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Editorial

The Glorious Depreciation- Lipofuscin Lymph Node

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Amalgamation of lipofuscin pigment is a commonly encountered feature within lymph nodes. Incidentally discovered, lipofuscin configures as a golden brown, finely granular, intracellular pigment.

The pigment is preponderantly derived from denatured products of lipids, especially lipid molecules derived from cellular membranes. Ceroid emerges as an acid fast variant of lipofuscin which may be appropriately discerned by auto-fluorescence.

Lipofuscin is accumulated within various cells denominating the post mitotic phase and is delineated as aggregates of oxidized protein with cross linkages [1,2].

The pigment is conspicuous and especially associated with disorders demonstrating visceral and organ atrophy [1,2].

Lipofuscin pigment is concordant with diverse inflammatory or neoplastic lesions expounding distinct foci of necrosis. Commonly, chronic cholestatic lesions as primary biliary cirrhosis or primary sclerosing cholangitis may concur with intracellular lipofuscin accrual.

Lipofuscin amalgamation within lymph nodes is clinically associated with enlargement of lymph nodes [3,4].

Lipofuscin is commonly amalgamated with lymph nodes draining hepatic parenchyma and portal zones. Besides, diverse lymph node groups may demonstrate aggregation of lipofuscin.

Grossly, the lesion represents with enlarged lymph nodes pervaded with brown pigment [3,4].

Upon microscopy, nodal parenchyma depicts sinus histiocytosis. Follicular hyperplasia appears variable. Macrophages confined to intra-nodal sinuses appear impregnated with granular deposits of brown pigment [4,5].

Ultrastructural examination depicts electron dense, spherical inclusions of pigment confined by a distinct membrane [4,5].

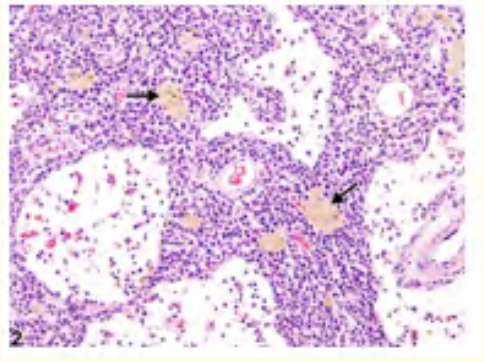

Figure 1: Lipofuscin pigment appearing as intracellular aggregates of golden brown pigment [11].

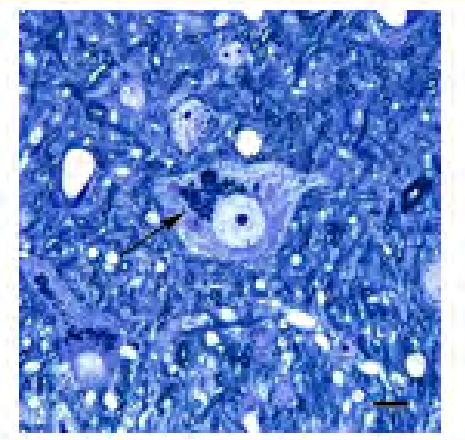

Figure 2: Lipofuscin demonstrating a bluish hue with toludine blue [12].

Clinical types of LyP	Histological Aspects	Phenotype	Differential diagnosis	Distinctive criterion
Type A	Wedge shaped, disseminated infiltrate or clusters. Large atypical CD30+ lymphocytes Admixed histiocytes, neutrophils, eosinophils.	Predominantly CD4+	Mycosis Fungoides Hodgkin's Lymphoma Anthropod bite reaction	Patches and plaques in MF versus papulo-nodular lesions in LyP Nodal Hodgkin's Lmphoma requires staging. Clinical presentation for assessing pruritis
Type B	Epidermotropic infiltrate of small to medium sized lymphocytes with variable CD30+ elucidation.	CD4+(CD30-may be non reactive)	Mycosis Fungoides (patch/plaque stage)	Patches and plaques in MF versus self regressing papulo-nodular lesions in LyP
Type C	Nodular cohesive infiltrate of large CD30+ atypical lymphocytes with a few reactive cells.	CD4+> CD8+	Anaplastic large cell lymphoma (primary cutaneous or systemic form) Mycosis Fungoides (transformation phase) Peripheral T cell lymphoma(NOS- primary cutaneous or nodal) Adult T cell lymphoma/leukaemia	Clinical presentation with solitary or grouped nodules in pc ALCL : staging in s ALCL. Patches and plaques preceding tumours in MF Lack of CD30 or a few CD30+ cells and staging of tumour (PTCL) Integration of HTLV1/2 in tumour cell genome
Type D	Prominent epidermotropism of atypical lymphocytes delineating CD8+ and CD30+.	CD8+(100%) CD30+(90%)	Pagetoid reticulosis (PR) Primary cutaneous aggressive epidermotropic CD8+ cytotoxic T cell lymphoma	Localized or solitary erythematous or scaly lesion Multiple rapidly evolving plaques and nodules with erosions and necrosis

