Giovanni Antioco Putzu, J Neurol Neurophysiol 2016, 7:4 (Suppl)
http://dx.doi.org/10.4172/2155-9562.C1.033

8th European Neurology Congress

September 21-23, 2016 Amsterdam, Netherlands

Giovanni Antioco Putzu

Casa di Cura Sant'Elena, Italy

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

GBS is an auto-immune life-threatening inflammatory polineuropathy 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 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 was immunolocalized in both myelinated and unmyelinated axons the sural nerve of GBS patients. We concluded that this substance may be directly responsible for axoal loss(G.A. Putzu et al, J. Neurol Sci, 2000). 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 plasmapheris and intravenous immunoglobulins in the treatment of GBS is nowadays clearly demostrated. The next frontier is the theoretical possibility to use monoclonal antibodies (i.e, anti-INF-gamma) as a therapeutic tool in GBS. We also rewied the literature on GBS-like conditions that may clinically mimick GBS.

Biography

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

puzzugio@gmail.com

Notes: