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Lyme Disease and Dementia, Alzheimer, Parkinson, Autism, an Easy Way to Destroy your Brain

Lapenta J[1*], Lapenta JM[2]

[1]Lapenta, J. Medic Surgeon, Specialty Dermatology, 24 years of exercise. University of Carabobo, Venezuela. Ceo Dermagic Express
[2]Lapenta, J.M. Medic Surgeon. University of Carabobo. Diplomat in Facial Aesthetics Occupational Medicine and Prehospital Auxiliary. Resident Doctor Ambulatorio Del Norte Maracay Aragua State. Coo Dermagic Express.

***Corresponding author:** Lapenta J, Medic Surgeon, Specialty Dermatology, 24 years of exercise. University of Carabobo, Venezuela, Email: www.dermagicexpress.blogspot.com

Abstract

Lyme disease or Chronic Erythema Migrans whose first clinical description was made by Afzelius in 1908, and its causative agent the spirochete *Borrelia Burgdorferi* was discovered 73 years later by Willy Burgorfer in 1981. Lyme disease is spread by a tick bite of the family *Ixodidae, Ixodes scapularis* and many others. Among the numerous species described, *Borrelia Burgdorferi* is disseminated mainly in the United States, *Borrelia garinii* and *Borrelia Afzelii* in Europe and Asia. In addition to producing skin lesions in infected people, and multi-organ side effects, the spirochete is able to reach the human brain and could produce dementia, Alzheimer, Parkinson and Autism. In this investigation we will make a chronological description of the events that lead to neuronal involvement. Just as the spirochete of syphilis, *Treponema pallidum*, produces neurosyphilis in its tertiary stage, also the *Borrelia* is able to reach the brain, and produce collateral damage, a term called neuroborreliosis, and among its most lethal effects may cause dementia, Alzheimer, Parkinson and Autism and hence the so-called post-treatment syndrome of Lyme disease.

Keywords: Lyme disease; Neuroborreliosis; Chronic erythema migrans; Borrelia burgdorferi; Dementia; Autism; Alzheimer; Parkinson; Post lyme disease syndrome

Main objective

The main objective of this research is to demonstrate that Lyme disease produced by the spirochete *Borrelia Burgdorferi* and its different species, is not just a skin disease. It has been scientifically proven to produce numerous organic manifestations, and in this work, we will demonstrate chronologically its neurological manifestations, which if not detected and treated in time, produce lethal sequalae, potentially dementia, Alzheimer's, Parkinson, Autism and as a terminal sequel, post Lyme treatment syndrome.

Secondary objectives

• Describe chronologically the definitive conclusion that the *Borrelia Burgdorferi* and its pathogenic species in humans can affect the brain in several of its stages and produce diverse neurological manifestations termed Neuroborreliosis, which can range from meningitis to dementia, Alzheimer's, Parkinsonism and Autism.
• Describe the neurological clinical manifestations in positive Lyme patients: children of positive Lyme mothers, adults, and adolescents not treated in time, or those resistant to treatment.
• The World Health Organization (WHO) in the update of the ICD-11 year 2018 (International Classification of Diseases) included the codes for Lyme borreliosis related to this research: "Lyme neuroborreliosis", "dementia due to Lyme disease" and "central nervous system demyelization due to Lyme borreliosis". After our review, we will urge the recognition by this entity.

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Introduction

The history of the term DEMENTIA produced by the *spirochaete Borrelia Burgdorferi*, is longstanding, beginning in 1922, when the French Garin and Bujadoux described a patient with neurological symptoms and meningitis after the bite of a tick; the skin lesion was similar to the Erythema chronicum migrans (ECM), which was first described in 1908 by Afzelius. A similar case was published by Hellerstrom in 1930[1-3].

Later in 1941 and 1944 Bannwarth described in 26 patients a clinical condition characterized by: lymphocytic meningitis, neuralgia and neuritis that mainly involved facial nerves. Only 3 of them were associated with a tick bite. Year's later European researchers found that this syndrome was associated with tick bites and Erythema Chronicum Migrans (ECM). This condition was described under several names including meningopolineuritis by tick bite (Garin-Bujadoux-Bannwarth), remaining with the definitive name of Lymphocytic meningoradiculitis or Bannwarth Syndrome.[4-6]

To date there are more than 700 articles published on the subject of Lyme disease. We will focus on the most relevant, about 200 referring to the issue we are developing, to keep a chronological time line.

Chronological Evolution

1922-1944: In 1922, the French Garin-Bujadoux[1], described a patient with neurological symptoms and meningitis after a tick bite with skin lesion similar to Erythema Chronicum Migrans (ECM), and then confirmed by Bannwarth in 1941 and 1944[4-6] with the description of 26 cases with a triad of symptoms: lymphocytic meningitis, neuralgia and neuritis, some associated with tick bites. This clinical picture was later named with the definitive name of meningopolineuritis or lymphocytic meningoradiculitis or Bannwarth syndrome[1-6].

