Evolution of pulmonary pathology in severe acute respiratory syndrome

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摘要

Abstract

Background and Purpose: Severe acute respiratory syndrome (SARS) is characterized by fever with rapid progression to acute respiratory distress and it is associated with substantial morbidity and mortality. Transmission patterns suggest spread by respiratory droplet or close person-to-person contact. To elucidate the correlation of clinical presentation to the pathogenesis and course of the disease, we reviewed the pulmonary pathological specimens of SARS patients taken at different stages of the disease. Methods: Four "probable" cases of SARS were studied. SARS-associated coronavirus (SARS-CoV) infection was demonstrated using reverse transcriptase-polymerase chain reaction in all four patients. Patient 1 was a 73-year-old man who was diagnosed at National Taiwan University Hospital and hospitalized at Taipei Municipal Hoping Hospital. Patient 2 was a 36-year-old woman who worked as a caretaker in Taipei Jen Chi Hospital. Patient 3 was a 55-year-old businessman who travelled from Guangdong Province, China via Hong Kong to Taiwan. Patient 4 was a 56-year-old man with an elder brother, a resident of Amoy Gardens Apartments in Hong Kong, who paid him a visit in Taiwan. The pulmonary specimens were taken on day 7 after symptom onset in patient 1, day 11 in patient 2, day 17 in patient 3, and day 21 in patient 4. Results: The autopsy lung tissue from patient 1, who died 7 days after symptoms onset due to the complication of acute myocardial infarction, revealed mild histological change in the lung. Only focal pulmonary oedema or haemorrhage was seen. In the 2nd patient, who died 11 days after symptom onset, severe acute alveolar damage was characterized by patchy or diffuse lung oedema, hyaline membrane formation, and scarce lymphocytic infiltration. The lymphocytes were mostly CD3-positive and CD20-negative. In the 3rd patient, biopsy specimen taken 17 days after symptom onset showed patches of organizing pneumonia with reactive fibroblastic proliferation, more abundant type II pneumocytes and clustering of CD68-positive macrophages within the alveolar spaces. Few CD68-positive syncytial multinucleated giant

cells were also seen in the specimens but no viral inclusion body could be identified in these cells. The lung biopsy specimen from patient 4 taken 21 days after symptom onset showed characteristics of the fibrotic stage, with significant myofibroblastic proliferation in the alveolar space and interstitium resulting in loss of pulmonary architecture. The number of CD3-positive lymphocytes and of CD68-positive macrophages in this specimen from a patient in the fibrotic phase of diffuse alveolar damage (DAD) whose condition deteriorated was less than in the specimen from case 3 who was in the early proliferative phase of DAD and eventually recovered. Conclusions: The histological evolution of SARS coincides with the different stages of DAD: acute, proliferative organizing, and fibrotic stages. SARS cannot be differentiated from the other aetiologies of DAD by morphological examination alone. The absence of DAD does not rule out the possibility of SARS-CoV infection, particularly in the early stage of the disease.