE: 3). The effect of IVIg started after 2 weeks and of PE after 4 weeks onset of weakness is unknown (LOE: 4).
	Mild forms	Consider treating mildly affected patients with a rapidly progressive course or with additional features such as autonomic dysfunction, bulbar or facial weakness (LOE: 2).
	Variants	Patients with typical MFS likely require supportive care only (LOE: 3). In complicated MFS (limb weakness, bulbar weakness) and BBE, treatment with IVIg or PE should be considered (LOE: 4). Other GBS variants should be treated according to local guidelines until results of specific treatment trials show otherwise (LOE: 4).
	Children	Treatment with IVIg is beneficial in children and IVIg is preferred over PE because it is easier to administer (LOE: 2).
Repeat or change of treatment	Insufficient clinical response	There is not enough evidence that switching to IVIg after PE is effective in patients who are severely affected (LOE: 2). IVIg followed by PE should probably be avoided (LOE: 4). The effect of a 2nd IVIg course in patients with a poor prognosis is currently investigated.
	TRF	Although there are no RCTs, there is some rationale to re-treat patients who experience a TRF with either IVIg or PE (LOE: 4). When a patient develops 3 or more TRFs or deteriorates 8 weeks after onset, A-CIDP should be considered.

A-CIDP = acute-onset CIDP; BBE = Bickerstaff's brainstem encephalitis; GBS = Guillain-Barré syndrome; IVIg = intravenous immunoglobulin; LOE =level of evidence; MFS = Miller Fisher syndrome; PE= plasma exchange; RCT = randomised controlled trial; TRF= treatment-related fluctuation.
Adapted from: C. Verboon, PA van Doorn, BC Jacobs. J Neurol Neurosurg Psychiatry 2017;88:346-352

Step 5 : treatment options AC +BKR

- Intravenous immunoglobulin (0.4g/kg daily for 5 days)
- Plasma exchange (200-250ml/kg for 5 sessions).
- IVIg is effective when started within 2 weeks of disease onset and plasma exchange when started within 4 weeks.
- *Because of evidence of complement activation in pathogenesis of GBS, effect of eculizumab was investigated (JET-GBS);2018, a small RCT study which did not show significant difference in the endpoint. It however indicated that Eculizumab potentially may be an effective treatment.*

	Specific patient groups
GBS variant	1.Pure MFS tend to have a relatively mild disease course and tends to recover with in 6 months hence not recommended , but requires close monitoring. 2.Severity of disease course in BBE justifies treatment with IVIg. 3. For other variants no evidence regarding treatment is currently available.
Pregnant women	Neither IVIg nor PE is contraindicated
Children	Same as in adults.

IVIG & Plasma Exchange in Pediatric GBS

- **Not Recommended for Mild Cases:**
  - Ambulatory children
  - Mild, nonprogressive, or stabilized symptoms
- **Consider Treatment If:**
  - **Rapid progression** within 1–2 weeks, even if stabilized
  - **Inability to walk unaided**
- **Timing Matters:**
  - **IVIG:** Best within **2–4 weeks** of onset
  - **Plasma Exchange:** Best within **4 weeks** if unable to walk

IVIG vs. Plasma Exchange in Pediatric GBS

- **IVIG Preferred** – Safer & easier to administer
- **No Clear Superiority** – Limited high-quality data
- **Study Findings (Egypt, 41 Children):**
  - No difference in **ICU stay** or **walking unaided at 4 weeks**
  - **Plasma Exchange → Shorter ventilation duration** (11 vs. 13 days)
    - **Adults:** IVIG & Plasma Exchange are equally effective

Hughes, R. A., Swan, A. V., & van Doorn, P. A. (2014). Intravenous immunoglobulin for Guillain-Barré syndrome. *The Cochrane database of systematic reviews*, *2014*(9), CD002063. https://doi.org/10.1002/14651858.CD002063.pub6

IVIG in Pediatric GBS

❖ **Efficacy**:

•IVIg shortens recovery time compared to supportive care.

•Small trials and observational studies suggest faster recovery.

•Similar beneficial effects seen in adults with GBS.

❖ **Recovery Timeline**:

•Muscle strength improves within 14 days; most walk within 3 months.

•Early transient relapse may occur after treatment.

❖ **Dosage**:

•2 g/kg total, either over 2 days (1 g/kg/day) or 5 days (400 mg/kg/day).

•Based on treatment for immune deficiency disorders.

•Evidence for second course is limited, but may help in treatment-related fluctuations.

Plasma Exchange in Pediatric GBS

❖ **Efficacy**:

- Superior to supportive care in adults and children ≥10 years.
- Best if started within 7 days of symptom onset.
- Benefits: Faster recovery of muscle strength, reduced need for ventilation.

❖ **Mechanism**:

Removes antibodies attacking nerves from the circulation.

❖ **Procedure**:

- Requires special equipment and trained personnel.
- 4-6 double-volume exchanges over 1 week.
- Cumbersome for children < 2yrs old .

❖ **Complications**:

- Risk of citrate-induced hypocalcemia, vascular catheter issues, or infection.
- Younger children may need central venous catheter placement.

Step 6: monitoring disease progression SB

- Regular assessment :

1. Respiratory function measurement - usage of accessory respiratory muscle , counting during expiration of one full capacity inspiratory breath, vital capacity, maximum inspiratory and expiratory pressure(20/30/40)

2. Muscle strength in the necks, arms and legs using MRC grading scale and functional disability using GBS disability scale.

