EDITORIAL

Lyme Disease and Mycotoxicosis: How to Differentiate Between the Two

Andrew W. Campbell, MD, Editor in Chief

Clinicians search for the right diagnosis for their patients which then leads to selecting the correct treatment. The masterpiece of medicine is the cure, and comes about as a result of the clinician's skill in applying their knowledge with the aid of diagnostic tools. When the presenting symptoms of two different diseases are similar, this may lead to erroneous diagnoses and treatments to the detriment of the patient's health.

The health effects of Lyme disease and of molds and their mycotoxins frequently present in the similar manner. Patients with either disorder, or both, have the same complaints:

- Fatigue, chronic
- Muscle and joint aches and pains
- Fever
- Memory disturbance
- Confused easily or a change in the ability to learn
- Spatial disorientation
- Frequently saying the wrong word
- Anxiety and/or depression
- Personality changes and mood swings
- Sleep disturbance and unusual nightmares
- Headaches
- Changes in visual acuity and/or blurred vision
- Seizures
- Numb or tingling feelings
- Lightheadedness, feeling "spaced out"
- Loss of balance
- Ringing in ears
- Twitching muscles
- Tremors
- Painful lymph nodes
- Intolerance to bright lights
- Intolerance to alcohol
- Decreased libido
- Sore(s) that will not heal
- Rashes
- Bruise easily
- Uncomfortable urination
- Hair loss
- Shortness of breath and/or cough
- Heart palpitations
- Heart murmur and/or mitral valve prolapse
- Allergies
- Chemical sensitivities including to foods, medications, and others
- Nose bleeds
- Testicular pain/pelvic pain,
- Menstrual irregularity and/or lactation
- Weight change: loss or gain
- Nausea
- Changes in bowel habits, i.e. constipation or diarrhea
- Abdominal discomfort

These symptoms may be mistaken and frequently are for other disorders such as:

- Multiple Sclerosis
- Fibromyalgia
- Chronic Fatigue Syndrome
- Infectious Mononucleosis
- Systemic Lupus
- Alzheimer's
- Guillain-Barre Syndrome
- Lou Gehrig's Disease (ALS)
- Rheumatoid Arthritis
- Thyroiditis
- Allergies
- Chemical Sensitivity

LYME DISEASE

Lyme disease (LD) is caused by the bite of an infected Ixodes tick with resulting bacterial infection with specific spirochetes from the genus Borrelia. The effects on human health of LD vary widely and depend on the stage and extent of the dissemination of the spirochete. A homogenous erythema may be the most common presentation in North America, with target lesions reported in less than 10% of instances. In the span of a few days to a few weeks after the onset of a local infection, the Borrelia bacteria may begin to spread via the bloodstream. The effects on human health of LD vary widely and depend on the stage and extent of the

dissemination of the spirochete. In the span of a few days to a few weeks after the onset of a local infection, the Borrelia bacteria may begin to spread via the bloodstream. This spread to distant sites and organs throughout the body generate a range of symptoms. Not all patients have all the symptoms, and many are not specific to LD, but can also occur in other disease states, including those encountered in patients affected by molds and mycotoxins. To complicate the clinical picture, the incubation period can give signs and symptoms in a few days or in some cases in months or years.

The prompt diagnosis and treatment of Lyme disease is essential to avoid chronic Lyme borreliosis and its long term harmful effects in humans. The abilities of Borrelia include antigenic changes on its surface to avoid the immune system and consequently infiltrate areas of the body that have low levels of drug distribution, such as the joints and nervous system. For the clinician it is essential to combine the symptoms and physical findings with the most sensitive laboratory methodology available to accurately diagnose Lyme disease. The multi-peptide ELISA (MPE) is more sensitive than Western Blot (WB). Many borderline positive or negative by WB can be correctly classified by MPE. MPE measures antibodies against different peptides that are presented during the life cycle of Borrelia. In addition, MPE not only detects antibodies to *B. burgdorferi sensu lato*, but also against *B. b. sensu stricto*, *B. afzelii*, and *B. garinii*. These are important in the diagnosis of patients with Lyme arthritis and neuroborreliosis. In addition, MPE also tests for the three most important co-infections that can exist with LD: Babesia, Ehrlichia , and Bartonella. These are essential for the complete analysis for LD.

