Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Fibroblast function in COVID-19

We read interesting short communication entitled COVID-19: Brief check through the pathologist's eye (autopsy archive) [1].

The deaths from severe acute respiratory syndrome coronavirus, also called COVID-19, are increasing as the number of cases increase. Diffuse alveolar damage seems to be major cause of death in COVID-19 infection.

At the core of tissue remodeling in COVID19 infected alveolar tissue may be fibroblasts. Fibroblasts in the lung interstitium are the common cells, producing extracellular matrix and active during the injury [2].

Normally, the fibroblast can be activated by interaction of alveolar epithelial cells, endothelial cells and inflammatory mediators [3]. Several viruses have been reported to infect fibroblasts [4]. ACE2, which is a binding receptor of the COVID-19 is expressed in fetal human lung fibroblasts [4]. In COVID-19, alveolar sac involvement may be patient specific and alveolar fibroblasts may have higher ACE2 receptor expression in some patients and virus may also infect the fibroblasts. This infection may cause proliferation of fibroblasts and extracellular matrix over production, with local adipogenesis may cause tissue expansion and edema. Also hyalin and collagen may absorb water and form thick hyalin membrane with cell debris. Fibroblasts produce cytokines and cause T cell infiltration which may increase the inflammation. Also thrombus may increase proliferation and function of fibroblasts. This thick wall cause oxygenization defect in severe cases [5].

Secondly; like in HIV, COVID-19 may hijack fibroblasts to cause viremia. Fibroblasts may transinfect the immune cells and make immune cells more prone to virus [6]. So viremia may occur in these patients.

After infection fibroblast function may be defective and activation of urokinase type

plasminogen activator by fibroblast may be disrupted [7]. Also Infected fibroblasts may not able do downregulate plasminogen inhibitör [8]. So infected fibroblasts may be in part responsible from increased risk of thrombus in COVID-19 patients.

Fibroblast dysfunction may also cause spagetti like messy unhealthy collagen accumulation which may cause virus entrance to deep tisue layers.

Viral hormones may affect diseases [9]. The viral insulin/insulin-like growth factor–I receptor (IGF-IR)-like peptides may have role on the pathogenesis of lung damage also by binding to IGF-1 receptors on fibroblasts.

Drugs that decrease the expression of ACE2, IGF-1 receptor inhibition and treatment with hyaluronidase may decrease the mortality in severe COVID-19 patients.

Declaration of Competing Interest

The authors report no declarations of interest.

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