

“Bell’s Palsy of the Gut” and Other GI Manifestations of Lyme and Associated Diseases



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Bell’s palsy signifies paralysis of facial muscles related to inflammation of the associated seventh Cranial Nerve. Physicians may not realize that this syndrome is caused by the *spirochetal agent of Lyme disease until proven otherwise*. Whether it is a full or hemi-facial paralysis, Bell’s palsy is cosmetically disfiguring when fully expressed. Sudden loss of normal facial expression terrifies patients who naturally fear they are having a stroke. When a smile is asked for, normal countenances warp into bizarre grimaces. The amount of tooth area exposed in this attempt to smile helps doctors evaluate the degree of paralysis and its change over time (Figure 1). In every case of Bell’s, doctors need to carefully investigate by history, physical, and laboratory work every shred of evidence that might suggest the presence of cryptic tertiary Lyme, a serious multi-system, gut and neuro-brain infection even though about half of fully diagnosed patients have no evidence whatsoever of having had a tick-bite.

Gastrointestinal Lyme disease may cause gut paralysis and a wide range of diverse GI symptoms with the underlying etiology likewise missed by physicians. *Borrelia burgdorferi*, the microbial agent often behind unexplained GI symptoms—along with numerous other pathogens also contained in tick saliva—influences health and vitality of the gastrointestinal tract from oral cavity to anus. Disruptions caused by GI borreliosis (Lyme) may include, amongst many others, distortions of taste, failure of other neural functions that supply the entire GI tract—paralysis or partial paralysis of the tongue, gag reflex, esophagus, stomach and nearby organs, small and/or large intestines

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(“ileus”), bowel pseudo-obstruction, intestinal spasms, excitability of gut muscles, inflammation of lumen lining tissues, spirochetal hepatitis, possibly cholecystitis, dysbiosis, jejunal or ileal incompetence with resultant small intestine bacterial overgrowth (SIBO), megacolon, encopresis and rectal muscle cramping (proctalgia fugax).

In cerebral hypothalamic and pituitary centers, usual sites of borrelial disruptions of the brain’s normal hormonal cascades, there are strong influences on human attitudes, ideation, and behavior relating to gastronomic issues. Newly discovered Lyme-endangered cerebral hormones and renegade cytokines regulate brain-gut interactions thus initiating behavioral tendencies such as anorexia or a failure of satiety with resultant obesity.

Ticks and other vectors of Lyme disease attract their own infections from many microbes, some known and some unknown (viruses, amoebas, bacteria, and possibly parasitic filaria), which they then also can pass on to humans. The GI tract is especially vulnerable to machinations of such co-infections as bartonellosis, mycoplasmosis, human anaplasmosis (HA), and human monocytic ehrlichiosis (HME). Syndromes exactly similar to Irritable Bowel Syndrome (IBS), Crohn’s Disease, and cholecystitis, for example, may not have readily suggested a borrelial etiology to the diagnostician but Lyme increasingly is known to be a potential contributor to each.

All known Lyme-gut syndromes are treated by combining several effective antimicrobials (including use of azole medications with specific antibiotics) with agents that boost gut lining repairs and overall immunity enhancement. Azole medications are borrelialcidal (against the anti-*Bb* spirochetal cyst form) medications such as metronidazole (Flagyl). Needed GI healing agents may include gut stimulants or relaxants, Ph agents, bile salts, nutraceuticals, immunity-enhancers, neurotoxin absorbents, and sterilizers of gut-specific microbes.

Parallelism between Lyme borreliosis-caused paresis of facial muscles supplied by Cranial Nerve VII and Lyme-caused gastrointestinal paralyses suggested a pseudonym to the author—*Bell’s palsy of the Gut*—despite the fact that these syndromes are related to different types of neural fibers and only occasionally occur together. Since similar injury to all sites may be etiologically related, however, otherwise unexplained *gastrointestinal symptoms should be considered as possibly related to Lyme borreliosis and/or its co-infections until proven otherwise.*

