Lyme Disease and Ophthalmic Manifestations

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What do you know about Lyme Disease?

Let's take a pre-test…

What do you know about Lyme Disease?

1. Lyme disease occurs throughout the United States: TRUE or FALSE?

What do you know about Lyme Disease?

2. The "two-tiered" blood test for Lyme disease is unreliable. TRUE or FALSE?

What do you know about Lyme Disease?

3. Lyme disease is transmitted from person to person: TRUE or FALSE?

What do you know about Lyme Disease?

4. The best way to remove an attached tick is:

(Choose one)

A. Burn it off with a hot match

B. Apply petroleum jelly

C. Grasp the tick with tweezers close to the skin and pull

D. All of the above work

What do you know about Lyme Disease?

5. A tick must be attached to a person's skin for more than 24 hours before it can transmit Lyme disease. TRUE or FALSE?

2011 Lyme Disease Incidence

In 2011, 96% of Lyme disease cases were reported from 13 states:

- Connecticut (56.0)*
- Delaware (84.6)*
- Maine (60.3)*
- Maryland (16.1)*
- Massachusetts (27.3)*
- Minnesota (22.2)*
- New Hampshire (67.3)*
- New Jersey (38.5)*
- New York (16.0)*
- Pennsylvania (37.2)*
- Vermont (76.0)*
- Virginia (9.3)*
- Wisconsin (42.2)*

*=incidence per 100,000 persons

Lyme Disease Statistics

- Lyme disease is the most commonly reported vector-borne illness in the US.
- In 2011, it was the 6th most common Nationally notifiable disease.
- Heavily concentrated in the Northeast and upper Midwest (*not* a nationwide disease).

Lyme Disease in History

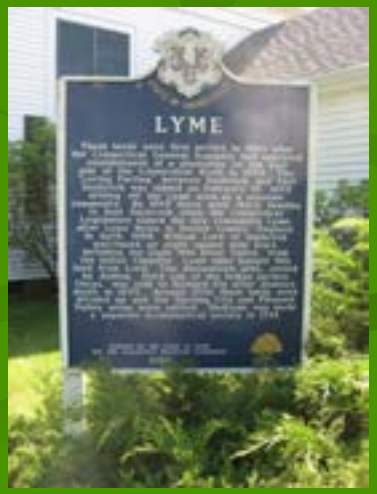
- First detailed description may have been as early as 1764 by Dr. John Walker as he describes the disease and the tick vector in the area off the west coast of Scotland.
- Preserved museum specimen in Germany from 1884 revealed the presence of the DNA sequence of *B. burgdorferi.*
- However…

Lyme Disease in History

An autopsy in 2010 on the Iceman Otzi, a 5,300 year old mummy, the found the DNA sequence of *B. Burgdorferi*. This makes him the earliest known human with Lyme disease.

Lyme Disease Discovered

- Connecticut in 1975-cluster of what was thought to be juvenile rheumatoid arthritis in Lyme and Old Lyme was investigated.
- Investigators recognized that the array of symptoms was similar to the tick-borne condition in Europe.



Discovery!

- 1980 - Willy Burgdorfer, a researcher for the Rocky Mountain Biological Laboratory reviewing ticks (samples from Shelter Island, NY) for rickettsiae noted "poorly stained, rather long, irregularly coiled spirochetes."
- Spirochetes were present in over 60% of the ticks.
- Ticks, cultures, and samples were obtained and Burgdorfer published the link in Science in 1982.
- The new spirochete was named in his honor.

Lyme Disease

- Caused by *Borrelia burgdorferi* in North America, but by other species in other countries around the world.
- Tick-born disease.
- Hard-bodied ticks of the genus *Ixodes* in nymphal stage are the main vectors.
- Nymphal stage ticks usually feed in the spring and summer months.
- Adult ticks can spread disease as well and are still active in cooler months.

Lyme Disease

- Lyme spirochetes have been found in other insects but there have been no reports of transmission to humans.
- No person to person transfer of Lyme disease has been reported from touching, kissing, or sexual contact.
- Lyme during pregnancy can lead to infection and stillbirth, but usually no harm to fetus if mother gets treatment.
- No transmission via breast milk.

Pathophysiology

- Untreated, the disease can become a systemic process.
- Spirochete has been found in the skin, heart, joints, peripheral and central nervous systems.
- B. burgdorferi is injected into the skin by the bite of an infected tick.
- Tick saliva contains anti-immune substances which permit the bacteria to establish itself.
- Days to weeks later the spirochetes spread and disseminate persisting for months or even years if untreated.

Isolates* of *Borrelia burgdorferi*

- Blood
- Synovial fluid
- Spinal fluid
- Retina
- Vitreous
- Brain
- Skin
- Other organs

***Borrelia burgdorferi* is notoriously difficult to culture from patients.**

Pathophysiology

- Disseminated disease leads to varying symptoms specific to the location of the infection.
- Spirochete causes the immune system and other cells to secrete products detrimental to the surrounding healthy tissue.
- Example: astrocytes undergo astrogliosis (proliferation followed by apoptosis/cell death) contributing to neurodysfunction.

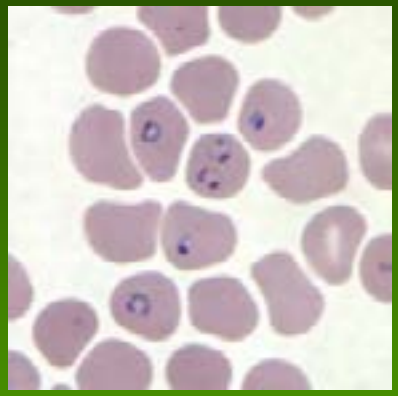
Associated Co-infections

- Ticks that transmit *B. burgdorferi* carry other parasites such as *T. microti* and *A. phagocytophilium*.
- *T. microti* causes babesiosis.
- *A. phagocytophilium* causes human granulocytic anaplasmosis (HGA) aka ehrlichiosis.
- In early Lyme patients, 2-12% had HGA and 2-40% had babesiosis. Overall studies suggest an association of approximately 10% co-infection rate.

Associated Co-infections

- Complicate the diagnosis of Lyme disease.
- Suspect co-infections if there is unexplained leucopenia, thrombocytopenia, or anemia.
- Suspect co-infections if there are more severe infectious symptoms such as a high fever greater than 48 hours despite appropriate medical therapy.
- Lyme disease patients that have a co-infection usually are more sick and have a more chronic form of the disease.

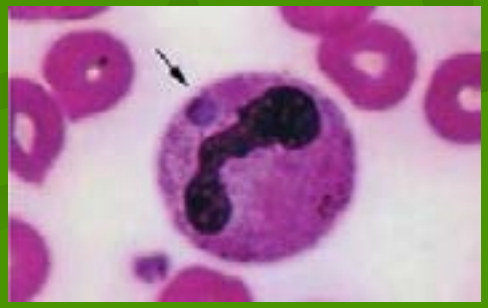
Babesiosis



- Fevers, chills, night sweats, arthralgias, and headaches.
- Can be fatal.
- Prominent vector is *Ixodes dammini*.
- Disease coexists with Lyme disease commonly in New England (especially in Rhode Island).
- No ocular involvement reported.

HGA or Erhlichiosis

- Acute febrile illness with myalgias, headaches, leukopenia, thrombocytopenia, elevated serum aminotransferase, and no associated rash.
- Can be fatal.
- Often coexists with Lyme disease in northern Midwest, but more recently has been identified in New York, Massachusetts, and Connecticut.
- No ocular involvement reported.



Diagnosis

- Symptoms.
- Physical findings.
- Serological blood testing.
- Clinical history of tick exposure.

