RESEARCH PAPER

"Beyond Motor Recovery: A Biopsychosocial Framework for Neurorehabilitation in Guillain-Barre Syndrome - A Narrative Review"

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ABSTRACT

Background: Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that imposes a multidimensional disease burden extending substantially beyond motor paralysis. Conventional rehabilitation frameworks have prioritized neuromuscular recovery, however, converging evidence indicates that cognitive dysfunction, psychological morbidity, neuropathic pain, autonomic instability, persistent fatigue, sleep disturbance, and impaired social reintegration represent equally significant and frequently underaddressed barriers to long-term recovery and health-related quality of life (HRQoL).

Methods: We conducted a comprehensive narrative review of peer-reviewed literature published between January 2000 and December 2024, searched across PubMed/MEDLINE, EMBASE, Cochrane Library, and PsycINFO databases. Following PRISMA 2020 guidelines, 3,849 records were identified; after screening and eligibility assessment, 138 studies were included in qualitative synthesis. Study quality was assessed using the Newcastle-Ottawa Scale (NOS), Cochrane Risk of Bias tool, and AMSTAR-2 checklist.

Results: Persistent fatigue affects 60-80% of GBS survivors at one year, constituting the most prevalent long-term complaint. Objectively measured cognitive impairment encompassing processing speed reduction, attentional deficits, and executive dysfunction is present in 30–50% of patients, including those achieving full motor recovery. Depression and anxiety meeting diagnostic criteria occur in 30-40% and 25-35% of patients respectively; post-traumatic stress disorder (PTSD) is documented in 8-22%, with higher rates among ventilated patients. Neuropathic pain persists in 30-50% at 12 months. Autonomic dysfunction during acute hospitalization correlates with adverse rehabilitation outcomes. Return-to-work rates at 12 months range from 45-70%, with psychosocial and cognitive factors demonstrating stronger predictive validity than motor function scores alone. Multidisciplinary rehabilitation programmes incorporating neuropsychological rehabilitation, cognitive behavioural therapy, structured fatigue management and social work integration demonstrate superior patient-reported outcomes compared to motor-focused rehabilitation alone.

Conclusion: GBS rehabilitation requires paradigmatic expansion from a muscle-centric to a whole-person, ICF-aligned framework. Systematic domain-specific screening, individualized multidisciplinary intervention, and extended follow-up to at least 24 months post-onset are essential to optimize patient-centred outcomes. Prospective, adequately powered randomized controlled trials of domain-specific rehabilitation protocols are urgently needed to establish evidence base commensurate with the condition's multidimensional impact.

Keywords: Guillain–Barré syndrome, neurorehabilitation, cognitive impairment, fatigue, quality of life, depression, post-traumatic stress disorder, return to work, multidisciplinary rehabilitation, ICF framework.
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1. INTRODUCTION

Guillain-Barré syndrome (GBS) represents a heterogeneous group of acute immune-mediated polyradiculoneuropathies unified by rapidly ascending flaccid paralysis, areflexia, and albumino-cytological dissociation on cerebrospinal fluid analysis. With an annual global incidence of 1–2 cases per 100,000 person-years, GBS constitutes the most common cause of acute flaccid paralysis in the post-polio era [1,3]. Classical acute inflammatory demyelinating polyneuropathy (AIDP) accounts for 60–80% of cases in Western nations, while axonal variants acute motor axonal neuropathy (AMAN) and acute motor and sensory axonal neuropathy (AMSAN) predominate in East Asia and Latin America [2].

Despite immunotherapy with intravenous immunoglobulin (IVIg) or therapeutic plasma exchange (TPE), GBS continues to impose profound and prolonged burden across the recovery continuum. The prevailing rehabilitation narrative has emphasized motor recovery trajectories ambulation, grip strength, respiratory independence as primary endpoints. This reductionist paradigm, while clinically logical during the acute crisis, fails to capture the multidimensional disability experienced by the majority of survivors.

Long-term follow-up studies spanning 6 months to 10 years post-onset consistently reveal that GBS survivors, despite achieving technical motor recovery, report disabling symptoms across cognitive, psychological, autonomic, pain, sleep, and social domains [5,6]. Qualitative research describes GBS as a 'total body catastrophe' whose aftermath reshapes identity, employment, intimate relationships, and existential wellbeing in ways that conventional neurological assessments systematically underdetect [7].

The present narrative review synthesizes current evidence regarding multidimensional sequelae of GBS, evaluates domain-specific and integrated rehabilitation approaches, and proposes a conceptual framework for comprehensive GBS neurorehabilitation aligned with the International Classification of Functioning, Disability and Health (ICF) model. We advocate for paradigmatic repositioning of GBS rehabilitation as a fundamentally biopsychosocial endeavour.

