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Review Article

Intrusive and Cankerous -Adenocarcinoma Lung

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Abstract

Adenocarcinoma lung manifests as a frequently discerned, non small cell carcinoma incriminating the pulmonary parenchyma. Neoplasm exhibits glandular differentiation or mucin production and exemplifies definitive morphological patterns as acinar, papillary, micro-papillary, lepidic or solid. Tumefaction is comprised of mucinous and non mucinous subcategories. Lepidic pattern represents as a non invasive component of an invasive adenocarcinoma. Additionally, tumour cells appear immune reactive to pneumocyte markers or thyroid transcription factor-1 (TTF-1).

Keywords: Intrusive; Cankerous; Adenocarcinoma Lung

Introduction

Adenocarcinoma in situ (AIS) and atypical adenomatous hyperplasia lung manifest as pre-invasive pulmonary lesions. Minimally invasive adenocarcinoma lung represents as a distinctive entity. Invasive mucinous adenocarcinoma is pre-eminently configured of mucinous bronchioloaveolar carcinoma. Along with, colloid adenocarcinoma appears to be articulated of clear cell, signet ring cell or mucinous cystadenocarcinoma. The terminology of bronchioloalveolar carcinoma and adenocarcinoma lung mixed subtype appears obsolete. The frequently expounded non small cell, adenocarcinoma lung exhibits a female predilection and is commonly observed within African Americans, in contrast to Caucasian population. Notwithstanding, adenocarcinoma lung frequently incriminates male, non smoker subjects. Neoplasm is enunciated within adult subjects between 60 years to 70 years [1,2].

Adenocarcinoma lung enunciates genetic mutation within EGFR, a manifestation which is exceptionally encountered within mucinous neoplasms. Besides, KRAS, BRAF or NTRK chromosomal mutation, ALK genetic rearrangement, ROS or RET genomic fusion, HER2 or MET genetic amplification or SMARCA4 loss of function may be encountered [1,2]. Generally, upper lobe and peripheral pulmonary parenchyma is implicated, in contrast to lower lobe or centric pulmonary zones. Distant metastasis is commonly exemplified within brain followed in frequency by bone, hepatic parenchyma or adrenal glands. Possible emergence of brain metastasis is contingent to tumour magnitude and regional lymph node metastasis [1,2].

Adenocarcinoma lung commonly arises in subjects with history of cigarette smoking or tobacco consumption and exposure to passive smoke. Besides, elements such as radon from soil, fumes of cooking oil or occupational asbestos exposure as with ship building or construction may contribute to disease emergence. Exposure to aforesaid toxic cellular substances engenders various genetic mutations with consequent proliferation of endobronchial cells [1,2].

Adenocarcinoma lung exemplifies cogent clinical symptoms as cough, haemoptysis, dyspnoea, loss of weight loss or chest pain. Mucinous adenocarcinoma is accompanied by productive cough. Paraneoplastic syndrome or endocrine syndrome is infrequently

discerned, in contrast to small cell carcinoma lung. Additionally, manifestations such as hypertrophic pulmonary osteoarthropathy with clubbing of fingers, symmetric polyarthritis or periostitis of long bones may ensue [2,3].

Cytological examination exhibits three dimensional clusters and cohesive cellular aggregates. Tumour cells are impregnated with foamy or vacuolated cytoplasm, fine nuclear chromatin and variable, prominent nucleoli. Tumefaction may commonly be discerned upon evaluation of pleural effusion or fluid obtained from aspirating needle. Grossly, adenocarcinoma lung appears as a well demarcated, non encapsulated neoplasm with a central scar. Foci of necrosis are observed. Cut surface is tan or grey/white. In contrast, minimally invasive adenocarcinoma emerges as a focal, solitary lesion with magnitude ≤ 30 millimetres [2,3]. Upon frozen section, majority (~85%) of neoplasms may be accurately categorized. Lesion may be classified as non small cell carcinoma or adenocarcinoma lung. Notwithstanding, neoplasms of advanced grade may be challenging to discern [2,3]. Upon microscopy, invasive mucinous adenocarcinoma is comprised of goblet cells or columnar cells imbued with abundant intracytoplasmic mucin. Tumefaction exemplifies tumour invasion > 5 millimetres into subjacent stroma. Invasive non mucinous adenocarcinoma preponderantly exhibits glandular differentiation. Neoplastic stromal invasion exceeds > 5 millimetres [3,4]. Foci of glandular differentiation are categorized as ~lepidic pattern wherein proliferating club cells and type II pneumocytes layer alveolar walls. Tumefaction is devoid of complex architecture. Lymphatic, vascular or perineural invasion is observed. ~acinar pattern is configured of spherical to elliptical glands which invade subjacent fibrotic stroma. Neoplasm may articulate complex, glandular subtypes and may represent advanced tumour grade. ~papillary pattern is comprised of papillary projections with fibro-vascular cores layered with malignant cuboidal or columnar epithelial cells. Papillary articulations supplant the alveolar layer. ~micropapillary pattern manifests with poorly defined cellular projections or epithelial tufting which appear devoid of fibro-vascular cores. ~solid pattern is configured of sheets of neoplastic cells. Infrequently enunciated subcategories emerge as ~colloid subtype which is composed of cuboidal or columnar epithelial cells encompassed within abundant pools of extracellular mucin which may deform alveolar spaces. ~foetal subtype which recapitulates pseudo-glandular foetal epithelium. Low grade tumour enunciates cellular component with minimal cellular and nuclear atypia whereas high grade neoplasm is constituted of significantly atypical cells. ~enteric subtype which is reminiscent of colorectal adenocarcinoma and appears immune reactive to intestinal biomarkers. ~minimally invasive adenocarcinoma which articulates a focal neoplasm demonstrating variable or predominantly lepidic configuration and tumour magnitude ≤ 30 millimetres with ≤ 5 millimetre magnitude of focal tumour invasion. Neoplasm may disseminate through air spaces, a feature which is commonly encountered within adenocarcinoma, in contrast to squamous cell carcinoma lung [3,4]. Tumour grade is contingent to combination of various histological patterns wherein up to 10% of tumour area appears representative of specific architectural pattern [3,4]. Adenocarcinoma lung is graded as ~grade I or well differentiated neoplasm comprised of lepidic predominant tumour along with an absence of or < 20% of high-grade tumefaction ~grade II or moderately differentiated neoplasm preponderantly constituted of an acinar or papillary pattern along with an absence of or < 20% of high grade tumefaction. ~grade III or poorly differentiated neoplasm composed of various architectural configuration with ≥20% of high grade tumefaction [3,4]. Ultrastructural examination exhibits tumour cells imbued with short microvilli. Besides, neoplastic cells appear devoid of cytoplasmic cytosomes [3,4].

