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Brain grieves when the gut sobs-observational case series of neuroimmunological disease relapses associated with leaky gut secondary to food sensitivities-an Indian neurology tertiary care centre experience

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Abstract

Environmental triggers are the major contributors to neuroimmunological diseases. Salient yet neglected trigger is diet. Gut microbial vexation and intestinal barrier tumult predispose to leaky gut, perturbing the gut brain axis. This can trigger either development of novel autoimmune neurological diseases or their relapse if condition is pre-existent. Patient tailored menu engineering, diet leverage in clinical practice and improvisation research bridges this gap.

Keywords: Leaky gut, neuroimmunological disease relapse, gut brain axis, food sensitivity

Introduction

Food sensitivity appears benign but is a validation of underlying immune-dysfunction. IgG microarray quantitative antibody test is a precise method of detecting chronic food sensitivity and must be incorporated in all neuroimmunological patients requiring holistic management as immunosuppression alone is insufficient to prevent relapses. Gut brain axis theory as the pathophysiological basis in neuroimmunological diseases is emphasized in our study. Large sample size is required to substantiate our findings and to identify specific food triggers and guide holistic approach towards neuroimmunological diseases.

Objectives

1. Observe clinical profile, disease pattern, common food epitopes and management of patients with dietary triggers causing relapse in demyelinating diseases.
2. Highlight implementation of leaky gut syndrome theory in clinical practice via food intolerance test and patient tailored diet.
3. Emphasize on implementation of basic science pathophysiological mechanisms & research in clinical practice for better patient management.

Design and Methods

Our observational case series compared diet and demyelinating diseases. Chronic food intolerance was detected by protein microarray IgG antibodies quantification for 215 common food epitopes (Different from the acute food allergy test which quantifies IgE antibodies). Baseline food intolerance test with respective patient tailored diet was prescribed for all. Patients who had disease relapse precisely attributable to dietary violations (Elimination diet and re-challenge) while on compliant immunosuppression were included in the study. Relapse assessment methods were comparison of worsening of clinical scores and other objective parameters, number of disease attacks and radiological relapses

Results

Eight patients (7 males, 1 female) with age range of 6-68 years were followed up for a mean duration of one year (1 year-2 years).

Profile included 1 seronegative LGI1-like encephalitis, 3 cases of MS, 2 combined central and peripheral demyelination (CCPD), 1 Myasthenia Gravis & 1 limbic encephalitis with VGCC antibodies.

Out of these, 6 patients were intolerant to dairy & almonds, 3 patients to peas and yeast, 2 to peanuts, egg white and flaxseeds. One patient had no food sensitivity. First episode of demyelinating disease occurred with variable duration of exposure to intolerant foods (6 months to one year). Clinical relapse was noted within 72 hours-2 weeks of consistent exposure to prohibited intolerant foods. All patients required either rescue immunosuppressant or dose escalation of maintenance immunosuppression to tide over the crisis along with avoidance of intolerant foods.

Conclusions: Food intolerance appears benign but is a validation of underlying immune-dysfunction. IgG microarray quantitative antibody test precisely detects chronic food intolerance. Incorporation of this in all neuroimmunological patients provides holistic management as immunosuppression alone might be insufficient to prevent relapses. Gut brain axis pathomechanism in neuroimmunological diseases- both CNS and PNS demyelination, is emphasized in our study. Larger case control studies are required to substantiate our findings, identify specific food triggers & guide holistic approach. Potential benefits of food sensitivity testing are as follows:

1. Tailor individual diet program
2. Reduce clinical relapses
3. Paves way to restore gut microflora and fix leaky guts

Limitations

Concept of antibody mediated food sensitivity is controversial.

Some studies found no relation between food specific immunoglobulins and perceived food sensitivity.

IgG is produced against all staple foods (foreign bodies) IgG are of 2 types, pro and anti-inflammatory IgGs and commercial tests can't distinguish the two.

Lack of reliability and less sensitivity are other limitations.

It is not used for diagnostic purpose

Future Research Avenues

Large scale and multicentre global research in AI diseases to include food sensitivities and leaky gut concept and its practical implementation for holistic management.