INTRODUCTION

Until proven otherwise, a patient’s unexplained facial paralysis is caused by the tick-borne spirochetes of Lyme disease (LYD) (1). The widely endemic bacteria are easily capable of inducing distal inflammation of the Seventh Cranial (Facial) Nerve (2). “Considering the incidence of Bell’s palsy in Lyme, it is improper to treat it as viral in origin without a work-up for Lyme disease” (3). In an early study with nearly 1000 LYD cases studied, Bell’s palsy occurred in at least 10% of validated cases (4). The fre-

quency of Lyme’s Bell’s palsy etiology is unfamiliar to many physicians. Likewise many physicians are unfamiliar with the spirochetal cause of paralyses of muscles that facilitate normal gastrointestinal transit. Yet, these vital muscles also may be greatly compromised by the same offending neurotropic spirochete, *Borrelia burgdorferi* (*Bb*) in patients who are totally unaware of having Lyme disease. Their physicians are often surprised to learn that persistent Lyme disease is outstandingly a disease of the brain as well as involving one or all components and sub-systems of the entire

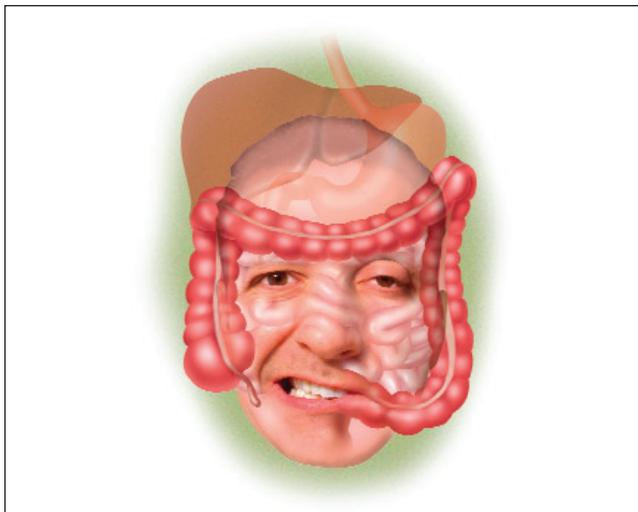


Figure 1. The author’s conception of a patient suffering with “Bell’s Palsy of the Gut.” Original art by Guiseppa Giampaolo, Willow Grove, PA.

nervous system (5). It is not yet widely understood by clinicians that at least 40% or more of Lyme-infected patients have major, handicapping, neurological manifestations (6,7) with the likelihood that 100% have some brain involvement. It remains to be clarified which *Bb* neuritides are involved in specific GI sequelae of the infection or if inflamed nerves are, indeed uniformly at fault.

“The vagi (10th Cranial Nerves) are major suppliers of the gut’s external nervous system and being very long and complex, are vulnerable to neuropathies such as Lyme disease or diabetes which can cause them serious damage.” (Personal communication from Neurologist, Richard Rhee, M.D., F.A.A.N., Neptune, NJ)

“Vagus nerve paralyzes are more commonly diagnosed when caused by Herpes (varicella) zoster or Herpes simplex viruses wherein most patients I have seen are nauseated and have no appetite. I have not observed paralytic ileus in these cases. Should vagal paralysis occur in a Lyme patient, I think the patient would complain of hoarseness and dysphagia.” (Personal communication from Dr. Hidecki Nakagawa, Japan) Indeed, both of these problems are common symptoms of neuro-Lyme.

“The autonomic nervous system supplies the gut . . . sympathetic fibers inhibiting peristalsis and secretion and parasympathetic fibers increasing

them . . . Functions of the sympathetic nerves include vasomotor, motor to the sphincters, inhibition of peristalsis, and transport of sensory fibers from all of the abdominal viscera. . . . Functions of the parasympathetic nerves comprise motor and secretomotor to the gut and glands” (8).

Borreliosis-caused, gastrointestinal tract paralysis and related abnormalities can occur anywhere along the entire length of the tract (9,10)—involving, for example, functionality of taste buds (11,12), muscular strength of the tongue, gag reflex, ability to swallow, gastroparesis, peristaltic retardation (or excitation) related to small bowel competency, dysbiosis, total arrest of peristalsis (“ileus”), pseudo-obstruction (sometimes associated with Bell’s palsy) (13), colon dysfunctions, encopresis, proctalgia fugax and the final act of defecation. “In 5%–23% of patients with early Lyme borreliosis, there can be gastrointestinal symptoms such as anorexia, nausea, vomiting, severe abdominal pain, hepatitis, hepatomegaly and splenomegaly. Diarrhea occurs but is seen in only 2% of cases” (14). Regardless of the site, spirochetes’ disturbing symptoms may come and go spontaneously, often temporarily resolving in a matter of hours to days, although resolution does not imply cure. As with Bell’s palsy of the face, these gastrointestinal conditions may endure or only partially remit (15).