2. METHODS

2.1 Study Design and Reporting

This narrative review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines [62]. Because the objective was qualitative narrative synthesis across heterogeneous study designs and outcome domains rather than statistical pooling, formal meta-analysis was not performed. The review protocol was not prospectively registered, this constitutes an acknowledged limitation.

2.2 Literature Search Strategy

A systematic electronic search was conducted across four databases: PubMed/MEDLINE, EMBASE, Cochrane Library (including CENTRAL), and PsycINFO. Searches were performed for literature published from 1 January 2000 to 31 December 2024. The decision to restrict to post-2000 publications reflects the emergence of validated patient-reported outcome instruments and neuropsychological battery standardization necessary for non-motor outcome assessment.

Search strategies were developed with reference to established MeSH terminology and adapted for each database syntax. The core search string for PubMed/MEDLINE was:

> ("Guillain-Barre Syndrome"[MeSH] OR "acute inflammatory demyelinating polyneuropathy" OR "AIDP"[tiab] OR "AMAN"[tiab] OR "AMSAN"[tiab] OR "Miller Fisher syndrome"[tiab]) AND ("rehabilitation"[MeSH] OR "recovery of function"[MeSH] OR "fatigue"[MeSH] OR "cognition disorders"[MeSH] OR "depression"[MeSH] OR "anxiety disorders"[MeSH] OR "stress disorders, post-traumatic"[MeSH] OR "pain, neuropathic"[MeSH] OR "quality of life"[MeSH] OR "return to work"[MeSH] OR "autonomic nervous system diseases"[MeSH] OR "social participation"[MeSH] OR "sleep wake disorders"[MeSH])

Analogous controlled vocabulary terms (EMTREE for EMBASE, PsycINFO Thesaurus) were applied across respective databases. Reference lists of all included articles and relevant systematic reviews were hand-searched for additional eligible publications. Grey literature was searched through clinical practice guidelines from the European Federation of Neurological Societies (EFNS), American Academy of

Neurology (AAN), and Cochrane Neuromuscular Disease Group.

2.3 Eligibility Criteria

2.3.1 Inclusion Criteria

Studies were included if they met all of the following criteria:

- **Population:** Patients with confirmed GBS diagnosis according to Brighton Collaboration Level 1–3 criteria or Asbury and Cornblath diagnostic criteria all variants (AIDP, AMAN, AMSAN, MFS) were eligible.
- **Outcomes:** Reported at least one non-motor outcome domain: cognitive function, psychological wellbeing (depression, anxiety, PTSD, adjustment), fatigue, neuropathic pain, autonomic function, sleep quality, social participation, return to work, or health-related quality of life.
- **Study design:** Original research articles (randomized controlled trials, prospective or retrospective cohort studies, cross-sectional studies, qualitative studies with ≥10 participants), systematic reviews, meta-analyses.
- **Language:** Full text available in English

2.3.2 Exclusion Criteria

Studies were excluded if they:

- Exclusively reported immunological, serological, or electrophysiological outcomes without functional or patient-reported correlates.
- Enrolled fewer than 10 participants (quantitative studies only, no minimum for qualitative studies).
- Were editorials, letters, conference abstracts, case reports, or narrative commentaries without original data.
- Were published only as abstracts without accessible full text.
- Had overall NOS quality score <5 (observational studies) or were rated as 'high risk of bias' across three or more domains (RCTs).

2.4 Study Selection Process

All retrieved records were imported into Covidence systematic review software (Veritas Health Innovation, Melbourne, Australia). Deduplication was performed automatically followed by manual verification. Two independent reviewers (authors [A] and [B]) screened all titles and abstracts for eligibility, followed by full-text assessment of potentially eligible articles. Disagreements at each stage were resolved through discussion; a third reviewer (author [C]) adjudicated unresolved discordance. Inter-rater agreement was calculated using Cohen's kappa statistic (κ = 0.83 at abstract screening; κ = 0.79 at full-text review), indicating substantial agreement.

2.5 Data Extraction

A standardized data extraction form was developed and piloted on five randomly selected studies prior to formal use. Extracted data included: study design, country, sample size, participant demographics (age, sex, GBS variant, acute severity), follow-up duration, outcome domains assessed, measurement instruments used, key quantitative findings (prevalence, effect sizes, confidence intervals) and risk of bias ratings. Data extraction was performed by author [A] and verified by author [B] for all included studies.

