Treating Lyme Disease

It's Much More Than Killing the Bugs

Three Essential Keys to Your Recovery

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Lyme disease was first identified in the U.S. in Old Lyme, Connecticut in 1975. In the 1980's Lyme disease as a chronic multisystem disease started becoming increasingly recognized. Historically, illnesses associated with ticks have been around for hundreds of years. However, the chronic Lyme disease that we see now is very different from the illness resulting from tick bites in the past.

Chronic Lyme Disease (e) today is a multisystem disease characterized by the presence of multiple infections including Mycoplasma, Babesia, Bartonella, Rickettesia, HHV-6, mold and various parasites, in addition to the Borrelia species commonly attributed to Lyme disease. There is frequently significant damage to the body brought on either directly or indirectly by these infections. This damage includes the following: significant immune compromise; large accumulation of heavy metals/chemicals/mold toxins/ & biotoxins due to impairment of detoxification pathways (due to genetic weakness and pathogen induced blocks); and debilitating endocrine impairments (especially involving the adrenal and thyroid glands). These catastrophic changes bring about symptoms in chronic LD patients that relate to practically every organ system in the body. They include debilitating fatigue, food sensitivities, bowel disturbances and malabsorption from GI inflammation, severe pain in muscles and joints from inflammation, and memory impairment along with depression, anxiety and even psychosis from neurological invasion by the infection and associated toxins.

In light of this background, it becomes obvious that successful recovery from chronic LD involves much more that simply killing the Borrelia organism supposedly causing Lyme disease. The first step, correctly identifying all the organisms infecting an individual, can be a daunting task as many organisms like Borrelia and Bartonella are very effective at decreasing or preventing the normal antibody immune response responsible for attacking them. Since many lab tests like the Lyme Western Blot rely on an intact immune system responding with antibodies, they can easily miss the infections. For this reason I strongly recommend using ART (Autonomic Response Testing), applied kinesiology, or electrodermal testing in addition to traditional blood and urine testing to avoid missing coinfections.

Cleaning up the damage that these organisms have caused over the years is as important, if not more important than eradicating the offending organisms. In fact, I will go as far as saying that if this damage is not repaired in a person suffering from chronic LD, they will not completely recover. With that overview in mind, I would like to share with you "Three Essential Keys" that are absolutely essential in successful recovery from chronic LD.

1. Biofilms

The concept of biofilms has been known in the scientific community for many years. However, it was Anju Usman, MD in working with children with autism who first recognized their significance in 2007 as being the culprit behind the frequent relapses she was seeing in the treatment of chronic Clostridium infections in these children.

Biofilms are extrapolymeric substance composed of mucopolysaccharides and protein along with calcium, magnesium and iron that are generated by an aggregate or community of bacteria. These biofilms allow bacteria to share genes for antibiotic resistance. They are also a way for bacteria to survive hostile environments including pH changes and antibiotics. In fact, these bacteria in biofilm can survive 100-1000 times the dose of antibiotics that would kill the same bacteria outside of the biofilms.[1] In addition, the bacteria in these biofilm colonies fail to express their outer membrane proteins (OMP) allowing them to evade the immune system. Dr. Alan MacDonald, a prominent researcher on Lyme disease, believes that biofilms are part of the biology of Borrelia in which they exist primarily as the cyst and L-form. Stephen Fry, M.D. (Fry Laboratory), in his groundbreaking research has demonstrated these biofilms in the peripheral circulation.[2] His findings show not only bacteria but also mold and parasites in these biofilms. In light of these findings, it is reasonable to assume biofilms maybe one of the primary reasons why frequently LD patients relapse soon after stopping antibiotics. It's likely the biofilms serve as a reservoir of Borrelia as well as other organisms that can begin multiplying and seed other areas in the body once treatment is stopped.

[1] "Testing the Susceptibility of Bacteria in Biofilms to Antimicrobial Agents" Antimicrobial Agents and Chemo, Nov 1990. [2] "Evidence of vector borne disease and epierythrocytic bacteria in Chronic Fatigue Syndrome, Fibromyalgia, Autoimmune disease, and Autism." Presentation at the Lyme Induced Autism Conference, 2009.

Treatment, per Dr Usman's protocol, involves taking enzymes on empty stomach to breakdown the proteins and polysaccharides in the biofilm. An hour later oral EDTA is taken with a snack to chelate the Ca, Mg, and Fe in the biofilms. Follow this half hour later with antibiotics/antifungals to eradicate the organisms liberated from the breakdown of the biofilms. A high quality fiber supplement and probiotics at the end can aid greatly in reducing the detoxification symptoms as well as helping to repair the gut lining. In my practice, I have found the treatment of biofilms almost consistently leads to an improvement in energy and sense of well being in my LD patients. Please be careful and do this under the supervision of your health care professional as the process can get uncomfortable. Start slowly and ramp up the dosages as potent Herxheimer reactions from the detoxification are fairly common.

