A healthy 42-year-old farmer lost his balance while riding a tractor, which then ran over his chest and upper abdomen. A computed tomographic scan of the abdomen demonstrated a grade III liver laceration that was treated nonoperatively. However, during the next 12 hours, he developed progressive shortness of breath and required intubation. An arterial blood gas measurement gave the following results: pH, 7.31; PaO₂, 57; and PCO₂, 46, while the patient was receiving a fraction of inspired oxygen (FiO₂) of 80%. Chest x-ray films obtained at admission (A) and after intubation (B) are shown in the Figure.

What Is the Diagnosis?

A. Aspiration pneumonia
B. Cardiogenic pulmonary edema
C. Acute respiratory distress syndrome
D. Bilateral pneumonia
E. Acute lung injury

From the Department of Surgery, University of Rochester Medical Center, Rochester, NY.
Acute Respiratory Distress Syndrome

A, Chest radiograph shows a nondisplaced left clavicular fracture and mild increase in vascular markings. B, Postintubation chest radiograph shows a displaced left clavicular fracture and early bilateral patchy infiltrates.

In 1967, Ashbaugh et al1 first described acute respiratory distress syndrome (ARDS) in 12 patients with acute respiratory distress, cyanosis refractory to O2 therapy, decreased lung compliance, and diffuse infiltrates that were evident on the chest radiograph. Initially termed adult respiratory distress syndrome, it was subsequently renamed because the condition affects patients of any age. In 1988, an expanded definition was proposed that included a 4-point lung injury score based on the extent of chest radiographic abnormalities, the severity of hypoxemia, the degree of lung compliance, and the amount of positive end-expiratory pressure (PEEP).2 In 1994, the American-European Consensus Conference Committee proposed the current definition of ARDS.3 These criteria included an acute onset, bilateral infiltrates evident on chest radiographs, and either a pulmonary capillary wedge pressure of ≤18 mm Hg or the absence of clinical evidence of elevated left atrial pressure.3 This committee also proposed the term acute lung injury to describe those patients who have a “lesser form” of ARDS. Acute lung injury is defined by a PaO2/FiO2 ratio of ≤300 mm Hg, whereas in ARDS, the PaO2/FiO2 ratio has been set as ≤200 mm Hg.

Numerous risk factors for the development of ARDS have been identified. These causes can be subdivided into those associated with direct lung injury and indirect lung injury. Direct causes include aspiration, pneumonia, pulmonary contusion, inhalation injury, fat emboli, and near-drowning. Indirect causes include sepsis, shock, severe extrathoracic trauma, cardiopulmonary bypass, and multiple blood transfusions.4

Often progressive ARDS is characterized by 2 distinct stages.5 The initial acute, or exudative, phase is manifested by a rapid onset of respiratory failure. Arterial hypoxemia that is refractory to O2 therapy is characteristic. Radiographic changes are sometimes delayed, and the patchy or asymmetric infiltrates can be indistinguishable from those of cardiogenic pulmonary edema. Often, these radiographic findings underrepresent the profound degree of arteriopulmonary shunt that may develop in these patients. During the acute phase, there is diffuse alveolar damage with hyaline membrane deposition and hemorrhage, as well as progressive neutrophil and macrophage infiltration.5 Protein-rich fluid leaks into the interstitial spaces and the alveoli. Resultant surfactant abnormalities lead to increased surface tension within the alveoli, causing alveolar disruption and atelectasis.

After the acute lung injury phase of ARDS, some will have an uncomplicated course and resolution.6 In others, the disease progresses to a fibroproliferative stage that can be observed histologically as early as 5 to 7 days after ARDS onset.7 The fibroproliferative stage is characterized by infiltration of the interstitium with fibroblasts and other mesenchymal cells.8 Collagen deposition with decreased lung compliance and increased dead space results.

Mechanical ventilation remains the central supportive intervention in managing ARDS. Historically, a volume of 12 to 15 mL/kg was recommended in patients with ARDS; however, this high tidal volume may cause further injury to alveoli and lead to hyaline membrane formation.9 A recent large ARDS Network trial10 compared traditional mechanical ventilation with ventilation at a lower tidal volume. The subsequent mortality was significantly lower in the group treated with lower tidal volumes (approximately 6 mL/kg) than in the group treated with traditional tidal volumes. Lung recruitment and alveolar stabilization through the use of PEEP are also mainstays of ARDS management.11 Other means of intervention, including surfactant replacement,12 inhalation of nitric oxide,13 glucocorticoid administration,14 and inverse ratio ventilation, have been investigated by various groups, but with conflicting results.

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