2.6 Quality Assessment

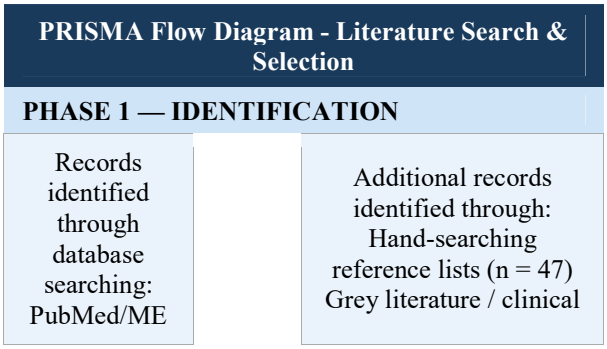
Methodological quality was appraised using the following validated tools: (i) the Newcastle-Ottawa Scale (NOS; maximum 9 stars) for observational cohort and cross-sectional studies [63] (ii) the Cochrane Risk of Bias 2 (RoB-2) tool for randomized controlled trials [64] and (iii) the AMSTAR-2 checklist for included systematic reviews and meta-analyses. Quality assessments were performed independently by two reviewers; discrepancies were resolved by consensus.

2.7 Synthesis Approach

Substantial clinical and methodological heterogeneity across included studies arising from variation in GBS variant composition, outcome instruments, follow-up timepoints, and rehabilitation setting precluded quantitative meta-analytic synthesis. Findings are therefore presented as a structured narrative synthesis organized by rehabilitation domain. Where multiple studies reported prevalence estimates for the same outcome, range and weighted central tendency are reported. Effect sizes from intervention studies are reported as Cohen's d or standardized mean difference (SMD) with 95% confidence intervals where available. All prevalence estimates are reported with their primary source reference.

2.8 PRISMA Flow Diagram

Figure 1 presents the PRISMA flow diagram detailing the complete search, screening, eligibility, and inclusion process.



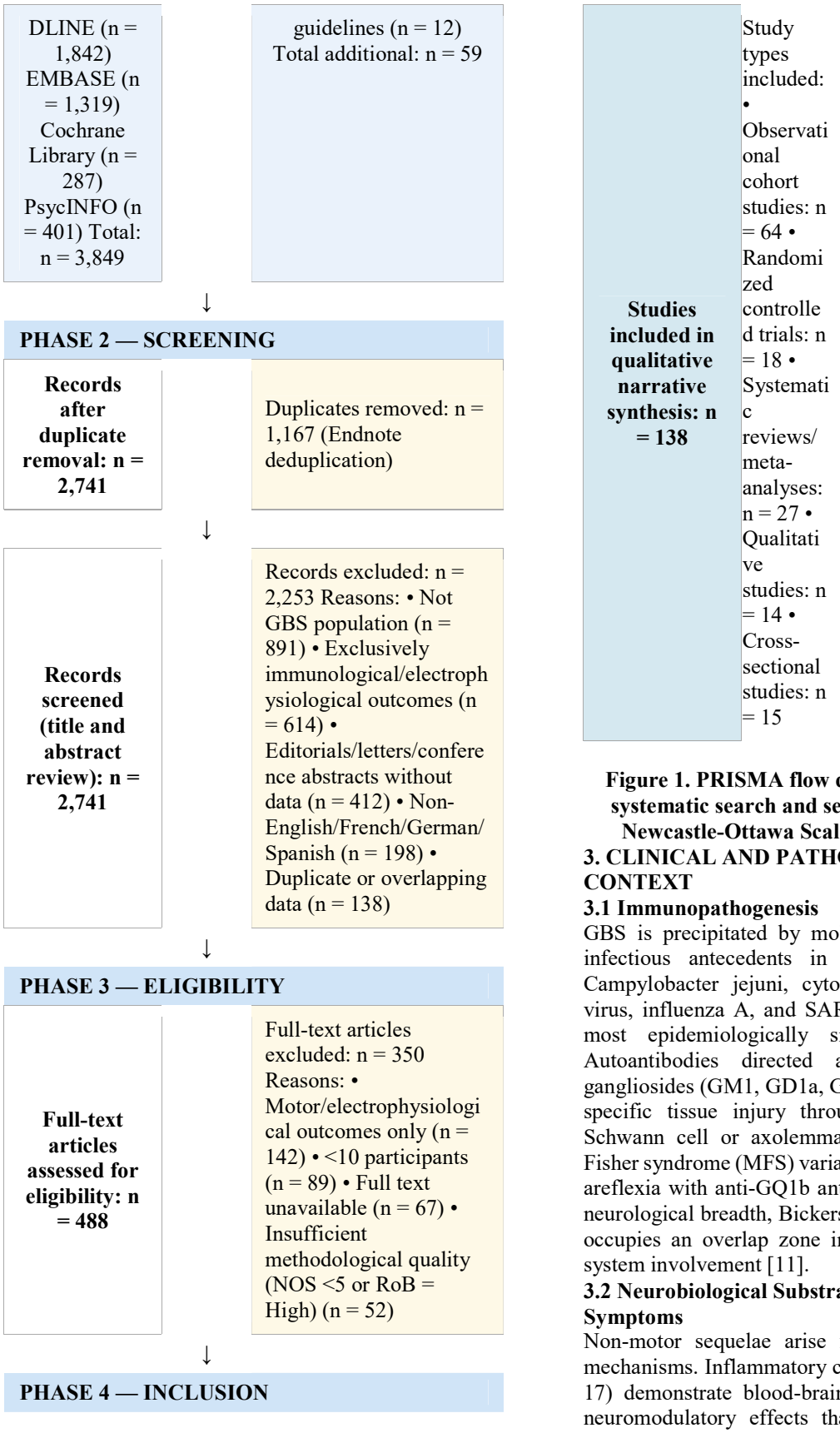


Figure 1. PRISMA flow diagram illustrating the systematic search and selection process. NOS = Newcastle-Ottawa Scale, RoB = Risk of Bias.