2. Detoxification

Successful detoxification is critical in recovery from chronic Lyme disease. While the use of heavy metal chelators like DMSA, DMPS and EDTA serves a useful purpose in patients whose detoxification pathways are intact, their efficiency is severely limited when these pathways are compromised. The methylation cycle and transsulfuration pathways *(see figure 1)* are frequently impaired in chronic Lyme disease patients. Currently studies reveal approximately 50% of the general population has one or more SNP's (single nucleotide polymorphisms) that will compromise these pathways.[3]

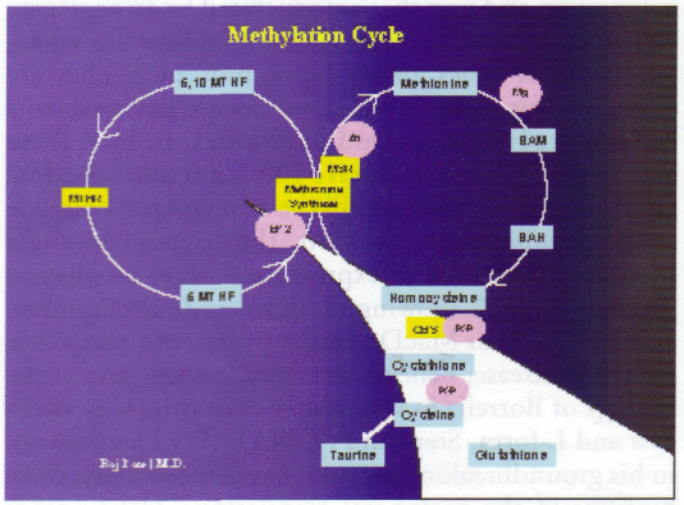

Figure 1

Methylation impairments have been shown to increase the risk of a variety of disorders including cardiovascular disorders,[4] renal disease,[5] and bipolar illness among others. In addition, heavy metals like mercury, lead and aluminum are well known to suppress these enzymes in a dose dependent fashion.[6] In chronic Lyme disease, Borrelia and the accompanying chronic viruses like HHV-6 may further impair the methylation cycle through glutathione depletion (as proposed by Konynenburg). Hypomethylation is also associated with compromise in the immune system's ability to respond to viral infections. Therefore, in a genetically susceptible individual with one or more SNP's in the methylation cycle who has active Borrelia with coinfections, heavy metals can accumulate rapidly. This leads to further impairment of the methylation cycle and even higher accumulations of heavy metals. It is precisely these individuals who also have difficulty with handling Herxheimer reactions and who frequently relapse when treatment is stopped. Urine amino acid testing (Doctors Data) combined with assessment of serum levels for SAM/SAH/Homocysteine (Vitamin Diagnostic Laboratories) can provide a comprehensive roadmap of those specific steps impaired in the methylation and transsulfuration pathways. Subsequent individualized nutritional support based on the work of Dr. Jill James and others can begin opening these pathways.[5,7] Monitoring fecal heavy metals will show heavy metals begin to emerge spontaneously without the use of chelators. As the heavy metals are excreted, their suppression of the detoxification pathways begins to reduce which then allows the body to eliminate more metals. It is important to note that both Borrelia and its coinfections (esp. viruses) need to be properly addressed in order to allow the body to begin spontaneously excreting heavy metals.

Therefore, I would propose that impairments in the methylation and transsulfuration pathways are a risk factor in the development of chronic LD. The high load of heavy metals as well as other toxins arising from SNPs in these detoxification pathways compromises these individuals and thereby making them a more susceptible host to the rapid establishment of borrelia infection upon exposure. Successful treatment involves individualized nutritional support to alleviate the genomic based weaknesses in these pathways coupled with aggressive treatment of Borrelia and associated viral coinfections.

3. Immune Dysfunction

Under this broad category I would like to highlight some of the immune changes brought on by the Borrelia infection either directly or indirectly. These immune changes include secondary hypogammaglobulinemia, depressed CD57 counts, and Kryptopyrroluria.

Secondary hypogammaglobulinemia is well documented in the research literature. Common causes include medications, systemic diseases (including lupus, autoimmune enteropathies like Crohn's, multiple myeloma and renal diseases among others), and chronic viral infections.[8]

[3] Ulrich CM et al. Cancer Epidemiol Biomarkers Prev. 1999 Aug;8(8):659-68 [4] Smulders YM et al. Folate metabolism and cardiovascular disease. Semin Vasc Med. 2005 May 5(2):87-97 [5] Friso S et al. Gene-nutrient interactions and DNA methylation. J Nutr. 2002 Aug;132(8 Suppl):2382S-2387S. [6] Waly M et al. Activation of methionine Synthase by insulin-like growth factor-1 and dopamine: a target for neurodevelopmental toxins and thimerosal. Mol Psychiatry. 2004 Apr;9(4):358-70 [7] Herrmann W et al. Hyperhomocysteinemia and response of methionine cycle [8] Jaffe EF et al. Secondary Hypogammaglobulinemia. Immunology and Allergy Clinics of North America. 2001 Feb Vol 21 Issue 1:141-63

