

Lung Histopathology: COVID-19 Compared to SARS, H1N1



The coronavirus disease has spread throughout the world and has resulted in many challenges for healthcare systems. It is a new disease with a significant burden of respiratory failure. This has led to questions about the pathogenesis and pulmonary pathology of COVID-19.

Patients with severe COVID-19 have respiratory failure with hypoxaemia, and acute bilateral pulmonary infiltrates. This is consistent with ARDS, which is the most severe form of acute lung injury. Acute lung injury is associated with diffuse alveolar damage (DAD), acute fibrinous and organising pneumonia (AFOP) and organising pneumonia (OP). Based on these clinical characteristics, it has been suggested that the coronavirus disease represents a novel pathologic entity.

Diffuse alveolar damage (DAD) is the traditional histopathologic correlate of ARDS. In a recent review by Polak and colleagues, histopathologic patterns consistent with ARDS were observed in patients with COVID-19. However, whether these patterns are unique to COVID-19 compared to other viral causes of ARDS still remains unknown.

In this systematic review, the researchers evaluate how the lung histopathology described in COVID-19 compares to the lung histopathology in SARS and H1N1 influenza. The review included case files of 171 COVID-19 patients, 287 H1N1 patients and 64 SARS patients from nine countries, including the U.S., Italy, Germany, Switzerland, China, Austria, Brazil, France, and Japan.

We are all well aware of the COVID-19 pandemic and its impact on the world. The H1NI influenza epidemic started in the spring of 2009 and resulted in 201,200 respiratory deaths within one year. SARS is a viral respiratory illness caused by SARS-CoV coronavirus. It spread across two dozen countries in 2003 and resulted in 8422 cases of infections and 916 deaths.

As per the findings of this review, diffuse alveolar damage (DAD) was reported in 88% of patients with COVID-19. This is similar to both H1N1 (90%) and SARS (98%). Pulmonary microthrombi was reported in 57% of patients with COVID-19. This was similar to SARS (58%) and also observed in H1N1 (24%). Organising fibrosis was observed in 52% of COVID-19 patients and was reported in 40% of H1N1 and 47% of SARS cases. 57% of COVID patients reported microthrombotic disease, and this was more similar to SARS with 58% and less similar to HIN1 with 24% of cases. Acute neutrophilic pneumonia was reported in 32% of COVID-19 patients, 30% of H1N1 patients and 31% of SARS patients. AFOP was observed in 4% COVID-19 patients and was a rare finding in H1N1 and SARS.

These findings suggest that diffuse alveolar damage is a predominant histopathologic pattern that is identified in lung pathology from COVID-19, H1N1 and SARS patients. However, research in this area must continue as future studies are critical to characterise the scope of lung pathology across the spectrum of COVID-19 severity and to determine which interventions could help prevent respiratory failure in COVID-19 patients.

Source: Chest

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Published on : Fri, 9 Oct 2020

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