Similarities between *Bb*-caused paralyzes of muscles supplied by the Facial Nerve and Lyme-caused GI neurogenic paralyzes suggested a pseudonym to this writer—*Bell’s palsy of the gut*—despite the fact that the two manifestations of the infection may not be synchronous. Yet, they are etiologically related, which suggests need for a high index of suspicion regarding presence of borrelial disease in all perplexing gastrointestinal syndromes.

LYME AND ITS POTENT MICROBIAL CO-INFECTIONS AS RELATED TO GEOGRAPHIC FACTORS

Endemic areas for tick-borne diseases include the entire Eastern and Western coasts of North America with their internally contiguous states as well as Midwestern states that support migratory bird North-South flyways

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Figure 2. *Ixodes scapularis* mating. Photo courtesy of the Lyme Disease Association’s photographer, Jim Occi.

(16). Infected deer ticks (*Ixodes scapularis* and similar hard-bodied ticks), vectors of many diseases including the ones discussed below, are thus most widely distributed by birds, geographically. There are few places in the United States that are totally safe from the risk of microbes thus ferried. In 2002, the CDC estimated the existence of nearly one-quarter million new cases in USA’s rapidly expanding LYD epidemic.

Very common co-infections from infected *Ixodes sp.* ticks (Figure 2) include the ehrlichioses—Human Granulocytic Ehrlichiosis, which recently was renamed Human Anaplasmosis (HA) and Human Monocytic Ehrlichiosis (HME). Human babesiosis, a tick-borne, one-celled parasite of erythrocytes, is



Figure 3. Nymphal tick (*I. Scapularis*) compared to a dime. Used with appreciation from the Iowa State university Entomology Department. Special thanks to photographer, Jim VanDyk.

widely misdiagnosed in its endemic, chronic form (17,18). A *Bartonella*-like bacteria, mycoplasma spp, and other viral and opportunistic infectors are now known to be tick-borne (19), existing in the full territorial range of *I. scapularis* and other ticks (20–22). Resultant illnesses include two that have been found to be the most common tick-borne invaders of children’s gastrointestinal tracts—the combination of bartonellosis and Lyme borreliosis gut infections (23).

As with the spirochetes of Lyme, *Bartonella* is an increasingly common (perhaps the *most* common) tick infector (21). “PCR analysis of *Ixodes scapularis* ticks collected in New Jersey identified infections with *Borrelia burgdorferi* (33.6%), *Babesia microti* (8.4%), *Anaplasma phagocytophila* (1.9%), and *Bartonella* spp. (34.5%). The *I. Scapularis* tick (Figure 3) is a potential pathogen vector that can cause coinfection and contribute to the variety of clinical responses noted in some tick-borne disease patients” (24). As more experience has been gained with *Bartonella henselae* and its related species, bartonellosis has been found capable of causing severe gastrointestinal pain and malfunction as well as specific skin eruptions. Both of these sites involve vasculopathy—enteric and dermal as well. Scar-like stripes on the patient’s torso are telltale “stretch marks” or “scratch marks” of the disease, easily notable. This external and visible sign (the seemingly mysterious but diagnostically pathognomonic striae) may make the GI bartonellosis diagnosis less complicated for gastroenterologists and other specialists (25).

Quite surprising to many physicians, bartonellosis can cause major central nervous system damage, similar in some aspects to the aforementioned Lyme neuroborreliosis. Lyme and bartonellosis symptoms may include encephalitis signified by headaches, major memory loss, rages, seizures, and coma, as well as inflammation of the heart, abdominal pain, bone lesions, and loss of vision. Until recent years, *Bartonella*, at onset of infection an endothelial and subsequent red blood cells infector, was considered to cause a relatively benign and common disease otherwise known as cat scratch disease (26–28). Now that ticks have become significant transmitters of *Bartonella* infections into humans, this vectoring appears to amplify victims’ general Lyme symptoms (26), and quite likely amplifies GI tract lining symptoms as well.

OFTEN UNSUSPECTED PRESENTATIONS OF GI TRACT LYME—DIAGNOSTIC USEFULNESS OF PCR TESTS ON SPECIMENS HARVESTED FROM ENDOSCOPY/COLONOSCOPY BIOPSIES (WITH ILLUSTRATIVE CASES)

One of the blessings of modern medical investigation is a positive PCR (A direct test—polymerase chain reaction—capable of pinpointing an offending microbe’s DNA). This test can be performed on specimens from the patient’s blood, serum, plasma, CSF, urine, mothers’ milk, and all biopsy tissues. PCRs can play a vital role in diagnosing tick-borne diseases especially those affecting any organs or associated tissues. “*Lyme disease is usually diagnosed and treated based on clinical manifestations. However, laboratory testing is useful for patients with confusing presentations and for validation of disease in clinical studies*” (29).

