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Editorial Article

Infringe and Entrench-Invasive Mucinous Adenocarcinoma Lung

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Invasive mucinous adenocarcinoma lung emerges as a distinct, exceptionally discerned ariant of pulmonary adenocarcinoma associated with an aggressive clinical course. ypically, neoplasm is well differentiated and homogeneous wherein a component of non-mucinous glands is absent. Tumour cells depict morphological features akin to goblet cells or columnar epithelial cells wherein neoplastic cells are imbued with abundant intracytoplasmic mucin. Intrapulmonary metastasis and tumour reoccurrence are frequently enunciated. Contingent to molecular and genetic disparity, colloid adenocarcinoma lung is currently contemplated to be a distinct entity.

Invasive mucinous adenocarcinoma configures ~0.2% of primary pulmonary carcinomas and ~ 10% of pulmonary adenocarcinomas. Invasive mucinous adenocarcinoma preponderantly incriminates inferior pulmonary lobes. A female predominance is observed with female to male proportion of ~2: [01, 02].

Invasive mucinous adenocarcinoma lung appears concordant with Ras-Raf-MEK-ERK pathway. Besides, KRAS genetic mutation and ALK genetic rearrangement is enunciated. EGFR genetic mutation is exceptional. Invasive mucinous adenocarcinoma lung expressing hepatocyte nuclear factor 4α (HNF4α) may exhibit genetic mutations within KRAS or EGFR genes. Few neoplasms devoid of HNF4α expression may enunciate ALK genomic fusion. Besides, neoplasm lacking KRAS genetic mutations may expound NRG1, ERBB4 and BRAF genetic fusions [01, 02]. In contrast to diverse subtypes of pulmonary adenocarcinomas, invasive mucinous adenocarcinoma is minimally associated with cigarette smoking [02, 03].

Invasive mucinous adenocarcinoma lung is frequently accompanied by intrapulmonary metastasis and tumour reoccurrence. In contrast, extra-pulmonary or distant metastasis is exceptionally encountered. Around ~80% neoplasms demonstrate lack of regional lymph node. metastasis(N0) and distant metastasis (M0) upon initial representation. Upon initial tumour discernment, enlarged neoplasms and advanced tumour (T) is exemplified [02, 03]. Cytological examination exhibits sheets and cellular clusters or papillary structures layered by tall columnar epithelial cells permeated with abundant intracytoplasmic mucin, designated as 'gold mucin'. Tumour cell nuclei appear miniature and expound minimal atypia [02, 03].

Grossly, tumefaction appears as a solid, grey/white to yellowish lesion with graded, poorly defined neoplastic perimeter. Tumour is circumscribed by and pervaded with mucus [03, 04]. Upon microscopy, neoplasm appears homogenous, well differentiated and is composed of goblet cells or columnar epithelial cells reminiscent of gastrointestinal epithelium. An admixture of non-mucinous glands is absent. Tumour cells are permeated with abundant intracytoplasmic mucin with basal nuclei and delineate minimal cellular and nuclear atypia. Adjacent alveolar spaces exemplify lumens impregnated with mucin, a feature which is non-specific of invasive mucinous adenocarcinoma lung [03, 04]. Goblet cells and columnar epithelial cells configuring invasive mucinous adenocarcinoma are pervaded with intracytoplasmic mucin. Cytological atypia is minimal to absent. Submucosal mucinous glands confined within bronchial tissue and neoplastic cells configuring invasive mucinous adenocarcinoma lung manifest with identical, bland cytological and morphological features Neoplasm may predominantly exhibit lepidic pattern of tumour evolution accompanied by mild distortion or destruction of alveolar architecture. Additionally, an admixture of minor papillary, solid, acinar or micro-papillary neoplastic configuration is observed Occasionally, depleted intracytoplasmic mucin and enhanced cytological atypia may occur, especially within areas of stromal invasion Tumour dissemination through air spaces is commonly enunciated. Besides, high grade transformation of pre-existing mucinous neoplastic components may ensue [03, 04].

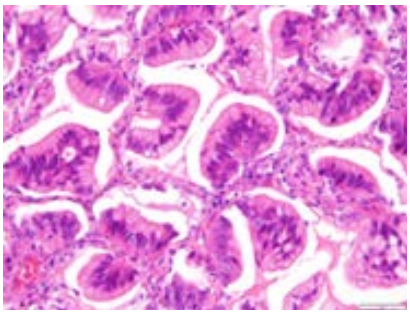

Figure 1: Invasive mucinous adenocarcinoma delineating papillary structures layered by tall columnar epithelial cells permeated with intracytoplasmic mucin. Cytological atypia is minimal. Lepidic pattern of tumour progression is observed [07].

