



MEDICAL UNIVERSITY – PLEVEN
FACULTY OF MEDICINE
**DEPARTMENT OF INFECTIOUS DISEASES, EPIDEMIOLOGY,
PARASITOLOGY AND TROPICAL MEDICINE**

Lecture № 12

LYME DISEASE

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Lyme disease – definition

- **Acute multisystemic infectious disease with tend to chronic course, that is caused by *Borrelia burgdorferi*, transmitted to human at tick biting.**

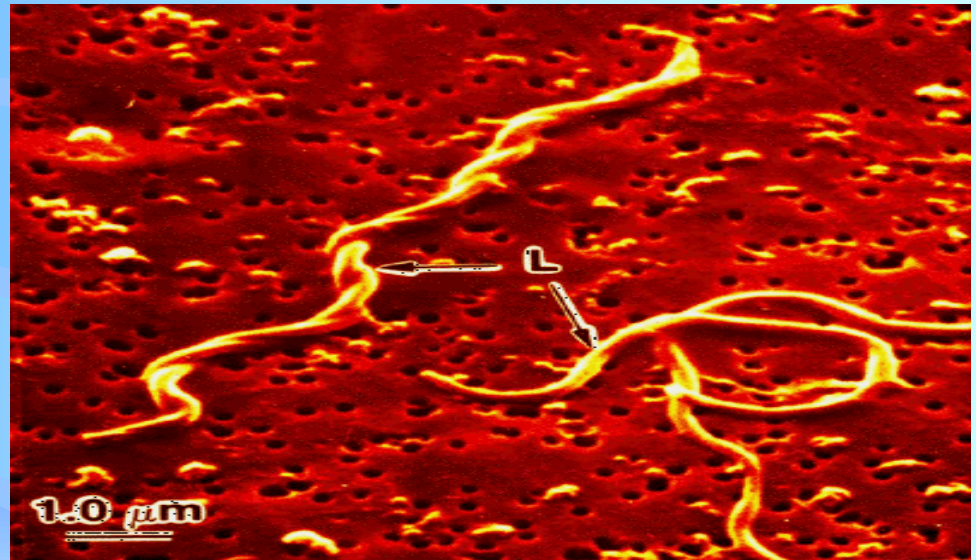
Lyme disease – etiology

- Causative agents – 3 species borrelia in the complex
Borrelia burgdorferi sensu lato:
 - ❖ **Borrelia burgdorferi sensu stricto**
 - ❖ **Borrelia afzelii**
 - ❖ **Borrelia garinii.**
- Gram (-) spirochetes with protoplasmic cylinder and 7-11 peritriches. Do not produce toxins, lead to infection by migration through the tissues of the host, avoiding the host's immune response and production of similar to toxin products.

From Burgdorfer W, et al. Lyme disease-a tick-borne spirochetosis? *Science*, 1982;216:1317. Reprinted with permission from AAAS. All rights reserved.