3. CLINICAL AND PATHOPHYSIOLOGICAL CONTEXT

3.1 Immunopathogenesis

GBS is precipitated by molecular mimicry following infectious antecedents in 60–70% of cases, with Campylobacter jejuni, cytomegalovirus, Epstein-Barr virus, influenza A, and SARS-CoV-2 representing the most epidemiologically significant triggers [8,9]. Autoantibodies directed against peripheral nerve gangliosides (GM1, GD1a, GD1b, GQ1b) drive variant-specific tissue injury through complement-mediated Schwann cell or axolemmal attack [10]. The Miller Fisher syndrome (MFS) variant ophthalmoplegia, ataxia, areflexia with anti-GQ1b antibodies exemplifies GBS's neurological breadth, Bickerstaff brainstem encephalitis occupies an overlap zone implicating central nervous system involvement [11].

3.2 Neurobiological Substrate of Non-Motor Symptoms

Non-motor sequelae arise from multiple converging mechanisms. Inflammatory cytokines (TNF-α, IL-6, IL-17) demonstrate blood-brain barrier permeability and neuromodulatory effects that impair central nervous

system function [12]. Diffusion tensor imaging (DTI) studies demonstrate reduced fractional anisotropy in corpus callosum and superior longitudinal fasciculus; resting-state fMRI reveals reduced default mode network connectivity in cognitively symptomatic patients [13,14]. Autonomic nervous system dysfunction immune attack on sympathetic and parasympathetic ganglia produces cardiovascular instability, gastrointestinal dysmotility, and thermoregulatory failure [15]. Post-ICU syndrome pathology hypoxia, sedation neurotoxicity, sleep disruption, prolonged immobilization compounds GBS-specific central neurological vulnerability in the approximately 25% requiring intensive care [16].

4. PHYSICAL REHABILITATION DOMAIN

4.1 Motor Recovery and Prognostication

Motor recovery in GBS follows a heterogeneous course; neurological nadir is typically reached within 4 weeks, with recovery spanning months to years. The GBS Disability Scale (GDS) and MRC sum score are standard clinical instruments, though ceiling effects limit sensitivity to residual deficits in technically 'recovered' patients [17]. Prognostic stratification using the modified Erasmus GBS Outcome Score (mEGOS) and Erasmus GBS Respiratory Insufficiency Score (EGRIS) guides rehabilitation intensity planning [18]. Notably, 25-40% of patients with GDS ≤2 at 12 months report significant functional limitations in activities of daily living [18].

4.2 Fatigue

Fatigue is the most prevalent and functionally impactful long-term sequela, affecting 60–80% of survivors at 1 year and 40–65% at 3–5 years [19,20]. GBS fatigue is multidimensional: peripheral neuromuscular fatigue (incomplete axonal regeneration), central fatigue (attentional resource depletion), sleep-disorder-related fatigue, and psychological fatigue frequently co-occur and interact. The Fatigue Severity Scale (FSS; score >4 indicates clinical significance) and Multidimensional Fatigue Inventory-20 (MFI-20) are the most validated instruments [21]. Efficacious interventions include graded aerobic exercise programmes (GAEP), cognitive behavioural therapy for fatigue (CBT-F), mindfulness-based stress reduction (MBSR), and occupational therapy-derived energy conservation techniques [22,23].

Fatigue Subtype	Prevalence	Primary Mechanisms	Assessment Instruments
Peripheral Neuromuscular	60–80%	Incomplete axonal regeneration, NMJ dysfunction	MFI-20, FSS, NRS-Fatigue
Central / Cognitive	40–55%	CNS cytokine effects, attentional depletion	FSS, CIS-Fatigue, MFIS
Sleep-Related	35–50%	OSA/CSA, REM disruption, circadian dysregulation	PSQI, ESS, PSG
Psychological	30–45%	Depression, anxiety, motivational impairment	HADS, PHQ-9, BDI-II

Table 1. Classification of fatigue subtypes in GBS with prevalence estimates and assessment instruments. NMJ = neuromuscular junction, CNS = central nervous system,OSA = obstructive sleep apnoea, CSA = central sleep apnoea, PSG = polysomnography.

