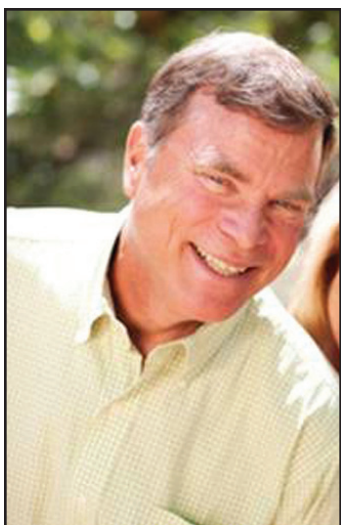


Waking Up the Nation,
One Reader at a Time...

PUBLIC HEALTH ALERT

Lyme Disease Management: Immune Health Part 3



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Let's summarize our prior two articles before launching into the final in this series on the extraordinary immune and gut relationship. The immune system is the most powerful means the body has to fight infection and its central role applies to Lyme disease as well.

Part one discussed how the immune system is divided into two types of sub-systems, the innate and the adaptive immune system. The innate system is a more primitive type of system and is seen in most life forms. The innate system is more generalized in its detection of a foreign sub-

stance. Its immediate response would be to destroy the substance by exposing harsh chemicals to the foreign substance. Major components of the innate system are dendritic cells, macrophages and natural killer cells. The adaptive immune system is seen in higher life forms and is more sophisticated in its specific reaction to an offending agent. The two major pathways of the adaptive pathways involve two different types of white blood cells or lymphocytes. These lymphocytes are known as B-cells and T-cells. The B-cells form antibodies. The T-cells can develop into a variety of forms depending on how they are programmed. The adaptive immune system is mostly programmed by the dendritic cells, a major component of the innate immune system.

Part two discussed how the gastrointestinal tract, GI tract, is a major contributor to the response of the immune system. Something foreign or irritating to the cells of the GI tract will trigger the innate immune system, causing a localized inflammatory response. If the localized inflammation is not kept under control it will progress to cause an inflammatory response throughout the

whole body. This whole body or systemic response is caused by the innate immune system's programming the T-cell lymphocytes to release pro-inflammatory chemical messengers called cytokines. The pro-inflammatory cytokines are managed by another type of programmed T-cell known as T regulatory cells. It is when our immune system cannot be controlled or it gets "stuck" in an expression of a repeating inflammatory response that tissue destruction occurs and it is felt as pain. The messengers for this chronic inflammatory response are certain cytokine expressions from specific T lymphocytes, Th1/Th17. An example of the over expression of Th1/Th17 is the joint pain and destruction seen in Lyme disease.

In view of the importance of the immune system and its intricacy it is clear that the management of a complicated disease such as Lyme requires utilization of a healthy immune system. A healthy immune system is dependent on a healthy gut. A healthy gut is dependent on a healthy bacterial flora and consumption of foods that are not inflammatory. An over-stimulated inflammatory response as a result of poor GI health can result in the

immune system getting "stuck" in a pro-inflammatory mode. The healthy immune system would benefit in balancing out the pro-inflammatory mode to a non-inflammatory mode through regulation or modulation of the immune system.

The over-usage of antibiotics will change the normal GI bacterial flora, which can lead to dysregulation of the immune system as described above. The dysregulation of the immune system will cause symptoms that are sometimes confused with that of Lyme disease. While antibiotics should be considered as a part of the treatment program for Lyme disease, it is important to maintain the delicate balance between the use of antibiotics and a healthy gut bacterial flora. Development of bacterial strains that are optimal for an individual's GI tract and immunity is one of the hottest topics undergoing investigational research. Today, we can only guess that we are improving the healthy flora in the selection of the specific probiotics selected for an individual. As research develops we look forward to being able to select the exact strains required by an individual.

What we eat has a

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great influence on our immune system response. Inflammatory foods or food sensitivities greatly change the internal milieu or environment of the GI tract. It can lead to severe dysregulation of the immune system, whereas a proper diet will lead to proper regulation of immune modulation by supporting and promoting regulatory T cells.

Note that food sensitivities are not the same as food allergies. Not all food sensitivities develop antibodies. As described above, the pro-inflammatory T-cell lymphocytes can be triggered off without developing antibodies. This response covers many situations where a person is just not getting better because of their diet. The problem is the reliance on a food to antibody test result. The test results could be negative yet the food sensitivity persists. Unfortunately, this misunderstanding leads to the mismanagement of individuals who have treatment directed only at inflammatory response and not the causes of the inflammation.

