Immunosuppresive/immunomodulating treatment in autoimmune limbic encephalities - when to stop? Based lab data.

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Over the last twenty years it has become clear that there are central nervous system disorders that are mediated by antibodies to neuronal or glial proteins. These disorders usually have a subacute onset and are associated, variably, with MRI, EEG and CSF evidence of neuroinflammation. Antibodies binding to the surface of neuronal molecules (e.g. LGI1, NMDAR) are diagnostic in forms of autoimmune encephalitis, and the patients benefit considerably from immunotherapies. In some cases, however, the response is slow or disappointing and the question is how to decide whether to escalate, continue or discontinue the potentially risky treatments with their many associated side-effects. Although all centres are dependent on their local laboratories measuring antibodies for diagnosis, many clinicians express doubts about the usefulness of measuring antibody levels during management. I will argue that this is the result of laboratory assays geared to diagnosis, but not geared to follow-up, and that accurate measurements can be very helpful in patient management. I will illustrate the results with examples of carefully-titred sera over months or years for different antibodies, and the relationship with clinical features. I will show how useful these titres can be in determining the effects of immunotherapies, and the need for further treatments.