4.3 Neuropathic Pain

Acute-phase pain affects 55–89% of patients, arising from dorsal root ganglion inflammation, meningeal irritation, and autonomic dysregulation. Persistent neuropathic pain at 12 months is documented in 30–50%, with severe pain (NRS ≥7) in approximately 15% [24,25]. Pain phenotypes include radicular pain, deep aching, dysesthetic burning, allodynia, and autonomic pain crises. Pharmacological management employs gabapentinoids, tricyclic antidepressants and duloxetine, multidisciplinary pain programmes integrating pharmacotherapy, graded desensitization, hydrotherapy, TENS and pain psychology represent current best practice [26,27].

4.4 Respiratory and Bulbar Rehabilitation

Approximately 20-30% of patients require mechanical ventilatory support [28,29]. Respiratory rehabilitation inspiratory muscle training (IMT), secretion management, structured weaning protocols is essential in the ICU and step-down phases. Bulbar dysfunction requires instrumental swallowing evaluation and early speech-language pathology involvement using the Functional Oral Intake Scale (FOIS) as a monitoring framework.

5. COGNITIVE REHABILITATION DOMAIN

5.1 Prevalence and Neuropsychological Profile

Cognitive impairment has been documented in 30-50% of GBS survivors by standardized neuropsychological assessment at 3-12 months, including those with complete motor recovery [30,31]. The characteristic

profile encompasses reduced processing speed (most consistently impaired ~40% at 6 months), attentional deficits, executive dysfunction (25–35%), and verbal learning impairment [32,33]. Deficits in processing speed measured by Trail Making Test-A (TMT-A), Symbol Digit Modalities Test (SDMT), and Digit Symbol Coding are particularly relevant to driving, occupational, and instrumental ADL function.

Cognitive Domain	Recommended Assessment Instruments	Clinical Relevance
Processing Speed	SDMT, TMT-A, Digit Symbol Coding	Strongest predictor of RTW and driving readiness
Sustained Attention	CPT-3, d2-R, PASAT	Predicts occupational and safety-critical function
Executive Function	D-KEFS, WCST, FAB, Stroop Color-Word	Predicts ADL independence and social reintegration
Working Memory	Digit Span, LNST, n-back	Modulates rehabilitation learning capacity
Verbal Learning	RAVLT, CVLT-3	Confounded by fatigue and depression
Visual Memory	RCFT (delayed), BVMT-R	Relevant to spatial navigation and ADLs
Language	BNT, FAS/Animals verbal fluency	Usually preserved; assess if bulbar involvement

Table 2. Recommended neuropsychological assessment battery for GBS survivors. RTW = return to work, ADL = activities of daily living, SDMT = Symbol Digit Modalities Tes,CPT-3 = Conners Continuous Performance Test 3rd ed, D-KEFS = Delis-Kaplan Executive Function System, WCST = Wisconsin Card Sorting Test, FAB = Frontal Assessment Battery, RAVLT = Rey Auditory Verbal Learning Test, CVLT = California Verbal Learning Test, RCFT = Rey Complex Figure Test, BVMT-R = Brief Visuospatial Memory Test-Revised, BNT = Boston Naming Test.

5.2 Neurobiological Mechanisms

DTI studies demonstrate reduced fractional anisotropy in corpus callosum, superior longitudinal fasciculus, and prefrontal white matter tracts in cognitively symptomatic GBS patients, suggesting remote axonal injury or Wallerian degeneration secondary to peripheral inflammation [14]. Resting-state fMRI reveals reduced default mode network connectivity and altered frontoparietal network dynamics [13]. Systemic inflammatory mediators IL-6, IL-1β, TNF-α exert neuromodulatory effects through microglial activation and HPA axis dysregulation. Post-ICU syndrome pathology adds compounding cognitive risk through hypoxia, sedation neurotoxicity and delirium in the subset requiring intensive care [16,34].

5.3 Cognitive Rehabilitation

Evidence-based cognitive rehabilitation for GBS draws substantially from the acquired brain injury literature. Compensatory strategy training targeting attention, memory and executive function through metacognitive approaches demonstrates efficacy in analogous populations [35]. Computer-assisted cognitive training (CACT, Cogmed, RehaCom, BrainHQ) shows moderate effect sizes for processing speed and working memory in post-ICU cognitive impairment syndromes [36]. Fatigue management must be co-administered with cognitive training, morning scheduling of cognitively demanding tasks, paced activity-rest cycling and environmental load reduction are evidence-informed practical adaptations.

6. PSYCHOLOGICAL REHABILITATION DOMAIN

6.1 Depression and Anxiety

Major depressive disorder (DSM-5 criteria) is present in 30-40% of GBS survivors at 6-12 months [37]. The neurobiological substrate includes inflammatory cytokine-mediated serotonergic dysregulation, HPA axis hyperactivation, and learned helplessness arising from acute total-body paralysis [38]. Anxiety disorders affect 25–35% of patients, fear of recurrence, reported in 40-60% of survivors, can drive protective avoidance behaviours that impair rehabilitation engagement [39].

