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Heteroclite and Turgid-Atypical Adenomatous Hyperplasia Lung

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Editorial

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Abbreviations: CT: Computerized Tomography; TTF: Thyroid Transcription Factor.

Editorial

Atypical adenomatous hyperplasia emerges as a miniature, localized proliferation of atypical pneumocytes which layer intact alveolar spaces. Atypical adenomatous hyperplasia is contemplated as a precursor lesion to invasive pulmonary adenocarcinoma.

Atypical adenomatous hyperplasia appears as a miniature, localized lesion ≤ 5 millimetre magnitude. Tumefaction appears disparate from alveolar parenchyma and is comprised of proliferation of atypical pneumocytes. Lesion is coated with non-contiguous monolayer of cells whereas circumscribing pulmonary parenchyma appears devoid of prominent inflammatory infiltrate or stromal fibrosis.

The terminology of atypical alveolar epithelial hyperplasia, atypical bronchoalveolar cell hyperplasia, atypical alveolar cell hyperplasia, bronchoalveolar cell adenoma or bronchial adenoma appears obsolete and is not recommended.

Atypical adenomatous hyperplasia lung is frequently discerned as an incidental finding within pulmonary resection specimens. Lesion may concur with female subjects selected for therapeutic surgical resection of pulmonary adenocarcinoma. Alternatively, tumour may be infrequently exemplified within autopsy specimens devoid of pulmonary adenocarcinoma. Atypical adenomatous hyperplasia is categorized as a condition amenable to antecedent morphological detection and a lesion which systematically progresses into pulmonary adenocarcinoma.

Atypical adenomatous hyperplasia may be associated with CYP19A1 genetic polymorphisms. A female predominance is observed. Atypical adenomatous hyperplasia lung is frequently confined to superior pulmonary lobes [1,2].

Of obscure aetiology, atypical adenomatous hyperplasia lung appears non concurrent with cigarette smoking or tobacco consumption. Neoplastic cells appear clone-specific. Proportionate copy number modifications and genomic mutations appear minimal, in contrast to adenocarcinoma in situ [1,2]. Atypical adenomatous hyperplasia lung is accompanied by driver mutations within KRAS, EGFR and BRAF genes. However, as lesions appear appropriately alleviated with surgical resection, molecular assessment appears superfluous [2,3].

Characteristically, tumefaction is discovered incidentally upon evaluation of surgical pathology specimens. Generally, atypical adenomatous hyperplasia appears non detectable upon adoption of cogent imaging techniques. High resolution computerized tomography (CT) may optimally discern enlarged lesions [2,3].

Upon frozen section, appropriate tumour discernment may be challenging as the lesion is composed of subtle morphological alterations. Besides, lesions demonstrating subtle atypia upon frozen section may be categorized as atypical adenomatous hyperplasia upon evaluation of formalin fixed paraffin embedded sections [2,3].

Grossly, singular or multifocal, poorly defined, grey/white, tan or yellow neoplastic zones are observed. Lesion magnitude is ≤5 millimetres and tumefaction is commonly confined to peripheral pulmonary parenchyma, abutting the pleura.

Upon microscopy, atypical adenomatous hyperplasia lung exhibits proliferation of atypical pneumocytes. Constituent cells delineate mild to moderate cellular and nuclear atypia, enhanced cellular magnitude and elevated nucleocytoplasmic ratio. Tumour cells are pervaded with hyperchromatic nuclei and intra-nuclear eosinophilic inclusions. Tumour cells layer intact alveolar spaces wherein atypical cells configure a non-contiguous cellular monolayer [4,5].

Lesion is constituted of an admixture of club cells and type II pneumocytes. Club cells represent as columnar epithelial cells permeated with eosinophilic cytoplasmic snouts and outpouchings. Type II pneumocytes manifest as cuboidal cells impregnated with finely vacuolated or clear cytoplasm. Characteristically, mitotic figures are minimal (Table 1).