This fact was 60 years before the discovery of *Borrelia Burgdorferi*, but the first neurological symptoms related to Erythema Chronicum Migrans (ECM) discovered by Afzelius in 1908[2,3], had already been described[3-6].

1955: The first confirmation that Erythema Chronicum Migrans (ECM) was and is produced by a living agent was discovered and published by Binder *et al* in 1955 who transplanted skin of three patients with ECM to 3 healthy volunteers. In the course of 1 to 3 weeks, the recipients developed the disease[7]. This occurs 11 years after the description of Bannwarth[4,5] and 33 years after the description of the first case by Garin and Bujadoux[1].

1956-1976: In 1976 Lefevre JP *et al*.[8], published 9 cases of meningoradiculitis after tick's bites and compared them with 56 previously published cases. They found as predominant clinical symptoms:

Pain: radiculitis; Paralysis: more than 50% of cases: unilateral or bilateral paralysis of the 7th cranial nerve; Pyramidal signs; Signs of brain irritation: meningitis, neuralgia; Cerebrospinal fluid (CSF): pleocytosis

This means that in these 20 years (1956-1976) about 65 cases of meningoradiculitis associated or not with ticks bite had already been published[8].

1978: In this year the French Goffinet AM *et al*. publish a paper on meningoradiculitis produced by ticks, where confirm the previously described symptoms and launch the hypothetical theory that the etiology is a transmission of a living agent, as Binder found in 1995[7], and suggested that it is a virus transmitted by the tick bite[9]. Still left 3 years for the discovery of the *Borrelia Burgdorferi*.

1981-1982: This year of 1981 marked a milestone, Wilheim "Willy" Burgdorfer discovered the causal agent of Chronic Erythema Chronicum Migrans (ECM), a bacterium belonging to the family of spirochetes as *Treponema pallidum* (syphilis), studying the stomach of a tick of the genus *Ixodes daminii* collected in the town of Old Lyme Connecticut, United States, which was named *Borrelia Burgdorferi* in honor of its name[10,11]. From the location where the causative agent was discovered (Lyme Village) the name of Lyme disease was born[10,11].

One year later in 1982. Hindfelt B *et al*.[12], publish 11 more cases of meningoradiculitis of Bannwarth, of which 50 % was associated with tick bites, describing the classic symptoms of this syndrome, (Meningitis, Neuritis and Radiculopathies),They found Cerebro Spinal Fluid (CSF) mononuclear pleocytosis (lymphocytes), defect of the blood-brain barrier and intrathecal IgG synthesis, expanding the clinical findings of this pathology.

All patients recovered almost completely from their neurological symptoms in 1-2 months, regardless of treatment[12].

1983: This year was also very important because Skoldenberg B *et al*.[13], published 21 cases of chronic persistent meningitis of which 4 (19%) had chronic erythema migrans as antecedent and 4 (19%) had been bitten by ticks. Their neurological symptoms included:

Aseptic meningitis; cranial neuropathy (mostly paralysis of the facial nerve); Peripheral motor and sensory radiculopathies; Myelitis; Cerebrospinal fluid alterations: mononuclear pleocytosis, protein increase, intrathecal IgG synthesis.

The symptoms coincide with those previously described for Bannwarth meningoradiculitis[4-9,12], but the authors report a dramatic improvement in all cases when treated with penicillin G for 14 days[13]. This fact confirmed what was stated by previous studies that it was a living agent that caused the disease[1-13]. Spirochetes (*Treponema pallidum*, syphilis) are sensitive to this antibiotic, and *Borrelia Burgorferi* is a spirochete[10,11].

1984-1985: In1984 published data of cases of meningoradiculitis of Bannwarth associated with Lyme disease, and Pachner AR *et al*.[14] published a study of 38 cases, emphasizing the neurological aspects from those patients studied between 1976 and 1983. Already began to speak of "Lyme meningitis" with the same characteristics of Bannwarth syndrome (Meningitis, Neuropathy and Radiculoneuritis); in addition to it, among other symptoms already described: headache, stiff neck, and lymphocytic pleocytosis in Cerebro Spinal Fluid (CSF), unilateral or bilateral facial paralysis and weakness of the extremities[14].

That same year Pfister *et al*. published 4 cases of meningoradiculitis of Bannwarth in Germany, Munich[15], and demonstrated the presence of antibodies against spirochetes of the *Ixodes daminii* tick in 100% of cases. In three patients, the

antibodies were also present in the Cerebrospinal Fluid (CSF). In one patient, they isolated CSF spirochetes, and demonstrated specific IgG antibodies in serum and CSF against spirochetes. In one case, the spirochete of Cerebrospinal Fluid (CSF) was isolated morphologically equal to the spirochete of the tick *Ixodes daminii*; and postulated that Lyme disease and Bannwarthmeningoradiculitis have the same etiology: spirochetes[10,11,15].

