A STUDY ON MICROVASCULAR CHANGES ON MONOCROTALINE-INDUCED
RAT LUNG BY CORROSION CASTING METHOD

모노크로탈린을 투여한 백서폐장의 미세혈관구조변화에 관한 부식주조법에 의한 주사전자현미경적 연구

Na Hye Myong, Eu Keun Ham*. Department of pathology, Dan Kook University and Seoul National University*, College of Medicine.

To investigate the microvascular structural changes in pulmonary hypertension, 24 Sprague-Dawley rats each were given one intraperitoneal injection of 2% monocrotaline(MCT) solution and then scanning electron microscopy(SEM) after microvascular corrosion casting on their lungs was performed. examination revealed medial thickening of pulmonary arteries. loss of pulmonary arterioles, and interstitial fibrosis since the 12th day. SEM on the normal lungs showed two kinds of microvascular structures. One showed a well-formed dimensional basket structure of uniform flat tubular alveolar capillaries which were connected with each other. The other revealed a two-dimensional reticular sheet composed of regular tubular branches mainly in the regions supplied bronchial arteries. The MCT-treated groups disclosed evident differences from these in their structures but more prominent changes were found in the bronchial capillaries, showing a characteristic appearance of their dense and thick encasement around pulmonary arteries. Alveolar capillaries generally showed loss of the basket-like appearance, which were replaced largely by angular or small ring-like structures. Quantitative examination revealed significant changes in diameter, density, and intercapillary distance. Both bronchial and alveolar capillaries were significantly increased in diameter but the density was increased only in the bronchial ones. Similar microvascular changes had already been reported in interstitial pulmonary fibrosis. In conclusion. morphological and quantitative microvascular changes be considered as a new finding of vascular remodelling in primary pulmonary hypertension. Also interstitial fibrosis and microvascular proliferation by bronchial arteries were thought to be important factors in the pathogenesis of irreversible pulmonary hypertension.