MOLDS AND MYCOTOXINS

According to the World Health Organization, disease caused by exposure to molds and mycotoxins is known as the "Great Masquerader" of the 21st century due to its ability to present with a number of nonspecific clinical signs and symptoms and not routinely suspected by clinicians.

It has been well documented in the literature that water intrusion from leaky roofs, pipes, windows, poorly maintained flashings, flooding from leaking washer, dishwasher, ice maker, etc. in the home as well as in the workplace cause mold growth with the subsequent accumulation and spread of mycotoxins. Reports of exposure include homes, office buildings, courthouses, hospitals, hotels, schools, and universities.

The Environmental Protection Agency, the Centers for Disease Control and Prevention, and the National Institutes of Health agree that molds start to grow and sporulate when they have been wet for 24 to 48 hours. Mold spores can be found in most indoor spaces; however, they are dormant until they come into contact with moisture or water and then they start to sporulate. Mold spores are like a packet of seeds: put them in a pot and water them and you get results.

Molds produce toxins known as mycotoxins. Molds are ALWAYS present in homes or workplaces that are water damaged and they are ALWAYS producing mycotoxins. These mycotoxins include satratoxin, T2 toxin, verrucarin, ochratoxins, trichothecenes, deoxynivalenol, gliotoxin, amongst others. It is common to find one mycotoxin being synthesized by different fungal species and one fungal species producing more than one mycotoxin.

Mycotoxins suppress the immune system through a balance of cytotoxicity and altered Th1/Th2 balance. The alteration of immune responses due to chronic mycotoxin exposures may also adversely affect the ability of the immune system to fight infections and other environmental exposures. This may explain patient complaints of concurrent susceptibility to infectious organisms and enhanced responses to chemical irritants. These effects give rise to symptoms in patients that can be vague and non-specific, just as in LD.

Blood serum testing for mycotoxin antibodies have been used for the last 20 years and are highly accurate. The specificity and sensitivity of blood serum testing for the presence of IgG and IgE antibodies to common mycotoxins in the blood are of the highest degree.

Urine testing for mycotoxins is inaccurate. According to the National Institute for Occupational Safety and Health (NIOSH), a part of the Centers for Disease Control and Prevention (CDC), low levels of mycotoxins are found in many foods. The United Nations Food and Agriculture Organization and the World Health Organization has estimated that 25% of the world's crops, such as nuts, cereals, and rice are contaminated by mold. Low levels of mycotoxins are found in many foods: cereals, meat, fruits, nuts, wine, beer, coffee, etc. For that reason, mycotoxins can be found in the urine in parts per billion in healthy people. Interestingly, albumin binds to the mycotoxin ochratoxin with unusual high affinity by binding 99.8% of ochratoxin, which is reabsorbed from all parts of the nephron by both active transport and passive diffusion, making elimination by glomerular filtration negligible. Yet laboratories providing urine mycotoxin levels routinely find high levels in urine, bringing into question the validity of their testing methodology. Mycotoxins can form adducts with human tissue and thus could be responsible for induction of autoimmunity. There are more than 80 different autoimmune diseases, and any one of these can be triggered by mycotoxins. This is another reason urine level of mycotoxins are a useless test, but measurements against mycotoxins antibodies is clinically much more meaningful.

LYME DISEASE AND MYCOTOXICOSIS

In approaching a patient with the above listed vague and general complaints, it is vital that the clinician make an accurate diagnosis. A number of these patients have been given little hope by conventional doctors, who sometimes tend to classify them as having psychosomatic disorders.

Can a patient have both LD and mycotoxicosis? Yes, and patients who are treated for one and have had a poor response or lingering symptoms may have the other. In my own clinical experience I have seen a number of patients who have been

treated for years for LD including with intravenous antibiotics do well after a diagnosis and treatment for mycotoxicosis has taken place. In the reverse, patients who after undergoing treatment for mycotoxicosis and who are still faring poorly did quite well after testing and treatment for LD. It may be difficult to determine which came first. In one case, a patient had water damage and moved out after several weeks of exposure to molds and mycotoxins to a camper while remediation was being done and had both diagnoses. The tick bite apparently may have occurred when the patient walked daily through a grassy area from the camper to where his car was parked.

