Viruses & Autoimmune Disease: The Infection Connection

Viruses

- Viruses consist of a nucleic acid (either DNA or RNA) associated with proteins encoded by the nucleic acid. The virus may also have a lipid bilayer membrane (or envelope) but this is acquired from the host cell, usually by budding through a host cell membrane. If a membrane is present, it must contain one or more viral proteins to act as ligands for receptors on the host cell.

- Since many viruses make few or no enzymes, they are dependent on host cell enzymes to produce more virus particles. Thus, viral structure and replication are fundamentally different from those of cellular organisms.

- Viral dependence on the host cell for various aspects of the growth cycle has complicated the development of drugs since most drugs inhibit cell growth as well as viral multiplication (because the same cell enzymes are used).

- Enveloped viruses do not necessarily have to kill their host cell in order to be released, since they can bud out of the cell - a process that is not necessarily lethal to the cell - hence some budding viruses can set up persistent infections.

Diagnosis

- Any history of autoimmune disease
- Check blood chemistries
- Specific antibodies for each virus
- Chronic fatigue
- Chronic fever of unknown origin
- Swollen lymph nodes
- Symptoms seem to go up and down
Blood Chemistry Patterns

- High WBC during acute phase
- Low WBC during chronic infection
- High lymphocytes and monocytes in chronic infection
- High C-reactive protein, ESR, Fibrinogen
- Check ANA, RF and other autoimmune markers based on clinical findings

Classic Chronic Viral Infection

<table>
<thead>
<tr>
<th>CBC, Platelet Ct. and Diff</th>
<th>Low</th>
<th>x1000/μl</th>
<th>4.9 - 10.5</th>
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<tbody>
<tr>
<td>Hb</td>
<td>4.65</td>
<td>x1000/μl</td>
<td>4.15 - 5.60</td>
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<tr>
<td>Hemoglobin</td>
<td>14.4</td>
<td>g/dl</td>
<td>12.0 - 17.0</td>
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<tr>
<td>Erythroid</td>
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<td>6</td>
<td>36.0 - 50.0</td>
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<tr>
<td>MCV</td>
<td>94</td>
<td>fL</td>
<td>80 - 94</td>
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<tr>
<td>MCH</td>
<td>28</td>
<td>pg</td>
<td>28 - 31</td>
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<tr>
<td>MCHC</td>
<td>31.1</td>
<td>g/dl</td>
<td>30.2 - 34.6</td>
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<tr>
<td>RDW</td>
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<td>6</td>
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<td>Eosinophil</td>
<td>100</td>
<td>x1000/μl</td>
<td>100 - 413</td>
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<td>Neutrophil</td>
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<td>Low</td>
<td>5</td>
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<tr>
<td>Lymphocytes</td>
<td>9</td>
<td>High</td>
<td>14 - 44</td>
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<tr>
<td>Monocytes</td>
<td>4</td>
<td></td>
<td>4 - 12</td>
</tr>
</tbody>
</table>


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A faulty human protein, abnormally phosphorylated tau, was recently publicized to spread "like a virus" from neuron to neuron in Alzheimer patients' brains. For several decades, we have been amassing arguments showing that herpes simplex virus type 1 (HSV-1), not p-tau, propagates this interneuronal, transsynaptic pathologic cascade.

METHODS: We reiterate convincing data from our own (and other) laboratories, reviewing the first anatomic foothold neurofibrillary tangles gain in brainstem and/or entorhinal cortex; the chronic immunosurveillance cellularity of the trigeminal ganglia wherein HSV-1 awakens from latency to reactivate; the inabilities of p-tau protein's physical properties to promote it to jump synapses; the amino acid homology between human p-tau and VP22, a key target for phosphorylation by HSV serine/threonine-protein kinase UL13; and the exosomic secretion of HSV-1-infected cells' L-particles, attesting to the cell-to-cell passage of microRNAs of herpesviruses.

RESULTS: The now-maturing construct that reactivated HSV-1 best accounts for the intracerebral propagation of AD changes in the human brain should at last seem highly attractive. This hypothesis might even explain statins' apparent mechanism in some studies for lowering AD incidence.

CONCLUSION: Provided that funding agencies will quickly ignite a new realm of investigation, the rejuvenated enthusiasm for testing this optimistic construct holds incalculable potential for rapid, efficacious clinical application, through already available and relatively safe antiviral therapeutics.
Role of early viral infections in development of multiple sclerosis.


Multinuclear sclerosis is a clinical manifestation of autoimmune demyelination. Disease is not a degenerative disorder with marked progressive course. Demyelination is located at various depths of brain tissue, including white matter, gray matter, and meninges. The disease progresses with specific inflammatory processes and chronic autoimmunity.

This study included patients (n=106) admitted to the Neurology Clinic in Sarajevo, Bosnia-Herzegovina, with a diagnosis of multiple sclerosis (MS) during the period January 2009-December 2011. To all patients beside history and neurological examination and tests to confirm the MS (brain MRI, evoked potentials and CSF examination) made serological tests for viruses, HSV, Rubella virus, cytomegalovirus and Epstein-Barr's virus, with reference to the previous parameters (old) and new viral infections. The study included patients treated at the Neurology Clinic in Sarajevo, with a diagnosis of multiple sclerosis (newly discovered) in the period January 2009-December 2011. To all patients beside history and neurological examination and tests to confirm the MS (brain MRI, evoked potentials and CSF examination) made serological tests for viruses, HSV, Rubella virus, cytomegalovirus and Epstein-Barr's virus, with reference to the previous parameters (old) and new viral infections.