Avenues to manipulate gut microbiome to treat food sensitivities and reduce AI relapses or severity.

Potential for new drug development* Zonulin inhibitor- prevent disruption of intestinal barrier function- reduces leaky gut- Larazotide acetate (AT-1001)* Zonulin agonists and analogues- reversibly increases paracellular drug delivery- absorptive enhancers.

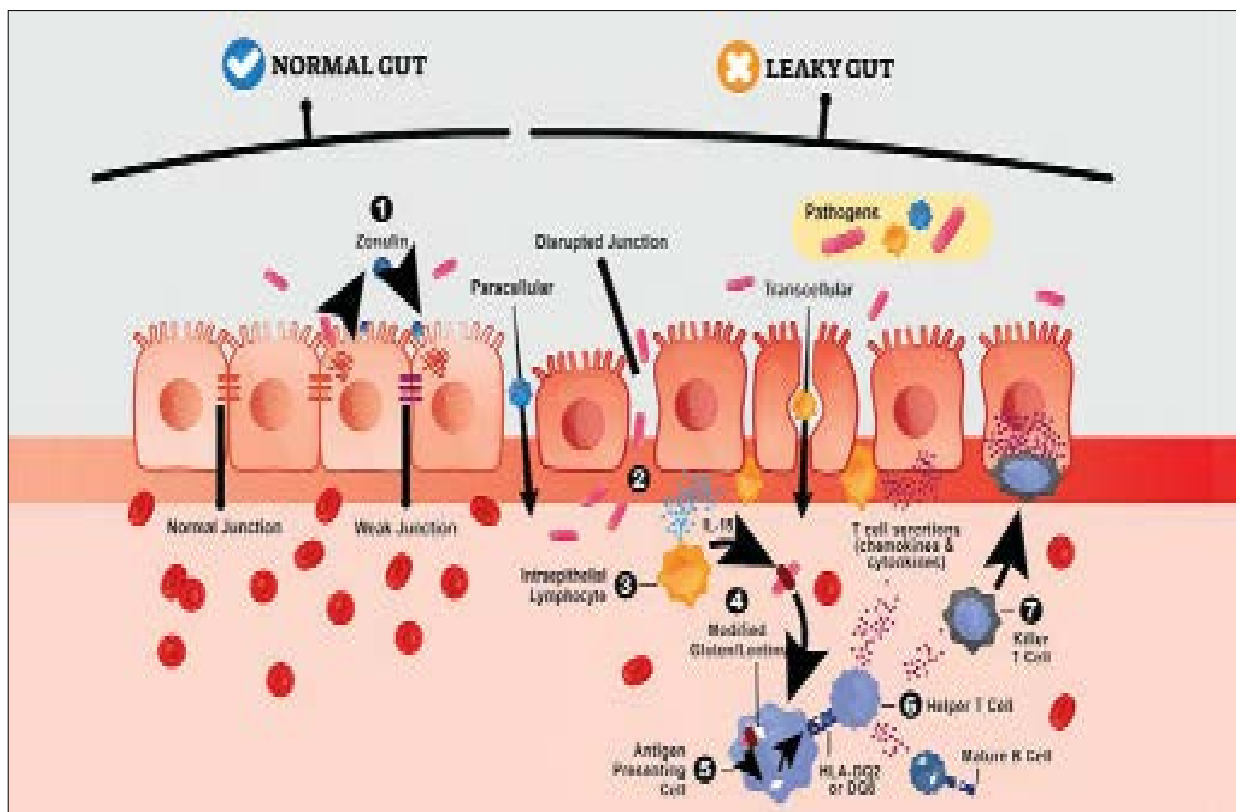


Fig 1: Leaky gut

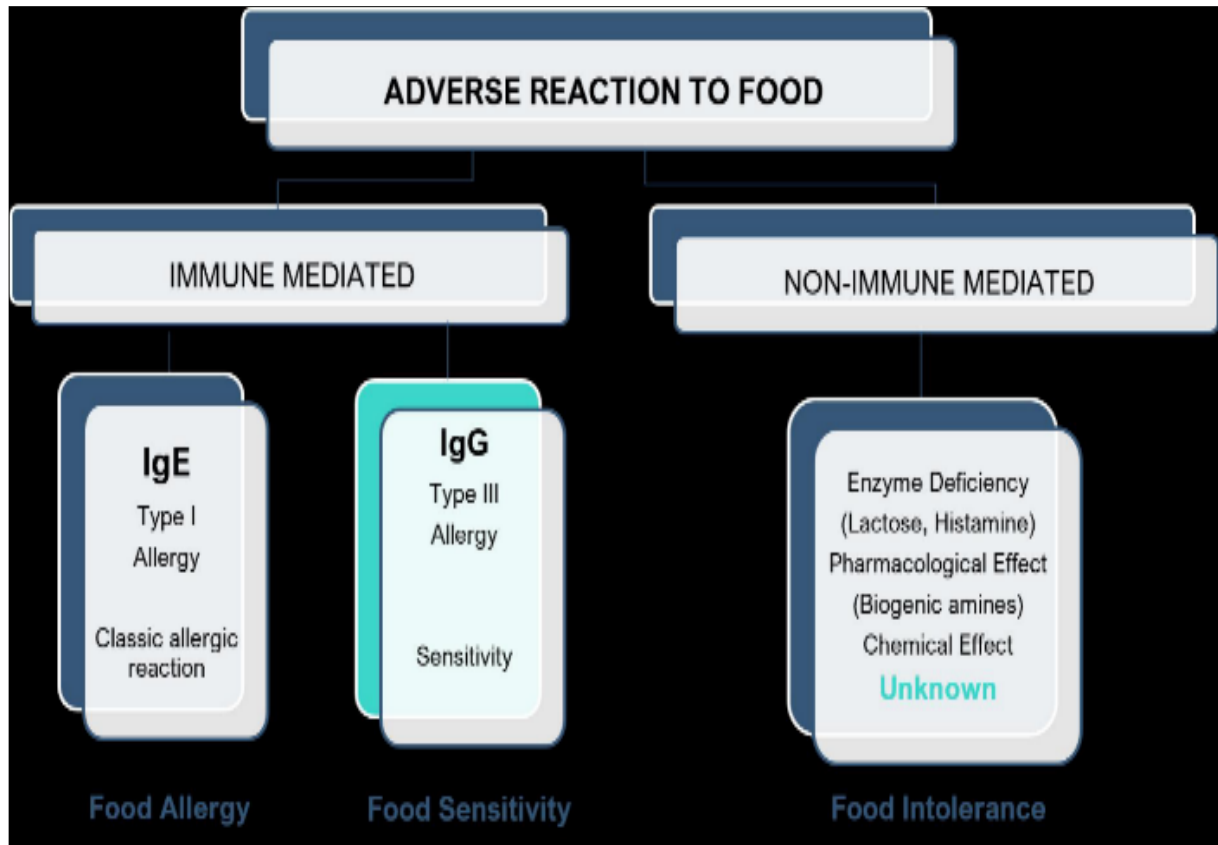