Surrounding pulmonary parenchyma is devoid of significant infiltrate of acute and chronic inflammatory cells or stromal fibrosis (Figures 1 & 2) [4,5].

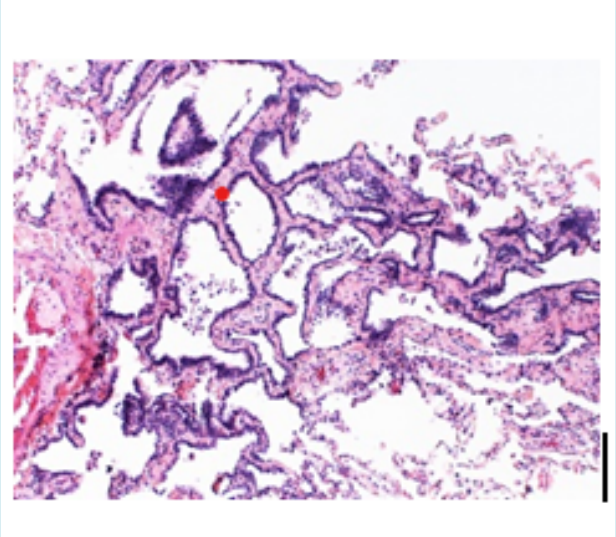

Figure 1: Atypical adenomatous hyperplasia demonstrating alveolar spaces lined by columnar epithelial cells incorporated with eosinophilic cytoplasm, enlarged, hyperchromatic nuclei with enhanced nucleocytoplasmic ratio and mild cellular and nuclear atypia. Surrounding pulmonary parenchyma appears unremarkable [6].

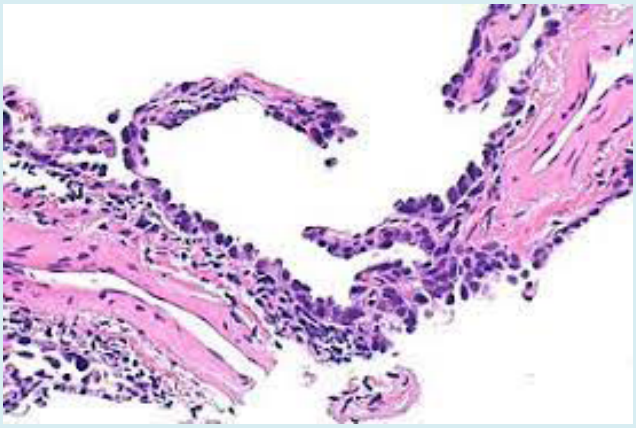

Figure 2: Atypical adenomatous hyperplasia delineating alveolar spaces lined by columnar epithelial cells permeated with eosinophilic cytoplasm, enlarged, hyperchromatic nuclei with enhanced nucleocytoplasmic ratio and mild cellular and nuclear atypia. Circumscribing pulmonary parenchyma appears mildly inflamed and fibrotic [7].

Epithelial Tumours
Papilloma
Squamous cell papilloma NOS
Squamous cell papilloma, inverted
Glandular papilloma
Mixed squamous cell and glandular papilloma
Adenoma
Sclerosing pneumocytoma
Alveolar adenoma
Papillary adenoma
Bronchiolar adenoma/ciliated muconodular papillary tumour
Mucinous cystadenoma
Mucous gland adenoma
Precursor glandular lesions
Atypical adenomatous hyperplasia
Adenocarcinoma in situ
Adenocarcinoma in situ, non-mucinous
Adenocarcinoma in situ, mucinous
Adenocarcinoma
Minimally invasive adenocarcinoma
Minimally invasive adenocarcinoma, non-mucinous
Minimally invasive adenocarcinoma, mucinous
Invasive non-mucinous adenocarcinoma

