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Editorial

Contagion and Defilement-*Yersinia pestis* Lymphadenitis

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Bacterial infection with gram negative *Yersinia pestis* is associated with mesenteric lymphadenitis. Infected individual demonstrates clinical symptoms simulating acute appendicitis and a probable abdominal mass. The condition is self limiting although concurrence with Kawasaki's disease is observed.

Gram's stain depicts polymorphic, coccoid or ovoid, motile gram negative bacteria. Administration of antibiotics as ampicillin may decimate foetal excretion of bacteria although amelioration of clinical symptoms remains absent.

Typically, *Yersinia pestis* is transmitted from an enzootic rodent to a flea vector and engenders the obstruction of gastrointestinal tract. Consequently, the flea feeds frequently and regurgitates *Yersinia pestis* into the host. Following host ingress, bacterium circumvents the immune system, traverses into lymph nodes and propagates within macrophages. Subsequent to egress from macrophages, the bacterium induces a pro-inflammatory response with emergence of cogent clinical symptoms [1,2].

Majority (~95%) of *Yersinia pestis* infections represent with lymphadenitis with suppuration, designated as the 'bubonic plague' [2,3].

Clinically, the bacterial infection is self limiting and confined to lymph nodes where it is designated as the 'bubo' or bubonic plague. Clinical symptoms recapitulate the occurrence of an acute appendicitis. However, surgical exploration demonstrates mesenteric lymphadenitis. Bacterial aerosols induce a 'pneumonic plague' [2,3].

Upon microscopy, lymph node cortex and para-cortical region expounds innumerable immunoblasts and plasma cells [3,4].

Germinal centre displays hyperplasia and large lymphocytes appear impregnated within lymphatic sinuses. Epithelioid cell granulomas appear absent. Lymph node parenchyma depicts aggregates of foamy macrophages and depletion of small lymphocytes. Focal necrosis and oedema may be encountered. The bacteria may ingress into vascular articulations. Lymph node capsule delineates thickening and oedema [5,6].

International Society for Cutaneous Lymphomas (ISCL)/European Organization of Research and Treatment of Cancer (EORTC) staging of cutaneous B cell lymphoma is designated as:

- Primary tumour (T):
- T1: Solitary cutaneous involvement:
- T1a: Solitary lesion < 5 centimetre diameter.

- T1b: Solitary lesion > 5 centimetre diameter.
- T2: Regional cutaneous involvement:
- T2a: All disease encompassed within < 15 centimetre circular zone.
- T2b: All disease encompassed within > 15 centimetre and < 30 centimetre circular zone.
- T2c: All disease encompassed within > 30 centimetre circular zone.
- T3: Generalized cutaneous involvement:
- T3a: Multiple lesions involving 2 non contiguous body regions.
- T3b: Multiple lesions involving ≥ 3 non contiguous body regions.
- Regional lymph nodes (N):
- N0: No clinical or pathological lymph node involvement.
- N1: Involvement of single peripheral lymph node region which drains an area of current or preceding cutaneous tumour site.
- N2: Involvement of ≥2 peripheral lymph node regions or any lymph node region which does not drain an area of current or preceding cutaneous tumour site.
- N3: Involvement of central lymph nodes.
- Distant metastasis:
- M0: No evidence of extra-cutaneous non lymph node disease.
- M1: Extra-cutaneous non lymph node disease present [5,6].

Yersinia pestis infection may be appropriately ascertained by smears stained with Gram's stain which delineate aggregates of gram negative, polymorphic, coccoid or ovoid, motile bacteria. Cogent cultures may be suitably adopted for bacterial isolation [7,8].

Yersinia pestis appears immune reactive to *Yersinia* immunostain.

Polymerase chain reaction (PCR) may be beneficial in detecting the bacterial organisms [7,8].

Bubonic plague or clinical representation with lymphadenopathy requires segregation from diseases contributing to lymphadenitis or lymphadenopathy as various bacterial, viral, fungal, oncological, iatrogenic, autoimmune or inflammatory disorders [7,8].

As bubonic plague is associated with sudden onset of pyrexia and buboes, intense inflammation and rapid progression of buboes with fulminant deterioration of the severe clinical condition, preliminary employment of antibiotics appears efficacious in treating the plague [9,10].

Commencement of therapy upon cogent clinical suspicion is beneficial as clinical outcomes are inferior. Antibiotics as aminoglycosides, gentamicin or streptomycin appear advantageous. Alternatively, doxycycline and tetracycline may be employed for up to two weeks. Additionally, trimethoprim-sulfamethoxazole combination, chloramphenicol or levofloxacin may be adopted for disease eradication [9,10].

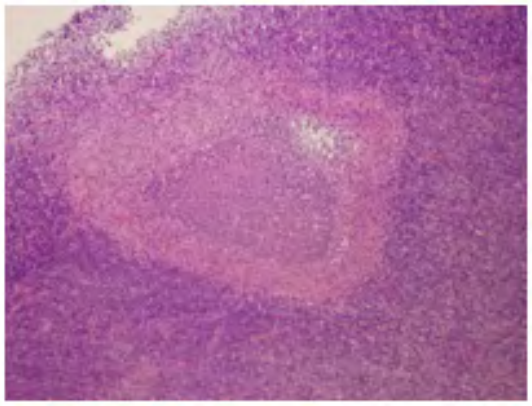

Figure 1: *Yersinia lymphadenitis depicting thickened capsule, plasma cells and immunoblasts in the paracortex, germinal centre hyperplasia and absence of epithelioid cell granulomas [11].*

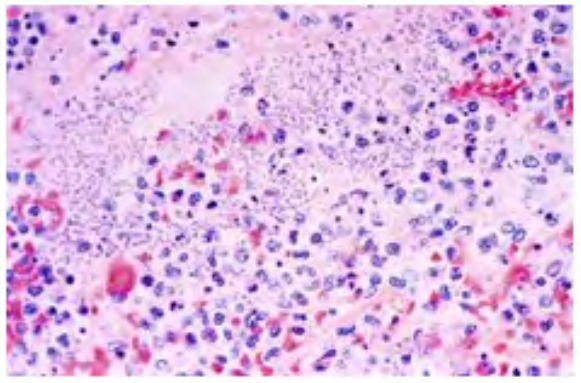

Figure 2: *Yersinia lymphadenitis delineating suppuration within lymph node parenchyma, disseminated plasma cells, immunoblasts, macrophages and small lymphocytes [12].*

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11. Image 1 Courtesy: Pathology outlines.

12. Image 2 Courtesy: Wikidoc.com.

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