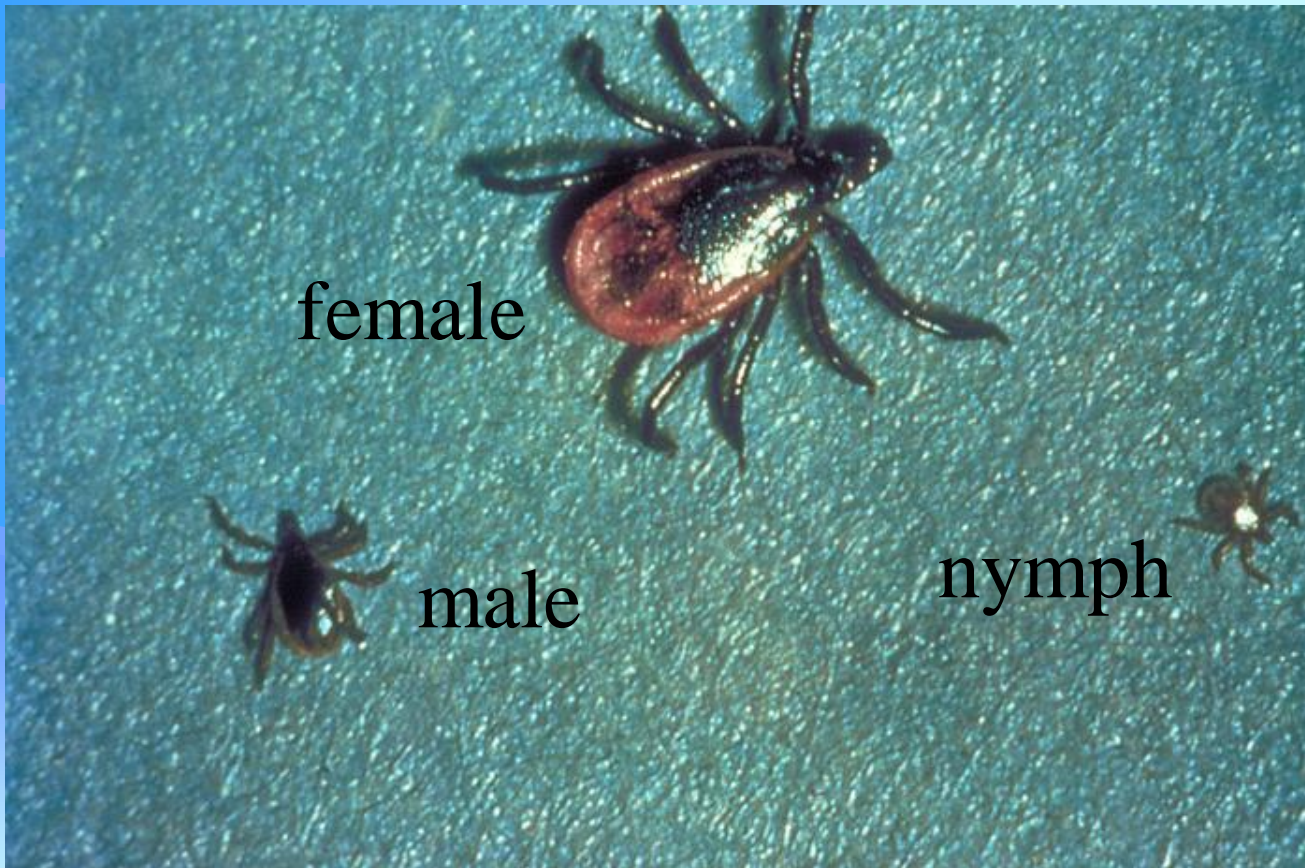
Borrelia burgdorferi



Lyme disease – epidemiology

- The most frequently transmitted by tick disease in the North America and Europe.
- Risk groups – forest workers, tourists, people in contact with a deer and livestock.
- The human' infection is by infected tick biting from the complex Ixodes – *Ixodes scapularis*, *I. pacificus* (in USA), *I. ricinus* (in Europe) and *I. persulcatus* (in Asia). The life cycle of the ticks is 2 years and includes 3 phases – larva, nymph and imago. Only the nymphs and imagoes transmit the borrelias. The nymphs have the most epidemiological significance.
- Spring-summer seasonal peak.
- The biting is painless. The transmission of borellia is within 24-48 hours after tick biting.

Ticks Ixodes scapularis



Lyme disease – pathogenesis

- After the inoculation in the skin *B. burgdorferi* migrates around the place of inoculation and causes erythema migrans or by the circulation disseminates to other organs. The dissemination in the skin and tissues facilitates by human plasminogen that transforms to plasmin. This increases the penetration ability of borrelia.
- *B. burgdorferi* stimulates production of cytokines (at lyme-arthritis interleukin 1 and tumor-necrotizing factor; at neuroborreliosis – interleukin 6). There are and autoimmune mechanisms in the pathogenesis.
- *B. burgdorferi* is able to cause persistent infection. It enters in endothelial cells and fibroblasts (despite of been extracellular pathogen) and avoids immune response and antibiotics.

Lyme disease – clinical manifestations

- The natural course of the infection (without antibiotic treatment) has 2 phases – early and persistent.

1. The early phase (acute infection) includes:

- Early localized (1st stage) – erythema migrans, flu-like Lyme borelliosis .
- Early disseminated (2nd stage) – dermatologic, neurologic, cardiac, joints manifestations etc.

2. Late phase (persistent infection, 3rd stage) – joints, dermatologic and neurologic manifestations.

This distinction is conditional because each stage is possible to be an onset of the disease.

Lyme disease – clinical manifestations

1. Early localized (1st stage) Lyme borreliosis – pathognomonic skin lesion – **erythema migrans** – centripetal enlargement of the peripheral margin within days and weeks (not within hours). Concentric circles are possible or as “bull eye”. Enlarged regional lymph nodes are palpable. Early localized flu-like Lyme borreliosis is rare – with fever, weakness, myalgia, arthralgia.
2. Early disseminated Lyme borreliosis:
 - Skin – multiple erythema.
 - Nervous system – neuroborreliosis (more frequent in Europe) – aseptic meningitis, cranial neuritis (most often facial nerve), in Europe – meningopolyneuritis (syndrome of Bannert).
 - Cardiac involvement – AV-blockage, more rare myocarditis, pericarditis or rhythm disorders.
 - Migrating pain in the joints, muscles and tendons within weeks.