3. Monitor for swallowing and coughing difficulties

4. Autonomic function

Guillain-Barré syndrome (GBS) Disability Score

The Guillain-Barré syndrome (GBS) disability score is a widely accepted scoring system to assess the functional status of patients with GBS. It was originally described in Hughes et al. (1978) and since then, various iterations have appeared in the literature. The adaptation best suited for use in the *Criteria* and BloodSTAR is from van Koningsveld et al (2007). The *Criteria* requires that the patient's level of disability be documented using the scale from 0 to 6 as below.

Guillain-Barré syndrome disability scale

Score	Description
0	A healthy state
1	Minor symptoms and capable of running
2	Able to walk 10m or more without assistance but unable to run
3	Able to walk 10m across an open space with help
4	Bedridden or chairbound
5	Requiring assisted ventilation for at least part of the day
6	Dead

EGRIS Kids score

EGRIS-Kids for predicting respiratory failure in children with GBS.

Predictors at hospital admission	Categories	Score
Age (years)	≤5	0
	6–10	1
	11–17	2
Cranial nerve involvement	Absent	0
	Present	3
GBS disability score	1	1
	2	2
	3	3
	4	4
EGRIS-Kids		1–9

EGRIS-Kids = Erasmus GBS Respiratory Insufficiency Score for children.

Step 7 : managing early complication AC+BKR

Table 2 | **Important complications of Guillain–Barré syndrome**

Complication	When to be alert
Choking	Bulbar palsy
Cardiac arrhythmias	All patients
Hospital-acquired infections (e.g., pneumonia, sepsis or urinary tract infection)	Bulbar and facial palsy, immobility, bladder dysfunction, mechanical ventilation
Pain and tactile allodynia	Limited communication
Delirium	Limited communication
Depression	Limited communication
Urinary retention	All patients
Constipation	Immobility
Corneal ulceration	Facial palsy
Dietary deficiency	Bulbar and facial palsy
Hyponatraemia	All patients
Pressure ulcers	Immobility
Compression neuropathy	Immobility
Limb contractures and ossifications	Severe weakness for prolonged period of time

Important complications of Guillain–Barré syndrome (GBS)[72]. Most of these complications can occur in any patient with GBS, at any time, but the second column shows when they are most likely to occur and/or when to be especially alert.

Step 8 : Managing clinical progression SR

Insufficient response to the treatment:

About 40 % of patients treated with standard doses of PE or IVig do not improve , it does not imply that the treatment is ineffective.

Observational study - the non randomised ISID study(oct 2019), second IVIg in patient with poor prognosis did not show better outcome.

Treatment related fluctuation

- To be considered in cases where there was initial improvement objectively by 1 grade in GBS disability score or by >/=5 in MRC grade scale within 4 weeks followed by reduction in the MRC score by > 5 and FDS by 1.
- The general view is that a TRF indicates wearing off the treatment effect while the inflammatory phase is still going on.
- These patient might benefit from repeating the treatment.

Step 9 : Predicting outcome. SB

- Most patients with GBS , even those who are tetraplegic at nadir or required mechanical ventilation show extensive recovery with in first year.
- Probability of regaining walking ability can be predicted by using mEGOS.
- Death can occur in 3-10% of cases , most commonly due to cardiovascular and respiratory complication.
- Risk factors for mortality include- advanced age and severe disease at onset.

Supplementary table 3. Modified Erasmus GBS Outcome Score (mEGOS)

Prognostic factors	*Categories*	*Score*
Age at onset	≤ 40	0
	41-60	1
	> 60	2
Preceding diarrhea	Absent	0
	Present	1
MRC sum score* at day 7 of hospital admission	60-51	0
	50-41	3
	40-31	6
	30-0	9
mEGOS 0-12		
0-6 = Low risk (>90%)		
7-9 = Intermediate risk (70-85%)		
9-12 = High risk (40-70%)		

Recovery in Children with GBS [AC]

- **Long-term Recovery**:
  - 85-92% achieve excellent recovery (symptom-free or minimal disability).
  - 90% are ambulatory within 6 months; nearly all walk within 1 year.
- **Residual Symptoms**:
  - 65% report sequelae (paresthesia, gait issues, fatigue).
  - Long-term deficits: sensory loss (14%), limb weakness (8%), areflexia (5%).
- **Risk Factors for Poor Recovery**:
  - Very young age (<2 years), severe weakness, rapid progression, cranial nerve involvement, ventilator support, and inexcitable motor nerves.

GBS Subtypes & Recurrence BKR

- **GBS Subtypes**:
  - Favorable outcomes in both AIDP and AMAN subtypes.
  - Delayed recovery more common in AMAN (80% walking within 6 months).
- **Recurrence & Fluctuations**:
  - Recurrence occurs in 2-5% of cases, months to years after the initial episode.
  - Treatment-related fluctuations in up to 7%, usually responsive to immunomodulatory treatment.
  - 2% may develop chronic inflammatory demyelinating polyneuropathy.

Step 10 : Planning rehabilitation SR

- Start rehabilitation programme early.
- Should aim to reduce disability in the early stages and later to restore motor and sensory function and physical condition to pre disease levels.
- Fatigue is found in 60-80%of patients with GBS and is often one of the most disabling complaints.
- Pain is reported in 1/3 of the patients of GBS 1yr after onset and persist for >10 yrs, characterized by muscle pain in lower back, arthalgias and radicular pain
- Early recognition and management of psychological distress is important as mental status can influence physical recovery.


THANK YOU!