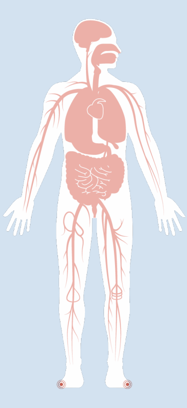

Health

Doctors keep discovering new ways the coronavirus attacks the body

Damage to the kidneys, heart, brain — even 'covid toes' — prompts reassessment of the disease and how to treat it

By Lenny Bernstein and Ariana Eunjung Cha

MAY 10, 2020

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Deborah Coughlin was neither short of breath nor coughing. In those first days after she became infected by the novel coronavirus, her fever never spiked above 100 degrees. It was vomiting and diarrhea that brought her to a Hartford, Conn., emergency room on May 1.

"You would have thought it was a stomach virus," said her daughter, Catherina Coleman. "She was talking and walking and completely coherent."

But even as Coughlin, 67, chatted with her daughters on her cellphone, the oxygen level in her blood dropped so low that most patients would be near death. She is on a ventilator and in critical condition at St. Francis Hospital, one more patient with a strange constellation of symptoms that physicians are racing to recognize, explain and treat.

"At the beginning, we didn't know what we were dealing with," said Valentin Fuster, physician-in-chief at Mount Sinai Hospital in New York City, the epicenter of the U.S. outbreak. "We were seeing patients dying in front of us. It was all of a sudden, you're in a different ballgame, and you don't know why."

[The girl who died twice: Juliet Daly was a healthy 12-year-old until the coronavirus invaded her heart]

Today, there is widespread recognition that the novel coronavirus is far more unpredictable than a simple respiratory virus. Often it attacks the lungs, but it can also strike anywhere from the brain to the toes. Many doctors are focused on treating the inflammatory reactions it triggers and its capacity to cause blood clots, even as they struggle to help patients breathe.

They have little solid research to guide them as they learn on the fly about a new disease with more than 78,000 U.S. deaths attributed to it. The World Health Organization's database already lists more than 14,600 papers on covid-19. Even the world's premier public health agencies, including the Centers for Disease Control and Prevention, have constantly altered their advice to keep pace with new developments.

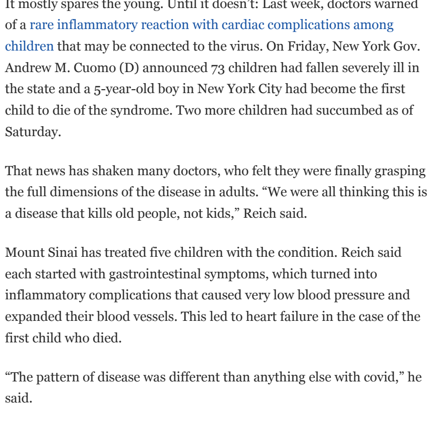
"We don't know why there are so many disease presentations," said Angela Rasmussen, a virologist at the Center for Infection and Immunity at Columbia University's Mailman School of Public Health. "Bottom line, this is just so new that there's a lot we don't know."

More than four months of clinical experience across Asia, Europe and North America has shown the pathogen does much more than invade the lungs. "No one was expecting a disease that would not fit the pattern of pneumonia and respiratory illness," said David Reich, a cardiac anesthesiologist and president of Mount Sinai Hospital in New York City.

It attacks the heart, weakening its muscles and disrupting its critical rhythm. It savages kidneys so badly some hospitals have run short of dialysis equipment. It crawls along the nervous system, destroying taste and smell and occasionally reaching the brain. It creates blood clots that can kill with sudden efficiency and inflames blood vessels throughout the body.

It can begin with a few symptoms or none at all, then days later, squeeze the air out of the lungs without warning. It picks on the elderly, people weakened by previous disease, and, disproportionately, the obese. It harms men more than women, but there are also signs it complicates pregnancies.

Symptoms of covid-19 appear to include:

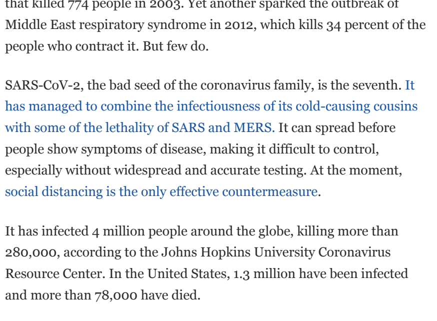


It mostly spares the young. Until it doesn't: Last week, doctors warned of a rare inflammatory reaction with cardiac complications among children that may be connected to the virus. On Friday, New York Gov. Andrew M. Cuomo (D) announced 73 children had fallen severely ill in the state and a 5-year-old boy in New York City had become the first child to die of the syndrome. Two more children had succumbed as of Saturday.

That news has shaken many doctors, who felt they were finally grasping the full dimensions of the disease in adults. "We were all thinking this is a disease that kills old people, not kids," Reich said.

Mount Sinai has treated five children with the condition. Reich said each started with gastrointestinal symptoms, which turned into inflammatory complications that caused very low blood pressure and expanded their blood vessels. This led to heart failure in the case of the first child who died.

"The pattern of disease was different than anything else with covid," he said.



