of MLC. CD68 and lysozyme, although not lineage specific for monocytes/macrophages, were found to be the most sensitive immunohistochemical stains in the detection of MLC in one study, regardless of the French-American-British subtype or transformation from myelodysplastic syndrome.[4]

While the molecular basis of homing to the skin by the myeloblasts in AML has been not completely elucidated, it is believed to be mediated by the interaction of chemokine receptors expressed on myeloblasts and ligands within skin. Some of the myeloid receptors involved in skin homing and retention include CCR5, CXCR4 and CCR2, via interaction with their ligands CCL3, CXCL12 and other unidentified chemokines, respectively.[5] In addition, expression of certain T cell antigens by myeloblasts facilitates skin homing, with expression of CD56, cutaneous leukocyte antigen (CLA) and lymphocyte function-associated antigen-1 all implicated.[6]

In our case the cutaneous myeloblasts were intimately associated with benign dermal naevus cells in a collision lesion. It is most likely that the collision between these tumours is simply a chance occurrence. Given the rare nature of this case, reasons for co-localisation of myeloblasts and naevus cells can only be speculative. Local paracrine effects from the dermal myeloblasts such as fibroblast growth factor (FGF) which is a melanocyte mitogen[7] and which is elevated in leukaemia[8] could have stimulated melanocyte proliferation. On the other hand chemokines secreted by the naevus cells may have resulted in accumulation of the neoplastic myeloblasts. For example, increased numbers of mast cells are typically seen within neurofibromata,[9] indicating that myeloid cells may accumulate within skin tumours.

While Keen previously described a single case of a combined melanoma *in situ* and leukaemia cutis,[10] to our knowledge, our case is the first to describe a collision between a benign intradermal melanocytic naevus and myeloid leukaemia cutis. In our case the histomorphology mimicked naevoid melanoma, but the correct diagnosis was reached using a broad immunohistochemical panel based on high index of suspicion. Further investigations revealed an occult primary haematological malignancy, allowing earlier treatment than would otherwise have been the case.

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Pulmonary epithelial myoepithelial carcinoma with papillary architecture: an uncommon morphology of a rare tumour

Sir,

Epithelial myoepithelial carcinoma (EMC) is a rare primary tumour of the submucosal bronchial glands. The literature encompasses only case reports of this entity from across the world. The common morphological patterns reported are those with tubules, glands or solid areas. Papillary architecture in EMC is rare. We present a rare case of EMC with papillary architecture.

A 50-year-old woman presented with chest pain and dyspnoea on exertion of 1-year duration along with occasional cough with haemoptysis. She had a background history of exposure to indoor smoke from a cooking stove ('chulha') for the previous 20 years. X-ray of the chest revealed patchy opacities in both lung fields and consolidation of the right paracardiac region. A contrast enhanced computed tomography scan (CECT) of the thorax showed a 2.6 × 1.9 cm soft tissue lesion at the right hilum (Fig. 1A) leading to blockage of the right middle lobe bronchus resulting in collapse and consolidation of right middle lobe. Fibre-optic bronchoscopy was performed, and biopsies were obtained which were diagnosed as EMC. The patient underwent a right lobectomy. The post-operative period was uneventful.

The lobectomy specimen revealed a 3.5 × 1.8 × 1.5 cm endobronchial tumour (Fig. 1B) with a greyish-white cut surface. Microscopically the sections showed an intraluminal proliferating tumour exhibiting a biphasic architecture comprising complex glandular architecture along with well-formed papillae (50% of each component) (Fig. 2A–F). There were focal areas with clear cell and oncocytic change also. The tubules/glands contained eosinophilic intraluminal secretions and were lined with a bilayer of inner epithelial and outer myoepithelial cells. The epithelial cells were cuboidal with eosinophilic cytoplasm and stained immunohistochemically (IHC) with cytokeratin CK7 (Fig. 2G). The myoepithelial cells were clear cells and exhibited positivity for P-40 (Fig. 2H), smooth muscle actin (SMA) (Fig. 2I) and S-100. Both the epithelial and myoepithelial cells were negative for thyroid transcription factor (TTF) 1. Focal

