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Editorial

Deadbeat and Groupie-*Leishmania* Lymphadenitis

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Leishmania associated lymphadenopathy is documented in infections where parasites continue to flourish within lymph nodes following a suitable clinical cure.

Leishmania parasites are transmitted through bite of sand-flies. Commonly, Phlebotomus and Lutzomyia vectors are encountered. Female sand-flies necessitate blood meals wherein *Leishmania* spp. are acquired by the sand-fly and transmitted to the host. Disease-naive sand fly ingresses an infected host with saw-like mouthparts inserted into the cutis in order to create a miniature wound from which blood pools into injured capillaries. Thereby, infectious promastigote infiltrates the sand-fly foregut with consequent parasitic replication. Sand-fly feeding upon canines, rodents, marsupials or humans induces disease transmission into the new host.

Protozoan with access to the host ingresses phagolysosomes. Contingent to subtype of phagocytic cells invaded, cutaneous leishmaniasis (CL) or visceral leishmaniasis (VL) may occur [1,2].

Cutaneous leishmaniasis exemplifies parasites which infect resident macrophages impregnated within the cutis. Singular cell imbued with amastigotes bursts and ensures a rapid release of parasites with consequent infection of surrounding macrophages [2,3].

Visceral leishmaniasis displays amastigotes associated with haematogenous dissemination into mononuclear cells of viscera as hepatic parenchyma, spleen, bone marrow or intestinal lymph nodes.

Cytological examination depicts commingled cellular exudate comprised of polymorphous lymphocytes, histiocytes, plasma cells, giant cells and tingible body macrophages. Extra-cellular accumulation of amastigote forms may concur with amastigote forms impregnated within histiocytes and multinucleated giant cells [3,4].

Scraping smears and fine needle aspiration cytology is associated with enhanced possible detection of amastigotes. Articulation of 'press-imprint-smear' is associated with significantly enhanced sensitivity for disease detection, in contrast to histopathological assessment.

Parasitic trophozoites may be suitably examined with cogent histopathological examination of paraffin embedded formalin fixed tissue or *in vitro* culture. Tissue sampling from edge of cutaneous ulcer or lymph node procures a suitable yield of parasites [3,4].

Category	Characteristics (age, incidence, gender, median survival)	Variants	EBV infection	Clinical features	Treatment
Hepatosplenic γδ T cell lymphoma	Young adults (35 yrs), < 1%, male, < 2 years	None	Yes	Cytopenia, liver, BM, spleen and rare LN infiltration	CHOP-like regimen, allo-SCT. Relapses-bendamustine, bortezomib, lenalidomide, vorinostat
Primary cutaneous γδ T cell lymphoma	Adults, <1%, none, 15 months	Aggressive, mycosis fungoides-like, subcutaneous panniculitis-like	No	Papule, plaque or nodule with ulceration, overlying epidermal necrosis	CHOP-like regimen, steroid, MTX, UV radiation, bexarotene
Mucosal γδ T cell lymphoma	>48 years, NK, none, 1 - 1.5 years	GIT, nasal, pulmonary, laryngeal	Yes	HSM, nasal destruction, GI perforation, LN and BM rarely involved	CHOP-like regimen, allo-SCT
Gamma/delta T LGL leukaemia	45 - 75 years, 2-3%, none, indolent	None	Yes, few cases	Cytopenia, splenomegaly, BM involved	Steroids, MTX, cyclosporine, cyclophosphamide, fludarabine, pentostatin
Nodal gamma delta T cell lymphoma	NK <2%, NK, <1 year	None	Yes	LN, BM infiltration, HSM	CHOP-like regimen, allo-SCT

Table: *Classification of gamma/delta T cell lymphoma [4,5].*

LGL: Large Granular Lymphocyte; LN: Lymph Node; BM: Bone Marrow; GIT: Gastrointestinal Tract; HSM: Hepatosplenomegaly; allo-SCT: Allogeneic Stem Cell Transplant; MTX: Methotrexate; CHOP: Cyclophosphamide; Doxorubicin Hydrochloride, Vincristine Sulphate, Prednisone; UV: Ultraviolet Radiation; NK: Not Known.

