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

FREE

PUBLIC HEALTH ALERT

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Laboratory Tests and Diagnosis for Lyme Disease and Co-infections

by Armin Schwarzbach, M.D., Ph.D.

It is important to detect Borrelia infections at an early stage. The earlier the detection, the simpler the treatment measures (usually antibiotics) and the shorter the ordeal of the infected patients.

This is how you can diagnose Lyme Disease - a Lyme disease infection progresses in 3 stages depending on symptoms and ailments (time specification after the tick bite):

1. Stage I (after days up to weeks): "bull's eye rash" ("Erythema chronicum migrans", only in 40-70% of all cases), Borrelia lymphocytoma, headache, fever, sweating, "Summer flu" (app. 20% of all cases), exhaustion and fatigue, facial palsy (especially with children/pupils).

2. Stage II (after weeks up to months): inflammation of the brain, meninges, spinal marrow, any nerve in the human body, inflammation of the joints ("arthritis"), joint and muscle pain, inflammation of the eye, liver and kidneys, myocarditis, pericarditis, cardiac arrhythmia.

3. Stage III (after months up to years): Thinning of the skin at the back of the hand, ("Acrodermatitis chronica atrophicans"), Borrelia lymphocytoma (ear, nose, scrotum), lethargy, fatigue, paraesthesia, cognitive dysfunction, muscle inflammations, joint inflammations and swelling, tendon inflammations, inflammation of the bursa, vasculitis, myocardinal diseases, depression.

Symptoms of Lyme disease occur in thrusts with alternating intensity and appearance in contrast to a classic organic illness. Many patients also suffer from slightly higher temperature during those thrusts. Co-infections with other bacteria and viruses have been increasing over the past years and often lead to a complicated course of the disease. Often the tick bite is not detected early enough or the acute treatment by the attending physician is not sufficient. The chronic Lyme disease patients often go through a true "martyrdom" as they do not only suffer from actual physical and mental ailments, but also from not receiving a reliable diagnosis and the fact that their illness is not taken seriously.

There are three main reasons why a Lyme infection is not immediately detected in daily practice, so that a treatment at an early stage is inhibited:

1. There is no bull's eye rash! ("Erythema chronicum migrans"). Research has proven that this classic symptom of Lyme disease only appears in 40% to a maximum of 70% of all cases.

2. No tick bite detected! Such a bite can be induced even by very small ticks (larvae). Or it was not detected because there was no specific skin reaction. In addition, the scientific community now assumes that Borrelia bacteria can also be transmitted by infected insects.

3. Only conventional blood tests have been performed. This was either too early (antibodies can be tested positive only after a period of up to six weeks after the tick bite) or there has been no antibody production in the body, or the cellular stage was not or not sufficiently tested (necessary tests are: Elispot®-LTT and CD3-/CD57+ cells).

First the **laboratory diagnosis** is crucial for the diagnosis of Lyme disease. There is a difference between the tests of the **humoral** (antibodies) and **cellular** level (lymphocytes, NK-cells). Both levels have to be examined at the same time when a Lyme disease or an apparent illness is suspected.

1. Laboratory testing of the humoral level (antibodies): Borrelia IgM- and IgG-EIA (Enzymimmunoassay) as well as Borrelia IgM- and IgG-Immunoblot

2. Laboratory-testing of the cellular level: Elispot®-LTT (Borrelia Elispot Lymphocyte Transformation Test), CD3-/CD57+ cells (NK-cells)

Explanations of these tests:

1. Antibodies - humoral level: In case of the Borrelia antibodies examinations up to 19% of the antibodies results in the EIA are falsely negative due to the minimal sensitivity (responsiveness) of the EIA in contrast to the Immunoblot. Therefore it is essential to check the Borrelia IgM- and IgG-Immunoblot along with the Borrelia IgM- and IgG-EIA (even with a negative EIA)! Important: The laboratory has to test for VlsE (Variable major protein-like sequence Expressed) in EIA and Immunoblot. VlsE describes the characteristics of the Borrelia as a "chameleon", which permanently changes the surface structure VlsE in vivo to resist the detection via the immune system. VlsE has the highest sensibility for the antibody search!

Beware of positive antibodies constellations! Borrelia IgM- as well as IgG-antibodies can remain in the body for months or years even without an active Lyme disease infection.
Hence a positive Borrelia antibodies test does not give any evidence of the activity of a Lyme infection, but gives only one conclusion: There must have been a tick bite or tick bites in the past during which Borrelia bacteria have been transmitted.

2. Cellular level:

a) **The Borrelia Elispot®-LTT** provides information about the current activity of the Borrelia bacteria and is 20 to 200 fold more sensitive than an EIA-antibodies test.

b) **The CD57+ cells** indicate the extent of immune suppression during a chronic Lyme disease and are the prognostic factor during and after the antibiotic treatment.

The complete laboratory diagnosis is very complex and - considering the co-infections - has to be put together like a mosaic. This demands experienced Lyme disease analysts who can diagnose and evaluate all other infections as well.

