

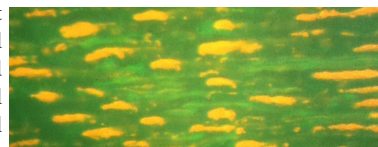


Giovanni Antioco Putzu

Casa di Cura Sant'Elena, Quartu Sant'Elena, Italy

Guillain-Barre Syndrome (GBS) and GBS-like Syndrome: Clinical, neuropathological and immunological correlations

GBS is an auto-immune life-threatening inflammatory polyneuropathy that may produce severe functional disability. Vaccines, viral, fungine or bacterial infections may trigger the disease. On the basis of neuropathological and neurophysiological findings, GBS is classified in demyelinating and axonal forms. In both features, functional disability is directly correlated to axonal loss. Involvement of amyelinic axons is responsible for autonomic disturbances, which, along with bulbar spread of the disease, represent a potential cause of death in GBS. A consistent number of patients both in the early or recovery phases may complain of neuropathic pain that requires an adequate treatment. Immunological aspect of the disease, i.e auto-antibodies directed against GM1 (Gangliosidosis) and recently to contactin-associated protein 1 (Caspr) of the paranodal region of myelinated nerves, have already been investigated. We have demonstrated that TNF-alpha (Tumor Necrosis Factor) was immunolocalized in both myelinated and unmyelinated axons the sural nerve of GBS patients. We concluded that this substance may be directly responsible for axonal loss(G.A. Putzu et al, J. Neurol Sci, 2000).



Immune localization of TNF-alpha on myelin and axons in the sural nerve of acute GBS, double immunostaining technique, longitudinal section, x 400

Interferon-gamma, which is a stimulator of IL28A was also easily detected in the sural nerve of GBS patients. The role of adhesion molecules like ICAM in the immune process of GBS will be also discussed. The therapeutic approach of GBS is aimed to avoid death in the acute phase (respiratory failure in Landry paralysis, cardiac rhythm anomalies in disautonomia). The efficacy of plasmapheresis and intravenous immunoglobulins in the treatment of GBS is nowadays clearly demonstrated. The next frontier is the theoretical possibility to use monoclonal antibodies (i.e, anti-INF Interferon-gamma) as a therapeutic tool in GBS.

We also reviewed the literature on GBS-like conditions that may clinically mimic GB.

Biography

The Author is a Medical Doctor since 1992, with specialization in Paediatric Neurology. He achieved his PhD in 1996. During PHD studies, He was a Research Fellow in Hammersmith Hospital of London, UK in 1992, then He moved to Marseille (France) to work at INSERM (Genetics) and in Neuropathology. The Author has published more than 15 papers in the field of Neuromuscular Disorders.

puzzugio@gmail.com