Upon microscopy, epithelioid cell granulomas are intermingled with abundant plasma cells. Foci of fibrosis and variable necrosis are encountered. Immunostaining for *Leishmania* amastigote may be advantageously employed [6,7].

Hence, the classic spherical to elliptical organisms of 2 to 4 micrometres with characteristic nuclei and kinetoplasts may be encountered [6,7].

Leishmaniasis inducing cutaneous ulcers and lymphadenopathy necessitate distinction from conditions as furuncular myiasis, staphylococcal infection, lepromatous leprosy with classic leonine facies, tuberculoid leprosy with hypo-pigmented patches and plaques or yaws delineating primary stage of ulcerative or nodular lesions confined to lower extremities [7,8].

The infectious disease may be suitably managed with circumventive measures. Avoiding endemic areas, exposure to animals and nocturnal activity of sand-flies are significant preventive manoeuvers [8,9].

Impregnation with permethrin appears advantageous. Dog vaccination and utilizing insecticide dog collars may decimate disease burden.

Limited disease may undergo spontaneous retrogression. Nevertheless, dermatologic disease is destructive and associated with secondary infections and permanent, disabling scars [9,10].

Limited disease may be managed with intralesional pentavalent antimonals as sodium stibogluconate and meglumine antimoniate.

Alternatively, agents as systemic miltefosine, amphotericin B, pentamidine isethionate, paromomycin or granulocyte macrophage colony-stimulating factor (GM-CSF) may be beneficially employed.

Azole therapies as fluconazole, ketoconazole or itraconazole may be employed singularly or in combination with amphotericin B [9,10].

Visceral leishmaniasis may be treated with agents as antimony sodium stibogluconate, amphotericin, paromomycin, and oral miltefosine [9,10].

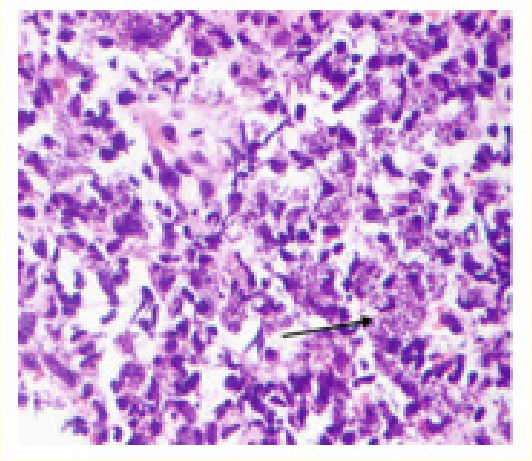

Figure 1: *Leishmania lymphadenopathy demonstrating epithelioid cell granulomas, abundant plasma cells, focal fibrosis and variable necrosis [11].*

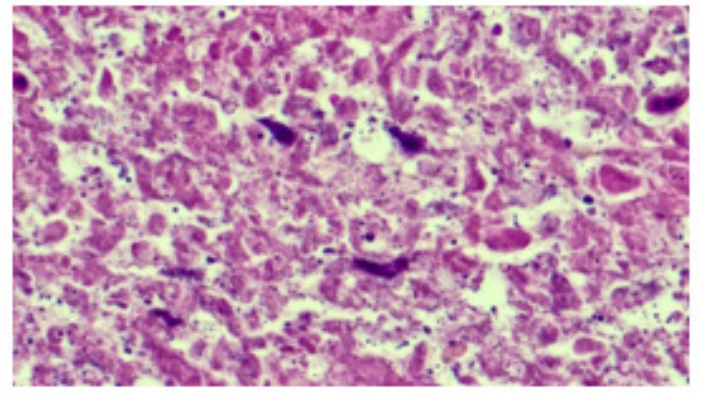

Figure 2: *Leishmania lymphadenopathy delineating epithelioid cell granulomas, several plasma cells, focal fibrosis and variable necrosis [12].*

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11. Image 1 Courtesy: Springer link.
12. Image 2 Courtesy: Pathology outlines.

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