Analysis of Characteristics in Death Patients with COVID-19 Pneumonia without Underlying Diseases

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Dear Editor,

Since December 2019, an outbreak of a novel coronavirus disease (COVID-19) in Wuhan, China has gained much attention (1−3). The World Health Organization (WHO) declared it a pandemic on March 11, 2020 and by March 21 the WHO's website announced 292,142 confirmed COVID-19 cases and 12,784 deaths world-wide (4). Previous studies have focused on the underlying diseases (hypertension, diabetes, and cardiovascular diseases) of the patients who have died (1,5). Surprisingly, little attention has been paid to patients without underlying diseases who have died.

Given the critical role of computed tomography (CT) in the diagnosis and monitoring of therapy for this global health emergency (6), a baseline characterization of COVID-19 cases is urgently needed in order to achieve a more accurate diagnosis. We retrospectively compared characteristics of representative patients who died without any underlying diseases. Our findings could provide understanding of chest morphometry of COVID-19 pneumonia and a helpful path toward improving of clinical strategies against the disease.

We describe two patients with no underlying disease from the Tongji Hospital in Wuhan, China. Real-time reverse transcription-polymerase chain reaction confirmed the infections of COVID-19, and both patients underwent two consecutive CT scans 5 days apart.

A 67-year-old man was admitted to a fever clinic with a low-grade fever for 3 days. Chest CT revealed multifocal ground glass opacities (GGOs) in both lungs. The diameter of the largest GGO measured 3.5 cm. The follow-up CT 5 days later showed progressive GGOs in right upper lobe, and new lesions in left upper lobe, with consolidation and fibrosis in both lungs. The diameter of the largest lesion was 6.7 cm and 46% of lung was involved. Five days later, his procalcitonin level was 0.13 ng/ml and his lactate dehydrogenase level was 1202 U/L. In another 5 days, his symptoms suddenly worsened, and his blood oxygen saturation decreased to 45%. Despite chest compression and adrenaline injection, he died.

A 62-year-old man felt fatigue and received a chest CT scan which showed multiple consolidative nodules and a GGO nodule with consolidation in the left lower lobe. These nodules were all less than 1 cm in diameter. A 5-day follow-up CT showed disease progression with multiple new patchy GGOs in the peripheral zones of all five lobes. The diameter of the largest lesion was 6.7 cm. Approximately 4.3% of his lung volume was involved. One week later, he was admitted to the hospital for continued fatigue, fever, and hemoptysis. His blood oxygen saturation was 62%, and he received bedside endotracheal intubation. His condition quickly worsened with a procalcitonin level of 0.59 ng/ml, plasma cytokines IL6 of 340.40 pg/ml, and lactate dehydrogenase of 1196 U/L. He received extracorporeal membrane oxygenation therapy but died 4 days later.

The inflammatory storm and its associated respiratory failure might synergistically contribute to the cause of death. Extensive GGOs and pulmonary consolidation on chest CT suggested acute respiratory distress syndrome and massive lung infections with alveolar damage. In the second patient, the CT scan and laboratory studies initially showed evidence of only mild disease. However, the disease developed rapidly. Excessive immune responses led to severe pulmonary damage, potential multiple organ dysfunction syndrome, and eventual death. Based on these observations, if the chest CT suggests acute respiratory distress syndrome, the prognosis is guarded. In contrast, if the chest CT shows only minor pulmonary inflammation, laboratory results (eg, proinflammatory cytokines) should be carefully monitored.

Sincerely,

FUNDING

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