DNA tests are especially handy because they can be utilized by way of biopsies harvested from inside the gut during otherwise routine colonoscopies and endoscopies in cases where the diagnosis is uncertain. PCR’s are highly *specific* although they are less than ideally *sensitive* so that a positive test is a reliable indicator of *Bb* infection while a negative test simply does not exclude Lyme and does not indicate a lack of infection (30).

An illustrative case history is that of “Mr. F,” a mature man thought to have been mentally retarded most of his life. His father had ascribed his youth’s sudden headaches, stiff neck, and cognitive losses to the will of God. No further evaluation or treatment was allowed. They lived in endemic tick territory at the time. Decades later the patient realized that his symptoms back then followed a series of bites by minute ticks). Now an adult, the patient’s chronic “ulcerative colitis” and depression kept him from his job as a school janitor. (Antidepressant medication had mostly just helped his anxiety) When a colonoscopy was needed, a generous gastroenterologist biopsied Mr. F’s luminal tissues, which the referring doctor then sent for testing to a reference lab specializing in tick-borne diseases. Specimen analysis returned as PCR positive for etiologies of 3 diseases that infected his colon: *Borrelia burgdorferi* (Lyme disease), *Mycoplasma fermentans* (suspected of causing GI injury via proinflammatory cytokines) (25), and *B. henselae* (bartonel-

losis). Each disease required its own unique treatment, all of which were successful and the patient’s GI symptoms resolved. Mr. F’s depression also cleared and in its place there was a kind of chronic good cheer, off and on resembling mild hypomania.

The case of “Mrs. M” illustrates another important method of detecting the presence of an active Lyme infection as well as uncovering a possible contributing cause of cholecystitis. Gall bladder (GB) tissue was tested for *Bb* spirochetal DNA following a cholecystectomy on this seronegative patient: A middle-aged woman with a known diagnosis of pre-existing, asymptomatic gallstones, experienced episodes of allergies, severe headaches and extreme chronic fatigue. She was treated for 2 tick-borne diseases—LYD and babesiosis, having had symptoms of both and a positive PCR blood test for babesiosis. The LYD was treated with oral antibiotics and then 3 months of IV ceftriaxone (Rocephin) following which she showed improvement.

About a year later, Mrs. M, again fatigued, developed right shoulder blade pain and afebrile nausea after eating greasy foods. Surgery to remove her diseased gallbladder was scheduled. Treatment (doxycycline) for suspected but unproven *persistent* Lyme was begun. The family physician asked that biopsy specimens of the removed gall bladder be tested in a reference laboratory specializing in tick-borne diseases (31). The resultant PCR test on her gall bladder tissue was *positive* for DNA of the causative *Bb* spirochete of Lyme disease. This PCR biopsy confirmation of a *seronegative* patient’s Lyme diagnosis illustrates that, while Western Blot and PCR *blood* sample testing, especially for active late stage LYD, may not show a positive antibody response, a *tissue* PCR analysis may confirm the diagnosis, even when the patient has previously been treated. PCR’s done on blood are less satisfactory since *Bb* prefers an in-tissue environment. Treatment of Lyme disease by IV Rocephin can lead to gall bladder sludging. In this case the GB stones were considered to have predated the IV treatment. Of interest, a similar spirochetal disease (leptospirosis) has been reported as simulating symptoms of cholecystitis (32). This may be the first confirmation of a diagnosis of Lyme disease performed on GB tissue to be pub-

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lished—its write-up has been submitted for publication. (Case and personal correspondence from Sabra Bellovin, M.D., Portsmouth, VA)

In another instance, “Mrs. E” was evaluated in a psychiatrist’s office for severe depression, anxiety, and fatigue some months following successful removal of a colonic polyp. She mentioned that she had been experiencing chronic, depleting, diarrhea and severe insomnia. Biopsy tissue was then obtained from a repeat colonoscopy by a cooperating gastroenterologist. The specimen was PCR positive for an unspecified *Mycoplasma*. *M. Pneumoniae* is a known gut epithelial lining pathogen (33) and *M. fermentans* has been found in inflamed gastro-enteric linings (19). Both potentially pathogenic mycoplasmas have been documented as carried by ticks. In addition, Mrs. E’s blood tests revealed the presence of high antibody titers for ehrlichiosis (Human Anaplasmosis—HA) as well as positive Western Blot (WB) tests for Lyme disease, indicating active cases of both when tested in a related specialty laboratory (34). Interestingly, Mrs. E’s family physician in Pennsylvania was willing to treat the ehrlichiosis but unlike some more southerly PCP’s (35) she thought Lyme was confined to New England and was unwilling to treat her patient’s borreliosis.