This study was confirmed by Rayberg B[16]. that same year in Sweden 9 more cases of Bannwarth syndrome where 6 cases (66%) had high titers of antibodies against the Lyme spirochete (Borrelia) and reaffirm the published data: Bannwarth syndrome and Lyme disease are caused by spirochetes[10,11,15,16]. 1984 also marks a milestone in the study of Lyme disease and its relationship with spirochetes when the German scientist Weber K.[17], published 4 cases of this disease that presented with Jarisch-Herxheimerreaction, after being treated with antibiotic therapy. This reaction was first described by the Austrian dermatologists Adolf Jarisch and the German Karl Herxheimer in 1895 and 1902 respectively, when they treated cases of syphilis with mercurials[18,19]. This reaction only occurs in cases of spirochetosis (syphilis, leptospira and others), and now the newly discovered spirochete *Borrelia Burgdorferi*[10-19].

The reaction occurs a few hours after administration of the antibiotic and is produced by endotoxins released by dying spirochetes, and is characterized by malaise, hypotension, chills, muscle spasms, headache, tachycardia, hyperventilation and exacerbation of lesions in skin[18,19]. This reaction was confirmed in later years in the treatment of Lyme spirochetosis.

That same year, 117 cases were described in New Jersey, studied between 1976 and 1982, of which 86 were adults and 31 children with Lyme disease. Chronic erythema migrans was present in 93% of the cases. The most common late neurological manifestations, in addition to the immediate organic ones (arthritis 26%, and febrile syndrome 45%), were meningitis (10%) and facial paralysis (8%) of the cases[20].

1985 the Germans Christen HJ, and Cols. describe 3 cases of meningoradiculitis of Garin-Bujaudox-Bannwarth in children, confirming the previous description, that the Erythema Chronicum Migrans (ECM) and its neurological manifestations can also affect children[20,21].

This same year, Stiernstedt GT *et al*. in 41 out of 45 cases of spirochetal meningitis (91%), prove that the Enzyme-Linked Immunosorbent Assay (ELISA) was highly sensitive to the diagnosis of spirochetal meningitis. While an indirect immunofluorescence method in serum and Cerebrospinal Fluid (CSF) with 98% of positivity[22].

It was in this decade that the term "Neuroborreliosis" began to be used in relation to the neurological manifestations of Erythema Chronicum Migrans (ECM) or the newly "named" Lyme disease, when the causative agent was discovered; and many of the works had already been described about meningoradiculitis or Bannwarth syndrome associated with tick bites, and its neurological manifestations. But really the term "Neuroborreliosis" was born in 1922 (63 years before) when Garin and Bujadoux described the first case[1], later confirmed by Bannwarth in 1941 and 1944[4,5].

This fact that the French Garin and Bujadoux were the first to describe Lyme neuroborreliosis has tried to be disqualified by some authors, but definitely the majority of the scientific community does recognize them.

Still left a year before the description of the first cases of dementia associated with Lyme disease being described[23], fact that marked a milestone and revealed that the neurological damage produced by *Borrelia Burgdorferi* went beyond the meningoradiculitis of Garin-Bujadoux-Bannwarth Classically described[1-12].

1986: MacDonald A.B[23]. published for the first time two cases where spirochetes were identified in brain tissue subcultures during an autopsy of two patients who died from dementia, using indirect immunofluorescence and monoclonal antibodies specific for Borrelia species, which had previously been identified by "dark field" light microscopy.

The cases were a 74-year-old woman with mild dementia of less than 1 year's duration living in Florida and New York, and the other a 69-year-old man who died in a nursing home in Texas after a history of progressive dementia. 4 to 5 years later he also presented with symptoms of Parkinson's[23].

1986 Willy Burgdorfer (discoverer of the spirochete) and Reik L. Jr[24]. who published 8 positive Lyme cases with neurological abnormalities, where they found high titers against Borrelia species. Neurological symptoms including: Aseptic meningitis; Encephalitis; Cranial neuritis; Motor and sensory radiculitis; Myelitis; Severe encephalitis that resulted in dementia in two (2) of these patients; Irreversible myelopathy in one (1) patient

These last two findings enlarged the neurological spectrum already known by *Borrelia Burgdorferi* infection;[12-24].

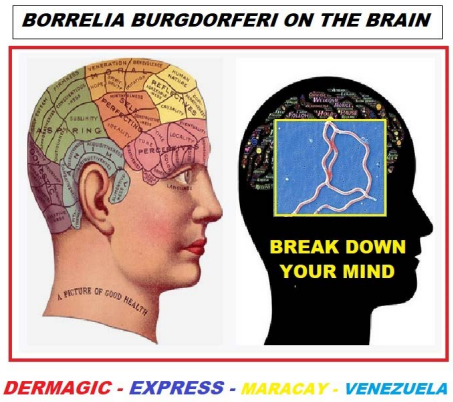
On the other hand no patient showed the classic marker of the disease that is the Chronic Erythema Migrans (ECM) and only 3 had arthritis, which is the other clinical marker of this pathology. This fact led them to conclude that Lyme disease can present as pure neurological forms, without extra neural findings[24].

