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ISBN 978-9969-589-98-6

March 2026

Optimizing the management of Guillain-Barré syndrome

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Introduction

Guillain-Barré syndrome (GBS) refers to a group of acute autoimmune polyradiculoneuritis, i.e. rapidly onset inflammatory neuropathies affecting the spinal roots and peripheral nerves simultaneously, and usually manifested by progressive, bilateral and relatively symmetrical weakness. It is the leading cause of acute flaccid paralysis globally since the near eradication of poliomyelitis, with heterogeneous clinical expression that reflects the diversity of its immunopathological mechanisms and antigenic targets within the peripheral nervous system (Bellanti & Rinaldi, 2024; Shahrizaila et al., 2021).

From a nosological point of view, GBS is defined less by a single clinical phenotype than by a common evolutionary framework: acute or subacute onset, progression over a few days to a few weeks, immunomediated involvement of the peripheral nerve, then variable recovery after a plateau phase. This syndrome unit nevertheless covers demyelinating subtypes, where the myelin sheath is the main damaged structure, and axonal forms, where the motor or sensory-motor axon is the dominant target; This distinction, which will be detailed later in the book, has major prognostic and pathophysiological implications (van Doorn et al., 2023; Bellanti & Rinaldi, 2024).

GBS represents a neurological and medico-organizational emergency, not only because of the risk of severe motor deficit, but also because of potentially life-threatening complications such as respiratory impairment, dysautonomia, and swallowing disorders. About a third of patients require mechanical ventilation in the most severe forms, contemporary mortality remains close to 5%, and up to a fifth of survivors retain a significant limitation of independent gait at one year, which immediately underlines the need for an integrated reading of GBS combining immunological foundations, evolutionary trajectories and functional consequences (Bellanti & Rinaldi, 2024 ; Shahrizaila et al., 2021).

Issue of diagnostic temporality. In the introduction to a book devoted to GBS, it is useful to recall that the value of the diagnosis lies as much in the identification of the syndrome as in the immediate estimation of its rate of evolution and its potential for visceral failure. In other words, an initially unspectacular form can be the result of intensive respiratory or autonomic monitoring within a few hours, which justifies considering GBS from the outset as a disease with serial reassessment rather than a diagnosis fixed once and for all (van Doorn et al., 2023; Leonhard et al., 2024).

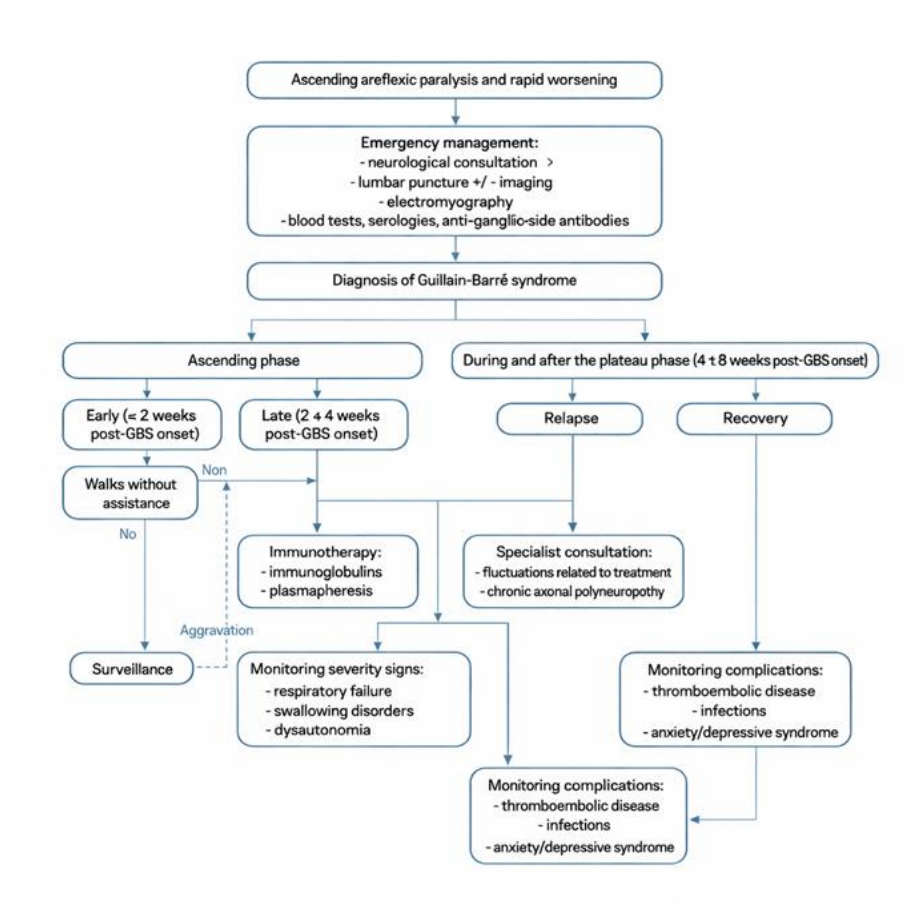

Figure 1. Summary of the initial clinical issues of Guillain-Barré syndrome

1 Epidemiology

GBS is a rare disease, but its health burden is disproportionate to its incidence due to its frequent initial severity, possible use of critical care, and residual functional burden. Contemporary syntheses converge towards an overall annual incidence close to 1 to 2 cases per 100,000 person-years, with a recent meta-analytic estimate of 1.12 cases per 100,000 person-years, while the population series of Europe and North America usually report values between 0.81 and 1.91 cases per 100,000 person-years; this variability is partly due to methodological differences, but also to regional exposure to triggering infections, the quality of diagnostic identification and the distribution of electroclinical subtypes (Xu et al., 2024; Wachira et al., 2023; Shahrizaila et al., 2021).

The epidemiology of GBS must be interpreted in the light of its most often post-infectious character. In nearly two-thirds to three-quarters of cases, a respiratory or gastrointestinal infectious episode precedes the neurological onset, lasting from a few days to a few weeks. However, the actual frequency of triggers varies by region, by clinical or serological documentation methods and by time period, which explains why incidence data cannot be dissociated from infectious exposure patterns or local health contexts, especially in low- and middle-income countries, where the burden of infection and constraints in access to care modify both the risk of occurrence and the conditions of infection. management (Leonhard et al., 2022; Papri et al., 2021).

1.1. Age and sex distribution

The incidence of GBS increases markedly with age. Cohort data and meta-analyses show a gradual increase in risk from adulthood, with peak rates in elderly subjects and, in several series, a peak beyond the age of 70. In the recent global meta-analysis, the mean incidence was approximately 0.48 cases per 100,000 person-years in subjects aged 0 to 18 years, compared with 1.59 cases per 100,000 person-years in subjects aged 70 years or older; in the journal Lancet, the risk increased by about 20% per decade of age. This age structure suggests the combined effect of immunosenescence, accumulation of infectious exposures and increased host vulnerability to peripheral immunological aggressions (Xu et al., 2024; Shahrizaila et al., 2021).

A male predominance is observed in a remarkably constant manner. Recent global estimates place the incidence at around 1.38 cases per 100,000 person-years in men, compared to 0.99 in women, and data from the Chinese urban population find the same gradient, with an incidence ratio close to 1.4. The reasons for this sexual asymmetry are not fully elucidated; They could associate differences in exposure to certain infectious agents, hormonal modulation of the immune response and biological susceptibilities that are still imperfectly understood. On a practical level, this male predominance does not in itself modify the diagnostic strategy, but it must be integrated into the interpretation of comparative series and prognostic models (Xu et al., 2024; Gorson, 2025; Shahrizaila et al., 2021).