



Upheaval and Transmutation-Splenic Metastasis

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Metastasis from diverse primary malignant neoplasms into splenic parenchyma is associated with emergence of extensive lesions. In contrast, isolated nodules of splenic metastasis are exceptionally observed.

Primary malignancies associated with distant metastasis are comprised of pulmonary parenchyma, breast, colon, ovary or malignant melanoma. Infrequently, primary neoplasms may arise from endometrium, brain, salivary glands or hepatic parenchyma.

Generally, distant metastasis occurring into splenic parenchyma is exceptional on account of precise immunological function of the organ. Distant metastases are articulated due to haematogenous or lymphatic dissemination of primary tumour. Besides, implantation into abdominal cavity or direct invasion from lesions confined to adjacent organs may engender splenic metastasis [1,2].

Notwithstanding, haematogenous or direct seeding of tumour cells is a commonly encountered mechanism of metastatic dissemination of various primary neoplasms.

Metastasis into splenic parenchyma from diverse primary tumours is devoid of specific clinical representation. Exceptionally, splenic rupture may occur [2,3].

Grossly, splenic metastasis configures as solitary nodules, multiple nodules or may represent as diffuse infiltration within splenic parenchyma. Alternatively, a cyst may be configured.

Exceptionally, the lesion may simulate follicular lymphoma and represent with multiple tumour nodules disseminated within the splenic parenchyma [2,3].

Upon microscopy, metastatic nodules represent with a morphological picture identical to the primary tumour. Red pulp expounds nodular transformation simulating sclerosing angiomatoid nodular transformation with the configuration of multiple, well demarcated, confluent angiomatoid nodules circumscribed by a stroma demonstrating variable fibrosis and sclerosis. Occasionally, a perimeter of fibrinoid tissue is encountered which appears as a granuloma-like structure [3,4].

Angiomatoid nodules display an admixture of slit-like, spherical or irregular vascular spaces layered by bland, plump endothelial cells devoid of atypia, necrosis or mitotic activity [3,4].

The nodules are comprised of three distinct varieties of vascular articulations reminiscent of blood vessels, configuring the normal red pulp wherein the vascular structures may be appropriately discerned with cogent immunohistochemistry. The vascular structures manifest as capillaries, sinusoids and miniature veins [4,5].

Stroma segregating the nodules appears as fibrous or fibromyxoid and represents as bland fibroblasts and myofibroblasts. Besides, the stroma is abundantly infiltrated by chronic inflammatory cells as lymphocytes, hemosiderin laden macrophages and polytypic plasma cells [4,5].

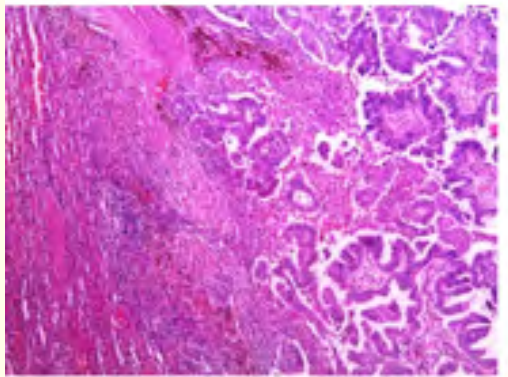


Figure 1: Splenic metastasis from a primary endometrial cancer comprised of neoplastic glands. Intervening splenic red pulp depicts capillaries, sinusoids and miniature veins surrounded by a fibrotic stroma infiltrated by chronic inflammatory cells [10].

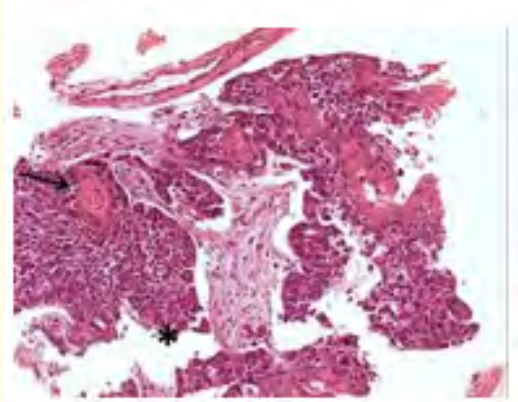


Figure 2: Splenic metastasis from a primary pulmonary squamous cell carcinoma composed of aggregates of malignant squamous epithelial cells. Splenic parenchyma is comprised of capillaries, sinusoids and small veins surrounded by a fibrotic stroma invaded by chronic inflammatory cells [11].

Attributes	CD30+ALCL	Borderline	LyP
Clinical Aspects			
Extent	Solitary > regional (exceptionally) diffuse	Regional	Regional/Diffuse
Lesion	Nodular, Tumours	Nodular	Papules/papulonodular
Regression	Infrequent	Frequent	Consistent
Extra- cutaneous disease	25%-30%	Absent	Absent
Histological Aspects			
Wedge shaped infiltrate	Absent	Infrequent	Prominent
Sheets of CD30+ cells	Persistent	Miniature aggregates	Absent
Subcutaneous infiltrate	Present	Absent	Absent
Immune reactive CD30+	>75% of tumour cells	Miniature aggregates	Scattered CD30+ reactivity

Table 1: Demarcation between Cutaneous CD30+ ALCL, Borderline Lesions and Lymphomatoid Papulosis [7].

ALCL: Anaplastic large cell lymphoma.

The distinctive vascular articulations configuring normal splenic red pulp emerge as capillaries which are immune reactive to CD34+, CD31+ and immune non reactive to CD8-. Sinusoids are immune reactive to CD8+, CD31+ and immune non reactive to CD34-. Miniature veins appear immune reactive to CD31+ and immune non reactive to CD34- and CD8- [6,7].

Myofibroblasts confined to the nodules appear immune reactive to smooth muscle actin (SMA). Histiocytes or macrophages appear immune reactive to CD68. A polytypic expression of kappa and lambda light chains is encountered within infiltrating plasma cells [6,7].

The vascular articulations appear immune non reactive to D2-40 and Epstein Barr virus (EBV) as detected by in situ hybridization techniques. Occasionally, innumerable IgG4+ plasma cells delineate elevated IgG4:IgG ratio. Nevertheless, serum IgG4 concentration appears unaltered. Adjoining splenic tissue appears unremarkable [7,8].

Metastasis into splenic parenchyma may be appropriately discerned by radiographic evaluation with subsequent morphological assessment of surgical tissue samples.

Ultrasonography delineates foci of distant metastasis disseminated into splenic parenchyma which appear as hyperechoic, hypoechoic or non-echoic.

Computerized tomography (CT) expounds hypodense lesions [8,9].

Cogent therapeutic options as chemotherapy or surgical intervention with splenectomy may be beneficially adopted. Diverse treatment manoeuvres are contingent to origin of primary tumour [8,9].

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10. Image 1 Courtesy: MDPI.com
11. Image 2 Courtesy: World Journal of Surgical Oncology.