Coronavirus: Heart damage emerges as yet another grim outcome in possible complications

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HMS physician-scientists Paul Ridker, left, and colleague Peter Libby have studied the interplay between the immune and cardiovascular systems for years. Credit: Harvard Medical School

Lung injury and acute respiratory distress syndrome have taken center stage as the most dreaded complications of COVID-19, the disease caused by the new coronavirus, SARS-CoV-2. But heart damage has recently emerged as yet another grim outcome in the virus's repertoire of

possible complications.

COVID-19 is a spectrum disease, spanning the gamut from barely symptomatic infection to critical illness. Reassuringly, for the large majority of individuals infected with the new coronavirus, the ailment remains in the mild-to-moderate range.

Yet, a number of those infected develop heart-related problems either out of the blue or as a complication of preexisting cardiac disease. A report from the early days of the epidemic described the extent of cardiac injury among 41 patients hospitalized with COVID-19 in Wuhan, China: Five, or 12 percent, had signs of cardiovascular damage. These patients had both elevated levels of cardiac troponin—a protein released in the blood by the injured heart muscle—and abnormalities on electrocardiograms and heart ultrasounds. Since then, other reports have affirmed that cardiac injury can be part of coronavirus-induced harm. Moreover, some reports detail clinical scenarios in which patients' initial symptoms were cardiovascular rather than respiratory in nature.

How does the new coronavirus stoke cardiac damage?

The ways in which the new coronavirus provokes cardiac injury are neither that new nor surprising, according to Harvard Medical School physician-scientists Peter Libby and Paul Ridker. The part that remains unclear is whether SARS-CoV-2 is somehow more virulent toward the heart than other viruses.

Libby and Ridker, who are practicing cardiologists at Brigham and Women's, say COVID-19-related heart injury could occur in any several ways.

First, people with preexisting heart disease are at a greater risk for severe cardiovascular and respiratory complications from COVID-19.

This is hardly a surprise. Research has shown that infection with the influenza virus poses a more severe threat for people with heart disease than those without cardiac problems. Research also shows that heart attacks can actually be brought on by respiratory infections such as the flu.

Second, people with previously undiagnosed heart disease may be presenting with previously silent cardiac symptoms unmasked by the viral infection. In people with existing heart-vessel blockages, infection, fever and inflammation can destabilize previously asymptomatic fatty plaques inside the heart vessels. Fever and inflammation also render the blood more prone to clotting, while also interfering with the body's ability to dissolve clots—a one-two punch akin to throwing gasoline on smoldering embers.

"It's like one big stress test for the heart," said Ridker, who is the Eugene Braunwald Professor of Medicine at Brigham and Women's Hospital.

Third, some people may experience heart damage that mimics heart attack injury even if their arteries lack the fatty, calcified flow-limiting blockages known to cause classic heart attacks. This scenario, called myocardial infarction type 2, can occur when the heart muscle is starved for oxygen, which in the case of COVID-19 may be triggered by a mismatch between oxygen supply and oxygen demand. Fever and inflammation accelerate heart rate and increase metabolic demands on many organs, including the heart. That stress is compounded if the lungs are infected and incapable of exchanging oxygen and carbon dioxide optimally. This impaired gas exchange can further diminish oxygen supply to the heart muscle.

Finally, there is a subset of people with COVID-19—some of them previously healthy and with no underlying cardiac problems—who develop fulminant inflammation of the heart muscle as a result of the

virus directly infecting the heart. This type of inflammation could lead to heart rhythm disturbances and cardiac muscle damage as well as interfere with the heart's ability to pump blood optimally.

The propensity of certain viruses to attack the heart muscle and cause viral myocarditis is well known, Libby said, adding that the most notorious viral offender has been the Coxsackie B virus. Nonetheless, a recent case report from Italy underscores the notion that the new coronavirus could also infect the heart and affect heart muscle function in healthy adults even after the acute phase of the infection has resolved and even in the absence of lung damage.