Fig 2: Good allergy, sensitivity and intolerance

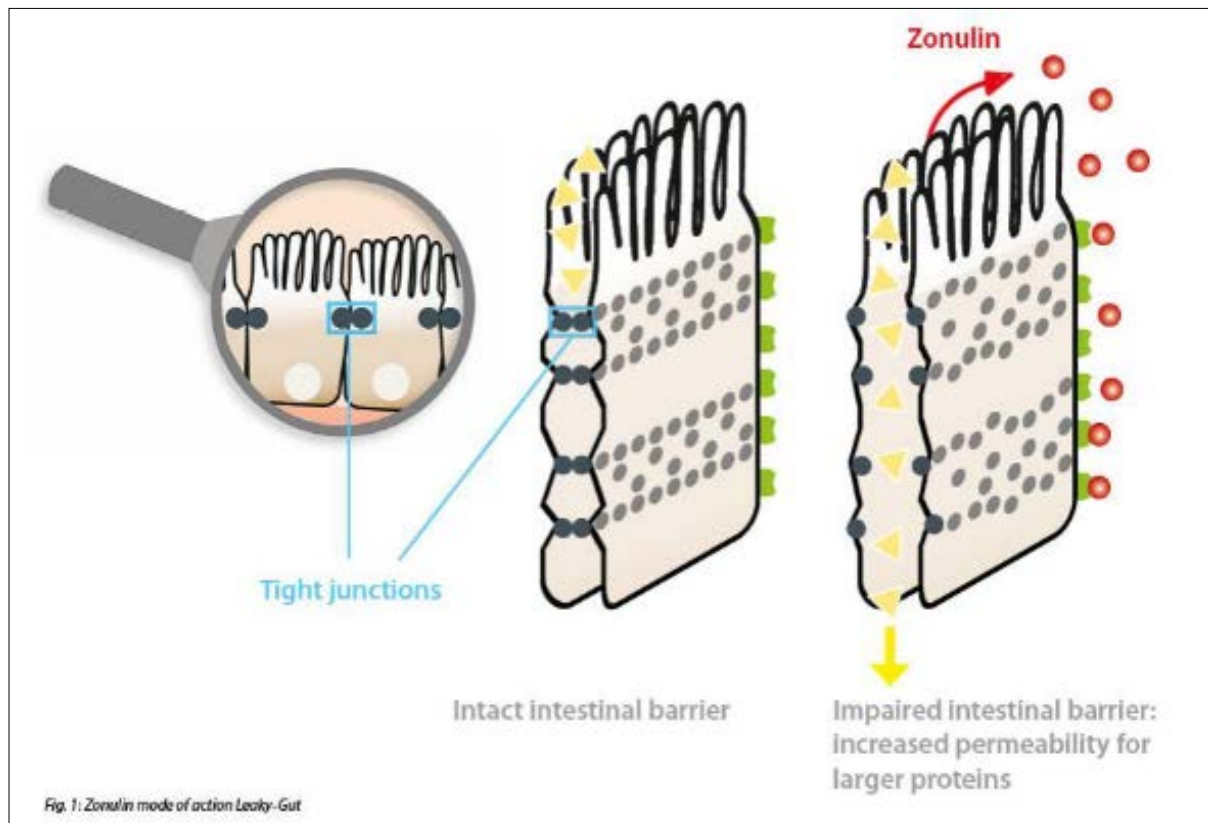


Fig 3: Zonulin microarchitecture

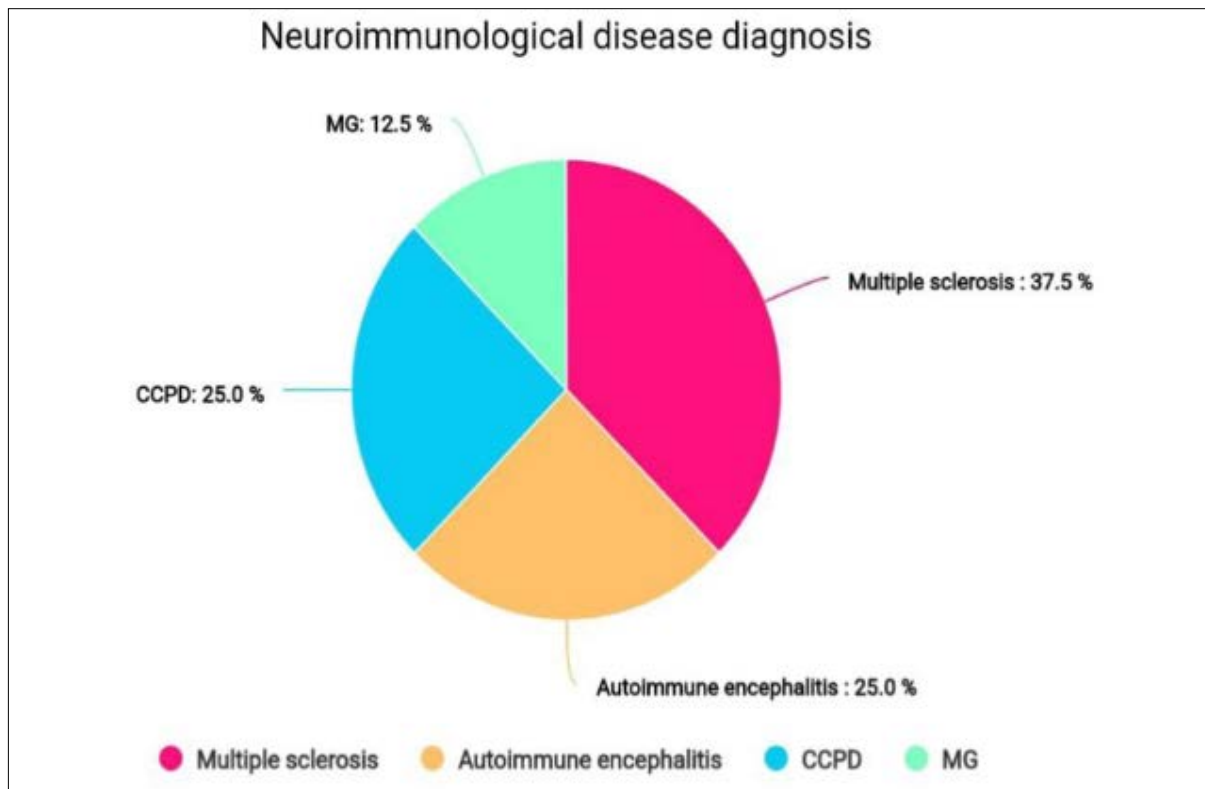


Fig 4: Diagnosis and ethology of autoimmune neurological diseases in our patients

