Lyme Disease and Co-infections

In 1975 an attentive MD from Old Lyme, Connecticut, reported a series of cases of patients with joint pains. He realized that all of them lived in the same area of town, suggesting an infectious cause. The causative microbe was identified as a spirochete seven years later, in 1982, by Dr. Willi Burgdorfer who named it Borrelia burgdorferi. Borreliosis quickly spread across the country and – via tourism and business travels – throughout the world. What is known today as “Lyme disease” – named for the town that saw the first outbreak – is however not just borreliosis alone. While borrelia is the primary microbe in Lyme disease, there is usually a whole host of co-infections and opportunistic infections that need also be addressed during the treatment. In the following article I will give a short overview over several of the infectious agents involved in Lyme disease.

First of all, Borrelia burgdorferi (Bb) itself: Borrelia is 1 of 8 genera of spirochetes which are a group of bacteria that under a microscope look like little cork screws. Other well-known spirochete genera are Treponema pallidum (the syphilis microbe) and Leptospira (common in Maui, New Mexico etc., transmitted by drinking water contaminated with animal feces). Spirochete genera have hundreds of subgroups, so-called species, one of which is Borrelia burgdorferi, the most commonly species found in the USA. Bb sensu strictu refers only to the B.burgdorferi species but includes many species causing identical symptoms, while Bb sensu lato includes B.afzelii and B.garinii, the most commonly found of the 5 species in Europe, as well as the 61 strains found in Japan. Since microbes constantly exchange DNA with each other via plasmids (DNA molecules that are separate from, and can replicate independently of the chromosomal DNA), there are Bb microbes with properties usually found only in Babesia or mycoplasma. There are no fixed boundaries between many of these microbes. Historically the microbes most difficult to treat were always those with the highest number of plasmids: The DNA found on plasmids often contains resistance mechanisms against antimicrobial therapy or genes for microtoxins suppressing the immune system in a more efficient way. While most bacteria have 1 or 2 plasmids, chlamydia has 4-6 plasmids and the syphilis spirochetes have 7, borrelia spirochetes are equipped with 21 plasmids (12 linear and 9 circular DNA sequences) and are therefore extremely adaptive. Most textbooks still report that Bb can only be transmitted by tick bite. However, recently the evidence of other ways of transmission has increased and now includes transmission via bites from mosquitos, fleas, stinging flies (horse flies).
and spiders as well as blood transfusions, sexual intercourse, kissing, trans-placentar transfer to the fetus, breast feeding and contaminated utensils like telephones. Therefore asking a patient if they remember a recent tick bite is not an adequate anamnesis. Borreliosis goes through several stages. At the first stage, patients will have flu-like symptoms. Only about 25 % of them report joint pains. Here antibiotics will be effective. Often patients enter the second stage after many near-asymptomatic years. They complain about muscle aches, fatigue, joint pain, “migratory arthritis”, meningitis and loss of appetite. Finally the third stage, often after many years of milder illness: Patients show severe chronic neurological symptoms, profound fatigue, memory loss, severe pain, depressions and even psychosis. All asymptomatic carriers are at risk of developing symptomatic borreliosis; often a temporary immune suppression by stress causes the activation of the disease.

There are 6 major sites of infection, each with characteristic symptoms:
1. Large joints and connective tissue: onset 4.3 months after insect bite, often self-limited (4 years). Flare-ups during Herxheimer reactions very common
2. Skin and connective tissue (B. afzelii): acrodermatitis chronica atrophicans, general collagen breakdown (premature ageing), collagen diseases, ...
3. CNS (B. garinii), PNS and ANS: after an insect bite it only takes a few hours until spirochetes are found in CNS even though it takes on average 2 years before symptoms are established. Most common symptom: brain fog and short term memory loss. Later stages: demyelination. Severe early changes can be found in SPECT scan (functional), MRI changes occur much later (physical); CNS: epileptic seizures, insomnia, tremor, ataxia, movement disorders (torticollis, etc.); irritability (key symptom in children), depression, bi-phasic behavior (manic depression), bouts of anger, listlessness; confusion, difficulty thinking, poor short term memory, increasingly messy household and desk, difficulty finding the right word, feeling of information overload; can resemble or imitate any known psychiatric illness. Chronic Fatigue (more severe in the early afternoon); Lack of endurance; Non-healing infections in the jaw bone, devitalized teeth, dental pain; Fibromyalgia; Multiple Chemical Sensitivity; loss of zest for life, sensitivity to electric appliances.
4. Heart: Lyme carditis is difficult to diagnose with current methods (PET scan positive early on) and has multiple symptoms from arrhythmia to angina. Has to be taken serious with first symptoms.
5. Kidney, bladder: the highest concentration of tissue spirochetes has been found in kidney and bladder. Symptoms often include interstitial cystitis, prostatitis (Babe-
sia often also involved), sexual dysfunction, loss of libido, pelvic pain, menstrual disorders, filtration problems in the kidney (low specific weight of urine) and urethritis after intercourse (the spirochetes are attracted during intercourse to the urethra and cause acute inflammation).

6. Immune system infections (white blood cells, thymus, brain, lymphnodes, adrenals, etc): non-healing infections in the jaw bone (also Babesia, Bartonella), devitalized teeth, dental pain, Immune system failure: with all known secondary illnesses such as herpes virus infection, intestinal parasites, malaise, hair loss, hepatitis C.