Treatment of active Lyme disease is often denied to very sick patients with or without the presence of positive test findings. Serologic testing for Lyme disease as routinely performed by local laboratories is well known for insensitivity. The CDC surveillance case definition *excludes*, for example, as many as 78% for IgG of known positive cases (36,37). More modern guidelines are currently available for diagnosis and treatment of tick-borne diseases (38,39).

Because the recommended first-use enzyme-linked immunosorbent assay (ELISA) test tends to miss at least 50 % of authentically positive Lyme cases, it is less likely to be relied on (29,40). ELISA tests were not performed in any of the cases presented here.

LYME-ASSOCIATED MOTILITY VARIATIONS AND OTHER BB RELATED GUT PROBLEMS

A suddenly spastic or immobile esophagus or similar paralysis of the stomach muscles may represent esophageal and/or gastric paresis or spasm from Lyme

neuropathies (5). Infection influencing the vagus nerves has been documented to cause paralysis in other diseases (8). Additional *Bb*-related symptoms may manifest as gastroesophageal reflux disease (GERD), early or absent satiety, GI bloating, nausea, vomiting, and atypical colitis wherein the pANCA test may be helpful. If Crohn’s and colitis are considerations, a Prometheus first step may help to support this diagnosis; however tissue biopsy is necessary to confirm the diagnosis. (Personal communication from Martin D. Fried, MD, FAAP, Colt’s Neck, NJ)

As noted, neuropathies can result from the immune (cytokine) system over-activation often seen in chronic Lyme cases. This may lead to prolonged inflammation with resultant damage to the enteric nervous system and/or the autonomic nervous system supplying the gut (5). In addition, possible spirochetal paralysis of the vagal nerve(s) may cause temporary or long-lasting disruption of normal small intestinal mobility, and that, in turn, may lead to Small Bowel (or Intestinal) Bacterial Overgrowth (SBBO or SIBO) (41). SIBO can be a serious and difficult-to-eradicate infection. The colon microbes involved usually have migrated backwards to small bowel areas from their original site of benign bacterial growth following loss of competent peristaltic rhythm in a now partially compromised small bowel. This overgrowth of upwardly mobile but misplaced bacteria may greatly interfere with the normal absorption of nutrients from the small intestines causing dysbiosis and various forms of malnutrition among other mischief. Bacterial overgrowth in the small gut can result in remarkable, intermittent, immense, abdominal bloating/distention with or without eructation or flatulence (42). Such disruption may occur despite the fact that small bowel muscles have their own enteric enervation and could function independently to some degree. In many cases, the diagnosis of SIBO is verifiable by the *Hydrogen-Lactulose Breath test*, which can reveal excess hydrogen production from the relocated colon bacteria. Related test kits are offered to outpatients upon physicians’ requisitions by Genova (aka Great Smokies) (43) and Doctor’s Data (44) Laboratories, thus allowing the unassisted patient to complete the test at home and mail it back to the lab.

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Another borrelial cause of massive increases in abdominal girth associated with “gasless” bloating may cause diagnostic confusion. Unrelated to gut symptoms from Lyme’s disruption of the body’s *internal* “wiring,” *Bb*-inflicted polyradiculopathies of T7-12 (nerve root inflammations) may result in paralysis of external abdominal muscles such as the rectus abdominus. This in turn can also lead to the appearance, not the reality, of extensive bloating. No exercise “crunches” will alleviate this distention even for a previously well-toned individual. Antibiotic treatment for borreliosis may resolve this symptom (45, 46).

A diagnostic tip-off to the presence of LYD (and/or bartonellosis) may be a concomitant hypersensitivity of the chest or waist area skin in combination with distended belly from weakened abdominal wall muscles (47). One may hear from a child with unrecognized tick-borne disease, “*I can’t stand anything touching the front of me.*” Or, “*My clothes have to be real tight*” or “*I will wear only these (very loose) clothes.*” Parents of children with Lyme disease are often bewildered by apparent compulsions such children may develop while trying to get dressed in the morning. Catching the school bus on time can result in chaos as the harried parent attempts to ready a child when the child is not known to be Lyme- or bartonella-compromised.