Lepidic adenocarcinoma
Acinar adenocarcinoma
Papillary adenocarcinoma
Micro-papillary adenocarcinoma
Solid adenocarcinoma
Invasive mucinous adenocarcinoma
Mixed invasive mucinous and non-mucinous adenocarcinoma
Colloid adenocarcinoma
Foetal adenocarcinoma
Adenocarcinoma, enteric type
Adenocarcinoma NOS
Squamous precursor lesions
Squamous cell carcinoma in situ
Mild squamous dysplasia
Moderate squamous dysplasia
Severe squamous dysplasia
Squamous cell carcinoma
Squamous cell carcinoma NOS
Squamous cell carcinoma, keratinizing
Squamous cell carcinoma, non-keratinizing
Basaloid squamous cell carcinoma
Lympho-epithelial carcinoma
Large cell carcinoma
Adenosquamous carcinoma
Sarcomatoid carcinoma
Pleomorphic carcinoma
Giant cell carcinoma
Spindle cell carcinoma
Pulmonary blastoma
Carcinosarcoma
Other epithelial tumours
NUT carcinoma
Thoracic SMARCA4-deficient undifferentiated tumour
Salivary gland-type tumours
Pleomorphic adenoma
Adenoid cystic carcinoma
Epithelial-myoepithelial carcinoma
Mucoepidermoid carcinoma
Hyalinising clear cell carcinoma
Myoepithelioma
Myoepithelial carcinoma
Neuroendocrine tumours of lung
Precursor lesion
Diffuse idiopathic neuroendocrine cell hyperplasia
Neuroendocrine tumours
Carcinoid tumour NOS/ neuroendocrine tumour NOS
Typical carcinoid/neuroendocrine tumour grade I
Atypical carcinoid/neuroendocrine tumour grade II
Neuroendocrine carcinomas
Small cell carcinoma
Combined small cell carcinoma
Large cell neuroendocrine carcinoma
Combined large cell neuroendocrine carcinoma
Tumours of ectopic tissue
Melanoma
Meningioma
Mesenchymal tumours specific to the lung
Pulmonary hamartoma
Chondroma
Diffuse lymphangiomatosis
Pleuropulmonary blastoma
Intimal sarcoma
Congenital peribronchial myofibroblastic tumour
Pulmonary myxoid sarcoma with EWSR1-CREB1 fusion
PEComatous tumours
Lymphangioleiomyomatosis
PEComa benign
PEComa malignant
Haematolymphoid tumours
MALT lymphoma
Diffuse large B cell lymphoma NOS
Lymphomatoid granulomatosis NOS
Lymphomatoid granulomatosis, grade I
Lymphomatoid granulomatosis, grade II
Lymphomatoid granulomatosis, grade III
Intravascular large B cell lymphoma
Langerhans cell histiocytosis
Erdheim-Chester disease

NOS: Not otherwise specified

Table1: Classification of Lung Tumours World Health Organization 2021 [4].

Atypical adenomatous hyperplasia lung appears immune reactive to thyroid transcription factor-1(TTF1).

Atypical adenomatous hyperplasia lung requires segregation from neoplasms such as non-mucinous adenocarcinoma in situ or reactive pneumocyte hyperplasia [8,9].

Characteristically, atypical adenomatous hyperplasia is incidentally discovered upon histological examination of pulmonary resection specimens. Appropriate discernment upon miniature core needle biopsy samples or trans-bronchial tissue specimens appears challenging as the entire lesion necessitates precise assessment [8,9].

Atypical adenomatous hyperplasia lung is devoid of pertinent biochemical and haematological parameters indicative of the lesion. Computerized tomography (CT) exhibits faint, fluid nodules which may assist lesion detection.

Atypical adenomatous hyperplasia can be appropriately managed by surveillance upon computerized tomography (CT). Frequency and duration of surveillance appears contingent to tumour magnitude [8,9]. Optimally, tumefaction may be subjected by comprehensive surgical eradication.

Prognostic outcomes are superior and incriminated subjects appear alleviated upon adequate extermination of lesion [8,9].

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