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Editorial

Adamantine and Indomitable - Hemosiderosis Lymph Node

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Hemosiderosis or iron deposition within lymph nodes emerges as an exceptionally encountered disorder. Nodal hemosiderosis may concur with hemochromatosis.

Contingent to primary contributing cause, hemosiderosis of lymph node arises within portal, splenic, mesenteric or axillary lymph nodes.

Hemosiderosis of lymph node may concur with contributory factors as parenteral iron administration with accompanying multiple blood transfusions. Lysis of red blood cells is associated with iron deposition within hepatic and splenic macrophages which eventually drain into adjacent lymph nodes.

Conditions as hereditary hemosiderosis delineate anomalies of iron transport, commonly chromosomal mutation within HFE1 gene. Besides, genetic mutations within TfR2, HJV or HAMP genes may occur. Additionally, ferroportin-V162del may ensue [1,2].

Anaemia of inflammation may concur with conditions as rheumatoid arthritis, gout or plasma cell Castleman's disease [1,2].

Hemosiderin is configured of degradation product of iron-storage complex and manifests as miniature, inconspicuous, granular, yellow-brown pigment upon haematoxylin and eosin stain [2,3].

Hemosiderin originates from denatured haemoglobin or from an anomalous metabolic pathway of ferritin. Damaged or denatured erythrocytes egress ruptured blood vessel with transfer of haemoglobin into extracellular space. Deteriorated red blood cells or haemoglobin extravasation may be phagocytosed by macrophages. As hepcidin prohibits intestinal iron absorption and blocks transposition of iron molecules from macrophages, upregulation of hepcidin is associated with enhanced tissue deposition of hemosiderin [2,3].

Besides, interleukin 6(IL-6) emerges as a predominant inducer of hepatic synthesis of hepcidin. Thus, enhanced expression of hepcidin appears concurrent with hemosiderin deposition within soft tissues of disorders as plasma cell Castleman's disease [3,4].

Plasma erythropoietin (EPO) may mediate hepcidin suppression as a consequence to altered hematopoietic activity. Enhanced erythropoietin (EPO) concentration induces downregulation of interleukin 6(IL-6)induced secretion of hepcidin [3,4].

Additionally, serum C-reactive protein (CRP) levels and hemosiderin deposition appear to concur. C-reactive protein (CRP) expression is induced by interleukin 6(IL-6) during inflammation and serves as a significant biomarker for interleukin 6(IL-6) bioactivity [3,4].

Clinical symptoms are contingent to primary disease engendering accumulation of iron pigment within lymph node parenchyma. Regional or generalized lymph node enlargement may occur [3,4].

Grossly, hemosiderosis is associated with enlarged lymph nodes [7,8].

Upon microscopy, lymph node depicts follicular hyperplasia and sinus histiocytosis. Golden brown pigment appears to pervade the nodal parenchyma [7,8].

Ultrastructural examination depicts preponderant dissemination of phagocytic reticulum cells which are impregnated with vacuoles, myelin figures, lipid droplets and significantly enlarged lysosomal bodies pervaded with fine, electron dense granules. Besides, the lysosomal bodies and intracytoplasmic zone is permeated with molecular fragments of ferritin [7,8].

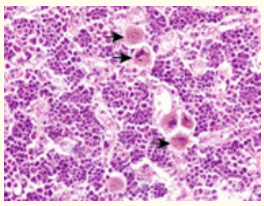

Figure 1: Hemosiderosis lymph node depicting intracellular aggregates of golden brown pigment disseminated within the nodal parenchyma [12].

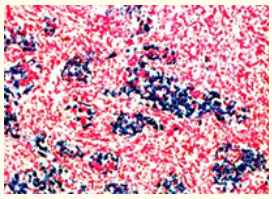

Figure 2: Hemosiderosis lymph node expounding intracellular aggregates of golden brown pigment dispersed within lymph node parenchyma [13].

Table 1: Evaluation of mucosa associated lymphoid tissue (MALT) lymphomas [5,6].

Comprehensive MALT lymphomas
History and physical examination
Bone marrow biopsy
Complete blood counts
Liver and renal function tests
Protein electrophoresis
Serum lactate dehydrogenase and β2 micro-globulin
Hepatitis B and C virus with human immune deficiency (HIV) virus
Computerized tomography (CT) scan of the chest, abdomen and pelvis.
Bone marrow biopsy assay
Biopsy of the affected tissue
Morphological and immune phenotypic (modified Giemsa, pan-CK, CD20, CD3, MIB-1) investigations
Gastric MALT
Helicobacter pylori serology and/or stool antigen test
Endoscopic ultrasound
Evaluation of t(11:18), BIRC3(APxx12-MALT1). Genetic evaluation is not mandatory for initial evaluation.
Non Gastric MALT
Endoscopic examination of the gastro duodenal tract to rule out concomitant gastric involvement
Endoscopic otorhinolaryngologic examination
Magnetic resonance imaging of the orbit
Computerized tomography of the parotid/salivary glands

Iron pigment confined to lymph nodes may be suitably discerned by Perls prussian blue stain [7,8].

Alternatively, melanin stains appear immune nonreactive and inadequate in staining the iron laden nodal parenchyma [7,8].

Hemosiderosis of the lymph node requires segregation from lesions as aggregates of charcoal laden macrophages, hemazoin

pigment as encountered within cells infected by malarial parasite, accrual of melanin pigment as observed with lesions of malignant melanoma or dermatopathic lymphadenitis and intracellular tattoo pigment. Computerized tomography (CT) depicts enlarged and hyper-dense lymph node [8,9].

Hemosiderosis of lymph node may be suitably discerned with evaluation of surgically excised tissue samples subjected to Perls Prussian blue stain [8,9].

Nodal hemosiderosis may be appropriately managed with manoeuvers as decimation of iron overload. Additionally, cogent therapy of primary cause of hemosiderosis appears beneficial [10,11].

Severity and extent of disease is contingent to primary disorder contributing to emergence of nodal hemosiderosis [10,11].

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12. Image 1 Courtesy: Science direct.

13. Image 2 Courtesy: Pathology outlines.