1987: In 1987, articles on Bannwarth'smeningoradiculitis and its relation to tick bite, enlargement of the neurological spectrum, (chronic encephalomyelitis, late neuroborreliosis, and findings of IgG anti Borrelia antibodies in the cerebrospinal fluid by ELISA) continue to be published;[26-32] but in addition to this, another important fact occurs:

This year in the timeline of Lyme Neuroborreliosis and its relationship with Alzheimer's disease was demonstrated, because the researcher MacDonald A.B and Col.[25], published a case of a patient who died of Alzheimer's which I performed an autopsy of the brain tissue and found the spirochetes of *Borrelia* in the cerebral cortex. The authors propose that, as in tertiary or late syphilis, Borrelia species invade the brain and remain there for years, subsequently causing dementia; state that an undetermined number of patients with Alzheimer's disease have late tertiary neuroborreliosis[25].

This fact: Lyme's relationship with Alzheimer's was later shown in other studies that we will mention later.

Also this year of 1987 Moore JA, reports again the reaction of Jarisch-Herxheimer in the treatment of Lyme Borreliosis[32].



1988: For this year, the French Dupuis MJ[33]. published a study describing the three stages of Lyme disease and its neurological manifestations, making a summary of the symptoms, mostly already described previously:

Stage 1 during the first month is characterized by chronic erythema migrans and associated manifestations.

Stage 2 includes neurological symptoms: Classic meningoradiculitis. (Garin-Bujadoux-Bannwarth syndrome); Lymphocytic meningitis with acute or recurrent course; Facial paralysis; Neuritis of other cranial nerves; Cranial polyneuritis; Sign of Argyll-Robertson; Affectation of peripheral nerves; Acute transverse myelitis; Severe encephalitis; Myositis.

Stage 3 of the disease: neurological symptoms, months or years after the disease has started:

Chronic neuropathy with mainly sensory or motor signs; recurrent cerebrovascular accidents; Cerebral angiopathy; Progressive encephalomyelitis; Ataxic and spastic March. (Parkinson's symptoms); Dysfunction of the cranial nerve that includes optic atrophy and hypoacusia, dysarthria, focal and diffuse encephalopathy; Simulation of other diseases: multiple sclerosis; Lesions of the multifocal and mainly; peri-ventricular white substance; Psychic disorders: acute presenile dementia

The author states that high doses of the antibiotic penicillin can stop the disease and sometimes induce regression.[33]; also affirms the great similarity between *Borrelia Burgdorferi* and *Treponema pallidum* (syphilis), considering it as the "great imitator" of other diseases[33].

1989: This year of 1989 the researcher Pachner AR and Col.[34], contribute in the classification of the neurological spectrum of Lyme disease by publishing an article where they consider that *Borrelia Burgdorferi* and its species are highly neurotropic and classify neuroborreliosis in two types:

Lyme meningitis in the second stage of the disease, which resembles aseptic meningitis; it is often associated with facial paralysis, peripheral nerve involvement and / or radiculopathies and may be the first manifestation of the disease even without the presence of Chronic Erythema Migrans (ECM)

The third stage: parenchymal disease which causes a multitude of nonspecific manifestations of the central nervous system (CNS) that can be confused with conditions such as multiple sclerosis, brain tumor and psychiatric disorders.

The authors consider that the "new great imitator" of the diseases is "Lyme borreliosis", a term attributed ancestrally to another spirochete, *Treponema pallidum*, causal agent of syphilis; confirming what was stated in the previous study[33,34] and also in relation to the fact that the neurological manifestations of Lyme disease occurs in stages 2 and 3 of the disease fundamentally.

The scientist Kristoferitsch W. and Cols. describe in that year the characteristics of the neuroborreliosis, type meningoradiculitis of Bannwarth in Europe and affirm that the characteristics are very similar to those presented by the American patients (US)[35].

Pizzarello LD, MacDonald and Cols[36]. published a case of loss of vision in a 71-year-old patient due to temporal arteritis, biopsy and blood culture were performed where spirochetes compatible with Borrelia spices were found. After treatment with IV ceftriaxone the patient showed a moderate improvement. The authors considered this case as the first one where Borrelia is found in a biopsy of the temporal artery[36].

That same year the Germans Bialasiewicz AA, and Cols[37]. published a case of a 25-year-old female patient who presented with a neurorretinitis in the left eye, later confirmed by *Borrelia Burgdorferi*, presenting affectation of both eyes, finding advanced atrophy of the optic nerve, visual acuity decrease; magnetic resonance shows multiple demyelinating lesions for ventricular and subcortical areas. This case was considered by the authors to be the first in the literature describing demyelinating lesions in the brain with bilateral optic neuritis verified serologically. The patient improved after receiving treatment with doxycycline[37].