However, the complexity of a Lyme disease infection is not sufficiently covered by this and needs additional laboratory testing. Therefore further internationally accepted methods are applied which are of considerable importance for the patient before and after the therapy (e.g. for the cellular level: Elispot®-LTT and the CD3-/CD57+ cells).

In practice, the following laboratory constellations indicate a Borrelia infection:

(1) Positive antibody detection and positive cellular results (Elispot®-LTT and/or CD57+) - often active infection

(2) Positive antibody detection without positive cellular test findings - What kind of indications are shown by the clinical findings (symptoms)?

Armin Schwarzbach M.D., medical specialist for laboratory medicine, head of the BCA laboratory and specialist for Lyme and co-infections.

(3) Negative antibodies detection, but positive cellular test results
- Indication for an active Lyme disease at an early stage, but also chronic stage - what kind of indications are shown by the clinical findings (symptoms)?

Attention: Negative antibodies by EIA and/or Immunoblot cannot exclude a Lyme infection.

Reason: the antibody production of Lyme disease in stage I needs several weeks, 10 to 14 days at a minimum. Thus the immediate measurement of cellular activity in Elispot®-LTT is a compulsory necessity, as normally the cellular activity precedes the humoral activity in stage I of a Lyme disease infection.

In practice the following combinations of blood tests have proven useful:

Laboratory diagnosis stage I

1. Borrelia IgG- and IgM-EIA incl. VlsE
2. Borrelia IgG- and IgM-Immunoblot incl. VlsE
3. Borrelia Elispot®-LTT

Laboratory diagnosis stage II and stage III

1. Borrelia IgG- and IgM-EIA incl. VlsE
2. Borrelia IgG- and IgM-Immunoblot incl. VlsE
3. Borrelia Elispot®-LTT
4. CD3-/CD57+ cells

Necessary **"Staging"** (evaluation of progression) of Lyme disease during and after an antibiotic or holistic approach to therapy: The above mentioned parameters also have to be checked during Lyme therapy.

Recommended laboratory diagnosis during the therapy:

Stage I

Performance of the 3 tests mentioned above 4 weeks after beginning of therapy and 8 weeks after the end of therapy.

Stage II and III

Performance of the 4 tests mentioned above every 8 weeks after the beginning of therapy as well as 8 weeks after end of therapy.

For explanation: Borrelia antibodies or titer cannot be "eliminated", but they can exist in the blood for months or even years. After a successful antibiotic therapy Elispot®-LTT as well as CD57+ cells should have come to a "normal" level 8 weeks after the end of therapy. But-- In spite of a symptom-free status after treatment there might still be positive results of Elispot®-LTT and/or CD57+ cells. Therefore the patient should be considered for a "monitoring" of the activity tests and possible future symptoms. Otherwise there might be the risk of a relapse, a new infection or a co-infection.

"Lyme" ...cont'd pg 4

Mother Buries Four Babies

by Shelley White

Missy Mossor has given birth to five infants. Out of those five, she has buried four. Her fourteen year old daughter MacKenzie survived, but is now fighting for her life alongside Missy. The underlying culprit? Missy had an unknown case of Lyme disease. This is an exceptionally intricate and heartbreaking story that every mother must hear. When beginning her story, Missy opened by saying, "I hope you will bear with me. I am writing this on the verge of tears ...I hope my brain will not let me down. From the time I was an adolescent, I suspected something was wrong with me, health wise. If I had only known what it was [Lyme disease], I could have saved myself, my ex-husband, and our extended families a lot of heartache and grief."

Marrying on her twentieth birthday, Missy had her whole life ahead of her and was excited to start a new family. After a mere six months of striving to conceive, her doctor performed the first of many invasive pelvic surgeries that she would endure over the next few years. She was then placed on hormones, resulting in her first pregnancy. Joy was short lived as red flags of an unhealthy pregnancy quickly surfaced.

Every mother's worst nightmare began for Missy early on when she experienced breakthrough bleeding. Although doctors advised her not to worry, the need to worry was confirmed by the time she was half way through her pregnancy. At twenty weeks, she was awoken by what she describes as "excruciating" back pain. Throughout the day, her pain worsened and she was rushed to a nearby hospital to no avail. Passing her mucous plug while in the hospital, Missy was shocked when the doctor dismissed it and sent her packing. Unable to bear the pain, she drove herself home at ten miles an hour. By 9:30 PM her symptoms had worsened and she was rushed back to the hospital. Arriving only fifteen minutes later, her OB/GYN was furious to learn that doctors failed to check Missy's cervix during her previous visit that day. When Missy's OB/GYN finally checked her cervix, she was dilated ten centimeters. Her water broke shortly after. Immediately, Missy said she could smell the infection as she gave birth to a still-born baby girl.

Doctors and nurses did not blink as Missy experienced this heart shattering tragedy. They tried to place her in a room with a mother who had just given birth to a healthy baby. As if they had not rubbed it in her face enough, they relentlessly pushed on. Weeks after returning home, Missy was forced to carry out the dreaded task of ridding her house of items she had collected to prepare for her baby's homecoming. While doing so, she received an envelope from the hospital containing pictures of her dead baby lying naked on a newborn blanket. When asked to further describe this event, Missy simply said, "No clothes. Not swaddled. No little pink toboggan. In one picture they had pulled a blanket halfway up her body. I was devastated. I couldn't believe how

heartless and cruel people could be."