Systemic Lyme Disease Manifestations

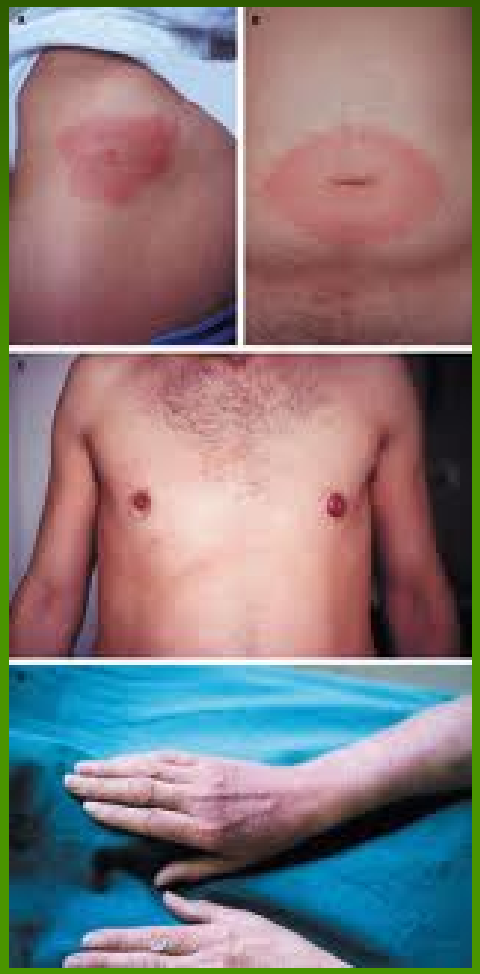
- Localized infection-early (Stage 1)
- Disseminated infection-early (Stage 2)
- Disseminated infection-late (Stage 3)
- Chronic Lyme disease or Post-treatment Lyme disease syndrome

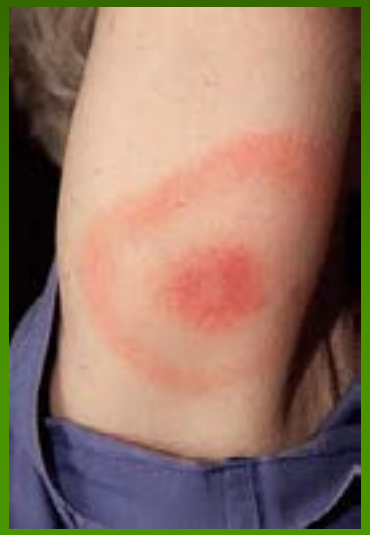
"The Great Imitator"

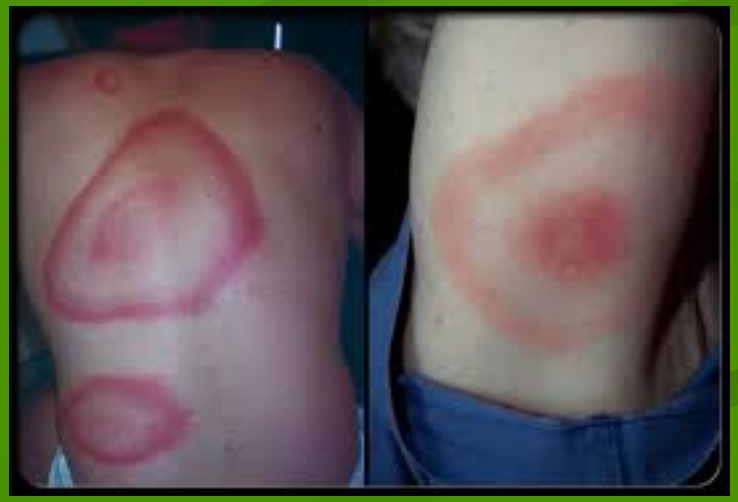
- Lyme disease has been misdiagnosed as:
  - Multiple sclerosis
  - Rheumatoid arthritis/JRA
  - Fibromyalgia
  - Chronic fatigue syndrome
  - Systemic lupus erythematosis
  - Crohn's disease
  - Mononucleosis
  - Amyotrophic lateral sclerosis (ALS)
  - Other autoimmune or neurodegenerative diseases

Localized-early

- Occurs 3-30 days post-tick bite.
- Disease has not yet spread into other areas of the body.
- Signs and symptoms include:
  - Erythema migrans (EM or ECM)
  - Flu-like symptoms including fatigue, chills, headache, muscle or joint aches and (+)lymph nodes



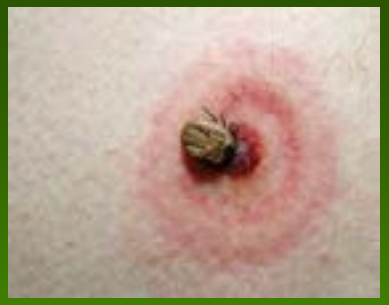




Erythema Migrans

- Classic sign of Lyme disease, but not always seen.
- Also called erythema chronicum migrans.
- Occurs at the site of the bite 3-30 days *after* tick bite.
- Rash seen in 70-80% of patients and on average shows up at 7 days and may last up to 1 month.
- Rash is red, painless, with the inner portion becoming indurated with a clearing portion followed by a red outer edge giving the appearance of the classic Bull's eye.

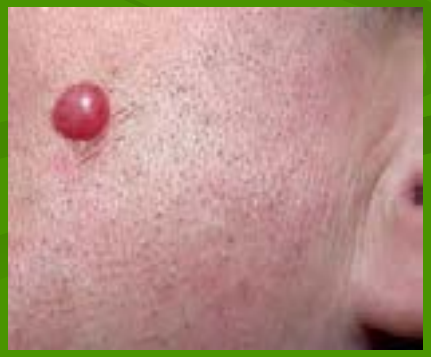
Erythema Migrans

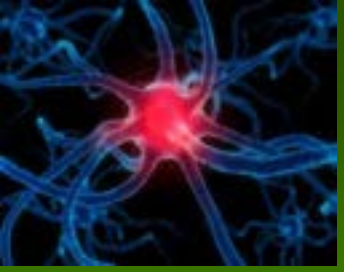


- Rash is usually greater than or equal to 5 cm in largest diameter showing up around days 7-14.
- But, a rash seen *within first day or two* represents a hypersensitivity reaction which is non-infectious.
- This early rash is less than 5 cm and has an urticarial appearance which often disappears in the first 24-48 hours.
- This is in contrast to the EM which will *increase* over this time period!

Disseminated Infection-Early

- Occurs within days to weeks after the onset of the localized infection.
- *Borrelia* begins to spread through the blood stream and may affect locations no where near the initial bite.
- A purplish nodule may form called a borrelial lymphocytoma. This lesion is more common in European cases.
- Systemic symptoms begin.





Disseminated Infection-Early

- Symptoms may include:
  - Migrating acute severe pain in joints, muscles, or tendons (usually the large joints are affected)- 60% of patients affected.
  - Acute neurological problems including Bell's palsy (Facial nerve palsy), meningitis, encephalitis, or radiculoneuritis (shooting pains and abnormal skin sensitivity)- 15% of patients affected.
  - Cardiac manifestations may include heart palpitations or dizziness, heart block or rarely myopericarditis-8% of patients are affected.

Disseminated Infection-Late

- Stage occurs months to even years post-tick bite.
- Untreated patients may have dysfunction of the brain, heart, nerves, eye, joints and heart.
- Acrodermatitis chronica atrophicans, disabling symptoms and even paraplegia.
- Some reports of chronic progressive encephalomyelitis with cognitive impairment, leg weakness, gait disturbances, bladder problems, vertigo and even psychosis.

Chronic Lyme Disease

*Best descriptive term is: Post Treatment Lyme Disease Syndrome or PTLDS.

*10-20% of patients have symptoms that last months to years after treatment.

*Muscle/joint pain , cognitive defects, sleep disturbances, and fatigue persist.

*Unknown as to why PTLDS occurs since there is no active infection.

