

Acute Pancreatitis and Pulmonary Complications

Murli Manohar*, Gulshan Singh

School of Medicine, Tulane University, New Orleans, USA

*Corresponding author: Manohar M, Department of Medicine and Section of Pulmonary Diseases, Tulane Eosinophilic Disorder Center, Tulane University School of Medicine, USA, Tel: +504-865-5000, E-mail: mmanohar@tulane.edu

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Editorial

Acute pancreatitis (AP) is an inflammatory disorder of pancreas characterized by the activation of intrapancreatic digestive enzyme and acinar cell damage followed by systemic inflammatory response [1,2]. Acute pancreatitis can be happened by several ways such as due to alcohol consumption, blockade of pancreatic ducts, food and mutation in trypsinogen genes [3,4]. However the severe form of acute pancreatitis causes multiple organs dysfunction syndrome (MODS) with a high rate of mortality about 15%-20%. During the pancreatitis progression, impaired airflow and difficulty to breathe (due to the narrowing of the airways, termed airflow obstruction) is the most common symptom of acute respiratory failure. Several other types of respiratory complications such early arterial hypoxia, pleural effusion, mediastinal abscess, pulmonary infarction, elevated diaphragm, empyema, acute lung injury and finally acute respiratory distress syndrome (ARDS) have been observed during severe acute pancreatitis [1,5]. Notably, acute lung injury (ALI) and ARDS are the two main respiratory failures; and among them, ARDS is the most severe and characterized by diffuse lung injury and severe hypoxemia [1,6-8]. During the pathogenesis of pulmonary complications of acute pancreatitis, variety of inflammatory mediators is released from different immune cells and digestive actions of pancreatic enzymes play a key role. Interestingly, an intricate network of molecular mechanism has been involved in acute pancreatitis associated acute respiratory failure, but the exact mechanism is poorly understood and unexplored. However, recent findings have identified several pro-inflammatory mediators such as tumor necrosis factor- α (TNF- α), interleukin (IL)-1 β , IL-6, IL-8 and macrophage inhibitory factor (MIF) playing a critical role in AP associated lung injury. Furthermore, involvement of several inflammatory cells has been reported during the disease pathogenesis. Notably, polarizations of macrophages (M2 and M1) regulate the migration and pulmonary infiltration of neutrophils into the pulmonary interstitial tissue, causing injury to the pulmonary parenchyma [1]. Additionally, activated mast cells induced by pancreatitis appear to play a critical role in the initiation of pancreatitis-associated lung injury and involves endothelial barrier dysfunction in both the pancreas and lungs [9]. Acute lung injury and acute respiratory distress syndrome in acute pancreatitis remains an unsolved issue and needs more research and resources to develop effective treatment therapy. Several studies have reported different molecules such as

caspase inhibitors, kynurenine 3-monooxygenase inhibitors, resolvin, and plants originated phytochemicals including resveratrol and apigenin in the experimental model that showed promising treatment options for the pathogenesis of AP-induced multiple organ failure [1].

Taken together, acute lung injury is a severe complication arise due to acute pancreatitis and a significant health problem associated with a high rate of mortality and need further attention to develop novel treatment options. Expert team of physicians and scientists in the area of pancreatic and lung disorder should work in close association while examination the patients of acute pancreatitis with lung injury. The outcome of this association may be helpful to identify the causes that may be helpful to reduce morbidity and mortality. Therefore, a more clear understanding of the progression of pathogenesis of lung injury and other organ dysfunction is expected to identify some novel treatment therapies for acute pancreatitis.

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