Both diseases share the commonality of symptoms and in many cases physical findings. The use of the proper laboratory test is essential to differentiate one from the other. (*Altern Ther Health Med.* 2019;25(4):8-11).

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PERSPECTIVES

The Gonzalez Best Case Series Presentation to the NCI: 25 Cases, 25 Years Later

Linda L Isaacs, MD

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(*Altern Ther Health Med.* 2019;25(4):12-14).

The publication of the monograph *Proof of Concept: 25 Best Cancer Cases Presented to the National Cancer Institute* by New Spring Press provides another facet of the accomplishments of the remarkable man who wrote it, my long-time colleague and friend, the late Dr. Nicholas Gonzalez.[1] Gonzalez had been successful in the world of journalism before he decided, in his late 20s, to become a physician. While in medical school, he encountered the work of Dr. William Donald Kelley, an orthodontist who had developed a nutritional method for the treatment of cancer and other illnesses, involving individualized diet and supplement protocols, high dose pancreatic enzymes, and various detoxification routines. After reviewing Kelley's records, Gonzalez dedicated his life to preserving this treatment method and to trying to get it properly scientifically evaluated.

Gonzalez' findings about Kelley's practice would eventually be published, 25 years later, as the book *One Man Alone*.[2] But at the time Gonzalez finished his investigation of Kelley's results in 1986, no journal editor or book publisher was willing to accept that the case reports were real, to examine the medical records supporting the cases, or to take the risk of antagonizing others in the medical world. I was already working with Gonzalez at that time; I remember the numerous submissions of case reports and manuscripts, and the disappointment building as the rejections came. Finally, Kelley began to mistrust us, and it became clear that Gonzalez and I could no longer work with him. Since Kelley was no longer seeing patients, Gonzalez decided that he should try to recreate the protocol, in the hope of collecting more data to further document the treatment's potential, and that someone other than Kelley could implement it.

In the fall of 1987, Gonzalez began seeing patients in New York City, in the office of a physician friend. I helped out administratively until I resumed my internal medicine residency in June 1989. Those were difficult times, with limited resources. Gonzalez wrote instructions for diets and detoxification protocols, decided what supplements to use, and figured out how to get them distributed. He got publicity through contacts from his journalism days such as Dr. Robert Atkins. Most challenging of all was patient care. We had to learn the hard way the limitations of the treatment method and of our own stamina. In the early days, Gonzalez was making house calls on patients who in retrospect were simply too ill to benefit. Both Gonzalez and I were tempted on many occasions to quit, but invariably, when we were despondent over one poor outcome, shortly afterwards we would get good news about another patient who was improving.

In 1991, I finished my internal medicine residency, passed my boards, and rejoined Gonzalez, but there was no room for me to see patients in the office he rented. While Gonzalez saw patients, I reviewed his charts, looking for remarkable outcomes and incomplete records, and sent out release forms to other treating physicians, radiology facilities, and hospitals. All this proved invaluable when Gonzalez was invited to present a Best Case Series in 1993 at the National Cancer Institute.

For his presentation, Gonzalez was determined that every detail would be in place. We felt a single bit of missing data could provide an excuse for someone to criticize his selected cases. It became my mission to track down actual X-ray and CT films, to get pathology slides, and to be sure every relevant document was included. Meanwhile, Gonzalez continued to work long hours seeing patients and returning phone calls, while writing a monograph describing the cases in his presentation. We had it printed and bound, and distributed it to the attendees at his presentation, with patient names and identifying information intact in the medical records included.

In his introduction, Gonzalez discussed supporting evidence for the treatment, including the work of Dr. John Beard, who first suggested that pancreatic enzymes could be used to treat cancer.[3] Beard's thesis centered on the similarity in appearance and behavior of cancer cells to the trophoblast, the earliest stage of the mammalian placenta. Beard had observed that the trophoblast moderated its aggressive, invasive nature around the time the fetus began making pancreatic enzymes. He speculated that pancreatic enzymes