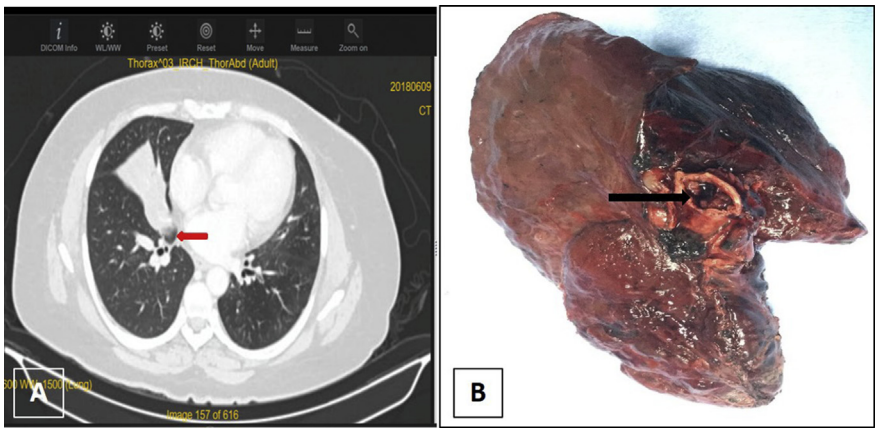

Fig. 1 (A) Contrast enhanced computed tomography image showing a hyper-dense endobronchial growth with irregular margins and few hypo-dense areas of necrosis within the right bronchus intermedius (red arrow) along with collapse of the right middle lobe. (B) Gross appearance of the lobectomy specimen revealing endobronchial growth (black arrow).

necrosis and few mitotic figures (3−4/10 high power fields) were also identified with Ki-67 proliferation index of about 25%. There was no infiltration of the surrounding soft tissue by the tumour cells. None of the lymph nodes dissected from the specimen showed metastatic tumour deposits.

The case was also tested for common driver mutations of lung cancer which included *EGFR* mutation testing by real time-polymerase chain reaction and IHC for *ALK* rearrangement on Ventana platform (Ventana, USA) and for HER2-neu manually. However, none of them were positive.

Salivary gland tumours of the lung form <1% of all lung neoplasms and originate from the submucosal seromucinous glands present in the airways.[1] The name EMC was coined by Donath *et al.* in 1972.[2] Various other nomenclature for this entity include adenomyoepithelioma, glycogen-rich adenoma, tubular solid adenoma, clear cell adenoma, monomorphic clear cell tumour, glycogen-rich adenocarcinoma and clear cell carcinoma.[3] The existence of this entity is only as case reports in the literature with approximately 50 cases reported to date. Primary lung EMCs are described as well circumscribed polypoidal endobronchial tumours with a tan cut surface. The histomorphology is that of a biphasic tumour with a predominantly tubular-glandular architecture.[1] Some reports have found solid architecture accompanying the