*First stage (localized Lyme disease) –
erythema migrans (“bull eye”)*



*First stage (localized Lyme disease) –
erythema migrans*



*First stage (localized Lyme disease) –
erythema migrans (“bull eye”)*



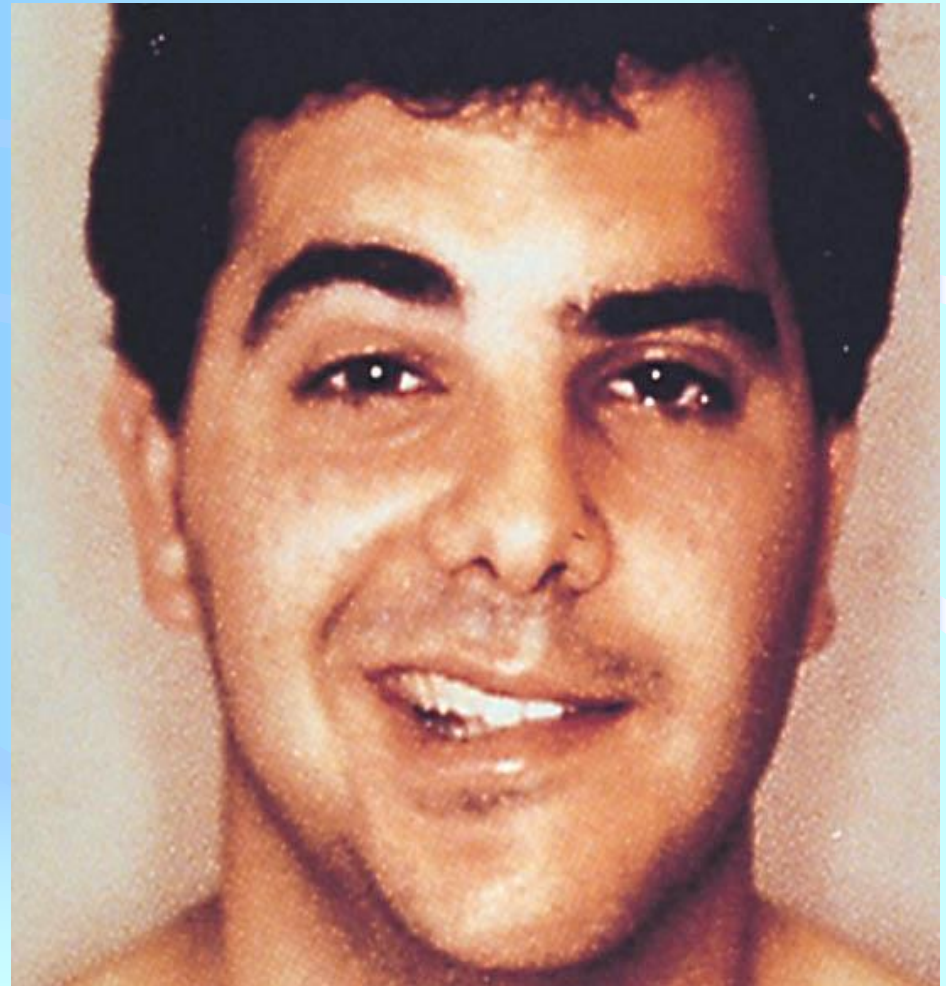
*First stage (disseminated Lyme disease) –
multiple erythema*



*First stage (disseminated Lyme disease) –
multiple erythema*



*Lyme disease –
unilateral Bell's palsy*



Lyme disease – clinical manifestations

4. Late phase (persistent infection) Lyme borreliosis.

- Skin – chronic atrophic acrodermatitis – blue-reddish skin lesion on the distal parts of the limbs evolving to the atrophy, sclerotic changes and ulcerations frequently combined with peripheral neuropathy.
- Lymphocytoma – blue-reddish painless node on the ears (children) or mammilla (adults) combined with regional lymphadenitis. It is possible to appear during 2nd stage as once clinical sign of the disease.
- Mono/oligoarthritis – at untreated patients in North America – most often knees, ankles and elbows. Undulating course with periods of exacerbations and remissions. Spontaneous recovery.
- Nervous system – most rare during the late phase – encephalomyelitis, encephalopathy, or peripheral neuropathy.
- Ocular involvement (in early and late phases) – uveitis, ceratitis, vitreitis, optic neuritis – casuistic.