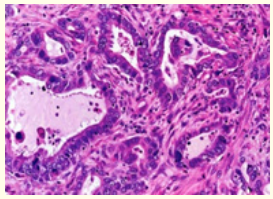

Figure 1: Adenocarcinoma lung demonstrating glandular articulations layered by columnar epithelial cells imbued with abundant intracytoplasmic mucin. Tumour cells display significant cellular and nuclear atypia. Surrounding stroma is fibrotic and exhibits focal tumour invasion [7].

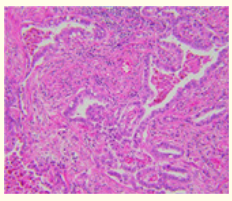

Figure 2: Adenocarcinoma lung delineating glandular structures layered by malignant columnar epithelial cells with abundant intracytoplasmic mucin and prominent cellular and nuclear atypia. Surrounding stroma is dense, fibrotic and infiltrated [8].

Subtype	Epidemiology	Immunohistochemistry	Molecular Alterations
PEAC	M>F, smokers (0.5%)	CK7, CDX-2, villin	KRASG12V, ERBB2, EGFR-del ex19, L8S8R
Foetal adenocarcinoma	Young female, smokers (0.5%)	Synaptophysin, vimentin	WNT signal
Colloid adenocarcinoma	F>M, smokers (0.1%)	CK7, CK20, CDX-2, MUC2	KRAS codon 12/13
NOS carcinoma	M>F (0.1%)	TTF-1, p40, none	EGFR, ALK-EML4

Table 1: Characteristics of Pulmonary Adenocarcinoma [4].

PEAC: Pulmonary Enteric Adenocarcinoma; NOS: Not Otherwise Specified; TTF-1: Thyroid Transcription Factor-1

Adenocarcinoma lung appears immune reactive to thyroid transcription factor-1 (TTF1), AE1 / AE3, CK7, β catenin or Napsin A. Tumour cells appear immune non reactive to p53, p40, p63, CK5/6, Wilm's tumour protein (WT1), D2-40, calretinin or epidermal growth factor receptor (EGFR) [5,6]. Adenocarcinoma lung requires segregation from neoplasms such as squamous cell carcinoma, small cell carcinoma, high grade neuroendocrine tumour, metastatic adenocarcinoma, atypical adenomatous hyperplasia, adenocarcinoma in situ, bronchial adenoma/ciliated muconodular papillary tumour, adenoid cystic carcinoma or metastatic papillary thyroid carcinoma. Besides, demarcation from benign pulmonary lesions as granuloma, hamartoma, pneumonia or various metastatic neoplasms is mandated [5,6]. Upon radiography, adenocarcinoma lung appears lobulated and demonstrates spicules with a well-defined perimeter. Neoplasm enunciates solid, dense zones with configuration of solid or acinar neoplastic configuration. Mucinous subtype or tumour with lepidic pattern may exemplify ground glass opacities. Besides, air bronchogram may be beneficially adopted for neoplastic determination. Adenocarcinoma lung can be appropriately discerned upon histological evaluation and cogent staining with immunohistochemistry [5,6]. Stage I, stage II, stage IIA and stage IIB of adenocarcinoma lung devoid of stromal invasion can be appropriately managed with surgical extermination in combination with adjuvant radiation therapy. Neoplasm within stage IIB delineating stromal invasion or stage IIIA and stage IIIB with absent stromal invasion may be subjected to surgical extermination in combination with precise chemotherapy and radiation therapy.

Conclusion

Neoplasms unamenable to surgical extermination or tumours with distant metastasis can be optimally treated with precise molecular or targeted chemotherapy in combination with radiation therapy [5,6]. Superior prognostic outcomes are encountered with lepidic tumour configuration of adenocarcinoma lung. Adverse prognostic outcomes are associated with contributory factors as ~neoplasm depicting dissemination through air spaces ~tumour magnitude > 2.5 centimetres ~tumour invasion of visceral pleura ~solid or micropapillary subtype of neoplasm [5,6].

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