Int. j. adv. multidisc. res. stud. 2026; 6(3):572-573

Received: 26-04-2026
Accepted: 06-05-2026

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

Reversible Conduction Failure in Guillain-Barre Syndrome Suggests Immune Nodopathy, Requiring Contactin and Neurofascin Antibody Determination

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DOI: https://doi.org/10.62225/2583049X.2026.6.3.6324
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Letter to the Editor

Yang *et al*.'s article on a retrospective study of the initial clinical and electrophysiological characteristics of 47 patients with Guillain-Barre syndrome (GBS), 19 of whom were diagnosed with acute inflammatory demyelinating polyneuropathy (AIDP), 18 with axonal degeneration (AMAN/AMSAN), and 19 with reversible conduction failure (RCF) is remarkable [1]. AIDP patients had higher cerebrospinal fluid (CSF) proteins than RCF and AMAN/AMSAN patients, and RCF patients had lower Hughes function scores than AMAN/AMSAN patients at admission and discharge [1]. It was concluded that RCF patients are easier to distinguish from AIDP patients than RCF patients from AMAN/AMSAN patients [1]. However, some inconsistencies need to be clarified.

Firstly, the RCF subtype of GBS is not frequently reported in previous publications. There are only few published studies on GBS with rapid clinical and electrophysiologic recovery. There is also no consensus on the definition of RCF. According to Chan *et al*., RCF is defined as 1. a 50% increase in the distal compound muscle action potential (CMAP) or sensory nerve action potential (SNAP) amplitude or as 2. the disappearance of a motor conduction block (MCB) with simultaneous shortening of the distal latency or the CMAP duration and acceleration of the conduction velocities [2]. According to Uncini *et al*. RCF is defined as 1. a continuum between transient MCB and axonal degeneration, 2. MCB caused by paranodal myelin detachment, nodal elongation, sodium channel dysfunction, disturbed water and ion homeostasis or abnormal axolemma polarisation, 3. rapidly reversible MCB without temporal dispersion, and 4. axonal degeneration caused by MCB, depending on the type and severity of the underlying disease [3]. An additional criterion for the definition of RCF could be the speed of improvement of RCF. Both axonal degeneration and segmental demyelination can regress or improve, but do so much slower than RCF. Furthermore, point 4 of the Uncini definition is not RCF itself, but rather a negative consequence of the same pathology that has reached a stage where it is no longer rapidly reversible and axonal degeneration has occurred. The authors used Uncini's criteria, but it would be interesting to know whether the 19 included patients with RCF also met Chan's diagnostic criteria.

Secondly, RCF is considered an indicator of a nodopathy [4]. Nodopathies are autoimmune disorders, of which four are currently distinguished: nodopathy with antibodies against 1. anti-contactin-1, 2. anti-contactin-associated protein-1, 3. anti-neurofascin-155 and 4. anti-pan-neurofascin (neurofascin 186) [5]. Since the initial description, more recent studies have expanded the clinical presentation of immune nodopathies. New IgG subclasses (e.g. IgG1/IgG3) have been discovered, particularly in acute anti-pan-neurofascin antibody disease (Fig 1) [6]. Experimental and clinical studies have confirmed the antibody-mediated pathogenicity of these IgG subtypes [6]. Different pathophysiological mechanisms are thought to be responsible for the action of antinodal antibodies, leading to a number of unique clinicopathological features [6]. The clinical profile and treatment of nodopathies may also vary depending on the antibody isotype [6]. B-cell-depleting therapies have been shown to be effective in some of these subtypes of nodopathy [6]. In view of these considerations, we should know whether the 10 patients with RCF were positive for antinodal or paranodal antibodies.

In summary, RCF in GBS indicates an immunonodopathy, which is why antibodies against contactin and neurofascin should be determined in RCF patients.

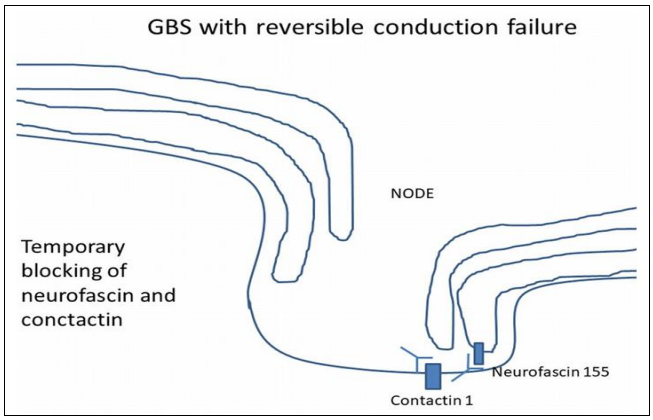

Fig 1: Action of contactin-1 and neurofascin-155 in reversible conduction failure

Declarations

Ethical Approval: Not applicable.

Consent to Participation: Not applicable.

Consent for Publication: Not applicable.

Funding: None received.

Availability of Data and Material: All data are available from the corresponding author.

Completing Interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author Contribution: JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. SM: contributed to literature search, discussion, correction, and final approval.

Acknowledgements: None.

Keywords: Guillain Barre Syndrome, Reversible Conduction Failure, Nodopathy, Neurofascin, Contactin

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