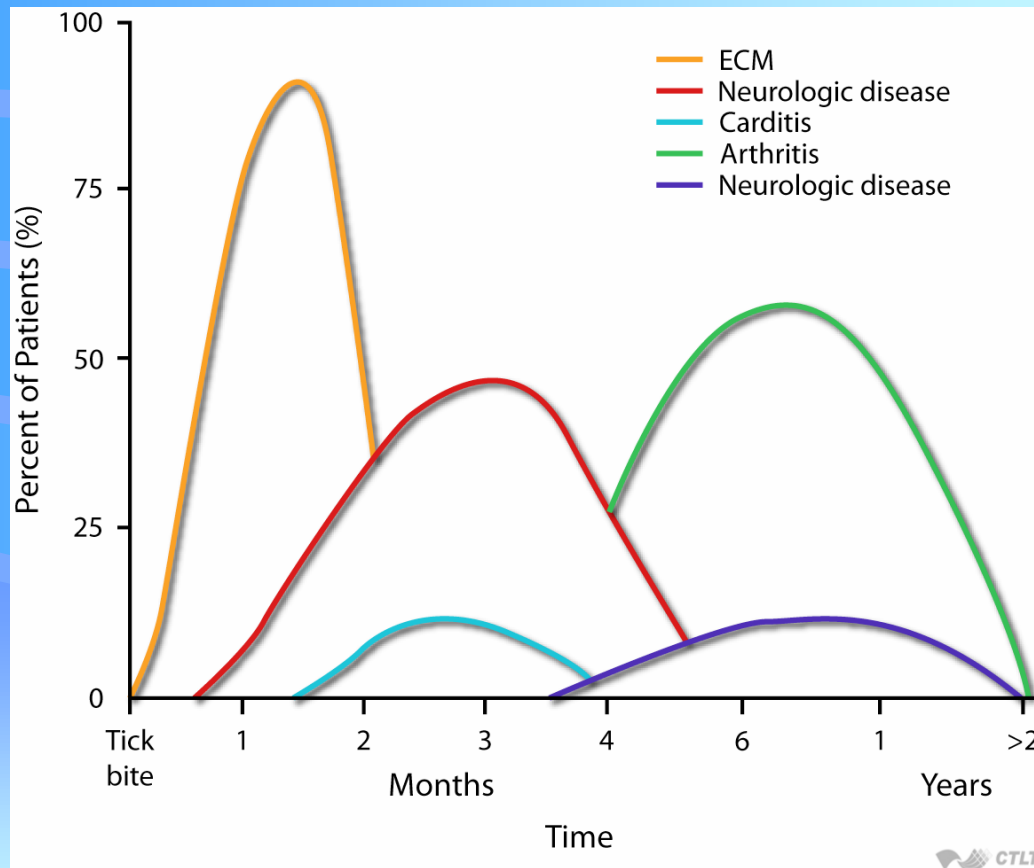
Late persistent Lyme disease – chronic atrophic acrodermatitis



*Late persistent Lyme disease –
monoarthritis*



Lyme disease – chronology of clinical syndromes



Lyme disease – laboratory findings

- The routine laboratory investigations are normal. Slightly elevated transaminases are found in 1/3 of the patients.
- At neuroborreliosis – common changes in CSF characteristic for aseptic meningitis – normal or slightly elevated protein level, mild mononuclear pleocytosis and normal glucose level.

Lyme disease – diagnosis

- Clinical and epidemiologic data.
- Microbiological – only in high-level reference laboratories.
- Serologic – taking of serum sample after 20-22 days after tick biting (no earlier). The negative result does not eliminate the diagnosis. It is necessary to repeat the investigation after 6-8 weeks. ELISA and Western blot are used.

Lyme disease – treatment

- **Etiologic** – doxycyclin, amoxicillin, cefuroxime. Course of treatment 14-21 days, at prolonged disease with/or systemic manifestations – 21 days. At disseminated forms – penicillin, ceftriaxon 21-30 days.
- It is not recommended a prolongation of the treatment to disappearing of the manifestations because the recovery is within months. Full recovery by peripheral neuropathy and encephalopathy is in the second year.

Lyme disease – prophylaxis



МАЙ ЮНИ ЮЛИ



Lyme disease – prophylaxis

- **Management of tick biting:**
- The tick removes by a pincers.
- Obligatory by glows!!!
- Do not turn!!!
- Disinfection with iodine and ethanol.
- If the tick's head is in the skin – surgery removing.
- Killing of the tick by burning or in a vial with ethanol. Do not crush!!!
- By this mode is removing of ticks from the pets.
- An observation for local manifestations within 30 days (erythema, swelling, pain on the place of biting) and general manifestations (fever, arthralgia, myalgia, rash).

**THANK YOU
FOR THE ATTENTION !**