detected in 6 patients (2.2%) who had not received positive-pressure ventilation.

Postmortem Findings

Postmortem examination of the lungs was performed on 2 patients. The first patient, a 49-year-old man, had an early case of SARS and did not receive steroids or ribavirin. He died of respiratory failure on day 16 of hospitalization. The salient findings were features of diffuse alveolar damage in both acute and reparative phases, present in about 80% of the lung tissue assessed. The alveolar septa were largely intact, but necrotizing alveolar septa and loss of septa were noted in focal areas. Areas in acute phase of damage showed fibrinoid alveolar exudate with mixed acute and chronic inflammatory cells. Examination also showed hyaline membrane formation. Focal alveolar hemorrhage was present, but alveolar edema was not prominent. In the reparative areas, alveolar spaces were filled with foamy histiocytic cells, occasional multinucleated histiocytes, hyperplastic type 2 pneumocytes, and fibroblasts. Many of these histiocytes and pneumocytes showed vacuolated cytoplasm. Examination identified focal areas with dilated terminal air space and collapsing alveoli suggestive of early honey-comb changes.

The second patient was a 34-year-old woman. She received ribavirin and corticosteroids immediately after admission, and pulse methylprednisolone was administered during the second week after she developed respiratory distress. She had sudden cardiac arrest and died on day 15 of hospitalization. Gross examination revealed a few occluded pulmonary vessels consistent with minor thromboembolism. Microscopic examination showed patchy involvement of the lungs by diffuse acute-phase alveolar damage, representing about 10% of the lung tissue assessed; other areas of lung tissue were well aerated and relatively normal. Reparative changes in the areas of diffuse alveolar damage were minimal. Examination confirmed minor pulmonary thromboembolism, which had contributed to her death.

In both patients, results of RT-PCR performed on lung tissue were positive for SARS coronavirus.

Hospital Courses and Outcomes

Only 1 patient in our cohort did not receive ribavirin and corticosteroid therapy. During hospitalization, diarrhea became increasingly prevalent, affecting up to 53% of patients. None of the patients with diarrhea had positive bacteriologic culture or detection of *Clostridium difficile* toxin in a stool sample. Serial monitoring of laboratory measures revealed increases in the incidence of anemia, from 16% to 53%. Incidence of lymphopenia also increased from 73% to 97%. Ninety percent of patients had hypokalemia, and 56% of patients developed elevated alanine aminotransferase levels during hospitalization.

Sixty-nine patients (26%) required intensive care because of the respiratory failure (defined as partial pressure of oxygen, measured in arterial blood samples, <8 kPa); 57 (21%) required mechanical ventilation. Other complications were subcutaneous emphysema, pneumothorax, and pneumomediastinum in 6 patients (2%); acute renal failure (defined as an abrupt decline in parenchymal renal function in a matter of hours or days) in 15 patients (6%); and acute liver failure (defined as rapid development of severe acute liver injury with impaired synthetic function and encephalopathy in a person who had a normal liver or previously well-compensated liver disease) in 2 patients (1%).

As of this writing, 234 patients have been discharged home. Only 1 patient was still hospitalized for convalescent care. Thirty-two patients died, and the following complications contributed to mortality: respiratory failure (100% of the patients who died), acute renal failure (44%), nosocomial sepsis in the form of focal infections (19%) or septicemia (25%), bilateral pneumothoraces in 1 patient (3%) undergoing mechanical ventilation, and acute liver failure in 2 patients (6%) (1 of the patients with liver failure had cirrhosis due to chronic hepatitis B; the other had septic shock leading to ischemic hepatitis). We followed all survivors for at least 3 months after their first day of hospitalization. The 3-month mortality rate, calculated by the Kaplan–Meier method, was 12% (95% CI, 8% to 16%). Figure 1 shows the survival curve.

Predictors of Mortality

Table 4 summarizes results of univariate analysis. We evaluated the effect of the following measures on mortality by using univariate and multivariate Cox regression: age older than 60 years, neutrophil count greater than $10 \times 10^9$ cells/L at presentation, and lactate dehydrogenase level greater than 3.8 $\mu$kat/L at presentation. In the univariate Cox regression model, risk for death was increased 4-fold in patients older than age 60 years (relative risk, 4.00 [Cl, 1.85 to 8.64]; $P < 0.001$). Lactate dehydrogenase levels greater than 3.8 $\mu$kat/L were also associated