"There are definitely some people who develop acute fulminant myocarditis—in which the virus infects the heart muscle itself or the cells within the heart—and causes a horrible inflammatory reaction," said Libby, who is also the Mallinckrodt Professor of Medicine at Brigham and Women's Hospital. "This can be life threatening, and it can happen in people who don't have any preexisting risk factors."

Libby and Ridker, however, say this out-of-the-blue scenario in otherwise healthy individuals is likely rare relative to the overall number of people with COVID-19 who experience heart problems.

The frenemy within

For Ridker and Libby, who have studied the immune pathways of cardiovascular disease for decades, the cardiac involvement in COVID-19 is yet another striking example of the widespread effects of inflammation on multiple organs and systems.

Inflammation is a critical defense response during infection, but it has a dark side. Infections can set off a cascade of immune signals that affect various organs.

Libby and Ridker hypothesize that any infection in the body—a festering boil, an injured joint, a virus—can become a source of inflammation that activates the release of inflammatory proteins known as cytokines and calls up armies of white blood cells and other messenger molecules that, in an effort to fight the infection, disrupt normal processes. When these inflammatory molecules reach the welcoming soil of a fatty deposit in the blood vessel wall—one that is already studded with resident inflammatory white blood cells—the cytokines can boost the local inflammatory response and trigger a heart attack.

"Our work has shown that cytokines can impinge on these cells in the plaque and push it through a round of further activation," Libby said.

The inflammatory chemicals released during infection can also induce the liver to ramp up the production of important proteins that defend the body from infection. These proteins, however, make the blood more prone to clotting, while also reducing the secretion of natural clot-dissolving substances. The tiny clots that may form can clog the small blood vessels in the heart and other organs, such as the kidneys, depriving them of oxygen and nutrients and setting the stage for the multisystem failure that can occur in acute infection.

Thus, immune-mediated injury to the heart and other organs could be collateral damage because of the body's overwhelming systemic immune response—a condition known as cytokine storm, which is marked by the widespread release of cytokines that can cause cellular demise, tissue injury and organ damage.

COVID-19 and blood pressure medications

SARS-CoV-2 invades human cells by latching its spike protein onto the ACE2 receptor found on the surface of cells in the airways, lungs, heart,

kidneys and blood vessels. The ACE2 protein is an important player in the renin-angiotensin-aldosterone system, which regulates blood vessel dilation and blood pressure. Two classes of drugs widely used to treat high blood pressure and heart disease—ACE inhibitors and angiotensin receptor blockers—interact with the ACE2 receptor. A possible concern related to COVID-19 stems from the notion that these blood pressure medications could increase the number of ACE2 receptors expressed on cells, possibly creating more molecular gates for the virus to enter. Some experts have wondered whether the use of such drugs could render people who take them more susceptible to infection. Conversely, others have postulated that the abundance of ACE2 receptors may enhance cardiovascular function, exercising a protective effect during infection.

The answer is far from clear, but a recent review suggests these medicines may play a dual role in COVID-19—on the one hand, enhancing susceptibility to infection and, on the other, protecting the heart and ameliorating lung damage from the disease.

Libby and Ridker cautioned that patients who take such life-saving medications should stay on them or at least have a careful discussion with their cardiologists. This is because these drugs have clear and well-established benefits in hypertension and certain forms of heart disease, while their propensity to make humans more susceptible to SARS-CoV-2 remains speculative for the time being.

But what remains speculative today will crystallize in the weeks and months to come, Ridker and Libby said, because the science is moving forward rapidly, with new papers coming out daily and a growing pool of patients to draw observations from.

"In 12 to 18 months we're going to have a great deal of information, but right now our job is to, number one, keep people from getting COVID-19 by strict adherence to now-familiar containment measures,"

Libby said. "Then, we need to get people who get the disease through this acute phase."

The need for rigorous randomized trials done quickly and effectively is acute, they said. Until the evidence from these trials begins to coalesce, clinicians will have to navigate the uncharted territory of delivering cardiac care in the time of pandemic with caution but also with resolve.

"We don't have the comfort of our usual databases, so we have to rely on our clinical skills and judgment. But we have to do so in all humility because often data don't bear out our logical preconceptions," Libby said. "Yet, we must act."

Provided by Harvard Medical School

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