This fact of the demyelination caused by the *Borrelia Burgdorferi* was confirmed that same year of 1989 when Meier C, and Cols[38]. publish a work on 8 patients with neurological complications of the peripheral nervous system produced by Lyme Borreliosis. More than 60% of the patients presented the classic meningoradiculitis of Garin-Bujadoux-Bannwarth. In the biopsy of the nerves it was found:

- Macroscopic infiltrations of epineural vasa nervorum and small infiltrations around of endoneurial capillaries: lymphocytes, histiocytes and plasma cells.
- Thrombosis and recanalization in some epineural vessels.
- Seven biopsies showed a significant loss of myelinated axons due to axonal degeneration.
- In one biopsy they observed segmental demyelination with axonal degeneration.

The authors conclude that the peripheral neurological manifestations of Lyme Borreliosis are angiopathic due to vasa nervorum vasculitis and mainly caused by axonal degeneration[38]. This fact, demyelination of the nerves, also in the brain appears for the first time in our timeline[37,38] and further broadens the clinical spectrum of neuroborreliosis. Still left nearby 3 decades in our timeline and already by this date (1989) the term "Neuroborreliosis" of Lyme had been recognized and among the neurological manifestations of our research: dementia, Alzheimer, Parkinson[23,33], they were already described; and in addition to this: central and peripheral demyelination of nerve cells[37,38] associated with *Borrelia Burgdorferi*.

1990-1999: Many studies were carried out this decade[39-62], since the potential damage that Borrelia can cause in both the Central Nervous System (CNS) and the peripheral nerves was already known, so the scientists focused their studies in this line; we will mention the most outstanding ones.

The investigator Judith Miklossy in 1990[39] describes the neuropathological findings of a case with Lyme neurobor-

reliosis, manifested as chronic meningitis; there were meningovascular and parenchymal occlusive changes similar to those that occur in tertiary syphilis or neurosyphilis; and suggest that the case described represents the meningovascular form of Lyme or tertiary neuroborreliosis, confirming previous studies.

In the year 1991 Krupp, L.B, and Cols[42]. describes 15 patients treated for Lyme Borreliosis who complained of persistent cognitive difficulty after 6 to 7 months of treatment with antibiotic therapy; they were compared with 10 healthy controls. Post-Lyme patients showed a marked deterioration in cognitive tests: memory loss mainly in selective recognition; attributed this finding to chronic encephalopathy produced by Borrelia. This study was confirmed in 1995 by Benke T. and Cols.[50], in 1997 by Gaudino EA. and Cols. who also made a differentiation between this syndrome and chronic fatigue syndrome (SCF).[55] In 1999 Elkins LE. and Cols. also confirmed this fact[61].

Perhaps this represents the first study[42], about what later was and is called Post-Treatment Lyme Disease Syndrome (PTLDS), or Post-Lyme Syndrome (PLS) because patients were evaluated months after receiving treatment and presented neurological damage symptomatology in this case chronic encephalopathy.

In the year 1993 Fallon B.A. and Cols.[44], published three cases (3) of neuroborreliosis associated with depression, previously described[33], but patients showed panic attacks and mania; considering these last symptoms as the first reported associated with Lyme neuroborreliosis[44].

In the year 1994 Schaeffer S, and Cols. Publish an article on Lyme disease associated with dementia[48], in 1995 Waniek C, and Cols., report another neuropsychiatric case[49] with fatal outcome of progressive frontal lobe dementia, where pathologically severe subcortical degeneration was found, and reaffirm the fact that Lyme disease can present as pure neurological forms[24,48]. the patient improved with antibiotic treatment, but then relapsed.

Now we will put a summary of the neurological findings described in this decade related to Lyme Borreliosis, apart from the classic triad of lymphocytic meningoradiculitis or Garin-Bujadoux-Bannwarth syndrome (meningitis, neuritis and radiculopathies), and the detection of spirochete in Cerebrospinal Fluid (CSF) and its changes, already widely discussed:

• Progressive dementia.
• Depression: 26 - 66% of cases. mania, panic attacks.
• Memory disorders: verbal memory, mental flexibility, associative, verbal and articulation functions; decreased consciousness, mental confusion.
• Encephalitis, encephalomyelitis, isolated transverse myelitis, vasculitic cerebral disorders.
• Paranoia, schizophrenia, bipolar disorders, obsessive-compulsive disorders[46].
• Hallucinations, nightmares, hypersexuality[52].
• Sleep disorders, fatigue[54,59].
• Jarisch-Herxheimer reaction, post-treatment with antibiotics (amoxicillin)[56].
• Hemiparesis, hemianopsia, stenosis of cerebral arteries[58].
• Sensitivity to light, touch and sounds[60].

The highlights in this decade were the appearance of new neurological symptoms associated with *Borrelia Burgdorferi* infection, which led to it being considered a "neuropsychiatric" disease, the appearance of the Post-Treatment Syndrome of the Disease (PTLDS) or (PLS)), and the appearance of new laboratory techniques to detect the causative agent[62].

2000-2009: Already for this decade Lyme neuroborreliosis was widely recognized by scientists worldwide. There were numerous studies[63-97] of which we will highlight the most important in relation to our research.