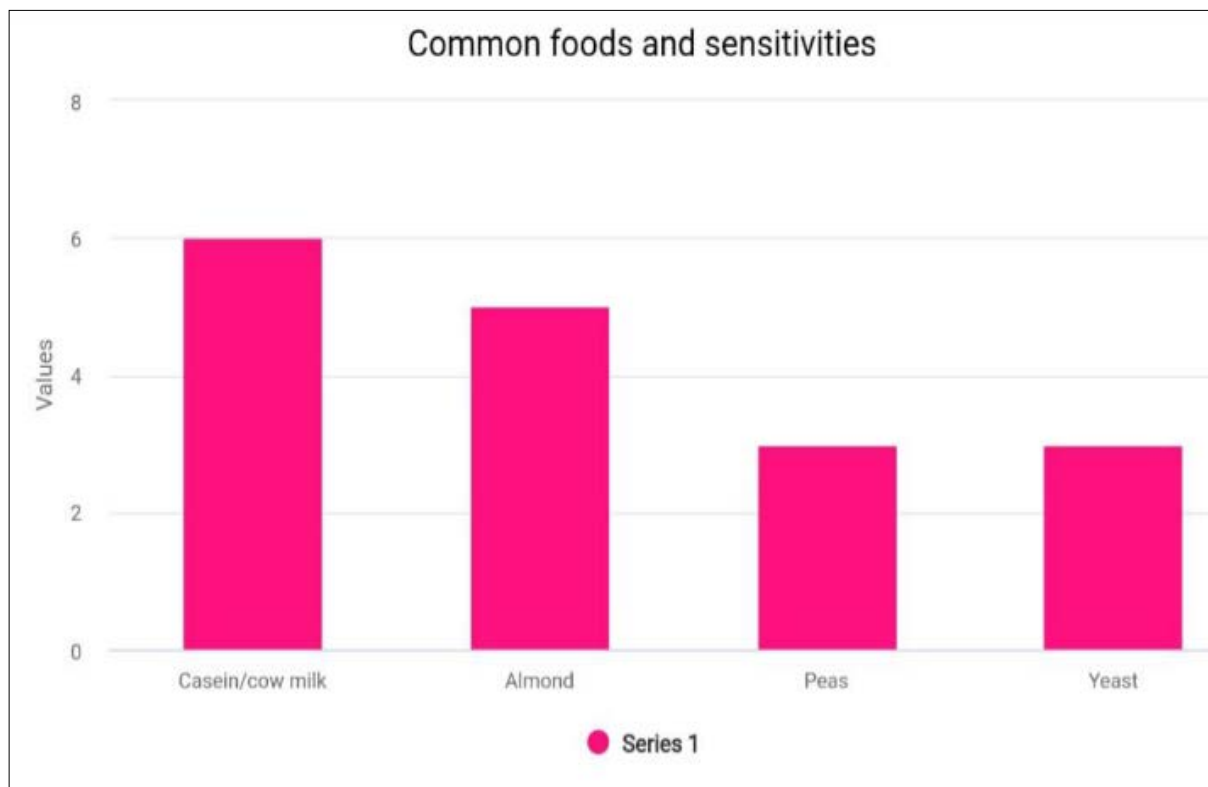
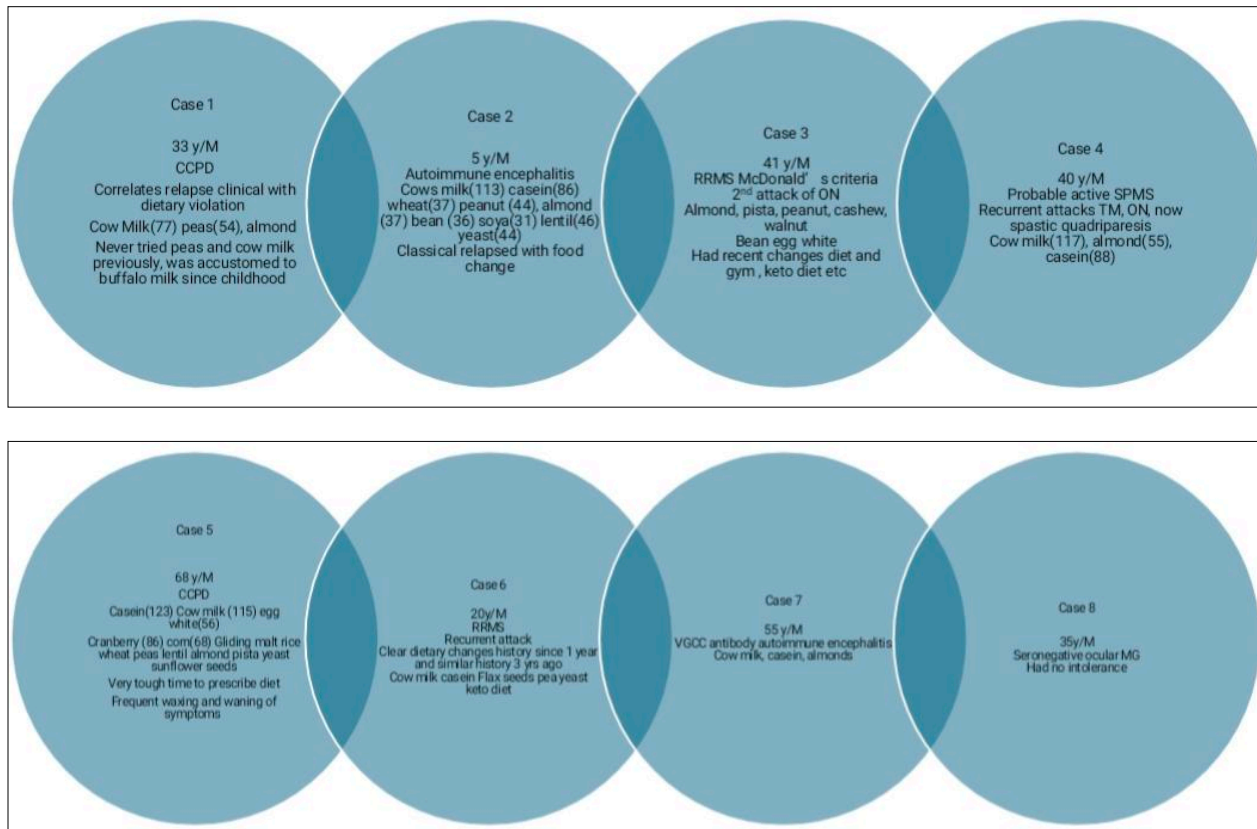


Fig 5: Common foods and sensitivities

**Fig 6:** Case reports**Disclosures and conflict of interest**

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