Adynamic or paralytic ileus, a non-obstructive motility failure (suddenly “silent” intestines), may occur as a result of neuroborreliosis on an intermittent basis, with resultant abdominal distention. As mentioned, these functional lapses and pseudo-obstructions from faulty gut motility may be due to direct spirochetal or other microbial invasion with resultant tissue inflammation, or to noxious influences of cytokine (immune system) reactions, or to microbe-produced neurotoxins that can affect Central, Somatic, Autonomic (parasympathetic or sympathetic), and Enteric nervous systems that supply the GI tract.

In children and in adults who unknowingly have been inoculated with *Bb* spirochetes, etc. from ticks or from bites of other less common Lyme disease vectors such as horseflies, deer flies, or even mosquitoes (48), the resultant altered gastrointestinal motility symptoms may be mild to life-threatening. (Ehrlichiosis has a 5% mortality rate in children.) Students are frequently

reported to the office as having persistent stomach pain (“belly aches”) (49), failure to thrive, reluctance to go to school (their behavior often incorrectly labeled psychosomatic, attention-getting or amotivational), or as adults, patients may be fearful of going out to eat or to work due to an apparent “Irritable Bowel Syndrome.” These latter borreliosis symptoms are a result of visceral hypermotility instead of paralysis. In addition, the patient may have bloody diarrhea reminiscent of Crohn’s disease, or of colitis (50). As in the case of *H. pylori*’s discovery as a cause of gastric ulcers, suspicion amongst researchers is growing in regard to “stress” as the cause of IBS. And, Crohn’s Disease is now considered etiologically related to a pre-existing (unspecified) gastroenteritis (51).

Constipation of an unusual type can occur in a LYD patient who is not prone to having sluggish bowel movements. The stool can suddenly become putty-like, unresponsive to usual laxative treatments. Even massive efforts to relieve this obstipation using all vigorous conventional methods may not suffice. In addition, many patients with gastrointestinal Lyme disease develop symptoms reminiscent of Sprue/ceeliac disease and/or lactose intolerance all of which may improve somewhat when treatment for the underlying infection(s) is successfully concluded.

THE MOLECULAR BRAIN AS A GUT-INFLUENCING ORGAN

Another site of *Bb* spirochete-caused neuron damage that likely affects the GI tract is the human brain—especially its Lyme-injured hypothalamic and brain stem melanocortin circuits. “*Melanocortins are small protein molecules that carry messages between nerve cells in the brain. They are involved in regulating a variety of complex behaviors, including social interactions, stress responses and—most importantly in this context—food intake. So it is easy to see how interference with them could cause anorexia and bulimia . . . Anorexia and bulimia may be autoimmune diseases—and so may several other psychiatric illnesses*” (52). This passage refers to the work of scientists from the Karolinska Institute in Stockholm, Sweden, who have been looking at possible connections between different gut bacteria

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and autoantibodies against melanocortins to see if they can determine which bacteria might be responsible for a variety of eating disorders. They are finding that the level of autoantibodies to melanocortins is positively correlated with anorexia, but inversely correlated with bulimia (53). When melanocortins are pathologically over or under-activated, either stimulation of hunger or of food avoidance may result. The former leads to hyperalimentation and obesity (54). The latter leads in some cases to anorexia nervosa and other health problems. Brian Fallon, MD, and other psychiatrists have long noted that when their neuro-Lyme patients are treated with antibiotics for the underlying chronic *Bb* infection, there is significant improvement in eating disorder symptoms (55). Bell’s 7th and the vagus’ (10th) Cranial Nerve pathologies, brain molecular distortions, gastrointestinal disruptions, and human behavioral idiosyncrasies are all perceived of as interrelated.

ADDITIONAL DIAGNOSTIC HINTS

Patients with a Lyme disease-related facial paralysis may not have positive antibody laboratory tests for borreliosis as is often also true of those with gastrointestinal neuroborreliosis. Despite those facts, it is imperative that the multi-organ infecting microbes associated with such dysfunctions be suspected and treated if they are likely to be present—but the prescription of immunity-lessening steroids should never be used *routinely* to decrease symptoms (56). Neuro-Lyme is mid-or-late-stage (tertiary) Lyme disease, which may account for the lack of positives on many antibody tests (antibodies having been depleted by *Bb*, an ace immune system disabler.) Commonly, active tertiary Lyme shows a diagnostic positive IgM response that is conventionally but mistakenly thought to be a marker accurate only in relatively early infection (57). Persistence of a positive IgG WB test is most often seen in those with predominantly arthritic forms of Lyme disease (58).

