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SHORT COMMUNICATION

Pulmonary Fissures–How do they form?

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Abstract

Variations of pulmonary fissures are a factor during pulmonary surgery. Data regarding their development is limited. Early fixation of the visceral pleura seems to be mediated by myofibroblasts and extracellular components like fibronectin. So far, no concepts explain sufficiently the origin of the shape and orientation of these.

The great variation of pulmonary fissures is often neglected in Anatomical textbooks. During pulmonary surgery and therapy, it is crucial to identify the anatomy prior to the intervention. Examples are thoracoscopic lobectomies for lung cancer treatment [1] or endoscopic emphysema therapy [2]. While several publications describe the variation of pulmonary fissures in different populations (morphologic [3-14], radiologic [15-17]), knowledge of their development is scarce (most developmental aspects are focused on the lung tissue proper [18-20]).

During the early phase of lung development, the two entodermal pulmonary buds are surrounded by an early vascular plexus and some mesenchymal cells. They contact the splanchnopleura of the intra-embryonal coelom which sends mesenchymal cells towards the formation of the bronchial trees and the visceral pleura covering the lung tissue [19]. While the dividing bronchi grow caudal and lateral, parts of the visceral pleura remain affixed next to the first bronchial divisions constituting the pulmonary fissures and subsequently the lobar arrangement. This arrangement is to a certain degree species-specific [21], but somehow preserved within a species. Humans usually show only one oblique fissure develop in the left lung, while an oblique and a horizontal fissure develop in the right lung.

The mechanism underlying the lobar separation is not fully understood. Both variations, lack fissure development [22-24] and accessory fissures [24], are known to occur in different syndromes. Selective absence of the right horizontal fissure has also been observed [25,26]. Beyond their description, two aspects are discussed in the literature: a mixture of biochemical factors, and a possible physical influence.

Factors like Fras 1 (Fraser Syndrome 1 protein) [27] or Isl1 (Insulin gene enhancer protein 1) regulation of homeobox protein Nkx 2.1 [28] are known to be essential for lobe formation of the lungs, while Wnt (portmanteau of wingless and int-1), Fibroblast Growth Factor 10 and Sonic hedgehog were only studied in for epithelial tip-splitting and further branching [29]. Fibroblast Growth Factor 8, which plays a role in fetal lung development, is not involved in lobation [30]. The difference between left and right lungs seems to be controlled by Lefty-1 [29].

In general, the extracellular matrix seems to play an important role in lung lobe formation [31]. Fibronectin (influenced by Wnt) and myofibroblasts are discussed as a physical resistance for epithelial spreading leading to branches next to/around this blocking [29]. Basement membrane proteins like QBRICK might also be essential components [32]. Hegazy added that slight shift of the heart tube towards the left side during embryonic development might result in the difference in lobation of the two lungs. Such shift might amalgamate the part of left lung corresponding to middle lobe of right lung into its upper lobe resulting in formation of only two lobes 'upper and lower' in left lung [33,34].

Both aspects do not sufficiently explain the morphogenetic shape and orientation of the pulmonary fissures. Hopefully, this short communication stimulates the community to raise further ideas and experiments concerning this basic aspect of pulmonary lobation.

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