Told it was rare for her previous pregnancy experience to repeat itself, Missy and her husband had high hopes when she became pregnant with triplets the following year. Once again, at twenty weeks pregnant, she was rushed to the hospital after excruciating back pain. She had dilated again. Attempting to delay labor, doctors inserted a "purse-string" cerclage around her cervix. One of her fraternal triplets had an infection surrounding it, much like her still-born baby girl, Angel, whom she buried only a little over a year before. At twenty-four weeks the purse-string cerclage broke, moving the bottom baby partially into the birth canal. Missy was taken into surgery for an emergency caesarean section and gave birth to three baby boys named Tristin, Logan, and Dakota. Tristin and Logan lived approximately twelve to fourteen hours. Dakota, the smallest of the three, passed away twenty-four hours later. According to doctors, Missy would never be able to conceive on her own again.

Despite what doctors said, Missy became pregnant again within a few months. This time she gave birth to her baby girl, Mackenzie. She was six and a half weeks early, and weighed six pounds and fifteen ounces. Missy could not believe it. Mackenzie was the healthiest looking baby in the unit, with little fat rolls and long dark hair. Unfortunately, this did not prove to be Missy's happy ending.

Last year Missy found out she has had Lyme disease the whole time, resulting in all her previous pregnancy complications. Without knowing, she passed Lyme to her daughter. Mackenzie, who is now fourteen, is unable to go to school due to the aggressive progression of her disease. Both her and Missy are debilitated and spend their days within four walls struggling to win their battle against Lyme. *pha*

A: They both do!

But the child is far less likely to receive proper diagnosis and treatment from a knowledgeable doctor.

See www.txlda.org for more info

Public Health Alert

The PHA is committed to researching and investigating Lyme Disease and other chronic illnesses in the United States. We have joined our forces with local and nationwide support group leaders. These groups include the chronic illnesses of Multiple Sclerosis, Lou Gehrig's Disease (ALS), Lupus, Chronic Fatigue, Fibromyalgia, Heart Disease, Cancer and various other illnesses of unknown origins.

PHA seeks to bring information and awareness about these illnesses to the public's attention. We seek to make sure that anyone struggling with these diseases has proper support emotionally, physically, spiritually and medically.

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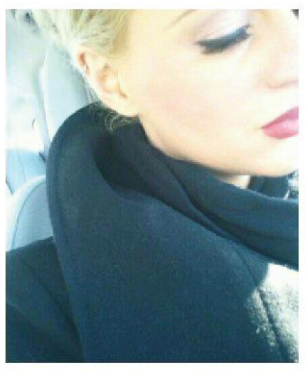

Kim's dream
is to become a doctor.
Let's join together and
help her reach her goal.

"Lyme" ... cont'd from pg 1

It should be noted that laboratory parameters alone do not give a definite proof of Lyme disease, but very important evidence and details about a possible infection. The parameters are also very important to make decisions about the duration and success of the therapy.

Laboratory tests of possible co-infections

Laboratory tests of possible co-infections have an increasing importance when adequate evidence exists. Especially Ehrlichia/Anaplasma, Babesia, Bartonella, Rickettsia, Chlamydia and Mycoplasma are to be named.

Reasons: numerous symptoms of the co-pathogens overlap the same symptoms of Lyme disease patients. Without an exact knowledge of a patient's possible co-infections the therapist cannot make an exact decision about the antibiotic therapy. Not all co-infections can be treated by the generally used antibiotics for Lyme disease. However, the testing for co-infections can induce extensive additional laboratory costs. But these are justified by the additional security in the diagnosis and especially by the correct antibiotic decision, leading to more success in antibiotic therapies as well as generally less expensive costs for medication during the antibiotic therapy (vs. the "classical" antibiotic therapy of chronic Lyme disease).

Apart from the LTT for Borrelia, further cellular activity tests for Ehrlichia/Anaplasma, Chlamydia pneunomiae and Chlamydia trachomatis have been developed in the meantime (Babesia cellular activity testing is coming soon) and can therefore be determined with Elispot-LTT-technique. With the help of this new Elispot®-LTT, many activities of Chlamydia and Ehrlichia have been discovered in the BCA! A serum examination concerning co-infections is perfomed at the same time. Well-standardized antibody tests for Chlamydia, Mycoplasma, Ehrlichia, Bartonella, Rickettsia, Babesia, Yersinia etc. are now available. These tests offer the same advantages as the Borrelia-LTT: the antibodies alone do not have any significance towards the activity of an infection - but the Elispot®-LTT is significant as it attests the high-specific interferon release against the respective co-pathogen in the blood.

Furthermore it is in some cases the co-pathogen itself which is responsible for the symptoms and not the Borrelia infection: e.g. Chlamydia cause diseases patterns such as Morbus Alzheimer, Multiple Sclerosis, fibromyalgia, Chronic Fatigue Syndrome (CFS), myocardial infarcts, strokes, vasculitides and visual disturbances.

