Study finds that SARS-CoV-2 can infect the arteries of the heart

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A recently published study demonstrated that SARS-CoV-2, the virus that causes COVID-19, can infect the coronary arteries that supply blood to the heart. These arteries are the ones involved in a typical heart attack, where blockage of the arteries results in oxygen depletion and consequent death of a segment of heart muscle.

The fact that the coronavirus can directly damage these arteries and induce the formation of so-called "plaques" is a surprise. Most theories about how SARS-CoV-2 results in the increase in heart attacks and other coronary events seen thus far in the pandemic have had to do with either inflammation or hypercoagulability. In both cases, it was presumed that there was an increased predisposition to forming clots that block the arteries.

The result is not entirely without precedent. Previously, a handful of viruses were associated with the development of atherosclerosis (plaques in the arteries), including hepatitis viruses, herpes simplex viruses, human immunodeficiency virus, human papillomavirus, human cytomegalovirus, and influenza virus. And of those, only herpes simplex viruses, hepatitis C virus, and human cytomegalovirus had evidence of directly infecting the cells in the walls of the coronary arteries.

Despite the pre-existing precedent, the result is still shocking. SARS-CoV-2 is now the only respiratory virus known to directly infect blood vessels. Influenza viruses have not been shown to do so. Their known and hypothesized effects on inducing atherosclerosis are indirect. Furthermore, the study showed that SARS-CoV-2 infection was a direct cause of both formation and growth of plaques.

The study examined the coronary arteries of eight patients who had died after testing positive for COVID-19. They found evidence of SARS-CoV-2 viral replication in the tissues making up the arteries in all eight patients. They localized the infection to the arterial wall, finding lower amounts of viral material in the fat tissue surrounding the arteries.

They further localized the virus to macrophages, a white blood cell that fights infections in virtually every tissue of the body. They found that muscle cells of the arterial walls were infected to a far lesser extent than macrophages. By elucidating the differential effects among cell types, the researchers also confirmed that the findings were not incidental.

The researchers then infected both normal macrophages as well as macrophages that had accumulated large quantities of fat or lipids inside their cell wall. These latter lipid-laden cells are called "foam cells" because of their appearance under a microscope. The researchers found that both kinds of macrophages had a high degree of viral replication, but foam cells had more rapid viral replication than normal macrophages. Furthermore, normal macrophages cleared the virus faster than foam cells. Notably, foam cells are the hallmark of all atherosclerosis, regardless of cause and stage of development.

The researchers then verified that SARS-CoV-2 infected previously uninfected tissue by introducing the virus to arterial specimens—called "explants"—taken from unrelated patients who had no history of COVID-19. They found that the virus also infected these cells and replicated in similar quantities, confirming the original findings.

The researchers then studied whether SARS-CoV-2 infection of the coronary arteries induced inflammation. They found that it did, and that the inflammation was characteristic of atherosclerosis both in the original patients and in the explants. This induced-inflammatory response also possibly explains the increase in heart

attacks seen during the COVID-19 pandemic.

The researchers then went on to identify the key mechanism of viral binding and entry into macrophages and foam cells. They found that the virus infects these cells through the neuropilin 1 (NRP-1) receptor, and not the ACE-2 receptor that is a common viral entry point in other tissue types. Nevertheless, NRP-1 was a well-known viral entry point prior to this study. They confirmed this finding by adding a compound that inhibits NRP-1 binding, which resulted in significantly reduced viral replication in both foam cells and normal macrophages.

The study adds to the already large body of evidence that COVID-19 is nothing like “the flu.” It is far worse, with considerably greater morbidity and mortality. Patients who survive COVID-19 infection have a greater risk of cardiovascular events for at least one year after infection, regardless of whether they have any pre-existing conditions that increase risk. This new study provides at least one significant mechanism by which this risk is conferred and sustained over time.

The ruling class policy of letting a novel virus infect and reinfect billions of people, with total indifference to the high potential of serious sequelae such as coronary artery infections, is further exposed as a vast social crime.