Current medical opinion is still uncertain regarding the usefulness of treating these cases with IVIG. However, a limited number of initial studies do show benefits.[9, 10, 11] In my medical practice, I have found approximately 30% of my chronic LD patients have depressed IgG or IgM levels. It is unclear whether Borrelia directly or the concurrent viral infections (like EBV and HHV-6) commonly found in chronic LD is the cause of this deficiency. Mild cases respond well to oral Transfer Factor.

CD57 is a marker on a subset of lymphocytes (natural killer cells) discovered by Stricker and associates.[12, 13] It is a useful marker in patients suffering from chronic LD. While it has its limitations suffering from significant fluctuations day to day, it can provide a general indication of the degree of immune compromise one is dealing with before starting treatment. It tends to bounce back late in the course of therapy. After aggressive treatment for LD, if the marker has increased significantly (60% or more of the upper end of the normal range), it can serve as one indicator of recovery.

Kryptopyrroluria (KPU) was originally identified by Abram Hoffer, MD and more recently pioneered by Dietrich Klinghardt, MD, PhD. The condition is found in a variety of diseases including schizophrenia, CFIDS, autism and Chronic LD among others. The disorder is characterized by elevated excretion of pyrroles in the urine as a result of abnormalities in heme synthesis. The pyrrloes bind to and leach out of the body large amounts of nutrients critical to proper immune functioning. These nutrients include Zinc, Pyridoxine/P5P, Manganese, Magnesium, Biotin, and Omega-6 fatty acids among others. Aggressive replacement of these nutrients improves immune functioning. Spontaneous excretion of heavy metals and associated Lyme toxins is frequently experienced by patients undergoing nutrient repletion as a result of improved immune functioning and lowering of infection load. The reader is referred to article on Kryptopyrrlouria by S. Forsgren for additional information.[14]

Conclusion

Chronic Lyme Disease is a debilitating, under-recognized, and under-treated condition. The traditional approach used by many doctors of antibiotics and/or antimicrobial herbs, though apparently successful initially, is often characterized by frequent relapses. The Borrelia organism and its many associated coinfections routinely wreak havoc on the endocrine and immune systems in the body. It is precisely this damage done to the body that allows these organisms to evade the immune system and systematically begin taking over the body. Treating a chronic LD patient with long term antibiotics without addressing this damage is a recipe for eventual disaster. A comprehensive approach to treating chronic LD should involve the proper identification and treatment of immune dysfunction, eradication of biofilms, & detoxification of heavy metals/Lyme biotoxins. In addition to these areas discussed in this paper, proper endocrine support, exercise, dietary/lifestyle modifications are critical to recovery. The judicial use of antibiotics in this context can result in recovery from chronic LD with minimal risk of relapse.

About the Author

Raj Patel, M.D. graduated from Robert Wood Johnson Medical School in New Jersey. He received his Board Certification in Family Medicine in 1991 and 1997. He currently practices in Los Altos, California ***www.DrRajPatel.net*** *specializing in the treatment of Autistic Specrum Disorders (ASD) and chronic Lyme disease. Dr Patel was one of several individuals involved in research on the use of Ampligen in CFIDS patients. He has been invited as a guest speaker at various corporations and medical conferences on Lyme disease and ASD. Most recently, he founded Lymelighters, a Lyme support network serving patients with chronic Lyme disease and their friends and family.*

[9] Leon F, Olivencia P, Rodriquez-Pena R, et al. Clinical and immunological features of adult-onset generalized autoimmune gut disorder. Am J Gastroenterol 2004; 99: 1563–1571. [10] De Giacomo C, Maggiore G, Scotta MS, Ugazio AG. Administration of intravenous immunoglobulin in two children with hypogammaglobulinemia due to protein losing enteropathy. Clin Exp Immunol 1985;60:447–448 [11] Ogi M, Yokoyama H, Tomosugi N, et al. Risk factors for infection and immunoglobulin replacement therapy in adult nephrotic syndrome. Am J Kidney Dis 1994; 24:427–436. [12] Stricker RB, Winger EE. Decreased CD57 lymphocyte subset in patients with chronic Lyme disease. Immunol Letters 2001;76:43-48 [13] Stricker RB, Burrascano J, Winger E. Longterm decrease in the CD57 lymphocyte subset in a patient with chronic lyme disease. Ann Agric Environ Med 2002;9:111-3. [14] Forsgren S. Kryptopyrroluria. Explore! 2009 Vol.18 No.16:11-17