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Editorial

Coiled and Radial-Luetic Lymphadenitis

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Luetic lymphadenitis describes lymph node pathological modifications induced by infection with spirochetes of *Treponema pallidum* or syphilis. Additionally designated as syphilitic lymphadenitis, the infective lymphadenitis displays characteristic morphological features as significant capsular fibrosis with peri-capsular dissemination of chronic inflammatory cells, follicular hyperplasia with innumerable plasma cells, phlebitis with endarteritis and occasionally discerned, inadequately configured epithelioid cell granulomas.

The bacterial organisms may be appropriately stained by Warthin Starry silver stain. Alternatively, specific immunohistochemistry and bacterial confirmation by serological techniques or polymerase chain reaction (PCR) may be employed.

Syphilis may emerge as a sexually transmitted infection or may configure as congenital syphilis acquired from vertical maternal transmission. Regular screening is recommended for pregnant females, men having sex with men and subjects with human immune deficiency virus (HIV) infection.

Regional lymphadenitis appears to be associated with secondary syphilis wherein inguinal, axillary, cervical or occipital lymph nodes are involved in decreasing order of frequency [1,2].

Spirochetes of *Treponema pallidum* disseminate from primary site of inoculation with configuration of painless ulcer with indurated margins or a 'chancre' and towards regional or distant lymph nodes with consequently emerging lymphadenopathy. The bacterium may be transmitted through sexual contact or vertical transmission of infection from mother to child [1,2].

Infection with *Treponema pallidum* evokes a 'robust' humoral and cellular immune response. However, control of infection is inadequate wherein relapse of infection is frequently encountered and may occur within weeks or months. Pre-eminently, macrophages confined to cutaneous region induce degradation of bacteria [2,3].

Cell membrane of spirochetes or lipoproteins are preponderantly involved with interaction of host immune system with initial induction of an innate immune response [2,3].

Lipoproteins of spirochetes as outer surface protein B (OspB) may prohibit neutrophil function and circumvent an oxidative burst. Additionally, activation of neutrophils with induction of neutrophil extracellular traps, activation of monocytes/macrophages and dendritic cells through CD14 and Toll-like receptor signalling pathways are encountered. Aforesaid manoeuvers induce a pro-inflammatory response and production of cytokines [2,3].

The singular, extraneous surface of *Treponema pallidum* is smattered with minimally exposed lipoproteins and delineates antigenic variation within lipoprotein expression. A comprehensively inferior immune reaction ensues with antibodies arising within serum of syphilitic subjects along with evasion of immune system by *Treponema pallidum* spirochetes.

Secondary syphilis may occur as a consequence of evasion of immune recognition by *Treponema pallidum* spirochetes while engendering inflammation. *Treponema pallidum* infection may trigger systemic immunologic anomalies [3,4].

Occurrence of syphilis enhances possible emergence of human immune deficiency virus (HIV) infection with augmentation of HIV viral load and decimation of CD4+ count within subjects infected with HIV.

As infection with gram negative spirochete *Treponema pallidum* engenders syphilis, clinical representation emerges as:

- Primary syphilis which demonstrates a painless chancre characteristically within 3 weeks to 6 weeks of spirochete inoculation upon site of infection along with or devoid of regional lymphadenopathy.
- Secondary syphilis demonstrates a disseminated maculopapular cutaneous rash or pustular scales. Predominantly localized to palms and soles of feet, the rash is characteristically discerned within 4 weeks to 10 weeks following emergence of the chancre. Well demarcated, hypertrophic papules or plaques or 'condyloma lata' are confined to diverse cutaneous surfaces. Lesion may be associated with diffuse lymphadenopathy, hepatosplenomegaly, hepatitis or nephrotic syndrome [3,4].
- Tertiary syphilis occurs in up to 20 years following infection with *Treponema pallidum* in untreated subjects.
- Cardiovascular syphilis induces an aneurysm or aortic insufficiency wherein granulomatous lesions appear to invade and engender tissue destruction with configuration of gummas and emergence of neurosyphilis.
- Latent syphilis expounds a lack of cogent clinical symptoms.

Cytological examination depicts branching or arborizing vascular articulations circumscribed with perivascular cuff of plasma cells, neutrophils and configuration of epithelioid cell granulomas [3,4].

Frozen section examination depicts features recapitulating morphological manifestations with occurrence of reactive follicles and enhanced plasma cell population. The spirochetes may be suitably discerned upon May-Grünwald Giemsa stain [3,4].

Upon microscopy, lymph node depicts a preserved or partially effaced architecture with distortion. Lymph node capsule is thickened with peri-capsular dissemination of chronic inflammatory cells and plasma cells. Peri-nodal fibrosis is extensive [4,5].

Significant follicular hyperplasia with plasma cell dissemination into inter-follicular, sinusoidal, perinodal and perivascular zones is observed. Lymph node capsule and nodal parenchyma depicts phlebitis with endarteritis. Infiltration with histiocytes is variable. Non-caseating granulomas may be encountered.

Non specific lymph node configurations are represented as articulation of abscess impregnated with spirochetes [4,5].

Ultrastructural examination displays a complex bacterial structure composed of dual layered extraneous wall [4,5].

