# COVID-19 lung damage caused by persistence of 'abnormal cells'

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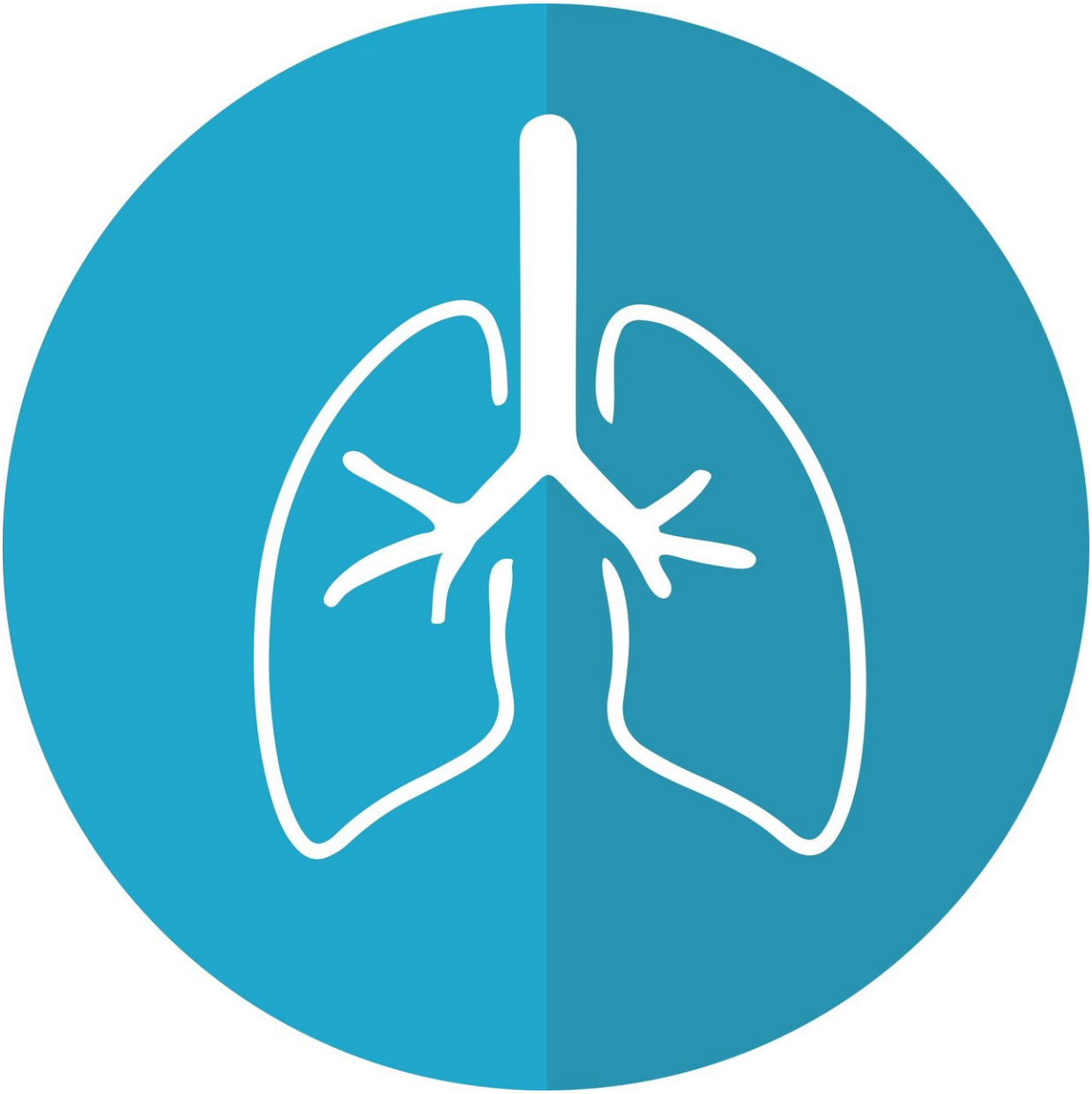

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Investigations of deceased COVID-19 patients have shed light on possible lung damage caused by the virus.

The study, published today in The Lancet's *eBioMedicine*, by King's College London in collaboration with University of Trieste and the International Centre for Genetic Engineering and Biology in Italy, shows the unique characteristics to the SARS-CoV-2 virus and may explain why patients suffer from 'long COVID'.

Patients with COVID-19 can experience symptoms such as blood clotting and loss of smell and taste. Some who survive the infection can experience the effects of the disease for months—known as 'long COVID' - with a feeling of fatigue and lack of breath. There have been a limited number of studies that have analyzed the organs of COVID-19 patients which means the characteristics of the disease are still largely unknown.

Researchers analyzed the organs of 41 patients who died of COVID-19 at the University Hospital of Trieste, Italy, from February to April 2020, at the start of the pandemic. The team took lung, heart, liver, and kidney samples to examine the behavior of the virus.

Findings show extensive lung damage in most cases, with patients experiencing profound disruption of the normal lung structure and the transformation of respiratory tissue into fibrotic material.

Almost 90% of patients showed two additional characteristics that were quite unique to COVID-19 compared to other forms of pneumonia. First, patients showed extensive blood clotting of the lung arteries and

veins (thrombosis). Second, several lung cells were abnormally large and had many nuclei, resulting from the fusion of different cells into single large cells. This formation of fused cells (syncytia) is due to the viral spike protein, which the virus uses to enter the cell. When the protein is present on the surface of cells infected by the COVID-19 virus, it stimulates their fusion with other normal lung cells, which can be a cause for inflammation and thrombosis.

Additionally, research showed the long-term persistence of the viral genome in respiratory cells and in cells lining the blood vessels, along with the infected cell syncytia. The presence of these infected cells can cause the major structural changes observed in lungs, which can persist for several weeks or months and could eventually explain 'long COVID'.

The study found no overt signs of viral infection or prolonged inflammation detected in other organs.

Professor Mauro Giacca, at the British Heart Foundation Centre at King's College London, said: "These findings are very exciting. The findings indicate that COVID-19 is not simply a disease caused by the death of virus-infected cells but is likely the consequence of these abnormal cells persisting for long periods inside the lungs."

The team is now actively testing the effect of these abnormal cells on blood clotting and inflammation and are searching for new drugs that can block the viral spike protein which causes cells to fuse.

Provided by King's College London