Acute Lung Injury: HRCT and Histopathologic Spectrum

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INTRODUCTION
Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are major pulmonary causes of morbidity and mortality. Acute pulmonary injury often results in acute hypoxic respiratory failure. Patients with acute pulmonary injury typically present clinically with ARDS. ARDS was formally defined in 1994 by the American-European Consensus Conference on ARDS as acute hypoxemia with a ratio of partial arterial oxygen to the fraction of inspired oxygen (i.e., PaO2/FIO2) of <200 mm Hg or less, bilateral infiltrates with a radiographic appearance that is consistent with pulmonary edema, and no evidence of cardiac failure clinically. ALI is less severe than ARDS and has similar diagnostic criteria with the exception of PaO2/FIO2 of ≥200 mm Hg.

DIFFUSE ALVEOLAR DAMAGE
Diffuse alveolar damage (DAD) is the most common histologic manifestation of ARDS and ALI. Patients present with severe hypoxemia and almost always require mechanical ventilation. While the precise mechanism of injury is unclear, capillary endothelial and alveolar epithelial damage result in exudation of fluid and products of cellular breakdown. With time, pneumocyte hyperplasia and fibroblast proliferation ensue.

The histologic appearance of DAD is divided into three phases: the acute or exudative phase, the organizing or proliferative phase, and the chronic or fibrotic phase. The acute phase occurs during the first week after the initial insult, and findings may include intra-alveolar edema, interstitial widening, and hyaline membranes (composed of cellular and proteinaceous debris). Thrombi may also be seen as a result of localized alterations in the coagulation pathway. The organizing phase follows features of interstitial fibrosis and type 2 pneumocytes. Eosinophils may be present in the interstitium, may infiltrate blood vessel walls, and may form eosinophilic microabscesses.

Histopathologic findings of DAD include intra-alveolar fibrin, macrophages, hyaline membranes (similar to the acute phase of DAD), and numerous eosinophils. Eosinophils may be present in the interstitium, may infiltrate blood vessel walls, and may form eosinophilic microabscesses.

HRCT findings of acute eosinophilic pneumonia are nonspecific and include consolidation and ground-glass opacities in a random (2/3) or peripheral (1/3) distribution. Smooth interalveolar septal thickening and small pleural effusions are frequently present and, in the absence of cardiomegaly, are helpful clues to the diagnosis.

HRCT findings of organizing pneumonia (OP) are airspace consolidation in a subpleural or peribronchial distribution. Ground-glass opacities may also be present, often with a bilateral asymmetric distribution. The reverse halo sign may be present in almost 25% of cases.

CONCLUSION
The patterns of acute lung injury and acute respiratory distress syndrome have different HRCT and histopathologic features. Knowledge of the characteristics and features of the different patterns in the spectrum of ALI and ARDS is important and helpful for guiding appropriate diagnosis and treatment of affected patients.

REFERENCES
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