Diagnosing Bb:
There are several lab tests available for borrelia that can be used to make a diagnosis: Direct microscopy (www.Bowen.org, www.BradfordResearchInst.org), Detection of antibodies (ELISA, Western Blot), Lymphocyte proliferation tests (MELISA and LTT), CD 57 Stricker panel, ART testing (www.neuraltherapy.com, www.INK.ag), indirect tests (FACT, different lab parameters). However, spirochetes can assume a cystic form which can lay dormant in tissues and escape detection from any diagnostic method. Also, the cells of the immune system responsible for making antibodies are usually sick and cannot produce antibodies. The Western Blot turns positive as soon as an effective treatment has been given – not before. Therefore you first have to treat before you can make the diagnosis.

Several lab parameter configurations are typical for an infection with Bb: Abnormal lipid profile (moderate cholesterol elevation with significant LDL elevation), elevated triglycerides (= early response) or very low triglycerides (late response), insulin resistance, borderline low wbc (3000-5000), normal SED rate and CRP, low-normal thyroid hormone tests but positive Barnes test and excellent response to giving T3, adrenal failure or weakness (high cortisol in early stage, low cortisol, DHEA and testosterone in late stage Lyme), low alkaline phosphatase (indicating low zinc levels, usually from lyme associated HPU), decreased urine concentration (low specific gravity), positive test for HPU.

An acute infection with borrelia may respond to the textbook antibiotic treatment (ceftriaxon or doxycyclin). For the treatment of chronic borrelia infections I recommend the Klinghardt Lyme Cocktail which has to be taken long term in order to catch the cystic forms.

As I mentioned earlier, most patients present with co-infections and opportunistic infections. The latter include viral, mold and yeast infections as well as parasite infestations settling in comfortably after the immune system has been compromised. The discussion of these I will save for another article while focussing on the most important of the co-infections in this one. We often find Babesia, Bartonella, and Rickettsia species such as Ehrlichia in Lyme patients. Babesia are intracellular Malaria-like protozoal organisms that infect the red blood cells. 2/3rds of Lyme patients are also infected with Babesia. Since there are 17 known antigen-different subspecies, each requiring a very specific lab test, they are often overlooked. The most common strains are B.microti in the Western United States and B.divergens in Europe. The best way to diagnose a Babesia infection is ART or long term observation of blood under the darkfield microscope: Babesia tend to leave dying cells while under darkfield observation. Symptoms of Babesia infection include: Vertigo, headache, fatigue, eye problems (floaters, blurry vision), dental problems (accelerated tooth decay, cavitation formation), TMJ problems, fibromyalgia, shortness of breath, malaise as well as drenching night sweats and fever/chills during Herxheimer reactions.
Bartonella henselae is the most commonly found intracellular co-infection in red blood cells, endothelial cells, bone marrow and in macrophages. An estimated 70% of the cats in Italy are infected (c"cat’s scratch disease"), and cat to human transfer is common. Symptoms include swollen lymph glands, endocarditis, hepatitis, neovascularization, fatigue, low grade fever, jaw bone cavitations, devitalized teeth, fibromyalgia and joint pain. We often find it as a co-infection in ALS patients.

Ehrlichia and other rickettsia are often found in patients who have contact with horses and farm animals. They are also common in dogs and their owners. Symptoms include fever (after initial infection, sometimes recurrent for years), myalgia, arthralgia, headaches, leukopenia, thrombocytopenia, hyponatremia, mental confusion, skin rashes, genital and oral ulcers, nausea, vomiting (acute flare-ups), and severe pain syndromes.

The Klinghardt Lyme Cocktail is designed to treat both the borrelia infection and the co-infections, as well as the surge of neurotoxins released during the die-off (Herxheimer reactions). There are of course many other microbes that can be transmitted by the same insect bite, and even more can be contracted secondarily. Many of them respond to the Lyme Cocktail as well. The parasite treatment may have to include conventional anthelmintic drugs such as Ivermectin. Here the recently discovered lungworm, Varestrongylus klapovi, seems to be one of the worst offenders and will certainly have to be addressed by scientists and practitioners in the future.

There are always new additions and slight changes to the protocols I’m currently using in response to any new scientific findings. I will continue to fine-tune the treatment protocols and share new information with you in my lectures.

The Klinghardt Lyme Cocktail

Ingredients per dose:
1 glass ½ water,
½ organic grapefruit juice,
1 tablespoon Phospholipid Exchange (PLE - BioPure/INK),
200-400 mg Artemisinin (source: internet),
20 drops propolis Incture 20%,
500 mg B5 as powder (open a capsule),
1000 mg ascorbic acid (Vit C),
1 heaping tsp bee-pollen,
20 drops Rizol Gamma (source: BioPure/INK),
100 mcg MicroSilica (BioPure),
Quintessence 5 dropperfull and Rizol Gamma 15 drops (BioPure),
1 tsp local honey,
1-2 tbsp Rechtsregulat (BioPure)

Use regular blender at high speed. Start with a little water just covering the blade of the blender, add the whole amount of PLE and Artemisinin. This creates over 5 minutes or so a liposomal artemisinin, the worlds most powerful antimicrobial for Babesia, Bartonella and many aspects of Lyme. Wait till this mix turns from watery to gel-like. Only then add the other ingredients. The MicroSilica (MS) is a powerful toxin binding and removal agent that binds to sulmydryl affinity metals and microbial biotoxins. Do not give minerals at the same time, since they would be bound up by MS. The HPU minerals should be given with a meal, away from the cocktail. The cocktail is given twice daily, 5 days on, 2 days off for many months. It may decrease iron levels, which should be monitored.