ECRONICON OPEN ACCESS

EC NEUROLOGY
Editorial

Peculiar and Heteroclite-Atypical Mycobacteriosis Lymph Node

Anubha Bajaj*

Department of Histopathology, Panjab University, A.B. Diagnostics, India

***Corresponding Author:** Anubha Bajaj, Department of Histopathology, Panjab University, A.B. Diagnostics, India.

Received: October 10, 2025; **Published:** October 24, 2025

Atypical mycobacterial infection of the lymph node commonly emerges as a granulomatous lymphadenitis. Additionally designated as atypical mycobacterial lymphadenitis or non-tuberculous mycobacterial lymphadenitis (NTML), the disorder represents with unilateral enlargement of anterior cervical lymph nodes.

Generally, immunocompetent paediatric population of up to 5 years or immunocompromised adults are implicated.

Granulomatous inflammation of the lymph node may be accompanied with or appear devoid of soft tissue necrosis. Besides, partial or complete effacement of lymph node architecture with dissemination of sheets of foamy macrophages may be encountered.

Atypical *Mycobacteria* may be ascertained with culture or with exclusion of *Mycobacterium tuberculosis* obtained in concurrence with an indicative histological picture.

Nontuberculous (non-TB) mycobacterial lymphadenitis necessitates definitive clinical ascertainment of infection with techniques as mycobacterial culture or polymerase chain reaction (PCR) assay obtained from purulent discharge or surgical tissue sample. Additionally, fine needle aspirate may be examined for occurrence of epithelioid cell granulomas disseminated within a zone of suppuration.

Atypical mycobacterial infection commonly arises within paediatric population wherein children between one year to 5 years appear susceptible. Besides, immunocompromised adults with human immune deficiency virus (HIV) infection may be involved [1,2].

Generally unilateral, lymph nodes within the head and neck or anterior cervical lymph nodes appear infected [1,2].

Infection is induced by mycobacterial agents which occur as intracellular organisms and appear to replicate within macrophages. Infected macrophages invoke bacterial propagation due to mechanisms concordant with tumour necrosis factor (TNF). Constituent *Mycobacteria* inculcate apoptosis of infected macrophages [2,3].

Fresh batch of recruited macrophages appear to engulf cellular debris with consequent expansion of configured epithelioid cell granulomas. The contemporarily recruited macrophages may extrude from primary epithelioid cell granulomas in order to configure secondary granulomas, especially within distal tissues. Commonly, organisms as *Mycobacterium avium intracellulare* complex, *Mycobacterium marinum*, *Mycobacterium fortuitum, Mycobacterium scrofulaceum* or *Mycobacterium kansasii* may induce the lesions [2,3].

Clinically, chronic, painless, gradually progressive lymphadenopathy is encountered. Generally, unilateral lymph node enlargement is observed within the head and neck [2,3].

Cytological examination depicts epithelioid cell granulomas interspersed within a background of suppuration. Typically, *M. tuberculosis* infection is associated with necrotizing granulomas. Nevertheless, atypical mycobacterial infection may display necrotizing granulomas. Epithelioid cell granulomas devoid of necrosis appear to indicate the occurrence of sarcoidosis [3,4].

Frozen section examination recapitulates morphological features of the lymph node [3,4].

Grossly, an enlarged, rubbery lymph node of tan hue is observed. Cut surface is glistening and expounds irregular, multifocal zones of soft tissue necrosis [3,4].

Upon microscopy, lymph node depicts granulomatous inflammation along with or devoid of soft tissue necrosis. Additionally, features as articulated micro-abscesses, inadequately defined granulomas, non-caseous granulomas and few giant cells appear to indicate the presence of nontuberculous mycobacterial infection [3,4].

The infection may represent with a population of bland appearing foamy histiocytes permeated within sinuses or occurring as diffuse sheets replacing the lymph node parenchyma partially or completely, thereby configuring a mycobacterial pseudo-tumour [3,4].

Morphological features	**Progressive transformation of germinal centre**	**Nodular predominant Hodgkin's lymphoma (A/B)**	**Lymphocyte rich classic Hodgkin's lymphoma, nodular variant**
Follicles			
Magnitude	Scattered large nodules	Nodules larger than PTGC	Moderately enlarged
Germinal centre	Variably disrupted with involution	Absent	Present in tumour nodules
Centrocytes and centroblasts	Decreased, scattered BCL2-	Absent	In residual GCs
Mantle zone lymphocytes	Inward growth into GC, BCL2+, IgD+	Absent	Expanded
RFH	Present	Focal/absent	Focal/absent
Immuno-architecture			
T follicular helper cell rosettes (PD-1+)	Absent	Present	May be present
IgG4+ plasma cells	~50% instances	Absent	Absent
Large B cells	Rare immunoblasts	LP (popcorn cells)	Present, HRS cells
Immunophenotype	IgG+	CD20+, CD45+, OCT2+, EMA+/-, PAX5+ strong, IgD-/+	CD30+, CD45-, CD15+, PAX5+ dim, OCT2-, EMA-, CD20

Table: *Differentiation between progressive transformation of germinal centres and lymphomas with large nodules [3,4].*

PTGC: Progressively Transformed Germinal Centre; RFH: Reactive Follicular Hyperplasia; GC: Germinal Centre; HRS: Hodgkin's Reed Sternberg; LP: Lymphocyte Predominant; EMA: Epithelial Membrane Antigen.