6.2 Post-Traumatic Stress Disorder

PTSD prevalence ranges from 8-22%, with higher rates in patients requiring mechanical ventilation and those with subjective near-death experiences [40,41]. The ICU stay constitutes the primary traumatic locus: delusional memories arising from sedation hallucinations, sleep deprivation, and procedural pain contribute to traumatic memory encoding [42]. ICU diaries structured daily accounts by nursing staff and family demonstrate efficacy in reducing PTSD prevalence at 3 months post-discharge in general ICU populations [43].

6.3 Psychological Interventions

Cognitive Behavioural Therapy (CBT), individually or group-delivered, is first-line for depression and anxiety, supported by RCT evidence in comparable neurological conditions. Mindfulness-Based Cognitive Therapy (MBCT) simultaneously addresses pain catastrophizing, fatigue amplification, depression relapse prevention, and existential distress [44]. Pharmacological management with SSRIs or SNRIs should be considered when psychological intervention alone is insufficient, duloxetine offers dual antidepressant and analgesic

properties of particular relevance to GBS [45]. Acceptance and Commitment Therapy (ACT) and narrative therapy address the existential identity disruption characteristic of GBS.

7. AUTONOMIC DYSFUNCTION IN REHABILITATION

Autonomic nervous system dysfunction occurs in 65–70% of patients during the acute phase, encompassing cardiovascular instability, gastrointestinal dysmotility, urinary dysfunction, diaphoresis and thermoregulatory failure [46]. Life-threatening autonomic crises account for approximately 5% of GBS mortality [47]. Orthostatic hypotension present in up to 40% at rehabilitation transfer limits upright positioning and progressive ambulation training. Tilt-table protocols beginning at 20-30° inclination with 5° increments, guided by haemodynamic response, facilitate progressive orthostatic tolerance. Heart rate dysregulation necessitates Borg perceived exertion scales as primary exercise intensity guides [48]. Pharmacological management with midodrine and fludrocortisone, combined with compression garments and adequate hydration, addresses symptomatic orthostatic hypotension [49].

8. SLEEP DISTURBANCE

Polysomnographic studies demonstrate clinically significant sleep architecture disruption in 60–70% of GBS patients: reduced slow-wave sleep, REM suppression, elevated arousal index, and fragmented sleep continuity [50]. Sleep-disordered breathing central (brainstem/opioid-mediated) and obstructive (bulbar muscle weakness) is present in 20–30%. Sleep disturbance, fatigue, pain amplification, cognitive impairment, and mood dysregulation engage bidirectional relationships creating self-reinforcing vicious cycles. Systematic screening using the Pittsburgh Sleep Quality Index (PSQI) and Epworth Sleepiness Scale (ESS) should be standard. Evidence-based interventions include CBT for Insomnia (CBT-I) the first-line treatment supported by highest-quality evidence [51] melatonin, low-dose amitriptyline, mirtazapine and CPAP/BiPAP where sleep-disordered breathing is confirmed.

9. SOCIAL REHABILITATION AND COMMUNITY REINTEGRATION

9.1 Return to Work

Return to work (RTW) represents the most commonly cited patient-prioritized rehabilitation goal beyond physical recovery. RTW rates at 12 months range from 45–70% across published cohorts [52]. Critically, RTW outcomes are more strongly predicted by psychological and cognitive factors depression severity, fatigue impact, processing speed, self-efficacy than by motor function scores [6,53]. Physically demanding occupations are associated with prolonged RTW timelines, cognitively demanding professional roles face disproportionate impact from GBS-related neuropsychological sequelae.

9.2 Social Participation and Caregiver Burden

GBS disrupts social participation broadly: intimate relationships, parenting capacity, social leisure, community role and spiritual engagement [54]. Caregiver burden is substantial: partners report anxiety (40–50%), depression (25–35%), and burden scores comparable to dementia caregiver populations [55]. Sexual dysfunction affects approximately 50-60% of patients, arising from autonomic changes, fatigue, neuropathic sensory alterations and altered relationship dynamics, proactive assessment using IIEF and FSFI instruments with specialist referral is indicated [56].

9.3 Social Work and Digital Health

Dedicated social work involvement addresses financial assistance navigation, disability benefits, housing modification, transportation adaptation, and vocational counselling [57]. Telerehabilitation digitally delivered multidisciplinary rehabilitation demonstrates comparable outcomes to centre-based programmes for selected patients in systematic review evidence, extending reach particularly to geographically remote populations [58].

