GUILLAIN-BARRE SYNDROME IN A PATIENT WITH CHRONIC LYMPHOCYTIC LEUKAEMIA. CASE REPORT

S. CEPPARULO, M.P. PRUDENZANO, A. MANNI, I. TEMPESTA, E. D'ERRICO, B. BRANCASI, I.L. SIMONE, P. LIVREA

Neurology unit, Department of Basic Medical Sciences, Neurosciences and Sense Organs
University of Bari "Aldo Moro", Italy

Background: Guillain-Barre Syndrome (GBS) is a paralytic demyelinating disorder of the peripheral nervous system characterized by massive lymphocytic infiltration and damage of peripheral nerves myelin sheath. We describe a case of GBS associated with Chronic Lymphocytic Leukaemia (CLL).

Case report: A 58-year-old man with a history of CLL, hypertension and obesity, presented progressive weakness at lower limbs, with onset two weeks before our observation and already treated with Betamethasone low dosage without any improvement. There was no history of recent viral or bacterial infection. CLL had been diagnosed about 2 years previously (Binet A, Rai II), for which he did not receive any treatment. At the time of admission he additionally complained of chest, dorsal and bilateral leg pain. Lower extremity reflexes could not be elicited and Babinski sign was absent. His tactile sensibility was slightly impaired, while pallesthesia was severely damaged at four limbs. He was restricted to wheelchair and presented urge incontinence. His complete blood count was consistent with CLL and other laboratory results were normal, included anti-ganglioside antibodies. Electromyography and nerve conduction study detected severe and diffuse demyelinating-axonal sensorimotor damage with rare acute denervation findings in one of the explored sites, most consistent with GBS. The CSF analysis showed a mild blood barrier damage (AQ= 12,3), normal glucose ratio and cell count; no oligo-clonal bands were detected. A diagnosis of GBS was performed and confirmed by a sural nerve biopsy. He underwent intravenous immunoglobulin treatment (0,4 gr/kg/die for five consecutive days). His clinical status improved immediately after the treatment, with a gradual and continuous improvement in symptoms.

