

Contagion and Spoliation-Tularemia Lymphadenitis

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Tularemia is engendered due to *Francisella tularensis*, an aerobic, gram negative, pleomorphic *Coccobacillus*. Animal vectors as rodents, rabbits and hares harbour the contagious organism whereas insects, ticks or deer flies transmit the bacterium. Additionally designated as rabbit, hare, deerfly or lemming fever, the virulent organism may be employed for biological warfare.

Bacterial infection commonly occurs due to aerosol, droplets and contamination of food and water by rodents, hunting hares or mosquito bites. Direct contact with contaminated animal vectors and tick bites emerges as a predominant mode of disease transmission. Infection may induce significant lifelong immunity although reinfection may occur. A vaccine for tularemia remains unapproved.

Contingent to clinical appearance, World Health Organization (WHO) denominates tularemia into distinctive variants as ulceroglandular, glandular, oropharyngeal, oculoglandular, respiratory and typhoid forms. Clinical representation pertains to factors such as bacterium's entry point, virulence of the organism, host's immune status and inoculated bacterial dose. Although varying from one to twenty one days, incubation period for tularemia is approximately three days to five days [1,2].

The infection may appear as an asymptomatic condition. However, sepsis and untimely demise may ensue. Swollen or inflamed palatine tonsils are encountered in > 50% subjects. Initially discerned within summer or autumn months, clinical symptoms as sore throat, dry cough, flu-like symptoms, headache, abdominal pain or initial diarrhoea, weight loss, muscle or body aches, joint pain, fatigue, chills, progressive myalgia, pneumonia, oral or cutaneous ulcers, lymphadenopathy, ocular involvement, rashes, soft tissue abscess, otitis media, meningitis, brain abscesses and rapid onset of pyrexia may be encountered [1,2].

Swelling of lymph nodes, suppuration and a prolonged recovery time may be exemplified. Unexplained lymphadenopathy unresponsive to beta-lactam antibiotics may be observed [1,2].

Ulceroglandular variant expounds prominent lymphadenopathy within axillary, cervical or inguinal regions. The ulceroglandular variant manifests as a miniature, localized, cutaneous lesion as a scratch, cut or insect bite, a breach which configures as an entry point for bacterial ingress. The variant represents as persistent, festering cutaneous ulcer associated with localized or regional lymphadenopathy confined to corresponding cutaneous area [1,2].

Glandular variant simulates the ulceroglandular variant although specific, primary, cutaneous zone of bacterial ingress appears absent. Pneumonic variant ensues due to inhalation of bacterium and delineates pneumonia with symptoms as breathlessness, coughing or painful breathing [1,2].

Plain X-ray chest depicts pulmonary infiltrates and hilar lymphadenopathy [2,3].

Oropharyngeal variant arises due to ingestion of contaminated food or water. Clinical symptoms as sore throat, signs indicative of pharyngitis, tonsillitis, enoral blisters, stomatitis and swollen, painful lymph nodes may appear [2,3].

The oculoglandular variant represents with conjunctiva as accidental, digital mode of bacterial ingress. Characteristically, conjunctival ulceration and nodules upon the conjunctiva may be enunciated. Absence of therapy is associated with bacterial dissemination into circumscribing lymph nodes and swelling of lymph nodes [3,4].

Typhoid tularemia emerges as a systemic disease associated with a severe clinical course and neurological symptoms in the absence of specific zone of bacterial ingress. Local signs and disease manifestations of diverse variants appear absent [3,4].

The conditions represents with nonspecific symptoms as pyrexia, asthenia, headache, myalgia and neurological symptoms as confusion, stupor or behavioural alterations, simulating symptoms of typhoid fever [3,4].

Upon microscopy, necrotizing granulomatous lymphadenitis may be observed [4,5].

Preliminary lesions demonstrate reactive follicular hyperplasia. Subsequently, lymph node parenchyma expounds abscesses along with a variable epithelioid cell reaction followed by emergence of caseous necrosis. Diffuse lymphadenitis may ensue [4,5].

Delayed reaction emerges as granulomatous reaction simulating lesions of tuberculosis. Extracapsular inflammation may be frequently discerned [4,5].

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Table 1: Significant differential diagnosis of cutaneous T cell lymphomas [6,7].

Tularemia requires segregation from conditions as sarcoidosis, toxoplasmosis, tuberculosis, chlamydial infections, diphtheria, bacterial or fungal endocarditis, Legionella infection, Lyme disease, malaria, Psittacosis or Q-fever [8,9].

Tularemia may be aptly ascertained by cogent clinical examination, imaging techniques and serological tests. Bacterial isolation and culture may be challenging. Serological assay with micro-agglutination test, indirect immunofluorescence test and enzyme-linked immunosorbent assay (ELISA) appear as contemporary diagnostic modalities.

ELISA may discern specific antibodies within two weeks of disease ascertainment [8,9].

Serological assay depicts increasing antibody titres. Alternatively, the bacterium may be discerned by polymerase chain reaction (PCR) or immunofluorescence [9,10].

Ultrasonography delineates significantly enlarged, homogeneous or fluid lymph nodes. Besides, a characteristic capsule-like growth indicative of inflammatory alterations within the lymph nodes may be exemplified.

Computerized tomography depicts centrally hypodense, inflammatory, melted or necrotic lymph nodes with contrast-absorption within the lymph node wall. Capsular invasion appears absent [9,10].

Tularemia emerges as a fatal illness which necessitates escalating treatment with surgical intervention or multiple antibiotics. Clinically efficacious and recommended antimicrobial agents as aminoglycosides, fluoroquinolones or tetracycline may be adopted for therapy [9,10].

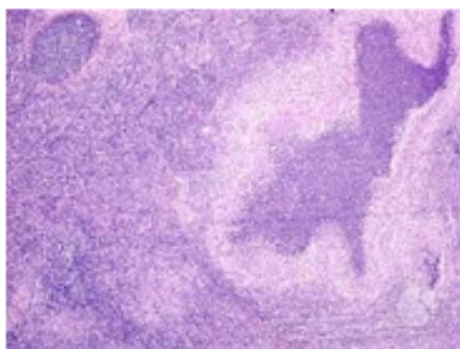


Figure 1: Tularemia expounding configuration of abscess with reactive follicular hyperplasia and variable epithelioid cell reaction [11].

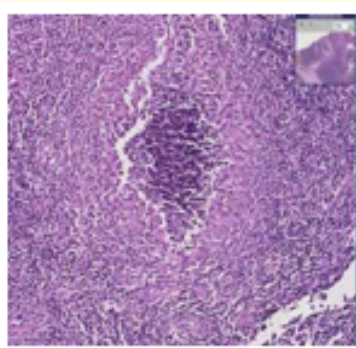


Figure 2: Tularemia enunciating configuration of abscess with reactive follicular hyperplasia and variable epithelioid cell reaction [12].

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11. Image 1 Courtesy: ASH image bank.
12. Image 2 Courtesy: You tube.

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