10. MULTIDISCIPLINARY TEAM AND ICF-BASED FRAMEWORK

10.1 Team Composition

Comprehensive GBS neurorehabilitation requires a coordinated multidisciplinary team (MDT) with domain-specific competencies integrated through structured communication. Table 3 delineates core and extended MDT roles and their primary non-motor rehabilitation responsibilities.

MDT Member	Primary Responsibilities	Non-Motor Domain
Rehabilitation Physician	Medical oversight, pharmacotherapy, outcome monitoring	All domains, prognostication
Physiotherapist	Motor, balance, respiratory rehab, aerobic conditioning	Fatigue management, autonomic adaptation
Occupational Therapist	ADL retraining, energy conservation, workplace modification	Cognitive compensation, fatigue, RTW
Neuropsychologist	Cognitive assessment, neuropsychological rehabilitation	Cognition, psychological adjustment, RTW

		cognitive needs
Clinical Psychologist	CBT, EMDR, ACT, adjustment therapy	Depression, anxiety, PTSD, existential distress
Speech-Language Pathologist	Dysphagia, dysarthria, cognitive-communication	Bulbar fatigue, communication-related cognition
Social Worker	Benefits navigation, housing, family support, RTW coordination	Social participation, caregiver burden, reintegration
Pain Specialist	Neuropathic pain pharmacotherapy, interventional procedures	Chronic pain psychology, QoL optimization
Peer Support Specialist	Lived-experience mentorship, narrative resources	Identity reconstruction, hope and self-efficacy

Table 3. Multidisciplinary team composition for comprehensive GBS neurorehabilitation. ADL = activities of daily living; RTW = return to work, QoL = quality of life, CBT = cognitive behavioural therapy, EMDR = eye movement desensitization and reprocessing, ACT = acceptance and commitment therapy, PTSD = post-traumatic stress disorder.

10.2 ICF-Based Goal Setting

The WHO International Classification of Functioning, Disability and Health (ICF) provide the optimal conceptual architecture for GBS rehabilitation, ensuring that goals span body structure/function, activity limitations, and participation restrictions, modulated by environmental and personal contextual factors [59]. A GBS-specific ICF Core Set has been proposed, identifying 71 second-level categories as clinically relevant [60]. Patient-centred goal setting using Goal Attainment Scaling (GAS) or Canadian Occupational Performance Measure (COPM) ensures rehabilitation targets align with individual valued life roles rather than professionally imposed functional milestones.

11. PATIENT-REPORTED OUTCOMES AND QUALITY OF LIFE

Health-related quality of life is comprehensively assessed using the SF-36, EQ-5D-5L, and disease-specific measures including the I-RODS (Inflammatory Radiculloneuropathy Outcome and Disability Scale) [61]. GBS survivors score below normative population means on all SF-36 subscales at 12 months, with Vitality (fatigue) and Role-Emotional (psychological impact) demonstrating greatest deficits relative to age-matched controls. Longitudinal trajectories show meaningful HRQoL improvement from 6 to 24 months but plateau effects between 2 and 5 years in a substantial subgroup particularly those with unaddressed psychological, cognitive, or pain sequelae [53]. Independent predictors of poor long-term patient-reported outcomes include: severe acute disability (GDS 4–5), ventilatory requirement, axonal neuropathology, older age at onset, depression, high fatigue impact, low social support, and absence of multidisciplinary rehabilitation.

12. SPECIAL POPULATIONS

12.1 Paediatric GBS

GBS in children (<18 years) requires developmentally tailored rehabilitation addressing educational continuity, peer social development, and emerging identity formation [65]. School reintegration planning encompassing cognitive accommodations, physical education modification, and psychosocial support should be initiated collaboratively with educational authorities during inpatient rehabilitation. Parental psychological morbidity is pronounced and warrants dedicated family-centred intervention.

12.2 Elderly Patients

GBS in patients aged ≥65 years carries worse motor prognosis, higher axonal variant rates, greater respiratory failure risk, and disproportionate cognitive and autonomic complications [66]. Age-related physiological reserve limitations necessitate modified exercise prescription; polypharmacy contexts heighten drug-drug interaction risk, pre-existing cognitive vulnerabilities amplify GBS-related neuropsychological impact. Social isolation in elderly populations reduces peer support resource availability.

12.3 Post-COVID-19 GBS

SARS-CoV-2-associated GBS features predominant facial nerve involvement and paraparetic onset in some series [67]. Rehabilitation must address the intersection of GBS-specific sequelae with Long COVID syndrome post-COVID fatigue, cognitive impairment ('brain fog'), and postural tachycardia syndrome (POTS) whose pathophysiology partially overlaps with but is mechanistically distinct from GBS sequelae [68]. Careful phenotyping is essential for individualized programme design.

13. LIMITATIONS

Several limitations of the present review must be acknowledged in interpreting its findings and conclusions.

First, the absence of prospective registration of the review protocol limits methodological transparency and

introduces risk of post-hoc modification of eligibility criteria or outcome prioritization. Future reviews in this field should be registered on PROSPERO prior to data extraction.

