and 1 with multiple cranial neuropathy as compared with 88 T1DM patients positive for anti-GAD antibodies.

Results: All anti-GAD antibody-positive neurologic disorders patients had elevated serum anti-GAD antibody titers (1,440 to 270,000 U/mL: normal ≤1). In most of T1DM patients, the anti-GAD antibody titers were between 1.0 and 100 U/mL. Five of six neurologic disorders patients were performed CSF analyses, and CSF anti-GAD antibody titers were also increased (30-770 U/mL). The anti-GAD antibody index in the four of five patients reviewed was >1.0 suggesting intrathecal synthesis of anti-GAD antibody. Three of six patients were associated with neoplasm (breast cancer, thyroid cancer, thymoma). Five of six patients were treated with immunotherapy, and showed clear effectiveness. The anti-GAD antibody titers of T1DM patients decreased over time. However, those of neurologic disorders patients tended to remain at a high level, and some cases showed re-elevation.

Conclusion: Neurologic disorders associated with anti-GAD antibodies were improved by immune therapy, and anti-GAD antibody titers and the anti-GAD antibody index would be correlated with disease activity and indicators of therapeutic evaluation.

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SHIFT 7 - AUTOIMMUNE DISORDERS

Gestational hypothyroxinemia increases permeability of the blood brain barrier and facilitates the early development of experimental autoimmune encephalomyelitis in the offspring

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Background: Hypothyroxinemia (HTX) is an asymptomatic condition characterized by low levels of plasmatic tetraiodothyronine (T_4). Gestational HTX has been largely associated with impaired neurological development in the progeny. Observations from our laboratory show that mice gestated in mothers with HTX develop earlier and more severe experimental autoimmune encephalomyelitis (EAE). Interestingly, these mice have higher basal levels of IL-17 and TNF-alpha in serum than controls, both of which are cytokines known to increase blood brain barrier (BBB) permeability.

Objective: To analyze the BBB permeability in adult mice gestated in HTX mothers.

Patients and Methods/Material and Methods: Maternal HTX was induced in pregnant mice by transient treatment with methimazole. Evans blue dye was used as a marker of BBB permeability to albumin. BBB permeability to immune cells was analyzed by adoptive transfer assays in mice under non-inflammatory and EAE conditions. Leukocytes were isolated from the central nervous system at different time points and analyzed by FACS.

Results: Maternal HTX offspring showed increased BBB permeability to albumin and immune system cells under non inflammatory and EAE conditions.

Conclusion: Our results support the idea that gestational HTX may be a risk factor for early development of EAE in offspring, an idea that could be confirmed in later studies conducted in children born to mothers with HTX. These findings emphasize the importance of screening for HTX in pregnant women, developing new approaches of treatment and prevention of complications in the progeny.

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SHIFT 7 - AUTOIMMUNE DISORDERS

Use of GnRH analog leuproreline as treatment of secondary progressive multiple sclerosis motor symptoms

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Background: Over 6 years ago Quintanar et al. showed that in EAE models, NFkB, IL-1B, IL-17, and TNFa were diminished by leuprolide acetate, a GnRH analog. GnRH receptors are expressed on motoneurons surface and its analogs lead to improvement of clinical signs of locomotion, axonal morphometry and diameter. Elevated neurofilament as well as myelin basic protein expression can be obtained by leuprolide administration. Increased regulatory T cells infiltrates have been reported with leuprolide acetate following EAE and could be part of the improvement of symptoms.

Objective: To prove that GnRH analog leuproreline is a promising therapy for improving MS motor symptoms.

Patients and Methods / Material and Methods: A 41 years old, hispanic male, with 15 years of MS, treatment naive, only taking vitamin D who presented with worsening in motor declined over the past months; unable to swallow liquids, bedridden with severe dysarthria, dysphagia with dystussia, bilateral horizontal gaze palsy, motor strength in upper extremities 2/5 bilateral, lower extremities 0/5, severe truncal ataxia. EDSS was 9.5

As large lesion burden was located at pons and cervical cord, GnRH analogs where proposed as a possible treatment to decrease EDSS. I obtained patient and proxy approval as necessary.

Results: After leuproreline 7.5mg IM q month, for 3 months, horizontal gaze, head stability and truncal ataxia improvement allowed wheelchair use most of the day, and upper motor extremities strength became 4/5 right and 3-4/5 left as well as improvement on dysarthria, dysphagia and microaspirations. EDSS was 8

Conclusion: We propose that GnRH analogs are promissing therapetic agents to improve MS motor symptoms.

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SHIFT 7 - AUTOIMMUNE DISORDERS

Ca(2+)-dependent anti-ganglioside antibodies in seronegative AMAN, AIDP, and SAN

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Background: We have reported Ca(2+)-dependent IgG anti-GQ1b antibody is present in the majority of GQ1b-seronegative patients with Fisher syndrome and its related disorders (FS-RD). Though the conventional assay for anti-ganglioside antibody is performed in the