Although the tests should be run, attempts to check for positive DNA is time consuming with results rarely coming back inside of several weeks. Yet, the patient needs immediate treatment. That same dilemma confronts both the patient with Seventh Cranial Nerve palsy as well as the enterically compromised patient. If paresis or spasm occurs and the esophagus stops func-

tioning, a patient may choke on recently swallowed food or fluid. If it occurs in the stomach, it may cause nausea and gnawing abdominal pain. If even a partial paralysis occurs in the small intestines, SIBO (SBBO) with bloating of immense proportions may ensue. Paresis of the colon may result in mega colon with severe constipation and/or encopresis even in very young children in Lyme-endemic regions. Diarrhea resembling an IBS-like syndrome can occur if there is *Bb*-sponsored gut hypermotility. Similarly, GI spasms may also result in a plethora of symptoms, including spastic colon and seeming occlusions. A trial on antimicrobials is helpful for those suspected of having tick-borne diseases despite negative tests. The “symptom intensification syndrome” known as a Herxheimer reaction needs to be anticipated by both doctor and patient as potentially distressingly difficult but is to be expected when immune systems over-respond to a spirochetal die-off. This reaction should not be confused with an allergic reaction to the antibiotic.

Most helpful diagnostic tests for Lyme disease are the direct or photographed observations of a “Bulls Eye’s” circular or oval skin rash. Unfortunately, it is only present in roughly 50% of known cases. If the lesion slowly expands (due to spirochetes multiplying in the outer edge, which fact allows easier biopsy and culture) it is perfectly diagnostic of Lyme disease or its associated “STARI” (Master’s disease—a form of Lyme disease.) In endemic areas, patients should be coached to photograph any suspect rashes and to keep the living tick for a doctor’s observation or *Bb* DNA testing. Western Blots (WBs) are best done in a reference lab specializing in tick-borne diseases with the doctor’s insistence that *all* antibody bands be counted and reported. The tests should employ the correct strains of *Borrelia* and also not depend on spirochetes that have lost DNA due to multiple passes through a series of hosts.

Acceptable tests have both high specificity and sensitivity. For example, the C6 Peptide/Lyme test has excellent specificity so that those tests that come back positive *are valid* and are confirmatory of Lyme’s presence. However, negative results from the C6 test merely show that the test was done—they do not show that *Bb* was absent. The negative test does *not* prove that the patient is free of Lyme disease.

Useful tests include a urine *Bb antigen* test with positive findings backed up by the highly accurate Southern Blot test. As noted, PCR tests on all appropriate tissues/fluids, especially serum, whole blood, urine, tears, mother’s milk and CSF are valuable diagnostically.

Choices of tests for several *Bb*’s co-infections are enhanced by awareness of the prevalent strain/species of the infection that is extant in the area where the patient was tick-inoculated. Tandem IFA and PCR tests are usually performed for co-infections. In addition, florescent microscopic views of stained slides can show babesiosis ring forms inside RBC and other tests can show cystic forms of *Bb* under black light. Bartonellosis can be tested for by PCR (blood and tissues) and its positive WBs are considered diagnostic when combined with history and physical evidence. As is true of *Bb*, however, bartonella patients may be seronegative and without PCR-DNA captured.

A BRIEF OVERVIEW OF SOME APPROACHES TO THE TREATMENT OF TICK-BORNE DISEASES AFFECTING THE GUT

Sensations of total, dire, overwhelming, unending, weakness or fatigue in most seriously ill Lyme patients lead many Lyme patients to consider suicide. Treatment begins with educating them about the treatable, underlying diseases and about realistic expectations in order to inspire hopefulness for recovery. The physician’s listening skills and willingness to give anxious patients extra time can be life-saving.

Prescription of skillfully combined oral antibiotics in an attempt to avoid IV treatment for all but those seriously afflicted with advanced neuro-Lyme (patients that manifest MS-like or ALS-type symptoms) is the next challenge (59). In addition to the usual antibiotics advised for Lyme disease, telithromycin (Ketek) used cautiously or azithromycin (Zithromax) may successfully accomplish blood-brain tissue barrier penetration that is needed. Such patients have to be monitored closely for liver, etc. side effects. In recent years, Lyme expertise has included the combining of antibiotic(s) with those in the azole family of drugs (such as metronidazole/Flagyl) that penetrate cell wall-less cyst forms of *Bb*, forcing spirochetes out

of cover as it were to their demise from the antibiotics. Regularly spaced “safety blood work” must be regularly ordered for all patients who require long-term use of any antibiotics. For those with Lyme-sluggishness of the gut with resultant SIBO, non-absorbable, intestinal “antimicrobials” likely will be needed (60). Current usage of rifaximin may include carefully monitored long term prescriptions.