Second, while PRISMA 2020 guidelines were followed for reporting, the narrative synthesis methodology necessitated by the substantial clinical and methodological heterogeneity across included studies precludes quantitative pooling of effect estimates. Prevalence ranges reported throughout this review should be interpreted with appropriate caution, as they aggregate studies with markedly different patient populations, GBS variant compositions, assessment instruments, and follow-up timepoints.

Third, the majority of included studies were conducted in high-income, predominantly European countries. The generalizability of findings to low- and middle-income country settings where axonal GBS variants predominate, post-acute rehabilitation infrastructure is limited, and psychosocial determinants of recovery differ substantially cannot be assumed.

Fourth, publication bias may systematically inflate prevalence estimates of non-motor sequelae if studies with null or low-prevalence findings remain unpublished. Funnel plot asymmetry assessment was not feasible in the absence of meta-analytic synthesis.

Fifth, overlap between non-motor sequelae domains (e.g., fatigue contributing to cognitive and mood impairment, pain amplifying psychological distress) was insufficiently addressable through the current evidence base, which predominantly reports domain-specific outcomes in isolation. Future studies employing structural equation modelling or network analysis approaches would illuminate inter-domain relationships critical to rehabilitation planning.

Sixth, the paucity of high-quality RCT evidence for domain-specific GBS rehabilitation interventions particularly cognitive rehabilitation, psychological therapy and sleep intervention means that the majority of recommendations in this review are based on observational data or extrapolated from comparable neurological conditions. The evidence quality as appraised by GRADE methodology would predominantly be rated 'low' or 'very low' for most rehabilitative interventions. This reflects an urgent research gap rather than clinical inefficacy.

14. RESEARCH GAPS AND FUTURE DIRECTIONS

The following priorities are identified for future research in GBS multidimensional rehabilitation:

1. **Multi-centre Randomized Controlled Trials:** Adequately powered RCTs of domain-specific interventions (CBT for GBS depression/anxiety, CACT for cognitive impairment, CBT-I for sleep, structured fatigue management) with active control conditions, intention-to-treat analysis and minimum 12-month follow-up are the highest-priority research need.
2. **Core Outcome Set Development:** Consensus adoption of a GBS Core Outcome Set (COS) incorporating cognitive, psychological, fatigue, pain, sleep, and social participation domains developed through COMET initiative methodology involving patient and clinician representatives would enable cross-study comparability and future meta-analysis.
3. **Biomarker Discovery:** Neuroimaging (DTI, resting-state fMRI), blood-based (neurofilament light chain, inflammatory cytokine panels), and electrophysiological biomarkers predictive of non-motor recovery trajectories would enable precision rehabilitation prescribing.
4. **Telerehabilitation Efficacy Trials:** RCTs evaluating digitally delivered multidisciplinary rehabilitation programmes are needed to address access inequity with particular attention to outcomes in low- and middle-income country contexts.
5. **Long-Term Prospective Registries:** Prospective registries with minimum 5-year follow-up incorporating validated patient-reported outcome measures across all ICF domains are required to characterize decade-long multidimensional disability trajectories.
6. **Health Economic Analysis:** Cost-effectiveness analyses comparing multidisciplinary versus standard rehabilitation models are needed to inform commissioning decisions and demonstrate the economic case for comprehensive GBS neurorehabilitation.
7. **Paediatric and Global South Research:** Dedicated studies in paediatric GBS populations and in low-resource settings are required to address the current evidence base's narrow demographic representativeness.

15. CONCLUSION

Guillain-Barre syndrome is a condition whose rehabilitation demands exceed what any muscle-centric paradigm can adequately address. The evidence synthesized herein establishes that fatigue, cognitive impairment, depression, anxiety, PTSD, neuropathic pain, autonomic dysfunction, sleep disturbance and compromised social reintegration are not peripheral concerns in GBS recovery they are central determinants of long-term functional outcomes and health-related

quality of life that affect the majority of survivors, including those achieving complete motor recovery.

The ICF framework provides the conceptual architecture for a paradigm-transforming approach to GBS rehabilitation one that systematically assesses and intervenes across body function, activity, participation, and contextual factors. Multidisciplinary teams incorporating neuropsychology, clinical psychology, occupational therapy, speech-language pathology, social work, and peer support alongside traditional physical rehabilitation represent the structural realization of this framework.

We advocate for the routine implementation of domain-specific screening protocols initiating at hospital discharge, individualized ICF-based goal-setting, and systematic follow-up extending to at least 24 months post-onset. The investment in this broader rehabilitation vision is justified by the epidemiological reality that GBS predominantly affects working-age adults whose full social and occupational reintegration is an achievable goal but only if rehabilitation looks beyond the gait laboratory and addresses the whole person.

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