

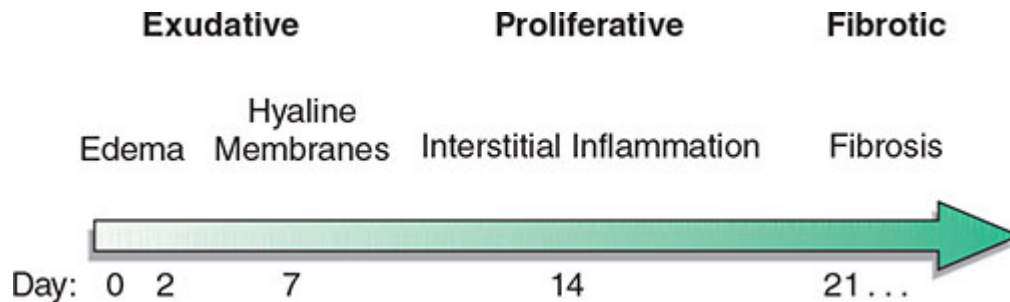


FIGURE 301-1 Diagram illustrating the time course for the development and resolution of acute respiratory distress syndrome (ARDS). The exudative phase is notable for early alveolar edema and neutrophil-rich leukocytic infiltration of the lungs, with subsequent formation of hyaline membranes from diffuse alveolar damage. Within 7 days, a proliferative phase ensues with prominent interstitial inflammation and early fibrotic changes. Approximately 3 weeks after the initial pulmonary injury, most patients recover. However, some patients enter the fibrotic phase, with substantial fibrosis and bullae formation.

Exudative Phase In this phase, alveolar capillary endothelial cells and type I pneumocytes (alveolar epithelial cells) are injured, with consequent loss of the normally tight alveolar barrier to fluid and macromolecules. Edema fluid that is rich in protein accumulates in the interstitial and alveolar spaces ([Fig. 301-2](#)). Proinflammatory cytokines (e.g., interleukin 1, interleukin 8, and tumor necrosis factor α [TNF- α]) and lipid mediators (e.g., leukotriene B₄) are increased in this acute phase, leading to the recruitment of leukocytes (especially neutrophils) into the pulmonary interstitium and alveoli. In addition, condensed plasma proteins aggregate in the air spaces with cellular debris and dysfunctional pulmonary surfactant to form hyaline membrane whorls. Pulmonary vascular injury also occurs early in ARDS, with vascular obliteration by microthrombi and fibrocellular proliferation ([Fig. 301-3](#)).