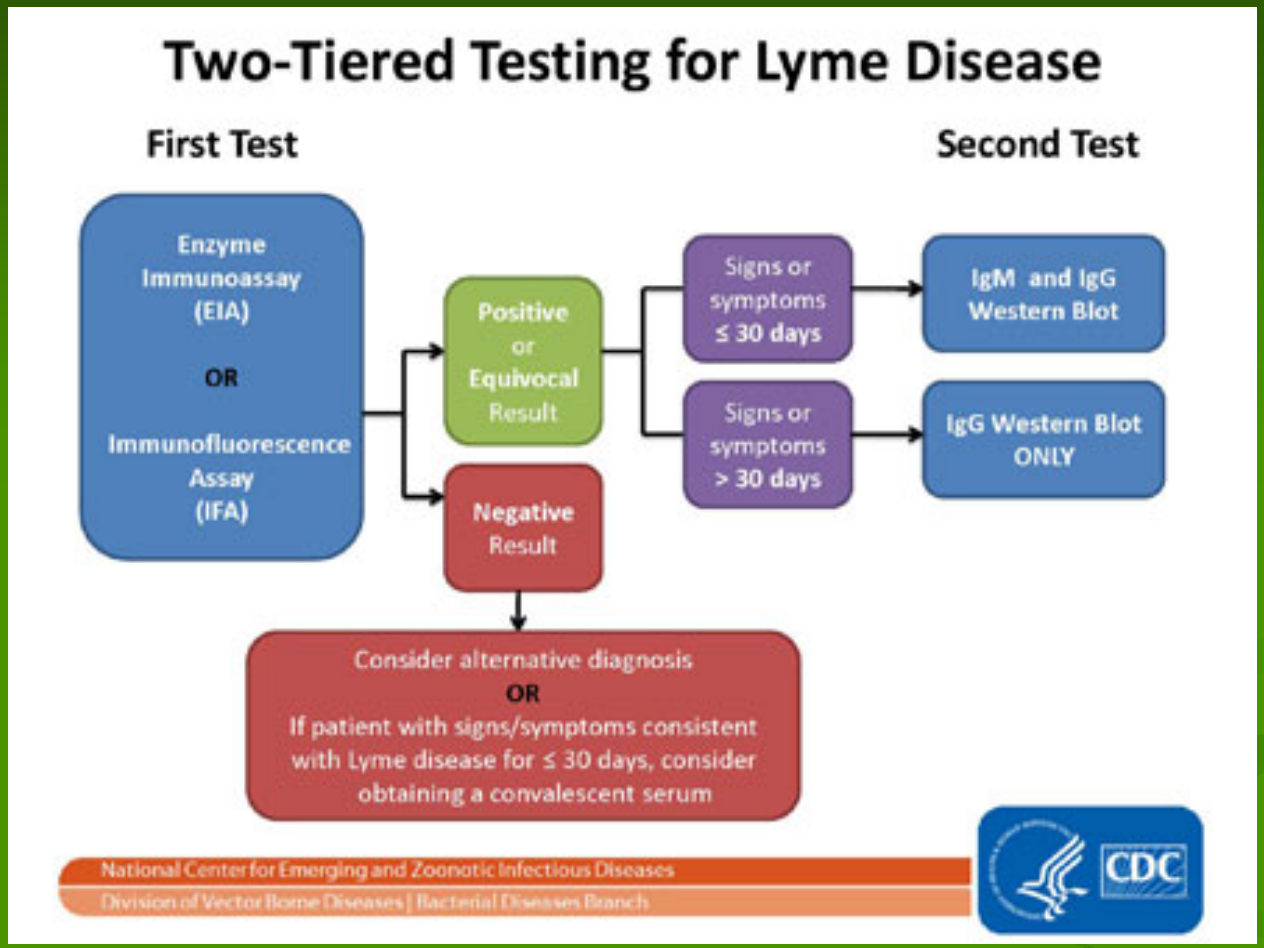
*Possible autoimmune response causing systemic damage.

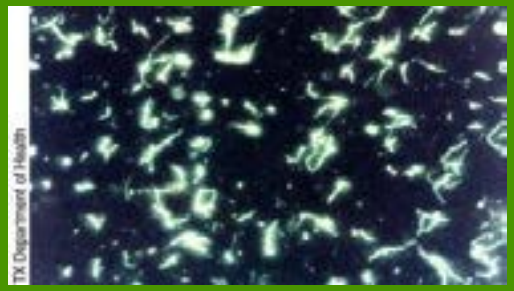
*Treatment for PTLDS is controversial.

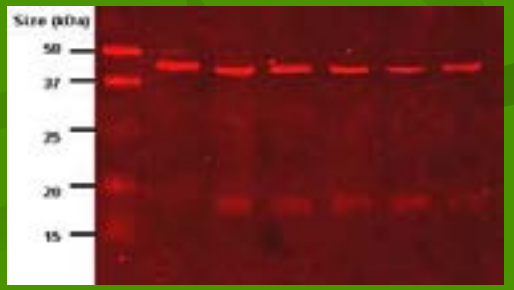
"It's Lyme disease again."

Lyme Disease Testing

Two-Tiered Testing for Lyme Disease

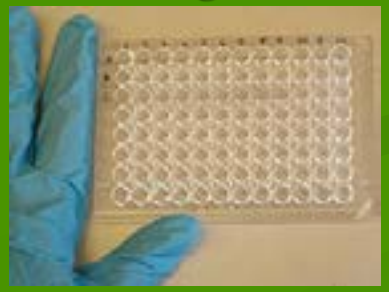






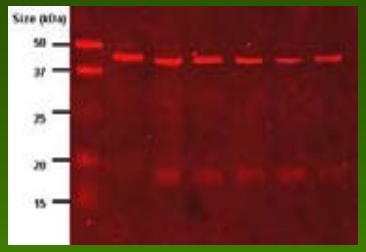
Enzyme Immunoassay (EIA)

- Antigens from the sample are attached to a surface. Then, a further specific antibody is applied over the surface so it can bind to the antigen. This antibody is linked to an enzyme, and, in the final step, a substance containing the enzyme's substrate is added. The subsequent reaction produces a detectable signal, most commonly a color change in the substrate.
- Performing an ELISA involves at least one antibody with specificity for a particular antigen. The sample with an unknown amount of antigen is immobilized on a solid support (usually a polystyrene microtiter plate) either non-specifically (via adsorption to the surface) or specifically (via capture by another antibody specific to the same antigen, in a "sandwich" ELISA). After the antigen is immobilized, the detection antibody is added, forming a complex with the antigen. The detection antibody can be covalently linked to an enzyme, or can itself be detected by a secondary antibody that is linked to an enzyme through bioconjugation. Between each step, the plate is typically washed with a mild detergent solution to remove any proteins or antibodies that are not specifically bound. After the final wash step, the plate is developed by adding an enzymatic substrate to produce a visible signal, which indicates the quantity of antigen in the sample.



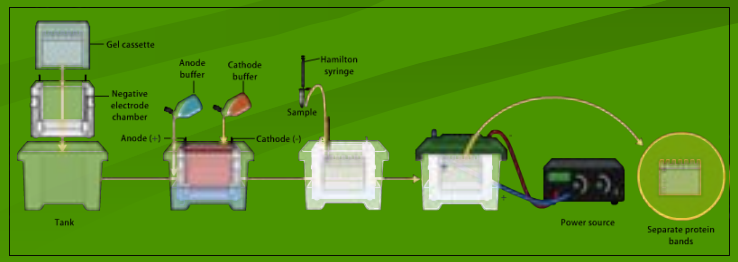
Immunofluorescence (IFA)

- **Immunofluorescence** is a technique used for light microscopy with a fluorescence microscope and is used primarily on microbiological samples. This technique uses the specificity of antibodies to their antigen to target fluorescent dyes to specific biomolecule targets within a cell, and therefore allows visualization of the distribution of the target molecule through the sample. Immunofluorescence is a widely used example of immunostaining and is a specific example of immunohistochemistry that makes use of fluorophores to visualize the location of the antibodies



Western Blot

- Western blotting identifies with specific antibodies proteins that have been separated from one another according to their size by gel electrophoresis. The blot is a membrane, almost always of nitrocellulose or PVDF (polyvinylidene fluoride). The gel is placed next to the membrane and application of an electrical current induces the proteins in the gel to move to the membrane where they adhere. The membrane is then a replica of the gel's protein pattern, and is subsequently stained with an antibody.



CDC Two-Tiered Testing

- First test is Enzyme Immunoassay (EIA) or Immunofluorescence Assay (IFA)
  - A negative test should prompt an alternate diagnosis OR if patient has symptoms for less than 30 days treatment may be considered with follow-up after with convalescent serum.
  - If testing is positive or equivocal, then further testing is recommended

CDC Two-Tiered Testing

- Second test after a Positive EIA or IFA:
  - Signs or symptoms <30days then test with Western Blot IgM and IgG
  - Signs or symptoms >30 days then test with Western Blot IgG only

Other Types of Laboratory Testing

Some laboratories offer Lyme disease testing using assays whose accuracy and clinical usefulness have not been adequately established. Unvalidated tests available as of 2011 include:

- Capture assays for antigens in urine
- Culture, immunofluorescence staining, or cell sorting of cell wall-deficient or cystic forms of *B. burgdorferi*
- Lymphocyte transformation tests
- Quantitative CD57 lymphocyte assays
- "Reverse Western blots"
- In-house criteria for interpretation of immunoblots
- Measurements of antibodies in joint fluid (synovial fluid)
- IgM or IgG tests without a previous ELISA/EIA/IFA

“I have the tick right here!”