An example of this misguided treatment is similar to someone who develops an intermittent autoimmune response. This person has a sensitivity to dairy and gluten but does not show an antibody response. Frequently, the main focus of treatment is a disease-modifying anti-rheumatic drugs, DMARDs (e.g. CellCept or Mobic), which are pro-inflammatory, initiated by Th1/Th17 immune-blocking drugs. There is no direction by the physician to tell the person to stop their routine consumption of dairy and foods which are contributing to the flare-up their T-cell response.

It is important to reduce the cause of an out-of-control T cell response in tandem with immune regulation which reduces the recurrence of auto-immune flare-ups. The use of such medications should be considered a secondary aid in treatment of an out-of-control immune system. The primary treatment should be the elimination of the irritant to the GI tract, in this case dairy and gluten, while supporting the competency of the digestive capacity.

A large portion of the immune system is affected by the GI tract. The management of Lyme disease is best directed at increasing the natural anti-body/antigen response, the adaptive immune system, while preventing an out-of-control inflammatory T cell response. Reducing a misdirected immune response sourced at the GI tract improves the directed immune response to management of Lyme disease.

Case Study

Toni was a 37-year-old top commercial banking executive with a specialty in acquisitions. She has a past medical history of progressive joint pain, which started approximately 2 years prior and was left untreated. The joint pains were debilitating and migratory with associated fatigue. She had developed significant fatigue to where she could not get out of bed four days out of the week. She also had associated with her illness significant muscle pain, insomnia and cognitive changes.

The noticeable cognitive changes include difficulty in thinking and concentrating, short-term memory loss, dis-

orientation, reversing numbers, word finding problems, and depression. The patient also states that she has had fever and night sweats; swollen and tender neck lymph glands; unexplained menstrual irregularity, PMS and loss of libido; stomach discomfort associated with bloating and daily diarrhea; heart palpitations with severe swollen ankles and feet; neck discomfort which includes stiffness, cracks and pain; and headaches with a slight dizziness similar to vertigo type symptoms. Her past medical history includes ongoing sinusitis and human herpes viral infections with prior increased liver enzymes and a history of asthma, which was triggered off by mold and pollution.

Skin problems included mild acne on the face with long standing history of treatment with doxycycline; bumps on the upper arms; increased cellulite; moderate amount of dark circles under her eyes; easy bruising; lackluster pale skin; sensitivity to bites; strong body odor and thick calluses on feet.

She has noticed an intolerance to milk and gluten products associated with nausea and severe upper abdominal pain.

She went to several top specialists and although some of the tests were positive for Lyme disease she did not fit the exact criteria and Lyme disease was excluded from her diagnosis.

She did start treatment with a Lyme literate physician who began treatment with Neurontin and Trileptal for joint and muscle pain; Trazadone for insomnia and Claritin for sinuses. She was to begin the antibiotic therapy in 2 weeks with the

use of three antibiotics (Omnicef, azithromycin and minocycline) to be stepped up quickly with the inclusion of Flagyl in two weeks. She was having difficulty in managing the above medication treatment and sought other medical advice.

Prior positive labs included:

2009 - Elisa and Western blot IgM for positive for Bb; total porphyrin elevated 173 August 2010 - CD57 @ 20/ul ; human herpes viruses (HHV) #1, #2, #3 and #6; D3@ 34 ; and C4a elevated at 4285.

Recent positive labs were as follows:

Infectious disease - Borrelia burgdorferi, HHV 1,2,3 and 6. Negative for typical co-infections and Chlamydia pneumonia

Gastrointestinal tract - The GI tract with small intestinal bacterial overgrowth (SIBO). Large intestine with Helicobacter pylori, yeast 2+/4+ and gluten sensitivity.

Immune system - The IL-6, C4a and the erythrocyte sedimentation rate (ESR) was mildly elevated. The fibrinogen, CCP antibodies and vascular endothelial growth factor were normal, CD57 @ 16/ul.

Coagulation profile - Plasminogen activator inhibitor type I (PAI-1) gene heterozy

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gous for the 4G/5G.

This signifies a decrease in fibrinolytic activity leading to persistent clot formation which could be associated with increased difficulty in breaking down Lyme's biofilm. Intermittent porphyria when physiologically stressed

Endocrine system - T3 (total) 77 ng/dl; T3 (reverse) 52.5 ng/dl; cortisol saliva test (showing an 18 hour cyclic pattern -6am through 12am) was below the lower limits of normal throughout the whole day; estrogen levels were sufficient for the luteal phase of the cycle yet progesterone deficiency levels were suggestive of anovulatory menstrual cycles.