In the year 2001 Tager F.A. and Cols. published a study on 20 children with a history of cognitive complaints after having Lyme disease compared with 20 healthy children. The Lyme positive children presented more cognitive and psychiatric disorders than the healthy ones, being the most relevant: anxiety, depression and fatigue.

In the year 2003, Cassarino D.S. And Cols. published a case of a 63-year-old male patient who presented Erythema Migrans rash, which was detected against *Borrelia Burgdorferi* antibodies in serum and Cerebro Spinal Fluid (CSF). Clinically I present joint pains and tremors. He was diagnosed with clinical Parkinsonism by several neurologists and subsequently had a fatal outcome. In the cerebral autopsy it was found: Mild atrophy of the basal ganglia; Depigmentation of the substantian-igra; Extensive neuronal loss; Extensive loss of the neuronal black substance; Astrogliosis; Absence of Lewy bodies; Ubiquitin-positive glialcytoplasmic inclusions in striatal and nigral oligodendroglia

The authors considered this case as the first report of striatonigral degeneration in a patient with *Borrelia Burgdorferi* infection of the central nervous system and clinical Parkinsonism associated with Lyme[72] Already the symptoms of Parkinson's had been previously reported.

In the year 2005, Jones CR. and Cols. present a study on the clinical manifestations of 102 children born to Lyme-positive mothers, the most notable neurological clinical symptoms being the following:

• Fatigue and lack of resistance: 72%
• Orthopedic disorders: sensitivity (55%), pain (69%) spasms and generalized muscle pain (69%), rigidity and / or retarded motion (23%).
• Neurological disorders:
  Headaches: 50%
  Irritability: 54%.
  Bad memory: 39%
• Delay in development: 18%
• Seizure disorder: 11%
• Vertigo: 30%
• Tic disorders: 14%
• Involuntary athetoid movements: 9%.
• Earning disorders and humor changes: 80%
  Cognitive speaking: 27%
  Speech delay: 21%
  Reading-writing problems: 19%
  Problems of vocal articulation: 17%.
  Auditory / visual processing problems: 13%
  Word selection problems: 12%
  Dyslexia: 8%
• Suicidal thoughts: 7%
• Anxiety: 21%
• Anger or rage: 23%
• Aggression or violence: 13%
• Irritability: 54% - 80%
• Emotional disorders: 13%

• Depression: 13%
• Hyperactivity: 36%
• Photophobia: 40 - 43%
• Ocular problems: posterior cataracts, myopia, stigmatism, conjunctive erythema (Lyme eyes), optical nerve atrophy and / or uveitis: 30%
• Sensitivity of skin and noise (hyperacuity): 36-40%
• Autism: (9%).

Autism related to Lyme disease appears in the timeline (2005), which was confirmed by subsequent studies

Bransfield, R.C. and Cols. publish a study in the year 2008 where they state that chronic infections, including *Borrelia Burgdorferi* and others transmitted by ticks, produce a weakened state in children, either in fetal development or during development that may promote an autistic state. Positive reactivity was found in several patients with autism spectrum disorder for *Borrelia Burgdorferi* in several studies of 22%, 26%, 20% - 30% and 50% for Mycoplasma; later the same author published in the year 2009 another work confirming this event and names as possible causal agent of this infectious spectrum:
Babesia, Bartonella, *Borrelia Burgdorferi*, Ehrlichia, Human herpesvirus-6, Chlamydia pneumoniae and *Mycoplasma* (in particular *Mycoplasma fermentans*).

In the year 2006, MacDonald A.B., publishes a paper in which he hypothesizes that the rounded forms of *Borrelia Burgdorferi* are the main cause of rounded structures called "plaques" in the brain affected with Alzheimer's disease (AD); these are emblematic and are observed as rounded amyloid forms of brain damage in Alzheimer's.

The same author (MacDonald) in the year 2007 publishes another study where he reaffirms his theory that the "neurofibrillary tangles" present in Alzheimer's disease are produced by a chronic infection, in this case the *Borrelia Burgdorferi*, mentioning a pilot study where it was demonstrated that seven (7) out of ten (10) Alzheimer's cases showed positive signs for infectious DNA in the neurons analyzed.; remember that this author in 1987 found *Borrelia Burgdorferi* in the brain of one patient who died of Alzheimer's disease.

In the year 2008, Judith Miklossy reaffirms what MacDonald presented, publishing a paper that concludes that bacteria, including spirochetes (Borrelia, Treponema) contain amylodogenic proteins; that cortical deposition of beta-amyloid-peptide (Abeta) and tau phosphorylation can be induced after chronic infections, which produce inflammation of the brain, cytokine release, apoptosis, generation of free radicals, release of nitric oxide and activation of complement, which generate a cascade of events that finally produce the amyloidogenesis typical of Alzheimer's disease. The author proposes that the treatment with antibiotics and anti-inflammatory in these cases is vital to avoid dementia.