Doxycycline has the advantage of being able to arrest both Lyme and the ehrlichioses in those who are multiply infected with each.

Bartonella (the tick-borne variant) usually responds, albeit slowly, to aggressive treatment by one of the quinolone family of antibiotics such as levofloxacin (Levaquin) or by rifampin (Rifampicin).

Mycoplasmas may respond best to tetracycline, rifampin, and erythromycin.

Babesia, the red blood cell parasite, requires different approaches for acute and chronic disease stages. In chronic babesiosis, the form incidentally seen by gastroenterologists, a combination of artemisinin, atovaquone (Mepron) or Malarone, a combination of atovaquone and proguanil hydrochloride, and azithromycin are still drugs of choice (61).

NUTRICEUTICALS AND ANTIMICROBIALS TO RESTORE THE IMMUNE SYSTEM AND THE GI TRACT

Restoration of gastrointestinal systems damaged by tick-borne diseases can be a formidable task depending on the presentation and severity of symptoms, antimicrobial or other treatments involved, and any side effects thus incurred. The goals are to enhance gut motility or reduce spasticity, remove toxins, improve patients’ general and gut-lining immunity while killing off invaders such as tick-borne microbes, fungi, and other gut opportunists (62,63).

Painful rectal area muscle spasms in Lyme patients usually respond to alprazolam (Xanax) 0.25 mg (½ to one tablet) best chewed for quick relief and *Natural Calm*, a formulary of instant release, water-soluble magnesium. Rectal cramps probably can be prevented most of the time by using the highest tolerated doses of daily magnesium—slow release is the recommended approach but many patients also need the quick-acting

powder at bedtime to prevent all kinds of Lyme-caused muscle cramping or spasms.

Dietary intake of all sugars and non-complex carbohydrates should be totally avoided while patients take antibiotics. **Probiotics**—high quality lactobacillus (2 enteric-coated pearls) once or twice daily or more as needed and bifidus (at least one cap) once daily are essential for gut protection during and following antibiotic treatment. **Immunity and energy enhancers** such as extract from reishi mushrooms, *Cordyceps sinensis* (at least one 740 mg capsule daily), Co-Enzyme Q10 (100 mg twice daily), green tea, acetyl L-Carnitine (500 mg at least twice daily), Vitamin B Complex-50 to 100, folate, sublingual B₁₂, magnesium (slow release tablets) taken to tolerance daily, gamma linolenic acid (GLA) as refrigerated Oil of Evening Primrose (½ tsp. daily) or borage oil (one 1,000 mg soft gel daily), Omega 3 EFA fish oil (one soft gel 3–4 times per day), selenium (200 mcg one cap daily), alpha lipoic acid (100 mg daily) and a comprehensive multivitamin (59)—all can be of great benefit.

Healing agents will be needed to repair the gut lining and restore functions damaged by Lyme-Bartonella-Mycoplasma infections. That list may include oral preparations of liquid Aloe Vera, Oil of Clove drops, *Uncaria* spp., anti-fungal tannins, garlic, chewable licorice tabs, betaine, Enteric-coated Oil of Peppermint, Conjugated linoleic acid CLA (1000 mg twice daily), a-lipoic acid (100 mg one daily), Slippery Elm demulcent capsules (325 mg 1–8 three times daily), and ursodiol bile acid tablets (64). Additionally, in the treatment of SIBO, complete stool analysis with culture and sensitivity of opportunistic bowel pathogens may elucidate the choice of antibiotic. Alternatively, a trial may be undertaken with rifaximin (Xifaxan) 200 mg three times a day until symptoms have cleared (60). Cholestyramine (Questran) may be useful in reducing the recycling neurotoxins produced by tick-borne diseases.

As tick-borne-diseased GI systems and their owners heal, relief will be palpable. Physicians will partner in that gratification as well when previously grim-faced patients move to the healthy side of a bell-shaped curve—a graph that would measure the degree to which both gastrointestinal tracts and lives have been restored to functional capacities. These satisfac-

tions will be re-experienced when wisely diagnosed and treated Lyme-sick patients will be able to smile broadly at last, knowing in their guts that zesty appetites for life really will be possible again. ■

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