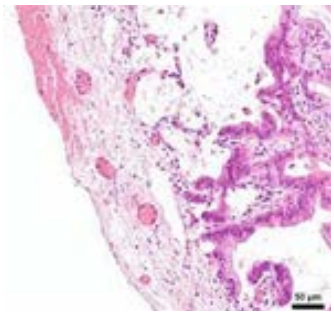

Figure 2: Invasive mucinous adenocarcinoma delineating papillary structures lined by tall columnar epithelium with abundant intracytoplasmic mucin. Cytological atypia is minimal [08].

Table 1: Histology of pulmonary mucinous adenocarcinoma(3,4).

Neoplasm	Characteristic morphology	Absent features	IHC
Mucinous adenocarcinoma in situ(AIS)	Mucin secreting cells lining alveoli. Magnitude>3 centimetres	Tissue invasion, necrosis, intra-alveolar tumour clusters, multiplicity	TTF-1-, CDX2- CK7+, CK20+
Minimally invasive mucinous adenocarcinoma	Mucin secreting cells lining alveoli. Magnitude>3 centimetres. Acinar invasion <5 mm	Tissue invasion, necrosis, intra-alveolar tumour clusters, multiplicity	TTF-1-, CDX2- CK7+, CK20+
Mucinous adenocarcinoma	Predominant mucin forming cells, an in situ component	Cuboidal or columnar cells admixed with abundant pools of extracellular mucin with distorted alveolar spaces	TTF-1+, CK7+, CK20-, CDX2-
Colloid adenocarcinoma	Cuboidal or columnar cells admixed with abundant pools of extracellular mucin with distorted, replaced alveolar spaces		TTF-1+, CK7+, CK20+, CDX2+ in TTF-1- tumours

IHC: Immunohistochemistry, TTF-1: thyroid transcription factor 1, CK: cytokeratin Tumour cells appear immune reactive to hepatocyte nuclear factor 4α (HNF4α), MUC2 or CK7. Periodic acid Schiff's stain (PAS) can be employed to highlight intracellular and extracellular mucin. Neoplastic cells appear immune non-reactive to thyroid transcription factor-1 (TTF-1), Napsin A, CDX2 or CK20 [05, 06]. Invasive mucinous adenocarcinoma lung requires segregation from neoplasms such as metastatic colorectal adenocarcinoma or colloid adenocarcinoma lung [05, 06]. Invasive mucinous adenocarcinoma lung can be appropriately discerned upon histological examination and precise immunohistochemistry. Primary invasive mucinous adenocarcinoma lung can be appropriately demarcated from colorectal adenocarcinoma with cogent endoscopic and radiological techniques. However, definitive discernment of a malignant neoplasm upon evaluation of surgical tissue samples may be challenging. Trans-bronchial biopsy is associated with limited results as the neoplasm is commonly confined to peripheral parenchyma of inferior pulmonary lobes. Percutaneous computerized tomography (CT) guided surgical tissue sampling is efficacious in obtaining diagnostic tissue samples [05, 06]. Upon plain radiography, incriminated parenchyma exhibits pulmonary consolidation with a variable countenance. Akin to diverse pulmonary carcinomas, invasive mucinous adenocarcinoma configures a solid lesion. Besides, lesion may exhibit a commingling of solid and ground glass opacities, reminiscent of pneumonia. An air bronchogram can be beneficially adopted for tumour discernment. Frequently, neoplasm is multifocal and exemplifies multi-lobar involvement [05, 06]. Fluorodeoxyglucose positron emission tomography (FDG/PET) demonstrates minimal tracer accumulation within the neoplasm. Stage I, stage II and stage IIA of invasive mucinous adenocarcinoma lung may be preferentially subjected to surgical extermination followed by adjuvant chemotherapy with platinum-based agents and radiation therapy. Neoplasms unamenable to surgical resection or exhibiting distant metastasis may be managed with targeted chemotherapy and variable quantities of radiation therapy [05, 06]. Stage I, stage II, stage IIA and stage IIB neoplasms

devoid of invasion can be optimally alleviated with surgical extermination in concurrence with adjuvant radiation therapy. Stage IIB tumefaction with invasion and stage IIIA or stage IIIB neoplasms lacking invasion are managed with surgical eradication along with adjuvant chemotherapy and radiation therapy. Invasive mucinous adenocarcinoma lung is associated with intermediate to inferior prognostic outcomes, in contrast to non-mucinous adenocarcinoma. An estimated 70% neoplasms disseminate through alveolar spaces, a feature associated with elevated tumour reoccurrence [05, 06].

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7. Image 1 Courtesy: Translational lung cancer research
8. Image 2 Courtesy: Nature