In that year of 2008, Judith Miklossy herself published another work on the neuropathology of dementia in syphilis and Lyme disease, where she emphasizes and reaffirms that both *Treponema pallidum* (syphilis) and *Borrelia Burgdorferi* (Lyme), both spirochetes, in later stages cause dementia, cortical atrophy and deposition of amyloid in the brain. These manifestations may occur years or decades after the primary infection and in the tertiary stage is where the dementia develops apart from other neurological symptoms.

Other studies conducted in this decade reported associated with neuroborreliosis: spontaneous hemorrhage of the temporal lobe, posterior column dysfunction, headache, loss of sleep, mild ataxia, dysfunction of perception, abnormality in the tendon reflexes, disturbances of sensory responses, memory deterioration, alteration of thoughts, personality disorders, anxiety and affect changes, paranoid and depressive syndrome.
Summary of this decade with Lyme neuroborreliosis: neurological manifestations in children, association with Alzheimer's, Parkinson's and Autism. The so-called Post-Treatment Lyme Disease Syndrome (PTLDS) also appears in the timeline.

2010-2018: In this last decade, Lyme disease became known worldwide and studies in relation to it and its consequences in the affected population increased markedly[98-208].

The dementia associated with Lyme neuroborreliosis was confirmed in several studies, corresponding to the years: 2011, 2014, 2016 and 2018.

In relation to Autism associated with Lyme disease, studies were published in this decade in 2012, 2013, 2014, 2017, 2018 with evident evidence that infection by the spirochete *Borrelia Burgdorferi* predisposes children of Lyme positive mothers to present the spectrum autistic, because it has been scientifically demonstrated that this spirochete crosses the placenta, and can conquer the fetal brain producing neurological damage in the short and long term in pregnant women, who did not receive adequate treatment, and even after receiving treatment with moderate answers; this fact had already been previously described.

Regarding the association of Alzheimer with Neuroborreliosis, highlight the researcher Judith Miklossy, who published 3 studies, years 2011, 2015, 2016, shows convincing evidence that infection by spirochetes such as *Treponema pallidum* (syphilis) and *Borrelia Burgdorferi*, (Lyme) chronically infecting the brain, produce the Beta-Amyloid peptide characteristic of this disease[107,135,165].

In 2017 we published a paper in which we demonstrated the effectiveness of the antibiotic Minocycline in Alzheimer's disease and other neurological disorders, such as Parkinson's and Multiple Sclerosis, which would confirm the infectious theory as part of the etiology of these diseases[192]; Other neurological disorders where Minocycline proves to be effective are: schizophrenia, bipolar disorders and autoimmune encephalomyelitis[192]. Here the reflection is unique: if an antibiotic improves a neurological condition, it is because you have an infection (bacteria) in your bloodstream and brain.

Lyme disease showed over time that it does not respect creeds, races, sports and professions by infecting movie celebrities, singers, presidents of nations, and athletes; we present them in this decade, because most of them were in recent years that manifested their illness when Lyme disease was already a notorious event in society. Among them are: Thalia (Mexican singer), 2007; Richard Gere (actor) 1999; Alec Baldwin (actor) 2011; Avril Lavigne (Canadian Singer); 2012; George W Bush Jr. (Former President of U.S.) (2007); Jennifer Capriati (tennis player) 2.013; Yolanda Hadid (TV Actress) 2012; Ben stiller (Actor) 2010; Kris Kristofferson (Actor and singer) 2006; Ashley Olsen (Actress) 2012, and many more[155-158,169]. These people are human beings; in no previous study they have been mentioned, they were omitted.

Of these cases, the singer and actor Kris Kristofferson deserves special mention. Since the year 2006 he was bitten by

a tick and later developed neurological disorders that led him to present loss of memory; He was misdiagnosed with Alzheimer's disease and received treatment with two medications without improvement. Later he was diagnosed with Lyme and after receiving antibiotic therapy began to "recover" the memory, considered by some as a true "miracle" and "come back". It was not a "miracle" his bloodstream and brain were infected with *Borrelia Burgdorferi*. Today recovered, is 82 years old and continues to sing[170,171].

In year 2017, the Ad-Hoc Committee for the recognition of the ICD-11 codes (International Classification of Diseases year 2.018) of Lyme disease was established by the social activist Luché-Thayer, J. and recognized scientists[177], who in March of that year describe the first spectrum of all the clinical manifestations of the disease. Subsequently, in august of the same year, published "The situation of human rights defenders of patients with Lyme disease and Recurrent fever." For that moment I joined the crew as an expert reviewer[208].

This fact led us to publish in January 2018 the full spectrum of Lyme disease under the name of "Understanding Lyme Disease, classification and codes"[190]; which we expanded with a second publication where we included aspects that were missing such as the post-treatment Lyme disease syndrome (PTLDS),[191] described in the 90's, and subsequently reviewed in numerous studies; and immunological aspects related to the HLA antigens (major histocompatibility complex), which shows the relationship of the HLA-DR-4, HLADR-2, and HLA DRB1 alleles, with greater susceptibility to Lyme disease, Lyme arthritis and resistance to treatment with antibiotic therapy.