Nodal lymphomas
Angioimmunoblastic T cell lymphoma
Follicular T cell lymphoma
Nodal peripheral T cell lymphoma with follicular helper T cell (TFH) phenotype
Extra-nodal lymphomas
Cutaneous CD4+ small/medium T cell lymphoproliferative disorder

Table: *World Health Organization (2017) classification of T follicular helper cell lymphomas [3,4].*

May-Grünwald Giemsa stain may be optimally employed to discern the organism. Spirochetes may be highlighted by silver stains as Warthin Starry or modified Steiner stain. Cogent immunohistochemistry may be beneficially adopted to discern the organism. However, precise detection of the organism with Gram's stain or acid fast Ziehl-Neelsen stain may be challenging [5,6].

Luetic lymphadenitis requires segregation from conditions as lymphogranuloma venereum, necrotizing granulomatous lymphadenitis, inflammatory pseudo-tumour, lymphadenitis due to cat scratch disease, toxoplasma lymphadenitis or lymphadenitis due to human immunodeficiency virus (HIV) infection [5,6].

Diagnostic manoeuvers as dark-field examination and molecular assays appear optimal for discerning *Treponema pallidum* organisms obtained directly from lesion exudate or infected tissue. Aforesaid manoeuvers appear pathognomonic for detecting preliminary stage of syphilis or congenital syphilis [5,6].

Precise serological assays applicable for diagnosing syphilis are contingent to antigen subtype towards which antibodies are directed and appear beneficial for ascertaining the presumptive diagnosis:

- Treponemal assays may discern antibodies to *Treponema pallidum* proteins and are ascertained as reactive or nonreactive. False 'positive' reactions may occur within various inflammatory diseases.
- Treponemal assays as micro-hemagglutination assay, particle agglutination, hemagglutination assay, fluorescent treponemal antibody absorbed (FTA-ABS) test, chemoluminescence immunoassays and enzyme immunoassays may be advantageous for detecting the organism [5,6].
- Nontreponemal assays are semi-quantitative, indicate disease activity and enunciate IgM or IgG antibodies directed against structures as lipoidal antigens, damaged host cell or possible treponemes. Nontreponemal tests as rapid plasma reagin (RPR), venereal disease research laboratory (VDRL) test and toluidine red unheated serum test may be beneficially employed for cogent diagnosis [6,7].

A specific or singular assay appears insufficient for precise detection whereas combination of treponemal and nontreponemal assays are advantageously adopted in order to confirm syphilitic infection or determine disease activity [6,7].

Peripheral smear depicts leukocytosis, anaemia and thrombocytopenia wherein aforesaid manifestations are especially encountered within neonates afflicted with congenitally acquired syphilis.

Cogent morphological assessment and immunohistochemistry appears adequate for diagnosis [6,7].

Silver stain as Warthin Starry stain or antibodies may be employed to detect spirochetes within formalin fixed paraffin embedded tissues obtained from subjects of primary or secondary syphilis.

Polymerase chain reaction (PCR) configures as an assay complimentary to serological techniques and appears beneficial in diagnosis of preliminary syphilis or congenital syphilis [6,7].

Dark field microscopy or immunofluorescence may be mandatory for disease determination and appears competent in demarcating *Treponema pallidum* from non-pathogenic treponemes.

Routine culture appears inadequate for precise diagnosis. Upon radiographic imaging, regional or distant lymphadenopathy is observed [6,7].

Therapy with specific antibiotics is associated with superior prognostic outcomes [6,7].

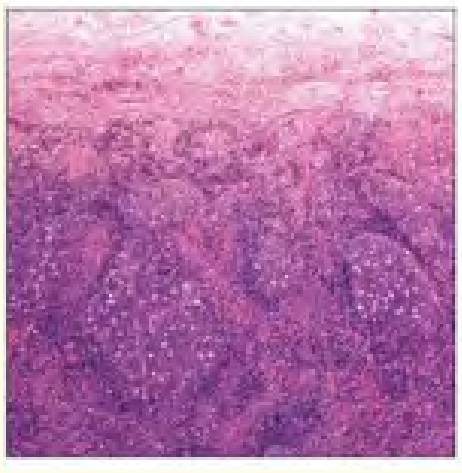

Figure 1: *Luetic lymphadenitis demonstrating epithelioid cell granulomas commingled with micro-abscess impregnated with spirochetes. A smattering of inflammatory cells as neutrophils, plasma cells and lymphocytes is encountered. Perinodal fibrosis is extensive [8].*

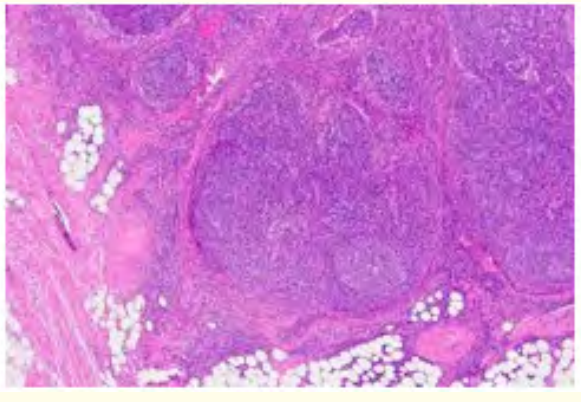

Figure 2: *Luetic lymphadenitis delineating epithelioid cell granulomas intermingled with spirochete laden micro-abscess. An admixture of neutrophils, plasma cells and lymphocytes is observed. Perinodal fibrosis is significant [9].*

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8. Image 1 Courtesy: Basic medical key.

9. Image 2 Courtesy: Pathology outlines.

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