The Lyme infection may have been treated successfully with antibiotics, but the co-infection might not have been cured by it. Therefore, an exact anamnestic documentation of the symptoms during the therapy is necessary before, during and after a Borrelia infection.

Not all possibly remaining symptoms are due to Lyme after an antibiosis, but could be due to a co-infection!

Infectiology will gain a considerable dominant position in medicine - among others due to climate change - and this does not only apply to tick-borne diseases.

Overview of the most frequent co-infections of Lyme Disease: Ehrlichia, Babesia, Bartonella, Rickettsia, Chlamydia, and Mycoplasma

Ehrlichia/Anaplasma

Pathogen: Anaplasma phgocytophilum (gram-negative, obligatory intracellular in granulocytes)

Vector: Ixodes ricinus

Spectrum of hosts: wild animals (e.g. deer), domestic animals, productive livestock, humans

Symptoms: (incubation period: days up to 4 weeks): Flu-like symptoms with fever, headaches and muscle pain with "sharp and stabbing, often located behind the eyes", neurologic symptoms (duration 1 up to 60 days) up to lethal ending, sometimes diffuse erythema (reddening of the skin) including palms of hands and soles of feet

Risk factors: elderly people, severe basic underlying diseases, immune suppression

Diagnostics:

- Activity test: Elispot®-LTT (Lymphocytes transformation test)
- Ehrlichia-PCR in blood (EDTA-blood): direct detection
- Pathogen detection in Giemsa stain
- Antibodies for Ehrlichia-IgM and Ehrlichia-IgG: indirect detection - control of progression!
- Leucopenia / Thrombocytopenia / Anemia

Transaminase increase Therapy:

Macrolides (Azithromycin, Clarythromycin)

Tetracycline (Doxycycline, Minocycline)

Quinolones (Ciprofloxacin, Levofloxacin)

Rifampicin (During pregnancy!)

Babesia

Pathogens: Babesia microti, Babesia divergens

Vector/Transmission: Ixodes ricinus, blood transfusion

Spectrum of hosts: wild animals (e.g. deer), domestic animals, productive animals, humans

Symptoms: (incubation period 5 days - 9 weeks): Sweating, neck stiffness, nausea, vomiting, loss of appetite, fatigue, feeling of weakness, constant exhaustion especially during stress, haemolytic anaemia, haemoglobinnuria, fever up to 40° C, shivering, sometimes hepatosplenomegaly, muscle pain, strong headaches, dizziness, coagulation dysfunction (blood-clotting disorder, hypercoagulability), stomach ache, emotional instability, mental dullness, kidney failure, dyspnoea, influenza-flu-like symptoms (up to a lethal level)!
Risk factors: Splenectomia, HIV, immune suppression, organ transplantation, elderly people.

Diagnostics:

- Babesia-PCR in blood (EDTA-blood): direct detection
- Blood smear: direct detection
- Antibodies of Babesia-IgM and Babesia-IgG: indirect detection - control of progression !

Rare:

- Hamolytic anemia (erythrocytes, haptoglobin)
- Thrombocytopenia
- Leucocytopenia
- Increase of liver parameters (sGOT, sGPT, sGGT)
- Increase of Creatinine, Urea

Hemoglobinuria Therapy:

- Clindamycin
- Malarone 250/200 mg 1x/day
- Malarone junior 65/25 mg 1x/day
- Atovaquone 750 mg 2x/day
- Lariam 250 mg

Bartonella

Bacteria: Bartonella henselae (gram-negative, optional intracellular in endothelial cells/ Erythrocytes) and/or BLO = Bartonella like organisms
Vector/Transmission: surface wounds/scratch from cats, Ixodes ricinus
Symptoms (incubation period 3 - 38 days): headache (80%), fatigue (100%), muscle twitches, tremors, cramps, shivering, fever in the mornings (30%, in thrusts up to 6 weeks, otherwise 1 - 3 weeks), swollen lymph nodes, arthralgia (often), myalgia, insomnia, depression, agitation, amentia, concentration and attention disorder, dizziness, restlessness, gastritis, intestinal problems, sore feet soles (especially in the morning), hypodermic nodules along the extremities, no or minimal joint pain (important according to J.J. Burrascano)
Severe progression: endocarditis, retinitis, epilepsy, aseptic meningitis, hepatosplenomegalia
Risk factors: immune suppression

Diagnostics:

- PCR on Bartonella in blood (EDTA-blood): direct detection
- Histology (hemangiome/lymphadenitis)
- Antibodies on bartonella henselae-IgM and bartonella henselae-IgG: indirect detection evidence - control of progression!
- Elevated vascular endothelial growth factor (VEGF), only rarely increased, but if so activity marker for monitoring

Therapy:

Macrolides (Azithromycin, Clarythromycin)

Tetracycline/Doxycycline

Quinolones (Ciprofloxacin, Levofloxacin)