- Tick testing is NOT currently recommended.
- Identifying the tick can be done, BUT there is no certainty the tick is infected or infectious.
- Results can take a long time.
- A negative result is not 100% accurate and may give a false sense of security.

Treatment of Lyme Disease

- Early infection
  - Tetracycline 250 mg PO qid x 10-30 days *OR*
  - Doxycycline 100 mg PO bid x 10-30 days *OR*
  - Amoxicillin 500 mg PO qid x 10-30 days
  - Alternative treatment: azithromycin/clarithromycin/erythromycin

  *(Note: Keflex is often used for soft tissue infections, but Keflex is *NOT* effective treatment for Lyme disease.)

- Severe disease
  - Ceftriaxone 2 gm qday IV x 14 days *OR*
  - Penicillin G 20-24 mil units qday IV x 10-14 days

Treatment

- Disseminated Infection-Late
  - Bacteria disseminated throughout the body and across the blood-brain barrier makes treatment challenging.
  - Oral antibiotics and intravenous antibiotics are used with ceftriaxone commonly used for a minimum of 4 weeks.

For further, more in depth treatment information see the web site below:

- From the Infectious Disease Society of America-

www.cid.oxfordjournals.org/content/43/9/1089.full

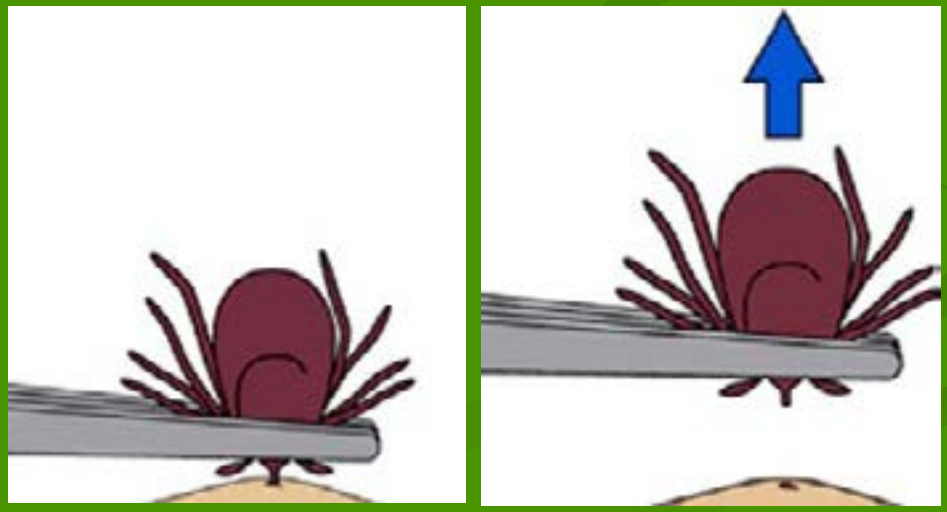
Prevention

Prevention

- Hat, long-sleeved shirts and pants tucked into socks or boots.
- Light colored clothing makes ticks more visible before it attaches.
- Inspect pets carefully.
- Permethrin sprayed on clothes kills ticks on contact.
- ***Remove ticks promptly!***

Tick Removal

- Pull out tick as close to skin as possible without twisting or crushing the body of the tick.
- Risk of infection increases with the time the tick is attached.
- Less than 24 hours, infection is unlikely.



Prevention

- In the community, reduce the number of primary hosts such as rodents, small animals, and deer in order to disrupt the life cycle.
- Unique approach may be to use domesticated guinea fowl.
- Guinea fowl are voracious consumers of insects and arachnids with a fondness for ticks!

Ocular Manifestations of Lyme Disease

Case Presentation

- 26 year old Caucasian male presents with:
  - Acute onset redness, pain OS.
  - OS +2 injection temporally with small elevation. Exam was otherwise normal.
  - Original diagnosis was nodular scleritis. Pt initially treated with indocin x 2 weeks.
  - At follow-up 2 weeks later, pain resolved; redness "90% improved."
  - OS still with moderate injection. More extensive work-up performed.

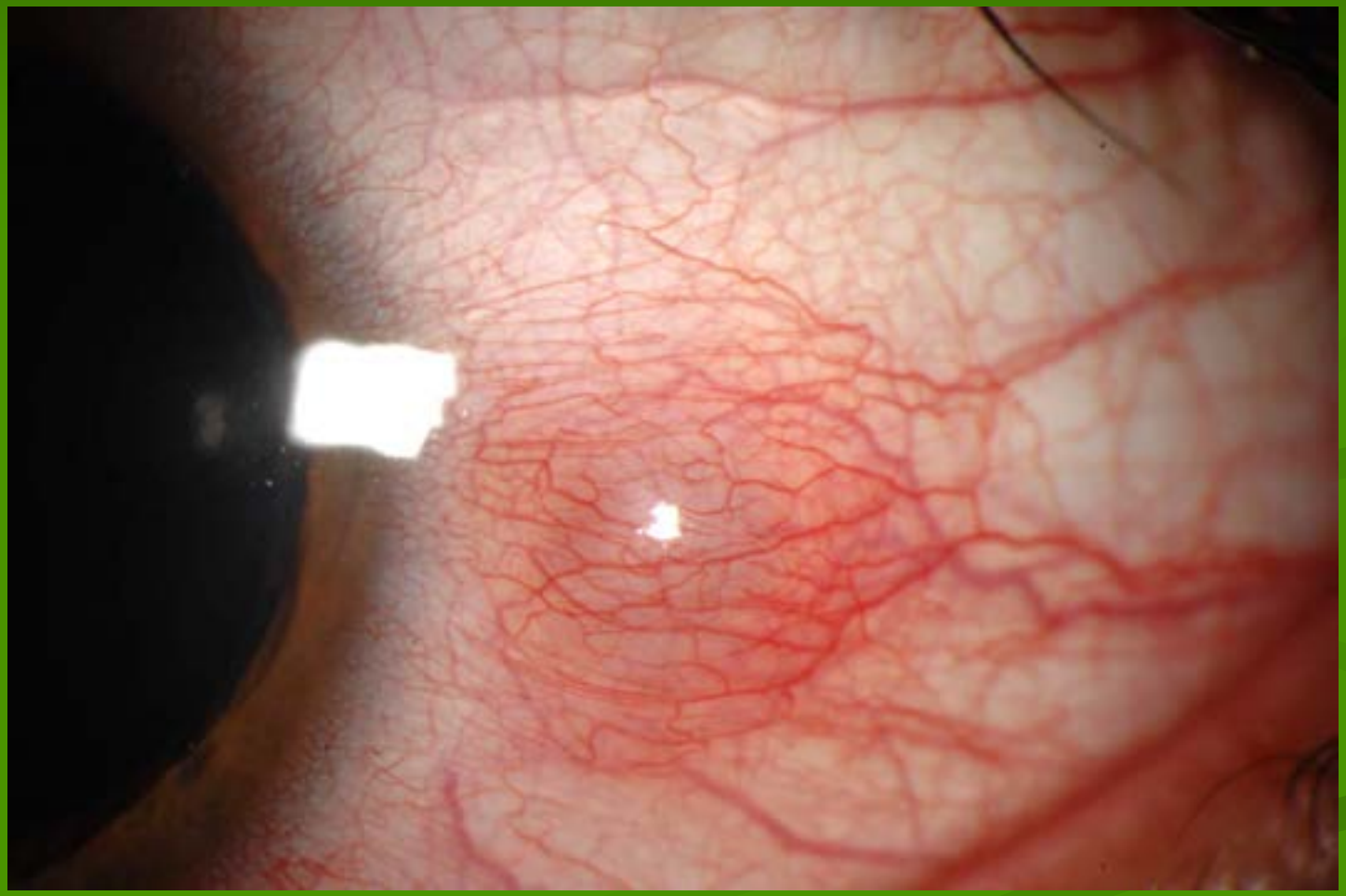
Case Presentation-2nd Visit

- ROS: Fatigue x 2 months and linear rash across chest "a few weeks ago."
- Medications: None.
- POH: Myopia.
- PMH: Attention deficit disorder and Mononucleosis (2002).
- FH: Glaucoma (mother) and Sarcoidosis (mother).
- SH: Lives in Allentown, PA, smokes 1 ppd, social ETOH, denies illicit drug use, single with no children, one dog, and employed by Norfolk Southern as conductor

