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The differences of apoptosis and pulmonary fibrosis following sulfur mustard-induced acute pulmonary injury via intraperitoneal injection and intratracheal instillation in rats

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Objective: Sulfur mustard (SM) is a vesicant alkylating agent. There are still biochemical mechanisms underlying SM damage that are unknown. This study was to determine the differences of apoptosis and pulmonary fibrosis underlying SM-induced acute pulmonary injury via intraperitoneal injection and intratracheal instillation in rats.

Methods: SM-induced changes in apoptosis and pulmonary fibrosis were observed in intraperitoneal and tracheal SM groups that were injected intraperitoneally and instilled intratracheally with 0.1 mL of diluted SM (0.96 LD50=8 mg/kg) and SM (0.98 LD50=2 mg/kg), respectively, and an untreated control group.

Results: In the alveolar septum, the positive expression ratio of apoptotic cells, Bax, Bcl-2, caspase-3, and caspase-9 by TUNEL and immunohistochemistry (streptavidin-perosidase method) in the intraperitoneal SM group was increased compared with the tracheal SM group ($P<0.05$). A significantly lower positive expression ratio of Bcl-2 was detected ($P<0.05$). Electron microscopic observations confirmed that the type I and II alveolar epithelial cells in lungs exhibited apoptotic morphologic features, such as cracked, lost, and disordered microvilli of membranes, fuzzy mitochondrial cristae, and detached, dissociated ribosomes from the surface of the rough endoplasmic reticulum. A significantly higher positive expression ratio of MMP-2, MMP-9, TIMP-1, TIMP-2, collagen type I, collagen type III, TGF-β1, and Smad7 by immunohistochemical staining in the alveolar septum were detected in the intraperitoneal SM group compared with the tracheal SM group ($P<0.05$).

Conclusions: These data demonstrated increased apoptosis and pulmonary fibrosis via intraperitoneal injection under similar SM LD50 doses in rats.

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