Rifampicin

Ceftriaxone/Cefotaxime

Rickettsia

Pathogen: Rickettsia conorii, R. rickettsii, R. helvetica, R. slovaca, R. prowazekii (not gram-stainable, obligatory intracellular in endothelial cells)
Vector/hosts: rodents, dogs, humans, Ixodes ricinus
Symptoms: (incubation period 5 - 7 days): fever, lymphadenitis, exanthema (roseola to macupapulous)
Complications (app. 13%):

"Lyme" ...cont'd pg 7

Lyme is a Brain Disease

by Virginia T. Sherr

Lyme borreliosis is a brain disease as well as a multisystemic disease caused by spirochetal bacteria.* Quite frankly, it is an infection that has been burdened with a thousand inaccurate medical diagnoses. The manner in which the current pandemic of tertiary Lyme disease, neuroborreliosis, has usually been handled---either angrily dismissed or strangely misdiagnosed--throughout the 30 years following its "discovery," has blemished the historic excellence of modern American Medicine.

After all the years, neuroborreliosis is still actually considered rare by a majority of physicians, most of whom are spirochetally naïve. Officially tallied patients (the numbers showing a dip down to 19,804 cases in 2004 after flawed reporting styles were instituted), when combined with uncounted cases may approach upward of an annual quarter million new borreliosis infections in the USA alone. And Lyme infections have been verified as present on all but one continent, globally. The disease is more often than not accompanied by several of a half-dozen or so of the other serious tick-borne co-infections that currently have been identified.

Losses of acuity in the human brain's visual cortex have been observed as early as 6 hours following the toxic bite of an infected tick. Lyme may persist after too brief a period of treatment or if there has been no treatment, and may result in chronic infections whereupon Lyme borreliosis becomes a potential cause of every symptom in medical and psychiatric lexicons. It is the "Great Imitator" of this Millennium, spirochetal paresis (neuro-syphilis) having been its precursor and its model.

Chronic or persistent Lyme disease--neuroborreliosis--seldom is identified by the symptoms of its most frequent form—subacute encephalitis--an infected/inflamed brain as well as an infected nervous system. However, this is the form in which it most commonly exists. Unfortunately, the syndrome that is falsely considered typical--a bull's eye rash, fever, positive Elisa test, and/or a swollen large joint--occurs in fewer then half of proven cases. Instead, Lyme borreliosis confirms itself in subtle to profound neuro-psychiatric symptoms, such as overriding confusion, loss of organizational skills, decreased concentration, memory loss, mood disorders, irritability, and unprovoked rages--to mention just a few. These symptoms can be very obvious to an experienced professional practicing in a Lyme-endemic area. However, cerebral-behavioral symptoms of neuro-Lyme remain invisible to those whose diagnoses are solely based on old-fashioned concepts limited only to the aforesaid doctor-viewed rashes, swollen knees with positive Elisa blood tests.

Blood tests completed by local labs most frequently show false negatives due to general laboratories' inadequate understanding of proper diagnostic technique and choices of poor quality spirochetal samples on which to base tests. Of course, insurance companies prefer their negative tests. As mentioned, Lyme can rapidly go from Stage One (Early borreliosis) to Late (Tertiary) Stage disease following attachment of an infected deer tick's or other vector's bite so that quick and competent treatment are of the greatest importance. Later, accurate findings by sophisticated laboratories may be helpful, especially if Late Stage symptoms appear many years after the infection.

Over the years, I have been asked to create a compendium of my published and unpublished works on the subject of Borrelia's neuropsychiatric epidemic. These literary contributions advocate for correction of medical neglect--the usually inadequate, sometimes cruel, diagnostic and treatment neglect experienced by victims of chronic Lyme borreliosis and its co-infections. I also have had articles published in an effort to attract attention from Organized Medicine—attention badly needed on behalf of a nearly invisible but serious epidemic that is more significant by far than anything this country has experienced since the Spanish Flu of 1918, the causative spirochete being less immediately deadly than was the virus of that epidemic, but deadly, nonetheless, cerebrally.

Sadly, Organized Medicine has mostly ignored or deserted the field of neuro-Lyme's immense proportions. The American public rapidly is becoming jaundiced toward doctors' lack of up-dated knowledge of spirochetal science and, having read the latest (indeed copious) peer-reviewed recent literature for themselves, are turning to other disciplines—even to veterinarians for accurate medical advice on the subject of Lyme disease and its co-infections. Veterinarians are more up to date on the diagnosis and treatment of human Lyme than the "Diagnose-and-treat-by-the-old-Guidelines" types of powerful but passé Academic physicians who cling to outdated medical dogma.