Clinical Examination-2nd Visit

- Va (cc) 20/20 OD, 20/16 OS
- Ta 12 OD, 14 OS
- PERRL; no RAPD
- Color 10/10 OU
- EOM full
- CF full to count fingers OU
- Normal DFE with no evidence of posterior involvement

Slit Lamp Exam



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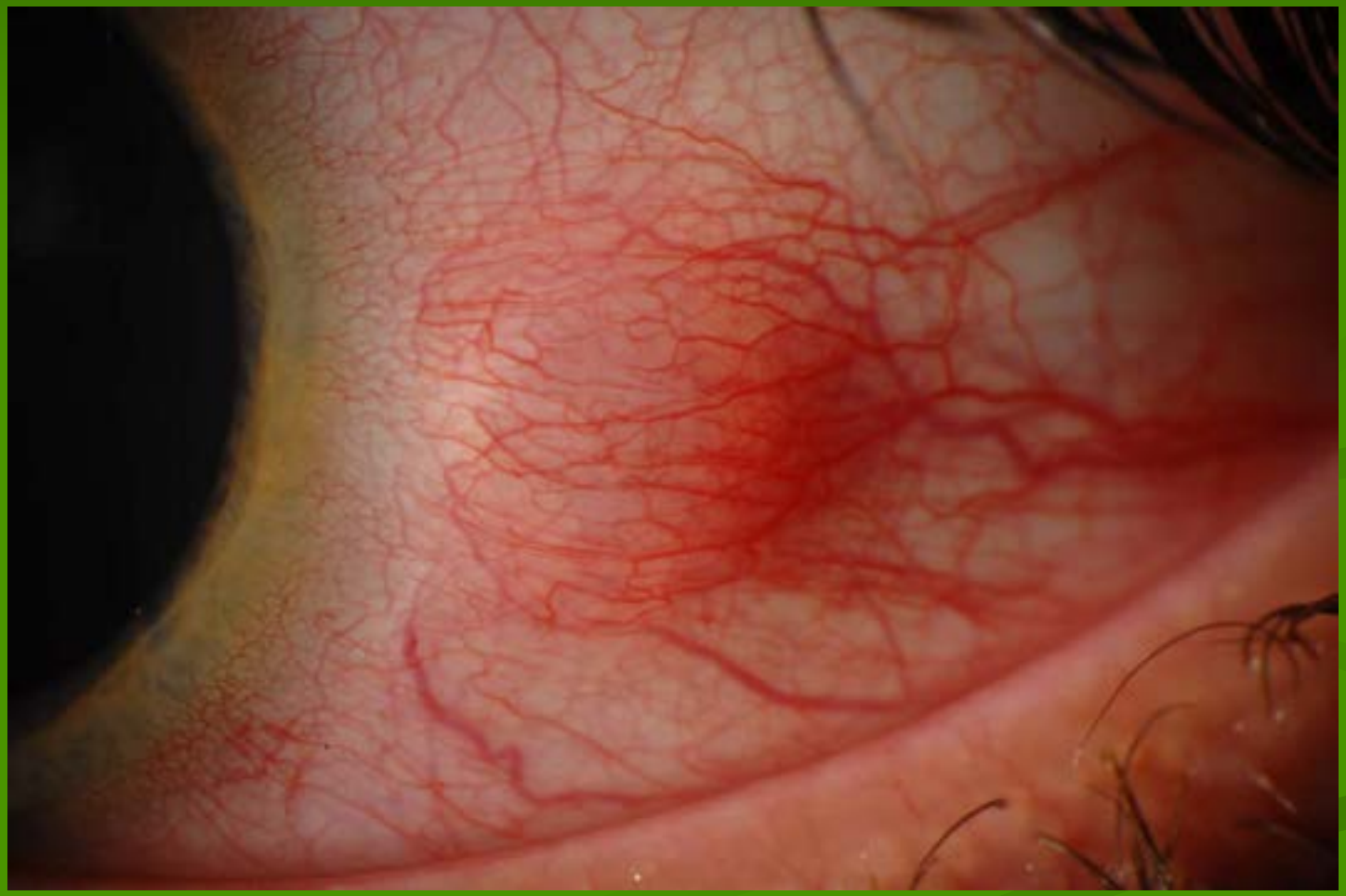

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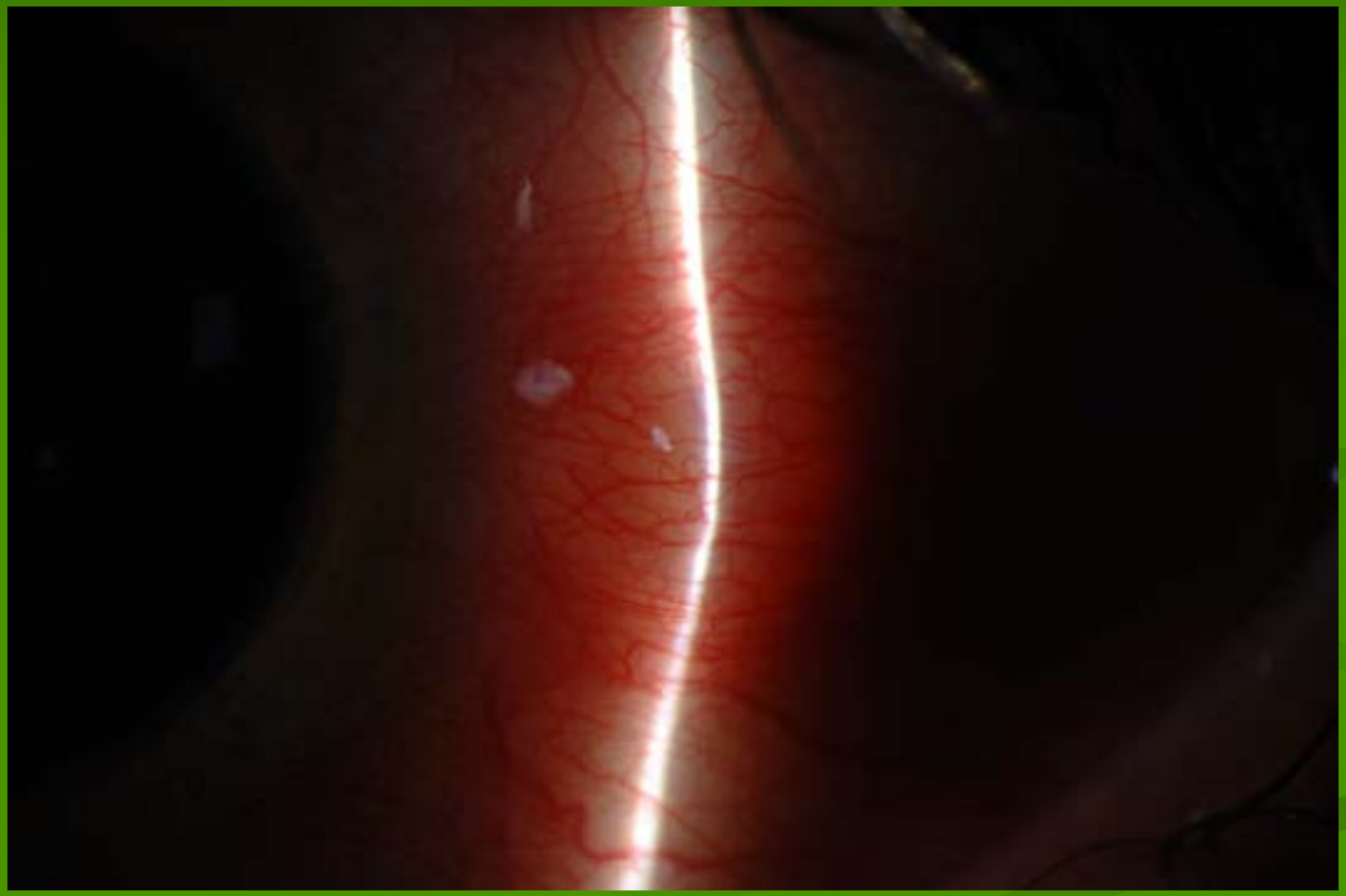


Laboratory Work-up

- RPR, FTA-Abs negative
- HLA-B27 negative
- P-anca, C-anca negative
- Angiotension converting enzyme (ACE) negative
- CXR normal
- Lyme
  - ELISA positive
  - 8/10 positive IgG bands
  - 2/3 positive IgM bands

Treatment Plan

- Doxycycline 100 mg PO bid x 1 month course.
- Indocin 50 mg PO bid.
- Patient referred to primary care for long-term follow-up.
- Symptoms resolved completely and patient feels he is “back to normal.”