In august of 2018 Bransfield Robert. C. publishes a compilation of practically the entire neuropsychiatric spectrum of Lyme disease[203], already described by him in other studies[83-203]. He concludes that there are three ways in which *Borrelia Burgdorferi* infects the brain causing neuropsychiatric symptoms:

The meningovascular form associated with cerebral vascular infarcts.

Infection within the Central Nervous System (CNS) which is the atrophic form of Lyme meningoencephalitis and is associated with cortical atrophy, gliosis and dementia.

Infection outside the Central Nervous System (CNS) that causes immunological effects within the Central Nervous System (CNS) and other effects that contributes to neuropsychiatric symptoms[203], among those who stand out: Developmental disorders, autism spectrum disorders, schizoaffective disorders, bipolar disorder, depression, anxiety disorders (panic disorder, social anxiety disorder, generalized anxiety disorder, posttraumatic stress disorder, intrusive symptoms), eating disorders, decreased libido, sleep disorders, addiction, opioid addiction, cognitive impairment, dementia, seizure disorders, suicide, violence, anhedonia, depersonalization, dissociative episodes, derealization[203]; most of them described previously.

This year of 2018 the World Health Organization recognized in the ICD-11 (International Classification of Diseases year 2018) the following codes for Lyme disease:[206]

- IC1G.0: Early Lyme cutaneous borreliosis.
- IC1G.1: Disseminated Lyme borreliosis.
- IC1G.10: Lyme neuroborreliosis
- IC1G.11: Carditis due to Lyme.
- IC1G.12: Ophthalmic Lyme Borreliosis.
- IC1G.13: Lyme arthritis.
- IG1G.14: Late cutaneous Lyme borreliosis
- IC1G.1Y: Other specified disseminated Lyme Borreliosis.
- IC1G.1Z: Disseminated Lyme borreliosis unspecified
- IC1G.2: Congenital Lyme Borreliosis.
- IC1G.Y: Other specified Lyme Borreliosis.
- 6D85.Y: Dementia due to Lyme disease.
- 9C20.1: Infectious panuveitis in Lyme disease.
- 9B66.1: Infectious intermediate uveitis in Lyme disease.
- 8A45.0Y: Central nervous system demyelination due to Lyme borreliosis.

Conclusion

- Lyme "neuroborreliosis" was not born in the 80s when it began to be mentioned in scientific studies after its causative agent, the *Borrelia Burgdorferi* was discovered by Willy Burgoderfer in 1981 and its presence in the human brain was later confirmed. He was born in 1922 when the French Garin and Bujadoux described for the first time the meningoradiculitis lymphocytic with its neurological manifestations.
- We demonstrate chronologically and scientifically that *Borrelia Burgdorferi* and its species can conquer the human brain and produce dementia, Parkinson's, Alzheimer's and Autism.
- Unlike *Treponema pallidum*, which only in the tertiary stage of syphilis is when it produces dementia (neurosyphilis); *Borrelia Burgdorferi* (Lyme) in its secondary and tertiary stage produces neuropsychiatric manifestations (neuroborreliosis).
- Lyme disease can present as pure neurological forms, without the presence of Erythema Chronicum Migrans (ECM); this fact is what in many cases makes the diagnosis difficult.
- Several highly specific diagnostic tests appeared to detect *Borrelia Burgdorferi*, even surpassing those proposed by the CDC.
- The World Health Organization(WHO) recognized the code "Lyme Neuroborreliosis", "Dementia due to Lyme" this year of 2018 in the ICD-11 and the "demyelination of the central nervous system due to Lyme borreliosis"
- Clinical diseases not recognized: "Alzheimer due to Lyme", "Parkinson due to Lyme", but assuming that "dementia" is a common symptom of Alzheimer's, its tacit recognition is understood; like the case of Parkinson's, because the demyelination of the central nervous system and the chronic infection of the brain by Borrelia species can cause symptoms of Parkinsonism. Also the "Autism due to Lyme" was not recognized but like the previously described, it is supposed to be included in the term "neuroborreliosis due to Lyme"
- The Post-Treatment Lyme Disease Syndrome (PTLDS), was not recognized by the WHO; it was extensively reviewed in this research and which was described more than 20 years ago, will be the subject of a forthcoming investigation.
- Finally, the Lyme disease, now it is not only a dermatological disease, is a neuropsychiatric illness that can easy destroy your brain, if it is not detected and treated at time.

Comments

If we include Alzheimer's disease, Parkinson's disease and Autism within the spectrum "Lyme neuroborreliosis" code, only Post-Treatment Lyme Disease Syndrome (PTLDS) will be left out in this investigation.

Acknowlegment

- To the scientists who with their consecration to the investigation since this disease were described, have managed to reach the bottom and clarify many questions about this disease.
- To organizations that struggle every day for society to understand how dangerous this disease is, the need for early diagnosis and adequate treatment; and how to prevent the tick bite.
- To my son, and father, motors of the real "machine" Dermagic Express of these investigations, which we dedicate to the entire world population.

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