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Healthy Choices for Mind and Body - Newsletter

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Putting medical and nutrition news in historical, scientific, and just plain practical context.

Editor's Note: I keep promising an article about vaccines but it's tough to write about a subject in the middle of its discovery. I've spent a lot of time bolstering my knowledge so that I'll be able to do a multi-article series about vaccines soon.

COVID-19 Pandemic Disease Panorama

by Ann Gerhardt, MD August 5, 2020

Science evolves.
We don't change facts, we learn more of them. That sometimes begets new conclusions.

Pneumonias that decimated Chinese patients and the fact that the virus generally infects via inhalation led to the assumption that SARS-CoV-2 was a respiratory virus. For that reason, we screen people for cough, fever and shortness of breath. Then infected people showed up with diarrhea, virus in their stool and a slew of other symptoms. We now know that only 77% of 393 New York COVID-19 patients sick enough to be hospitalized initially had fever and/or cough. Many had diarrhea, muscle aches, loss of immune blood cells, abnormal liver tests, kidney failure, cardiac inflammation and abnormal heart rhythm.

When scientists discovered early on that SARS-CoV-2 attaches to cells' surface angiotensin converting enzyme (ACE2), we should have known that it would infect many other tissues besides lung, ACE2 is part of a regulatory system for blood pressure, wound healing and inflammation. Cells of lung, heart, kidney, liver, and gastrointestinal tract tissue, as well as cells lining the inside of the nose and blood vessels, all bear ACE2.

SARS-CoV-2 attachment to blood vessel lining ACE2 is scary, since a profusion of blood vessels supplies most body parts. This explains patients appearing with a great of variety of problems, ranging from loss of smell to sudden death. Infected blood vessel cells can lead to inflammation and clotting which clogs blood supply to the tissue supplied by those vessels.

Some people with COVID-19 become hypercoagulable, meaning they clot very easily. Some start to mimic lupus patients by making an antibody that predisposes to clots. Reports of clot in the lung (pulmonary embolism) and veins (venous thrombosis) are trickling into the literature. Between ACE2 being present on blood vessels and coronavirus causing clotting defects, the man whose primary symptom was a stroke (caused by a blocked artery) shouldn't have been a surprise.

Obesity, especially severe obesity, poses the second most significant risk for severe COVID-19 disease and death, behind advanced age and ahead of the increased risk in males. People speculate that the underlying inflammation and reduced lung capacity common in obesity contribute to worse disease, but we don't know for sure if they are the link to worse disease.

People in China, Italy and the U.S. have had different symptoms and patterns of illness. As an RNA virus, SARS-CoV-2 mutates, giving rise to slightly different strains that might explain those differences. The exceptionally severe COVID-19 inflammation seen in New York children may have been caused by a different strain. Serial mutations could continue to generate new illness and transmission patterns over time. This bodes poorly for vaccine development, since we'll want a vaccine that works for all SARS-CoV-2 viruses. The only good news is that this virus is large enough to have a gene that repairs mutations, so most of the changes are fixed before they become permanent.

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