Ocular Manifestations of Lyme Disease

1992

Infectious scleritis: Report of four cases

MAITE SAINZ DE LA MAZA, RAMZY K. HEMADY &
C. STEPHEN FOSTER

Ocular Immunology Service, Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, MA, USA

Accepted 30 December 1992

Key words: Antimicrobial therapy, Corneoscleritis, Infection, Scleral biopsy, Scleritis

Abstract. While systemic autoimmune diseases are the main possibilities in the differential diagnosis of scleritis, other less common etiologies such as infections must also be considered. The authors report four cases of infectious scleritis to review predisposing factors, clinical characteristics, methods of diagnostic approach, and response to therapy. Two patients had primary scleritis and two patients had secondary scleritis following extension of primary corneal infection (corneoscleritis). Diagnoses included three local infections (one each with *Staphylococcus*. *Acanthamoeba*, and herpes simplex) and one systemic infection (Lyme disease). Stains, cultures, or immunologic studies from scleral, conjunctival, and/or corneal tissues, and serologic tests were used to make the diagnosis. Medical therapy, including antimicrobial agents, was instituted in all patients, and surgical procedures were additionally required in two patients (scleral grafting in one and two penetrating keratoplasties in another); the patient who required two penetrating keratoplasties had corneoscleritis and underwent eventual enucleation. Infectious agents should be considered in the differential diagnosis of scleritis.

2000

The Expanding Clinical Spectrum of Ocular Lyme Borreliosis

Helena O. Mikkilä, MD,[1] Ilkka J. T. Seppälä, MD,[2] Matti K. Viljanen, MD,[3] Miikka P. Peltomaa, MD,[4] Anni Karma, MD[1]

Objective: To delineate the clinical manifestations of ocular Lyme borreliosis, while concentrating on new symptoms and findings and the phase of appearance of ophthalmologic disorders.

Design: Observational case series.

Participants: Ten patients with Lyme borreliosis-associated ophthalmologic findings previously reported from the Helsinki University Central Hospital in addition to 10 new cases that have since been diagnosed.

Intervention/Testing: The patients underwent medical and ophthalmologic evaluation. The diagnosis of Lyme borreliosis was based on medical history, clinical ocular and systemic findings, determinations of antibodies to *Borrelia bugdorferi* by enzyme-linked immunosorbent assay and immunoblot analysis, the detection of DNA of *B. burgdorferi* by polymerase chain reaction, and exclusion of other infectious and inflammatory causes.

Main Outcome Measures: Ocular complaints, presenting ophthalmologic findings, and the stage of Lyme borreliosis were recorded.

Results: Four patients presented with a neuro-ophthalmologic disorder, five had external ocular inflammation, 10 patients had uveitis, and one had branch retinal vein occlusion. One patient developed episcleritis and one patient developed abducens palsy within 2 months of the infection incident. In the remaining 14 patients in whom the time of infection was traced, the ocular manifestations appeared in the late stage of Lyme borreliosis. Two patients with a neuro-ophthalmologic disorder and one with external ocular inflammation experienced severe photophobia, whereas the main reported symptom of the patients with uveitis was decreased visual acuity. Four patients with external ocular disease and one with a neuro-ophthalmologic disorder experienced severe periodic ocular or facial pain. Retinal vasculitis developed in seven patients with uveitis.

Conclusions: Lyme borreliosis can cause a variety of ocular manifestations, which develop mainly in the late stage of the disease. Photophobia and severe periodic ocular pain can be characteristic symptoms of Lyme borreliosis. In the differential diagnosis of retinal vasculitis, Lyme borreliosis should be taken into account, especially in endemic areas. *Ophthalmology 2000;107:581–587 © 2000 by the American Academy of Ophthalmology.*

2002

Posterior Scleritis Associated with *Borrelia burgdorferi* (Lyme Disease) Infection

Doreen Krist, MD, Hartmut Wenkel, MD

Objective: To report on the clinical findings in a patient with posterior scleritis associated with infection with *Borrelia burgdorferi*.

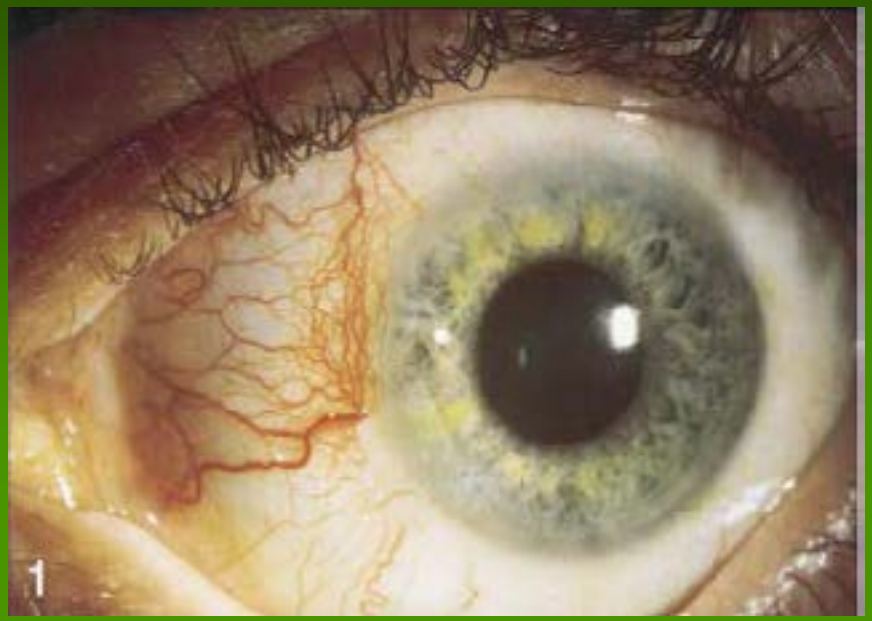
Design: Interventional case report.

Participant: A 39-year-old male ranger who experienced posterior scleritis after several tick bites with erythema migrans.

Testing: Extensive ophthalmic and systemic workup, including serologic testing and imaging techniques.

Results: Sonography and contrast-enhanced computed tomography showed a large scleral mass (16 × 12 × 13 mm) in a patient with painful proptosis in the left eye with episcleral vascular dilation, reduction in bulbar motility, and chorioretinal folds in the upper temporal quadrant. Treatment with high-dose corticosteroids resulted in rapid regression of clinical symptoms and of the scleral mass. Intensive workup revealed immunoglobulin M antibodies (enzyme-linked immunoassay, Western immunoblot) and a positive lymphocyte transformation assay against *B. burgdorferi*. No other cause for posterior scleritis could be identified.