RESULTS:

In this period there were 118 newly diagnosed multiple sclerosis from which 69.5% (82) female and 30.5% (36) male patients aged 23-56 years. IgG and IgM antibodies to herpes simplex virus, cytomegalovirus and rubella virus.

CONCLUSION:

Early infection by herpes simplex virus, cytomegalovirus, Epstein-Barr and Rubella virus could in certain cases be an initiating factor in multiple sclerosis. Early viral infection, with its characteristic immune response, can be observed in multiple sclerosis patients with a high level of antibodies to certain viruses, which confirms the high titer of antibodies to certain viruses in patients with the MS. To first of all herpes simplex virus, Epstein Barr virus, cytomegalovirus and Rubella virus, which suggests that viral factors could be becoming more and more important in the etiology of multiple sclerosis. Additionally, early viral infection of NS is relevant in patients with autoimmune diseases, with a significant risk for the development of multiple sclerosis. The findings strongly suggest that HCV infection of thyrocytes may play a role in the association between chronic HCV infection and thyroid autoimmunity. Furthermore, for the first time, we have demonstrated that HCV can infect human thyroid cells in vitro. These findings indicate that HCV-infected thyroid cells can serve as an antigenic reservoir for HCV virus replication, thus playing a role in the perpetuation of viral infection and in the development of thyroid autoimmunity.
CD8+ T-Cell Deficiency, Epstein-Barr Virus Infection, Vitamin D Deficiency, and Steps to Autoimmunity: A Unifying Hypothesis.

Pender MP.

Source
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Abstract
CD8+ T-cell deficiency is a feature of many chronic autoimmune diseases, including multiple sclerosis, rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome, systemic sclerosis, dermatomyositis, primary biliary cirrhosis, primary sclerosing cholangitis, ulcerative colitis, Crohn's disease, psoriasis, vitiligo, bullous pemphigoid, alopecia areata, idiopathic dilated cardiomyopathy, type 1 diabetes mellitus, Graves' disease, Hashimoto's thyroiditis, myasthenia gravis, IgA nephropathy, membranous nephropathy, and pernicious anaemia. It also occurs in healthy blood relatives of patients with autoimmune diseases, suggesting it is genetically determined. Here it is proposed that this CD8+ T-cell deficiency underlies the development of chronic autoimmune diseases by impairing CD8+ T-cell control of Epstein-Barr virus (EBV) infection, with the result that EBV-infected autoreactive B cells accumulate in the target organ where they produce pathogenic autoantibodies and provide costimulatory survival signals to autoreactive T cells which would otherwise die in the target organ by activation-induced apoptosis. Autoimmunity is postulated to evolve in the following steps: (1) CD8+ T-cell deficiency, (2) primary EBV infection, (3) decreased CD8+ T-cell control of EBV, (4) increased EBV load and increased anti-EBV antibodies, (5) EBV infection in the target organ, (6) clonal expansion of EBV-infected autoreactive B cells in the target organ, (7) infiltration of autoreactive T cells into the target organ, and (8) development of ectopic lymphoid follicles in the target organ. It is also proposed that deprivation of sunlight and vitamin D at higher latitudes facilitates the development of autoimmune diseases by aggravating the CD8+ T-cell deficiency and thereby further impairing control of EBV. The hypothesis makes predictions which can be tested, including the prevention and successful treatment of chronic autoimmune diseases by controlling EBV infection.

Autoimmune disease: A role for new anti-viral therapies?

Dreyfus DH.

Source
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Abstract
Many chronic human diseases may have an underlying autoimmune mechanism. In this review, the author presents a case of autoimmune CIU (chronic idiopathic urticaria) in stable remission after therapy with a retroviral integrase inhibitor, raltegravir (Isentress). Previous reports located using the search terms "autoimmunity" and "anti-viral" and related topics in the pubmed database are reviewed suggesting that novel anti-viral agents such as retroviral integrase inhibitors, gene silencing therapies and eventually vaccines may provide new options for anti-viral therapy of autoimmune diseases. Cited epidemiologic and experimental evidence suggests that increased replication of epigenomic viral pathogens such as Epstein-Barr Virus (EBV) in chronic human autoimmune diseases such as rheumatoid arthritis (RA), systemic lupus Erythematosus (SLE), and multiple sclerosis (MS) may activate endogenous human retroviruses (HERV) as a pathologic mechanism. Memory B cells are the reservoir of infection of EBV and also express endogenous retroviruses, thus depletion of memory b-lymphocytes by monoclonal antibodies (Rituximab) may have therapeutic anti-viral effects in addition to effects on B-lymphocyte presentation of both EBV and HERV superantigens. Other novel anti-viral therapies of chronic autoimmune diseases, such as retroviral integrase inhibitors, could be effective, although not without risk.

Lupus & Hashimoto's

- 39, female
- Fatigue/malaise, muscle pain, swollen cervical lymph nodes, insomnia, weight gain, joint pain, kidney pain, bloating, anxiety, depression, mood swings.
- Rosacea
- ANA positive
- Blood in urine
- Regular herpes outbreaks
**Nutrition**

- An alkaline-forming diet is high in Lysine which is anti-viral.
- Sugar devastates the immune system for approximately 6 hours after consumption.
- Coconut, garlic and onion have anti-viral properties.
- Foods high in arginine may feed viruses.
- Ensure adequate protein intake for immune system health ie. Whey protein to boost glutathione.
Conclusion

- More and more research is emerging on the connection between viruses and autoimmune diseases as well as disorders related to neurodegeneration such as Alzheimer's disease.
- Identify the virus through blood testing
- Look for chronic fevers and abnormal CBC's
- Treatment can take a few days to months
- Results can come very quickly when the virus is addressed
- www.infectionconnection.net