I have written about the rampant epidemiology of neuro-Lyme disease and its potent co-infections (especially the red cell parasite that causes babesiosis) and the fact that these are being systematically ignored, minimized, or distorted by this nation's overseeing healthcare agencies. Astoundingly, there are agencies that, in ignorance or arrogance, may actively persecute the victims of such borrelial, pansystematic illness, traumatizing parents and children as well as their treating physicians. There are those in authority who sponsor the official separation of children from parents whose

"Brain Disease" ...cont'd pg 7

Methylcobalamin Treatment in Lyme Patients (Methyl Vitamin B12)

by Erin Era, PharmD Candidate at the UMES School of Pharmacy Class of 2013

Lyme disease is a potentially devastating condition which can perpetuate many concurrent clinical abnormalities, including a deficit in Vitamin B12. Lyme disease is caused by the spirochete Borrelia burgdorferi which is most commonly transmitted by a bite from an infected tick[1], but recent studies do show that there is a chance that the spirochete may be transmitted by infected mosquitos as well.[5] Once this spirochete has entered the bloodstream, it can take as little as 12 hours for it to enter the central nervous system. There is now evidence that Borrelia burgdorferi produces potent neurotoxins that can cause the symptoms of encephalopathy and cause chronic inflammation, causing neurologic degeneration. There have also been studies that show Borrelia burgdorferi inhibits and kills B and T cells and decreases the count of the CD-57 subset of the natural killer cells, all of which are imperative for an adequate immune response, not only to Lyme disease which is then allowed to perpetuate, but also to other opportunistic infections that may arise.[2] Along with vital immune system components, other nutrients become depleted in the patient with chronic Lyme disease, among these is Vitamin B12.

Vitamin B12 is a supplement that has been used in the treatment of neurodegenerative diseases such as peripheral neuropathy, diabetic neuropathy, multiple sclerosis, and for the preliminary treatment of amyotrophic lateral sclerosis (ALS or Lou Gehrig's disease). A recent look Lyme disease treatment includes the addition of Vitamin B12 to help heal problems within the central and peripheral nervous system, increase immune function, regulate sleeping patterns and increase energy levels.[2] Vitamin B12 is available as Cyanocobalamin, Hydroxycobalamin, and Methylcobalamin. Cyanocobalamin is available as an over-the-counter supplement or as a prescription intramuscular or subcutaneous injection. This form of Vitamin B12 is artificially manufactured by a process that includes bacterial fermentation and the addition of potassium cyanide for stability; so, as the name would indicate, Cyanocobalamin contains cyanide. The cyanoform is an appropriate form of Vitamin B12 for most people looking to supplement their B12 intake; however, this is not the active form in the body. In order for our body to be able to use Cyanocobalamin, it needs to be converted to Methylcobalamin in the liver, the active "vitamer" and the most highly reduced form of Vitamin B12.[3] If this conversion does not happen, our brain cannot reap the benefit of methlycobalamin. For those affected by Lyme disease, using the activated methylcobalamin assists a body already in distress by avoiding hepatic conversion.

Vitamin B12 in its active form, Methylcobalamin, along with activated folic acid (L-methylfolate), are both used to remethylate the amino acid homocystine to methionine. Since adequate amounts of methionine cannot be achieved through diet alone, increasing the conversion of homocystine to methionine, through Methylcobalamin supplementation helps to supplement the amount of methionine that can be used for SAM-e (S-adenosyl methionine) production. SAM-e production is accomplished by the condensation of methionine and ATP.[4] Some of methylcobalamin's neuroprotective properties have been proposed to be dependent on this increased production of SAM-e which seems to aid in nerve regeneration and protection.[3] Along with the neuroprotective aspects of methylcobalamin, it has been shown to not only increase sleep quality, but also concentration and feeling refreshed after sleep.

Methylcobalamin is the most highly reduced form of Vitamin B12 and is already in its active form, this makes it a potent antioxidant. A patient with Lyme disease is undergoing oxidative stress, which may inhibit the ability to produce Methylcobalamin.[3] Using methylcobalamin and L-methylfolate in their "methylated/active" forms is also beneficial for a small population of patients who have a mutated MTHFR gene. A patient with a mutated MTHFR gene does not methylate well and would not be able to synthesize in vivo active methylcobalamin from cyanocobalamin or activate folic acid to L-methylfolate. For these reasons the use of methylcobalamin already reduced and in its active form may be preferred over cyanocobalamin. Methylcobalamin is a prescription medication that will need to be compounded. It is not absorbed when taken orally, so intramuscular or subcutaneous injections are the preferred delivery routes. Although injection is the preferred route of administration, sublingual tablets as-well-as intranasal sprays are available. Urine is expected to turn red shortly after each dose. If red urine does not occur, the current dose may need to be evaluated and possibly elevated.[2] For more information on how Methyl B-12 might be beneficial for you, call Community Compounding Pharmacy at 1-855-LymeRxs or send your inquiry to compounding specialist, Melissa Ruark at melissa@communitypharmacymd.com.

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Resources:

1. Lyme Disease. Center for Disease Control. http://www.cdc.gov/lyme/signs_symptoms/index.html

2. Burrascano MD, Joseph. Diagnostic Hints and Treatment Guidelines for Lyme and Other Tick Borne Illness. Advanced Topics in Lyme Disease. 2008 October, Edition 16.

3. Sharpe, Ed. Methylcobalamin and the New Story of Vitamin B12. http://health101.org/art_methylcobalamin.htm.

4. Kikuchi M, Kashii S, Honda Y, Tamura Y, Kaneda K, Akaike A. Protective effects of methylcobalamin, a vitamin B12 analog, against glutamate-induced neurotoxicity in retinal cell culture. Invest Ophthalmol Vis Sci 1997;38(5):848-54.