Conclusions: Posterior scleritis should be added to the list of ocular manifestations associated with Lyme disease. Because corticosteroids alone resulted in rapid improvement of clinical symptoms, the scleritis might be mediated by autoimmunologic mechanisms. *Ophthalmology 2002;109:143–145 © 2002 by the American Academy of Ophthalmology.*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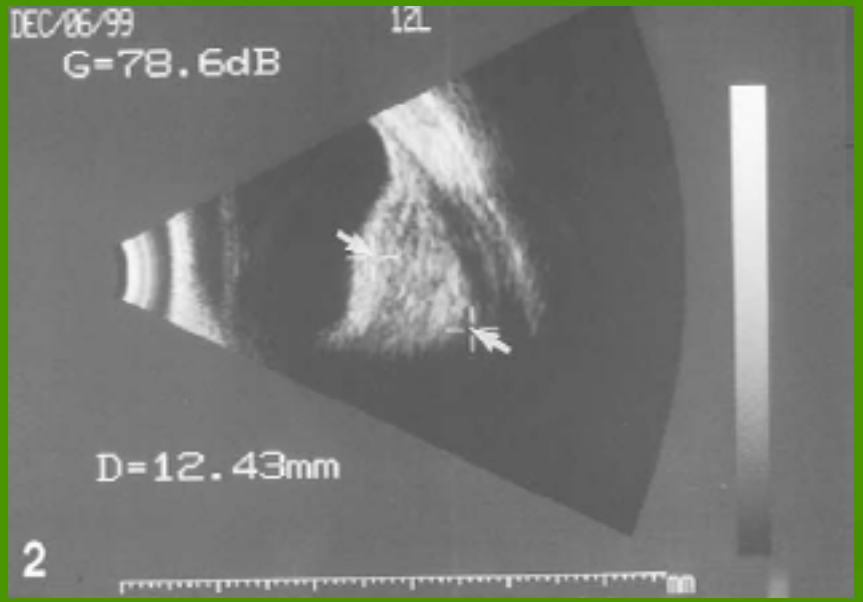
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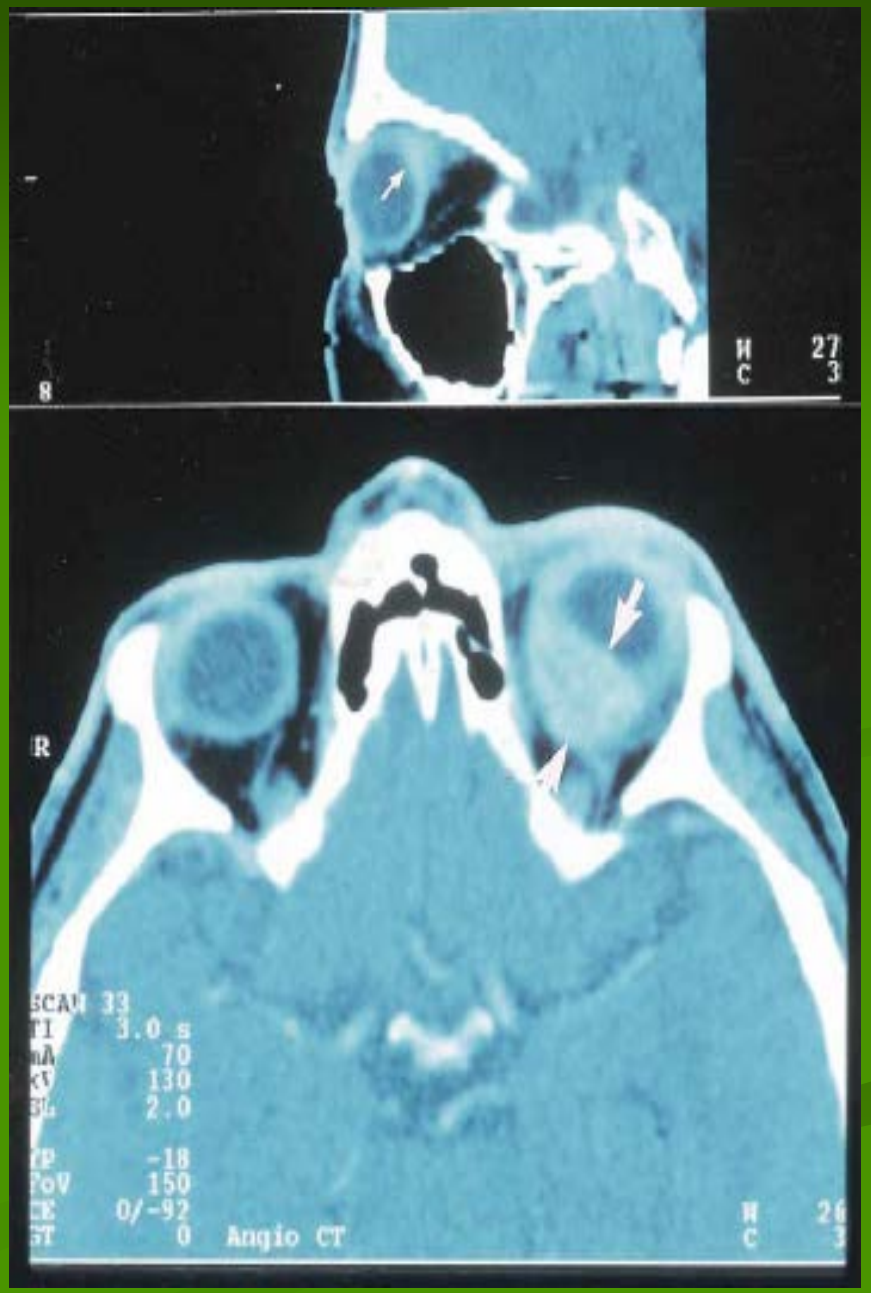
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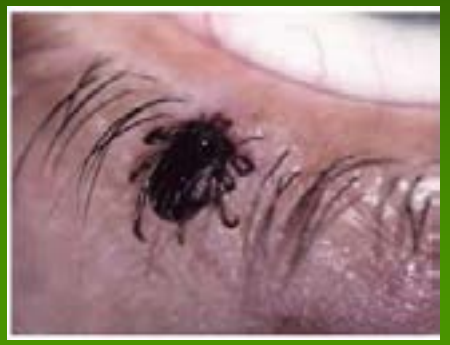
Ocular Manifestations of Lyme Disease

- Ocular Lyme disease cases are uncommon and therefore definitive treatment regimens may vary.
- As in the case presented, the usual treatment failed to correct the problem and thus an investigation was undertaken-suspicion of Lyme disease was high on the list.
- Treatment of ocular conditions may take longer than normal.