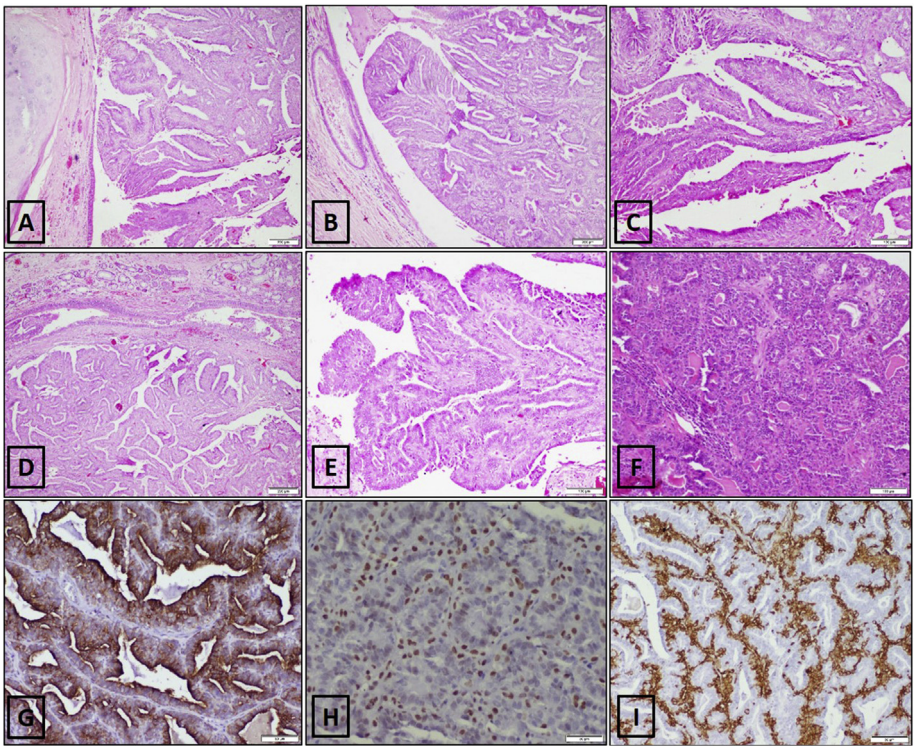

Fig. 2 Histomorphological and immunohistochemical (IHC) features of the case of epithelial-myoepithelial carcinoma. (A−E) Various areas of the tumour with well-formed papillary architecture. (F) Areas of the tumour showing tubulo-glandular differentiation. (G) The epithelial cells show CK7 expression in their cytoplasm. (H) Nuclear positivity of P-40 in the myoepithelial cells nuclei lining the epithelial cells. (I) Smooth muscle actin (SMA) staining the myoepithelial cells along with the stromal components.

tubular pattern.[4–6] The case described by Muñoz *et al.* has cystic architecture also.[7] Seethala *et al.* have described the variants of EMC of the salivary gland in their study; namely, the dedifferentiated EMC, oncocytic EMC, EMC ex pleomorphic adenoma, double-clear EMC, and EMC with myoepithelial anaplasia.[3] They reported a biphasic papillary pattern in a minority of salivary gland EMCs (7/61; 11.5%) in their study.[3] Our case had well-formed true papillae lined by TTF-1 negative and CK7 positive epithelial cells along with tubulo-glandular architecture. The abluminal myoepithelial cells can have varied morphologies including clear cells, spindled cells and amphophilic cytoplasm. Those seen in our case were predominantly clear cells which stained brightly with S-100, P-40 and SMA. These myoepithelial cells were also seen lining the papillary epithelium.

EMC has a broad range of differential diagnoses which depends on the predominance of the cells forming its architecture. The biphasic nature of this entity entails a differential of pleomorphic adenoma.[8] The absence of a chondromyxoid stroma and predominance of clear myoepithelial cells in our case helped in differentiating from this neoplasm. The second differential which arises is adenoid cystic carcinoma which can be distinguished by its infiltrative nature and cribriform architecture. The other close differential which may be considered is a metastatic salivary gland EMC to the lung. However, the patient had no such history in the past nor did she have any parotid or submandibular mass. In addition to these salivary gland type malignancies, endobronchial papillary tumours were added to the list of differential diagnoses due to a prominent papillary architecture in the current case. The primary endobronchial papillary tumours are rare and are commonly papillomas, cystadenomas or adenocarcinomas.[9,10] The index case, which was lined with TTF-1 negative epithelial cells, could be easily differentiated from the above lesions which originate from the surface epithelium and are in contrast TTF-1 positive. Rarely, the epithelial component of EMC shows pneumocytic differentiation giving rise to positive staining for TTF-1. The metastatic papillary tumours are the next common differentials, the likelihood of which was nullified by the histomorphology and the IHC characteristics of the present case. Some salivary gland tumours also rarely display a papillary architecture, such as mucoepidermoid carcinomas[11] and polymorphic low grade adenocarcinomas (PLGA).[12] Lack of squamous/transitional cells and mucin ruled out mucoepidermoid carcinoma and presence of a prominent abluminal myoepithelial component easily distinguished it from PLGA.

In conclusion, the present case highlights an unusual histomorphology of an endobronchial epithelial myoepithelial carcinoma bearing a papillary architecture. We also describe the gamut of differentials which needed consideration during the diagnosis of this lesion. We emphasise the importance of careful assessment of histomorphology and supportive immunohistochemistry which help in diagnosing complex cases.

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PSA-positive urethral adenocarcinoma of female genital tract

Sir,

A 75-year-old female presented with macroscopic haematuria and on examination was found to have a mass at the external urethral meatus. Her past medical history included severe emphysema, hypertension, dyslipidaemia and a 50 pack-year smoking history. A cystoscopy revealed a urethral tumour and also a separate exophytic bladder lesion overlying the right ureteric orifice. Contrast magnetic resonance imaging (MRI) of her pelvis showed a 3 cm mass with an enhancing peripheral rim in the posterior distal urethra extending both into the anterior vaginal wall inferiorly and into the urethra anteriorly.

The biopsy from the urethral tumour showed an invasive adenocarcinoma. Subsequently another cystoscopy was