5. Koski-Boqacka DI, Zuzna-Grygiel W, Jaborowska M. Ticks and mosquitoes as vectors of Borrelia burgdorferi s. l. in the forested areas of Szczecin. Folia Biol (Krakow). 2007;55(3-4): 143-6.

communitypharmacymd.com
1-855-LymeRxs

Sex & Lyme Disease

by Dr. Robert C. Bransfield

How does chronic Lyme disease affect sexual functioning, and how can it be treated? Lyme can affect sexual functioning by its effect upon the central nervous system, the endocrine system, the autonomic nervous system, the peripheral nervous system, and/or the body.

It is well recognized that Borrelia burgdorferi (Bb) causes depression, obsessiveness, panic disorder, and phobias that are functions of the emotional aversive pathways of the brain. However, we can also see dysfunction of the reward pathways as well, which affect capacity for pleasure, feeding, bonding and sex. Since Lyme disease alters the aversive pathways which affect what and who we are repelled from, it is understandable that Lyme can also alter sexual attraction and behavioral patterns as well. With this in mind, I shall begin with some patient accounts and observations.

Sexual arousal:

Most patients report a decline in both libido and overall sexual functioning. Some state that their interest in sex and sexual functioning remain normal while a few report increased libido. One such patient described a greatly increased libido, but was frustrated because the multitudes of chronic Lyme disease symptom made it painful to be touched and/or hugged. Others describe increased libido associated with hypnagogic hallucinations. A patient with this symptom was described in the medical literature two years ago. She displayed sexual obsessions, sexual hallucinations, and a tendency to compulsively masturbate in a dream-like state eighteen hours per day if left undisturbed.*

Some patients develop an obsessive-compulsive disorder with sexual obsessions, compulsions, intrusive images, and vivid dreams following the onset of chronic Lyme disease. Of particular interest, a few patients report a change in the content of sexual imagery. A change to more violent sexual themes is sometimes noted. This, in turn, sometimes alters sexual behavior.

Could Borrelia burgdorferi or other infectious diseases sometimes alter sexual orientation or contribute to gender dysphoria, or altered patterns of sexual arousal? There is evidence that sexual functioning is altered by a number of other parasites, including Wolbachia, Spiroplasma, Rickettsia and Microsporidia. When Bb infections begin in childhood, are there some cases where it may have an effect upon sexual development? Is infectious disease one of the many factors that may affect sexual development? When changes in sexual imagery occur in adults, most are upset by the changes, which result in a decline of sexual interest. However, there are times when some individuals act out these fantasies.

**Stein Sara L., MD. Et al, American Journal of Psychiatry 153:4, April 1996, Clinical Case Conference "A 25- Year-Old Woman With Hallucinations, Hyper sexuality, Nightmares, and a Rash."*

Fertility:

Patients complain of infertility with surprising frequency. Is infertility more common in chronic Lyme disease patients?

Atrophy of genitalia:

A few patients who have been infected for over ten years report atrophy of the genitalia. Males have reported atrophy of the penis and testicles, a change that is reversed by IV antibiotics. Females report lack of vaginal lubrication, painful intercourse, and anorgasmia. One female patient reported atrophy of one breast.

Anesthesia of genitalia:

On occasion, some patients complain of a loss or sensation of the genitalia. I have also seen this symptom in a few chronic fatigue patients.

Orgasm-induced migraine headaches: Although uncommon, this is sometimes seen in chronic Lyme disease patients.

Lymphocytoma of the nipple: This has been reported In Europe, but I have never seen such a case in my practice.

Menstrual irregularity: A common symptom in about 50% of menstruating patients.

Breast swelling, tenderness, and lactation: Some patients complain of this symptom.

Premenstrual Syndrome:

There is a significant tendency towards worsening of the chronic Lyme disease symptoms in the premenstrual period.

Besides these symptoms associated with Lyme disease, there are many other symptoms which indirectly affect sexual functioning, i.e. - fatigue, chronic pain, depression, paranoia, hypervigilance, mood swings, low frustration tolerance, temper outbursts, apathy, etc. These mood symptoms often alienate their partners. It is no surprise that many chronic Lyme disease patients report marital discord.

Treatment:

A well-planned treatment approach for chronic Lyme disease can help the overall prognosis, thereby possibly helping any of these symptoms. The treatment of sexual dysfunction is one of the last frontiers in medicine. Three drugs for male erectile dysfunction have been approved for marketing. One was Viagra, developed by Pfizer. Loss of libido and a loss of sexual functioning are treated by a number of methods. Testosterone treatments are sometimes effective for loss of libido in both men and women. Dopamine agonists such as Wellbutrin and Parlodel are also used as treatment modalities.

More interesting than the treatment of sexual dysfunction is the question - can some individuals with abnormal patterns of sexual arousal be treated with antibiotics?

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"Brain Disease" ...cont'd from pg 5

only sin is that they persist in seeking help for their ailing children. Tragically, those authorities are empowered to permanently remove sick or partially healed young ones from their devoted families.