Atypical *Mycobacteria* appear acid fast and stain red with Ziehl-Neelsen stain, Kinyoun stain or Fite stain. The sensitive Auramine O stain demonstrates the organisms as bright yellow, luminous rods disseminated within a dark background with fluorescent microscope. Also, tuberculous and non tuberculous mycobacteria may be detected equivocally. Acid fast bacilli are not discerned by Gram's stain [4,5].

Atypical mycobacteriosis may be detected by assays such as 16S rRNA sequencing. Polymerase chain reaction (PCR). Restriction fragment length polymorphism (PCR-RFLP) appears competent in rapidly discerning various (~28) species of *Mycobacteria* observed clinically.

For cogent bacterial detection, multiple samples of culture or polymerase chain reaction (PCR) assay are recommended [5,6].

Atypical mycobacteriosis requires segregation from conditions as tuberculous mycobacterial infection, infection with various fungi, sarcoidosis or foreign body giant cell reaction [5,6].

Organism culture is precise and diagnostic with a specificity of ~100%. Alternatively, polymerase chain reaction (PCR) can be beneficially employed with a specificity of ~100%. Cogent immunoassays may be adopted with a sensitivity and specificity of up to 100% [5,6].

Cutaneous testing with purified protein derivative S (PPD-S) may be employed for discerning the organism whereas multiple species may be detected with variants as PPD-A and PPD-K [5,6].

Polymerase chain reaction appears appropriate for detecting *Mycobacterium tuberculosis* and six dominant nontuberculous mycobacterial species [5,6].

Nucleic acid amplification (NAAT) appears relevant for discerning *Mycobacterium tuberculosis* complex, *M. tuberculosis* and *M. bovis* with a sensitivity of ~90%. However, the assay appears inappropriate for precise disease monitoring although ribonucleic acid (RNA) levels may be ascertained for up to six months following commencement of therapy.

Ultrasonography depicts a nodule with decimated echogenicity, intra-nodal accumulation of liquefactive or cystic necrosis, matted adjacent lymph nodes or soft tissue oedema of adjoining tissue [6,7].

Infected lymph nodes may be managed with comprehensive surgical excision. However, possibility of facial nerve palsy is significantly augmented with adoption of site-specific surgical manoeuvers [6,7].

Antibiotics as macrolides and rifampin are frequently employed for a duration of six months. Adoption of cogent therapeutic strategies as surgical eradication or antibiotics for extended duration appears beneficial, irrespective of site and quantifiable lymph nodes.

Spontaneous retrogression of disease may occur following six months of disease occurrence [6,7].

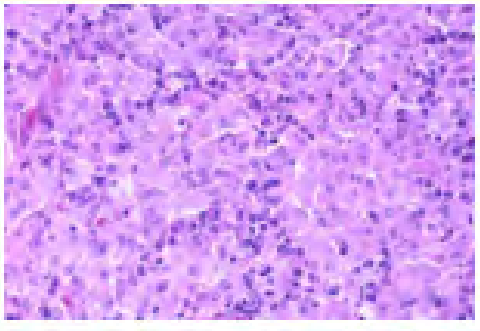

Figure 1: *Atypical mycobacterial infection depicting ill formed granulomas, few giant cells, and focal suppuration [8].*

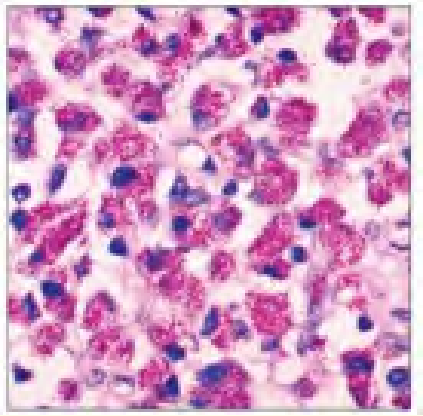

Figure 2: *Atypical mycobacterial infection delineating chronic inflammatory cells as plasma cells, lymphocytes and focal suppuration [9].*

Bibliography

1. Winburn B and Sharman T. "Atypical mycobacterial disease". Stat Pearls International. Treasure Island, Florida (2025).
2. Michaelides I., *et al*. "Cervical *Mycobacterium genavense* infection in a patient with lymphadenitis and previously unknown anti-IFN-γ IgG autoantibodies". *Infection* (2025).
3. Freeman AM and Matto P. "Lymphadenopathy". Stat Pearls International. Treasure Island, Florida (2025).
4. So M., *et al*. "Pulmonary lymphomatoid granulomatosis in a patient with long-term use of a tumour necrosis factor-α inhibitor". *BMJ Case Reports* 16.5 (2023): e254211.
5. Bethencourt Mirabal A., *et al*. "Lung nontuberculous mycobacterial infections". Stat Pearls International. Treasure Island. Florida (2025).
6. Denicolò S., *et al*. "Sarcoid-like lesions obfuscating the diagnosis of disseminated *Mycobacterium genavense* infection in a patient with IL-12Rβ1-associated immunodeficiency". *BMC Infectious Diseases* 22.1 (2022): 770.
7. Shinohara K., *et al*. "Cervical abscess caused by *Mycobacterium tilburgii* in a patient carrying anti-interferon gamma autoantibody: A case report and literature review". *Journal of Infection and Chemotherapy* 28.5 (2022): 699-704.
8. Image 1 Courtesy: Wikimedia commons.
9. Image 2 Courtesy: Basic medical key.

Volume 17 Issue 11 November 2025