General working diagnosis is composed of:

1. Infectious disease
2. GI dysbiosis with inflammation and leaky gut syndrome resulting in over-stimulation of the Th1/Th17 response with over-production of pro-inflammatory cytokines
3. Difficulties with hepatic and cellular detoxification
4. Biotoxin histocompatibility
5. Genetically impaired fibrin or clot break down, which increased difficulty with the Lyme biofilm breakdown
6. Hormonal imbalance
7. Euthyroid sick syndrome, poor metabolism of T4 to T3(active) secondary to inflammation
8. Adrenal fatigue resulting from a chronic inflammatory state
9. Dysregulation of sex hormones, including estrogen, testosterone and progesterone.

The approach to such a complicated patient is to begin with correcting the GI tract at the same time as balancing out the hormones. The GI tract had several variables which needed to be addressed. Gluten sensitivity treatment would be to stop all gluten products including food and personal hygiene prod-

ucts.

Toni has a propensity for high yeast growth in the large intestine, *Candida albicans*. This factor needs to be brought under control prior to starting any antibiotics. If ignored, a bloom of the *Candida* with its subsequent toxic release would confuse the infectious profile.

Treatment with nystatin, diet change, and probiotics were instituted immediately. Zinc carnosine was used to eliminate *H. pylori*. There is significant research out of Japan showing that this is an effective method without causing additional imbalance of the GI flora.

Properly balancing the hormones increased her resilience and management of immunological stress. The immune system was balanced by first addressing the cortisol levels. Hydrocortisone was utilized to maintain physiologic levels of cortisol throughout the day. After one week of supplementing hydrocortisone, Cytomel, T3 active, was also introduced taking care that the patient was not symptomatic of the T3 active excess in view of possible low cortisol levels. The hormonal balancing helped to improve her fatigue in conjunction with the length and quality of sleep.

The pain and depression medications, Neurontin and Trazadone, were transitioned to Lyrica and a serotonin based SSRI, Zoloft. 5-HTP was added to help increase the serotonin production. The depression resolved, and the muscle and joint pain started to lessen shortly thereafter.

After the GI tract was staged for control of an exacerbation of a possible fungal

overgrowth, antibiotics were introduced. She had significant difficulty with the antibiotics which required them to be slowly introduced. This was accomplished over a 2.5 month progression until tolerating a therapeutic daily intake of antibiotics, Azithromycin and minocycline. With the antibiotics in place and the GI tract and hormonal system supported it was time to add the fibrolytic, lumbrokinase, to start breaking down any fibrin encasement protecting the Bb.

During this time metronidazole, Flagyl, was introduced. After one month of using metronidazole she developed thrush, nausea and vomiting, and ankle swelling. The liver enzymes started to elevate slightly out of normal range. The metronidazole was discontinued and her symptoms improved. Focus was directed towards improving her phase 1 and 2 liver detoxification and elimination while maintaining proper GI balance. Once accomplished, the Flagyl was reintroduced without any problem.

After four months she was taking daily walks and saunas with drinking plenty of water. The swelling in the legs were gone. The pain medication was reduced as the discomfort subsided. Muscle weakness improved. Cognitive improvements continued with "feeling better to almost being back to "herself".

She had continued improvement of the muscle and joint pain yet the fatigue remained a continued problem. Using a scale of 1 to 10, with 10 representing the most severe, joint pain, dental pain, and muscle pain completely resolved from a 9 to a 3 out of 10. The antibiotics were then

pulsed and she continued to improve.

The swelling resolved and the fatigue had improved from a score of 9 to a 5 out of 10. She only felt a little achy with excess activity. The pulsing the antibiotics revealed an associated cyclical pattern of increased cognitive difficulties would worsen to an 8 out of 10 with diarrhea, and without the antibiotics, cognitive difficulties improved to a 3 out of 10 without diarrhea. The antibiotics were discontinued. The antibiotics leveled the playing field. It was time to move from the antibiotics to a more naturopathic regimen.

The antibiotics were transitioned to an herbal and homeopathic program while continuing to maintain GI balance and immune modulation.

Currently, the patient is off all pain medications; she is exercising to the level of not causing fatigue; the cognitive changes have improved, her menstrual cycles have returned to normal w/o any PMS symptoms and no GI issues. She remains on primarily a naturopathic regimen and is improving every day, without any GI problems. The only symptoms are a little fatigue at the end of the day, which she contributes to spending 60% of her day in helping her elderly parents relocate to their new home. The fatigue continually improves.

The success in the Lyme treatment for Toni was based on optimizing the immune system, balancing the major hormones and removing inflammation in the GI tract. The GI tract is the most forgotten yet most influential in absorption of nutrients, improvement of immune response and the pathway to

SPECIAL REPORT

health.

For any further information please visit our website page on Lyme disease: <http://www.alternativemedicinehealthcare.com/immune-health/lyme-disease>. Peter J. Muran, MD, practices Integrative Medicine in San Luis Obispo, CA, specializing in immune conditions such as Lyme disease.

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