Type E	Angio invasive infiltrates of atypical CD30+ lymphocytes. Haemorrhage, extensive necrosis and ulceration.	CD8+(70%)	Extra-nodal NK/T cell lymphoma; nasal type Cutaneous gamma/delta lymphoma Anaplastic large cell lymphoma (angio invasive form)	Association with EBV, secondary cutaneous involvement (staging) IHC : Elucidation of TCR gamma delta with absence of TCR alpha /beta Clinical presentation with solitary or grouped nodules in pc ALCL : staging in s ALCL

Table 1: Current terminology: Categories of Lymphomatoid Papulosis [6,7].

MF: Mycosis Fungoides, pc ALCL : primary cutaneous anaplastic large cell lymphoma, s ALCL: systemic anaplastic large cell lymphoma, NK: natural killer, EBV: Epstein Barr virus, HTLV 1/ 2: Human T lymphotropic virus type 1 and 2, TCR : T cell receptor, TCL: T cell lymphoma, IHC: Immunohistochemistry.

Diagnosis	Histology inflammation/architecture	Phenotype	Appendix 1: genotype	Appendix 2: clinical presentation
Lymphomatoid papulosis	Mixed cellular	CD4+	6p25.3	Generalized
	Epidermotropic	CD8+	NPM1-TYK-2gene fusion	Localized
	(pagetoid/ nonpagetoid)	CD4-/CD8-		Acral
	Cohesive			Mucosal/Oral
	Angio-invasive	CD56 (optional)		Pustular
	Folliculotropic	TCR (optional)		
	Syringotropic			
	Granulomatous			
	Intra-lymphatic			
	Spindle cell			

Table 2: Proposed contemporary terminology of Lymphomatoid papulosis [6,7].

Lymph nodes associated with lipofuscin pigment deposits may be highlighted by stains such as Sudan Black B, Schmorl's reaction, oil red O, carbol lipofuscin stain, periodic acid Schiff's stain, Ziehl-Neelsen acid fast stain or lysosomal acid phosphatase and esterase stains. Besides, auto- fluorescence may emphasize the intracellular pigment [8,9].

Stains such as Perls iron stain or melanin stains appear superfluous and non confirmatory in highlighting the pigment [8,9].

Lipofuscin pigment amalgamated within lymph nodes necessitates segregation from intra-nodal deposits of iron or hemosiderosis, melanin pigment or tattoo pigment [9,10].

Although associated with preceding consideration of a non significant intracellular molecule, lipofuscin molecule may occur due to and induce augmented intracellular oxidant stress wherein intracellular proteasomal and lysosomal functions may be impaired [9,10].

Bibliography

1. Snyder AN and Crane JS. "Histology". Lipofuscin. Stat Pearls International. Treasure Island, Florida (2025).

2. Deng Y., *et al*. "Comprehensive characterisation of age-related changes in cell subpopulations and tissue structural properties in secondary lymphoid organs". *Cell Death and Disease* 16.1 (2025): 679.

3. Fu Y., *et al*. "Immuno-senescence: signalling pathways, diseases and therapeutic targets". *Signal Transduction and Targeted Therapy* 10.1 (2025): 250.

4. McHugh D., *et al*. "Senescence as a therapeutic target in cancer and age-related diseases". *Nature Reviews Drug Discovery* 24 (2025): 57-71.

5. de Jong TA., *et al*. "Senolytic treatment to rescue hallmarks of senescence in lymph node fibroblasts from patients with rheumatoid arthritis: Implications for premature aging and potential therapeutic intervention in early rheumatoid arthritis". *Clinical and Experimental Immunology* 219.1 (2025): uxaf029.

6. Kempf W., *et al*. "Cutaneous CD30 positive T cell lymphoproliferative disorders: clinical and histopathologic features, differential diagnosis and treatment". *Seminars in Cutaneous Medicine and Surgery* 37 (2018): 24-29.

7. Kempf W., *et al*. "Lymphomatoid papulosis: making sense of the alphabet soup: a proposal to simplify terminology". *Journal der Deutschen Dermatologischen Gesellschaft* 15.4 (2017): 390-394.

8. Liu Z., *et al*. "Immunosenescence: molecular mechanisms and diseases". *Signal Transduction and Targeted Therapy* 8 (2023): 200.

9. Admasu T D and Yu J S. "Harnessing immune rejuvenation: advances in overcoming T cell senescence and exhaustion in cancer immunotherapy". *Aging Cell* (2025): e70055.

10. Li X., *et al*. "Inflammation and aging: signaling pathways and intervention therapies". *Signal Transduction and Targeted Therapy* 8 (2023): 239.

11. Image 1 Courtesy: National toxicology programme.

12. Image 2 Courtesy: Wikipedia.