

12 Autopsy Cases Reveal TRUTH About How Patients Die From Coronavirus

This is the link to the main study in this video: <https://www.acpjournals.org/doi/10.73...>

12 Autopsy Cases Reveal TRUTH About How Patients Die From Coronavirus | COVID-19 [#coronavirus](#) [#covid19](#) [#covid_19](#)

Coronavirus | COVID-19 YouTube Video Playlist: https://www.youtube.com/playlist?list=PLggCliyXQhezro4JBt2zJDWo7XdCTn_45

The vitamin D that I take: <https://amzn.to/36u3F0R>

In all 12 cases, the cause of death was found within the lungs or the pulmonary vascular system. For the ones who did not die of large pulmonary emboli, they died of the extensive inflammation within the lungs, meaning pneumonia with ARDS. In these cases, the lungs were wet and heavy, much like a sponge that is saturated with water. The surfaces of the lung often had a distinct patchy pattern, with pale areas alternating with slightly protruding and firm, deep reddish-blue hypercapillarized areas. This is indicative of areas of intense inflammation, with endothelial dysfunction that can be seen at the microscopic level. When they look at slices of the lungs under the microscope, they found diffuse alveolar damage in 8 cases. Specifically, they saw hyaline membrane formation, and tiny clots in the capillaries, and capillaries that were engorged with red blood cells, and other inflammatory findings. All these findings represent ARDS. They also found lymphocytes, a type of white blood cell, infiltrated these areas of infiltration. This fits the picture of viral pathogenesis.

They also looked at the pharynx of these patients, meaning in their throat. The lining of the throat, or mucosa, was hyperemic, meaning very red and irritated, and at the microscopic level, they saw lymphocytes invading there, which is consistent with a viral infection. In one case, a patient had lymphocytes invade his heart muscle, findings that are consistent with what we call viral myocarditis. More than half of the patients in this study had large blood clots. One-third of the patients had pulmonary embolism as the direct cause of death. All the others died of intense inflammation in their lungs related to pneumonia with ARDS (Acute Respiratory Distress Syndrome). Recently there's been studies showing that about 1/3rd of patients with severe COVID have blood clots. Another study of 191 patients with coronavirus aka COVID-19, half of those who died had clots, compared with 7% of survivors. And levels of D-dimer that were greater than 1000 µg/L were associated with a fatal outcome. So it's pretty clear now that the SARS-CoV-2 virus is causing a lot of clots to form in moderate to severe COVID disease.

How is this happening? It's likely a combination of reasons, that has to do with downregulation of the ACE2 receptor in the lung alveoli, with a subsequent shift towards having more angiotensin II in the lungs, and less angiotensin 1-7 and 1-9 in the lungs, and when this happens, this leads to more cytokine storm with more inflammation, more constriction of pulmonary arteries, and more clots that develop. That, in turn, leads to more endothelial dysfunction in the capillaries that surround the alveoli. Also, there is evidence that the virus attaches to the ACE2 receptors of those endothelial cells that line those capillaries, which further propagates inflammation and clotting. And in the cytokine storm that develops there, RANTES, a chemokine, binds to the CCR5 receptor of CD4 and CD8 lymphocytes, and that causes those lymphocytes to infiltrate those areas of inflammation, and in doing so, further contributes towards the inflammatory reaction. This is why we are seeing low levels of CD4 and CD8 lymphocytes in severe COVID. Endothelial damage can also lead to the development of antiphospholipid antibodies, and these antibodies are bad because they trigger the formation of blood clots. That's why patients who have clots with the diagnosis of antiphospholipid antibody syndrome need to be on blood thinners.

Also, 11 out of the 12 patients in this study had underlying heart disease and were obese. These are known risk factors not just for cardiovascular disease, but also known risk factors for endothelial dysfunction, and are known risk factors for COVID. So the big takeaways from the findings in this study are that most people who die of COVID, it's primarily a lung problem. Either related to inflammation with ARDS and/or blood clots.

Antiphospholipid syndrome might be a commonality among patients with thrombosis in COVID-19 patients.

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