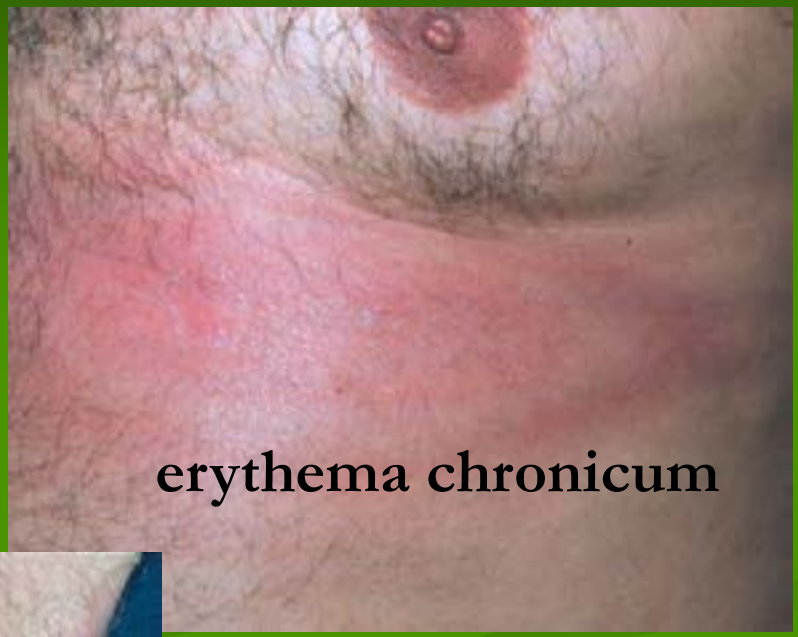
Ocular Manifestations of Lyme Disease

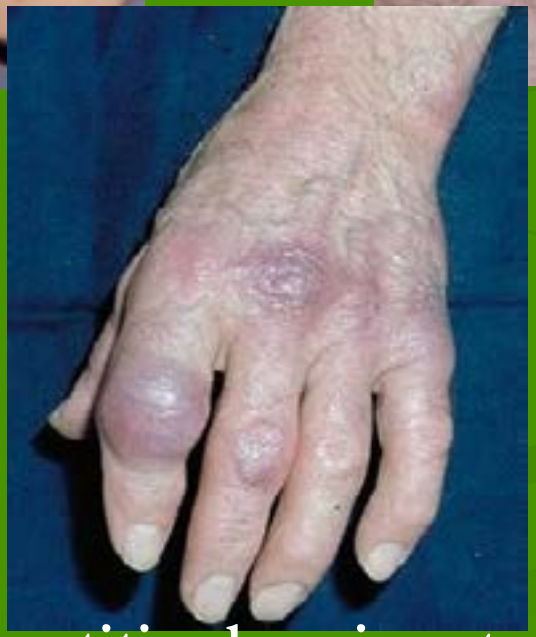


- Conjunctivitis
- Photophobia
- Bell's Palsy
- Cranial neuropathy/diplopia
- Disc edema and blurred vision
- Headache
- Episcleritis
- Symblepharon
- Keratitis
- Iritis
- Pars planitis
- Vitritis
- Choroiditis
- Panuveitis
- Retinal vasculitis
- Exudative retinal detachment
- Branch retinal artery occlusion
- Birdshot chorioretinopathy

Clinical Manifestations of Lyme Disease

	Systemic	Ocular
Stage 1	erythema migrans (80%)	follicular conjunctivitis
	fever, malaise, arthralgias	
Stage 2	mono/oligo arthritis (80%)	any signs
		uveitis/vitritis
	erythema chronicum	choroiditis
	CNS: meningitis, Bell's (40%)	episcleritis
	cardiovascular	neuro-ophthalmic
Stage 3	episodic arthritis	keratitis
	acrodermatitis chronica atrophicans	
	chronic CNS: fatigue, memory loss	





acrodermatitis chronica atrophicans

Ocular Manifestations of Lyme Disease

- Early Lyme disease affecting the eye is mild, brief, and largely resolves on its own.
- Eye care professionals will most likely encounter the Lyme disease patient in Stage 2 with the disseminated disease.
- Neuro-ophthalmic complaints are the most common reasons patients seek eye care.

Ocular Manifestations of Lyme Disease

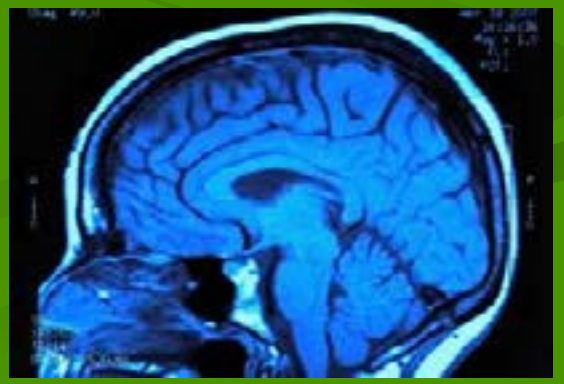
- Bell's Palsy or Facial nerve palsy very common manifestation of Lyme disease
  - One report in an endemic area reported Lyme disease was responsible for 25% of new-onset Bell's palsy.
  - Most cases are unilateral but bilateral involvement is not uncommon.
  - Paralysis largely resolves in about a month with treatment of Lyme disease.

Ocular Manifestations of Lyme Disease

- Diplopia is most commonly caused by a VI nerve palsy, but III and IV nerve palsies have been reported.
- Usually diplopia resolves without permanent sequelae.

Ocular Manifestations of Lyme Disease

- Blurred vision should lead to the search for optic nerve involvement such as papilledema, atrophy, neuritis.
- May be unilateral or bilateral.
- Can have associated systemic symptoms with optic nerve findings as they may indicate central nervous system involvement.

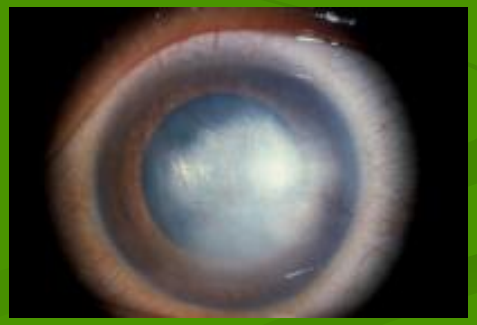


Ocular Manifestations of Lyme Disease

- In Stage 2 and 3 the presence of inflammatory disorders are most common.
- Keratitis, vitritis, and par planitis appear frequently.
- As the disease progresses, posterior segment inflammatory changes become more prevalent.

Ocular Manifestations of Lyme Disease

- Keratitis
  - Usually seen only in Stage 3.
  - Patient complains of blurred vision, foreign body sensation, and photophobia.
  - Presents as bilateral patchy, stromal keratitis with focal nummular opacities.



Remember…Think of Me!

Let's Go Over the Quiz…

Answers

1. Lyme disease occurs throughout the United States: ***FALSE.***

   Although Lyme disease cases have been reported from all 50 states, these reports reflect where the patient lives, which is not necessarily where he or she became infected. In truth, infected ticks of the type that transmit Lyme disease are not found in many states. In the states without infected ticks that spread Lyme disease, infections are usually the result of travel to a state where the disease is common, especially states in the northeast and upper Great Lakes regions.

2. The "two-tiered" blood test for Lyme disease is unreliable.

FALSE.

The "two-tiered" blood test measures antibodies that the human body naturally makes to "fight off" infection. The blood is analyzed first with a test known as ELISA or EIA. If the result is positive or borderline, then a second test, known as a Western Blot, should be performed.

It will typically take up to several weeks after a person is infected for the test to produce a positive result. This delay is common for antibody tests. In particular, patients with a pink or reddish "bulls-eye" rash (erythema migrans) may have negative test results early in the illness. However, patients who have been ill and infected for more than a few weeks will test positive 85-100% of the time.

3. Lyme disease is transmitted from person to person:

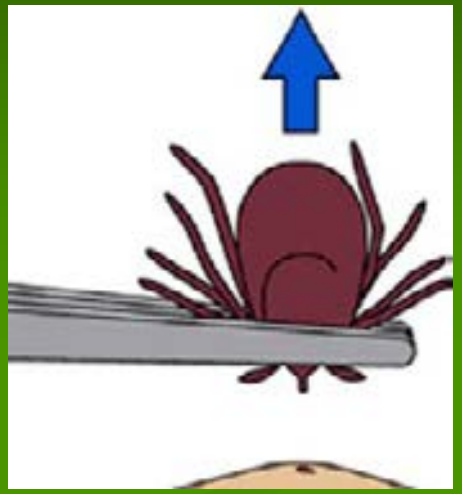
FALSE.

The only proven means of Lyme disease transmission is through the bite of a *Borrelia burgdorferi* infected tick. Although you may have heard that Lyme disease can be transmitted from person to person through breast milk or sexual contact, there is no scientific evidence for either of these routes.

The ticks that transmit Lyme disease are very small and often go unnoticed. Because family members usually share the same environment where infected ticks may be present, it is possible for more than one family member to become infected. This does not mean, however, that the disease is spread from person to person.

4. The best way to remove an attached tick is:
(Choose one)

A. Burn it off with a hot match
B. Apply petroleum jelly
C. Grasp the tick with tweezers close to the skin and pull
D. All of the above work



C. Correct.

The best way to remove an attached tick is: Grasp the tick close to the skin with tweezers and pull straight away from the skin.

5. A tick must be attached to a person's skin for more than 24 hours before it can transmit Lyme disease.

TRUE.

Ticks that transmit Lyme disease can take 3 or more days to feed fully. If the tick is infected, the chances of transmission increases with time, from 0% at 24 hours, 12% at 48 hours, 79% at 72 hours and 94% at 96 hours. This is the reason it is important to conduct tick checks after working or playing in tick infected areas, removing any ticks you find promptly.

Concluding Statements

- Worldwide disease increasing in frequency as populations increase and recreation activities take people into high risk areas.
- Early disease represents the infectious process.
- Late disease likely represents an immune mediated response.
- Systemic and ocular Lyme disease may mimic many other diverse clinical entities, but with a **high index of suspicion** Lyme disease can be correctly diagnosed.

Thank you!