To their everlasting shame, medical authorities have stood by while innocent mothers have been sent to jail for insisting that their children were ill and again have stood by while the parent's belief was verified by the death of their sick child while under state "care." The rights of patients and their treating physicians have been trampled by governmental and insurance agencies in ways reminiscent of the era when AIDs was trivialized and its victims spurned as "psychosomatic." Today's infected millions worldwide show how wrong they were. The phenomenon of that epidemic is being repeated with the spread of Lyme borreliosis. My writing is an effort to illuminate this dark and now vast expanse of medicine and to inspire activism and compassion for those patients who are suffering in agony while having to hear caretakers say, "I don't know what you are worried about--you look just fine--maybe you are just depressed." Or as one unknowing, dismissive and flippant doctor joked to a frightened patient who came to him for treatment and reassurance, "Well, we all have to die of something, sometime."

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**Alan G. Barbour, MD:*

"These tick-borne infections are notable for multiphasic antigenic variation through DNA recombinations in the case of relapsing fever, the occurrence of chronic arthritis in the case of Lyme disease, and invasion of and persistence in the brain in the case of both diseases." www.ucihs.uci.edu/microbio/ .

"Lyme" ...cont'd from pg 4

peri-/myocarditis, renal insufficiency, pneumonia, encephalitis, gastrointestinal bleedings, anemia, hepatitis, myalgia

Diagnostics:

❖ PCR on Rickettsia in blood (EDTA-blood): direct detection
❖ Antibodies Rickettsia-IgM and -IgG: indirect detection - control of progression !

Therapy:

Doxycyclin/Tetracycline
Ciprofloxacin
Chloramphenicol
Erythromycin (Children)

Chlamydia pneumonia

Pathogen: Chlamydophila pneumoniae (gram-negative, intracellular)
Transmission: airborne infection (aerogen), human to human, affection of epithelial cells of air passages
Symptoms: slight throat pain, hoarseness, sinusitis, atypical pneumonia, meningoencephalitis, bronchiolitis obliterans, myocarditis, Guillain-Barre-Syndrome Post-infection (4-6 weeks): arthritis, tendovaginitis
Associations: e.g. Morbus Alzheimer, Multiple Sclerosis, Fibromyalgia, Chronic Fatigue Syndrome (CFS), problems with prostate gland, heart attacks, apoplectic stroke, arteriosclerosis
Risk factors: immunosuppression

Diagnostics:

❖ Activity test: Elispot-LTT (Lymphocytes transformation test)
❖ PCR on chlamydia pneumoniae in sputum/secretion of the throat: direct detection
❖ Antibodies on chlamydia pneumoniae-IgA and chlamydia pneumoniae-IgG: indirect detection - control of progression.

Therapy:

Macrolides (Azithromycin, Clarythromycin)
Doxycycline
Levofloxacin

Chlamydia trachomatis

Pathogen: Chlamydophila trachomatis (gram-negative, intracellular)
Transmission: sexual contact, human to human
Symptoms: cervicitis, sterility, urethritis, trachoma, acute conjunctivitis ("swimming pool conjunctivitis"), lymphogranuloma venereum
After infection(4-6 weeks): arthritis, tendovaginitis
Risk factors: immunosuppression

Diagnostics:

❖ Activity determination: Elispot-LTT (Lymphocytes transformation test)
❖ Chlamydia trachomatis PCR in urine/urogenital smear: direct detection
❖ Antibodies on Chlamydia trachomatis-IgA and Chlamydia trachomatis-IgG: indirect detection - control of progression !

Therapy:

Macrolides (Azithromycin, Clarythromycin)
Doxycycline
Tetracycline
Quinolones (Levofloxacin, Ciprofloxain, Moxifloxacin)

Mycoplasma

Pathogen: Mycoplasma pneumoniae/fermentans (gram-positive, intracellular)
Transmission: airborne infection (aerogen), human to human
Symptoms: tiredness (100%), fever, joint pain, swollen joints, muscles pain, headache, insomnia, anxiety, emotional instability, lack of concentration, lack of alertness and memory, confusion
Risk factors: immunosuppression (e.g. AIDS), Chronic Fatigue Syndrome (CFS), "Gulf War Syndrome"

Diagnosis:

❖ Bacterial culture of special nutriment medium
❖ PCR on chlamydia pneumoniae in sputum/secretion: direct detection
❖ Antibodies on Mycoplasma pneumoniae-IgM, Mycoplasma pneumoniae-IgA and Mycoplasma pneumoniae-IgG: indirect detection - control of progression.

Therapy:

Macrolides (Azithromycin, Clarythromycin)
Doxycycline
Quinolones (Levofloxacin, Ciprofloxacin)

Other possible co-infections

Yersinia enterocolitica
Toxoplasma
Hepatitis C-Virus
Herpes simplex Virus Type I/II
Epstein-Barr-Virus
HIV-Virus
Cytomegalie-Virus
Borna-Virus

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Memberships
* International Lyme and Associated Diseases Society (ILADS):
* Chairman of the international and laboratory test committee of ILADS
* German Borreliosis Society
* Swiss Association for Tick-borne diseases
* German Association of Clinical Chemistry
* German Association of Laboratory Medicine