Figure 1.Pathogenesis of GBS

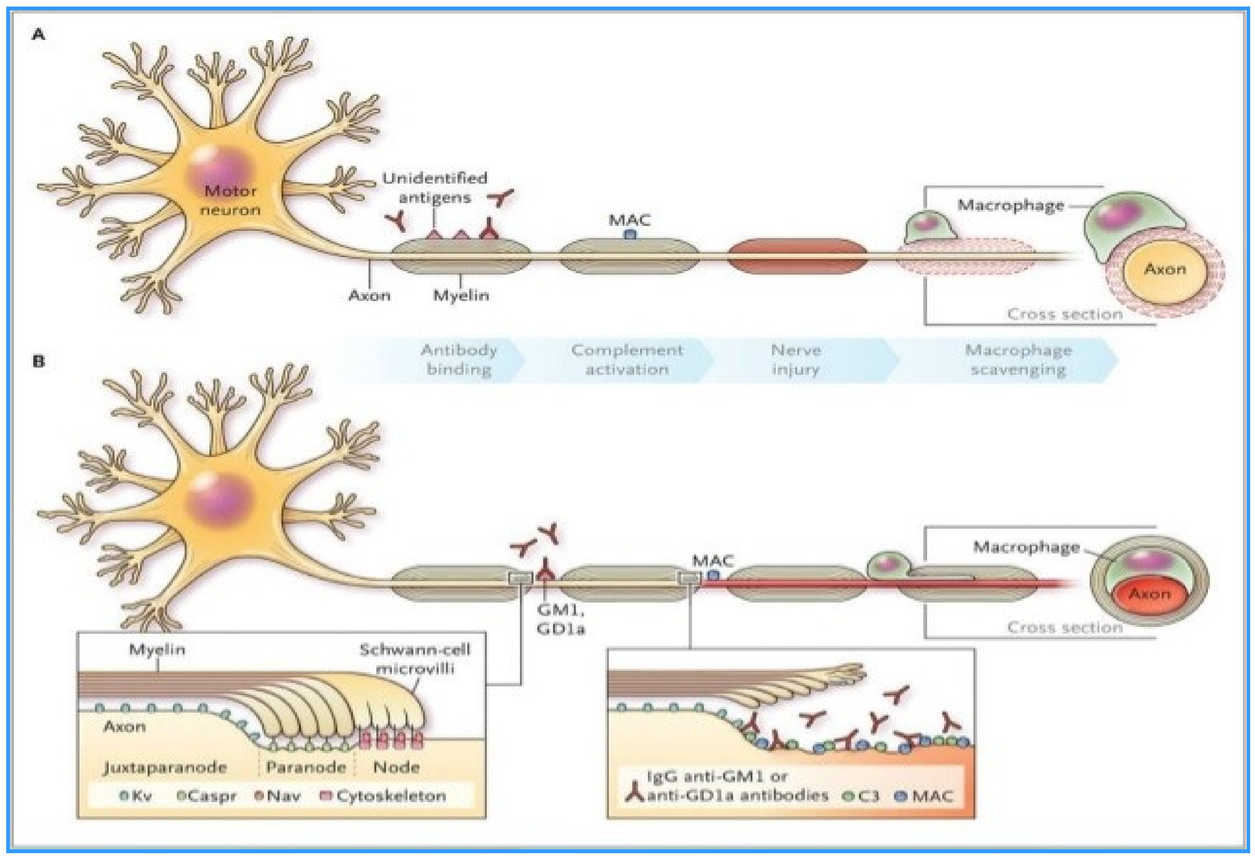
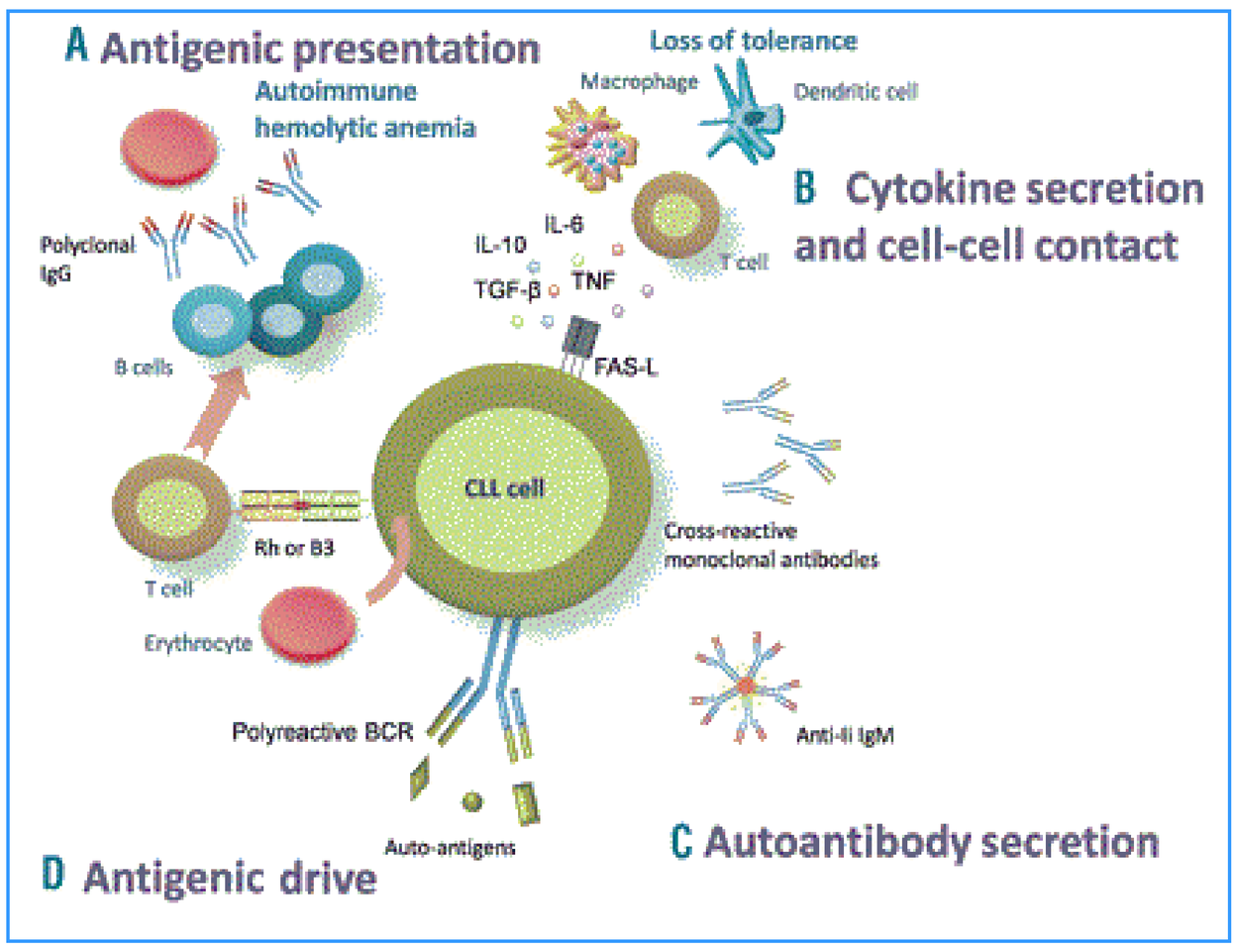


Figure 2.Mechanisms of autoimmune disease in CLL.



Discussions: CLL is associated with systemic autoimmunity, but neurologic autoimmune phenomena have been rarely described. The majority of previously described cases has been reported after treatment with Chlorambucil, but whether the occurrence of GBS was directly related to CLL or to a viral reactivation due to immunosuppression was undefined. In our case the patient was free of immunosuppression therapy so we can't relate the event to the activation of an antigen-specific T and B-cell clone during drug induced aplasia. We speculate an aberrant immune response directed against some components of the peripheral nerves due to an inefficient self tolerance during CLL (***Fig 2***).

Sin

XLVII CONGRESSO NAZIONALE
22-25 OTTOBRE 2016 – VENEZIA

WebPoster