"We were all thinking this is a disease that kills old people, not kids," said David Reich, president of Mount Sinai Hospital in Manhattan. (Jeenah Moon/Reuters)

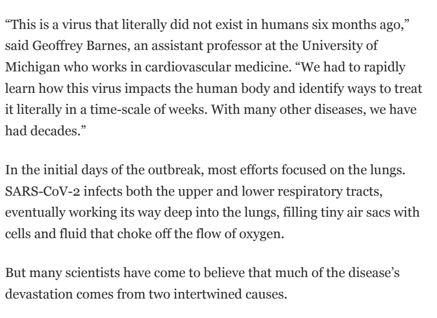
Of the millions, perhaps billions, of coronaviruses, six were previously known to infect humans.

Four cause colds that spread easily each winter, barely noticed. Another was responsible for the outbreak of severe acute respiratory syndrome that killed 774 people in 2003. Yet another sparked the outbreak of Middle East respiratory syndrome in 2012, which kills 34 percent of the people who contract it. But few do.

SARS-CoV-2, the bad seed of the coronavirus family, is the seventh. It has managed to combine the infectiousness of its cold-causing cousins with some of the lethality of SARS and MERS. It can spread before people show symptoms of disease, making it difficult to control, especially without widespread and accurate testing. At the moment, social distancing is the only effective countermeasure.

It has infected 4 million people around the globe, killing more than 280,000, according to the Johns Hopkins University Coronavirus Resource Center. In the United States, 1.3 million have been infected and more than 78,000 have died.

Had SARS or MERS spread as widely as this virus, Rasmussen said, they might have shown the same capacity to attack beyond the lungs. But they were snuffed out quickly, leaving only a small sample of disease and death.



Paramedics bring home a woman with covid-19 who underwent an emergency C-section because she was gravely ill. After extensive care, including time on a ventilator, she was released from a hospital in Stamford, Conn., and she has a healthy newborn. (John Moore/Getty Images)

Trying to define a pathogen in the midst of an ever-spreading epidemic is fraught with difficulties. Experts say it will be years until it is understood how the disease damages organs and how medications, genetics, diets, lifestyles and distancing impact its course.

"This is a virus that literally did not exist in humans six months ago," said Geoffrey Barnes, an assistant professor at the University of Michigan who works in cardiovascular medicine. "We had to rapidly learn how this virus impacts the human body and identify ways to treat it literally in a time-scale of weeks. With many other diseases, we have had decades."

In the initial days of the outbreak, most efforts focused on the lungs. SARS-CoV-2 infects both the upper and lower respiratory tracts, eventually working its way deep into the lungs, filling tiny air sacs with cells and fluid that choke off the flow of oxygen.

But many scientists have come to believe that much of the disease's devastation comes from two intertwined causes.

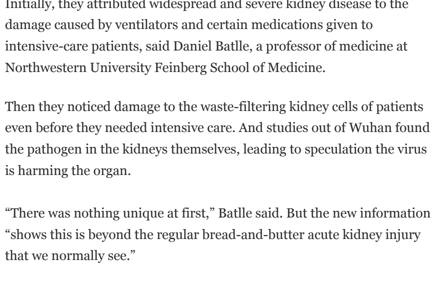
The first is the harm the virus wreaks on blood vessels, leading to clots that can range from microscopic to sizable. Patients have suffered strokes and pulmonary emboli as clots break loose and travel to the brain and lungs. A study in the Lancet, a British medical journal, showed this may be because the virus directly targets the endothelial cells that line blood vessels.

The second is an exaggerated response from the body's own immune system, a storm of killer "cytokines" that attack the body's own cells along with the virus as it seeks to defend the body from an invader.

Research and therapies are focused on these phenomena. Blood thinners are being more widely used in some hospitals. A review of records for 2,733 patients, published Wednesday in the Journal of the American College of Cardiology, indicates they may help the most seriously ill.

"Things change in science all the time. Theories are made and thrown out. Hypotheses are tweaked. It doesn't mean we don't know what we are doing. It means we are learning," said Deepak Bhatt, executive director of interventional cardiology at Brigham and Women's Hospital in Boston.

Inflammation of those endothelial cells lining blood vessels may help explain why the virus harms so many parts of the body, said Mandeep Mehra, a professor of medicine at Harvard Medical School and one of the authors of the Lancet study on how covid-19 attacks blood vessels.



That means defeating covid-19 will require more than antiviral therapy, he said.

"What this virus does is it starts as a viral infection and becomes a more global disturbance to the immune system and blood vessels — and what kills is exactly that," Mehra said. "Our hypothesis is that covid-19 begins as a respiratory virus and kills as a cardiovascular virus."

The thinking of kidney specialists has evolved along similar lines. Initially, they attributed widespread and severe kidney disease to the damage caused by ventilators and certain medications given to intensive-care patients, said Daniel Batlle, a professor of medicine at Northwestern University Feinberg School of Medicine.

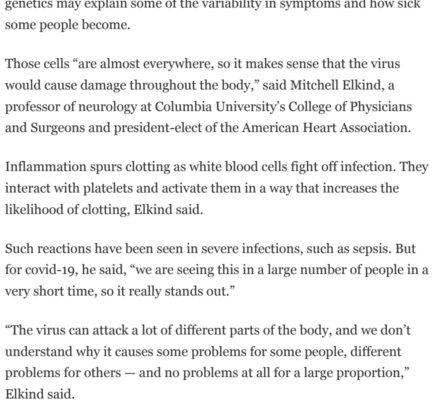
Then they noticed damage to the waste-filtering kidney cells of patients even before they needed intensive care. And studies out of Wuhan found the pathogen in the kidneys themselves, leading to speculation the virus is harming the organ.

"There was nothing unique at first," Batlle said. But the new information "shows this is beyond the regular bread-and-butter acute kidney injury that we normally see."

Like other coronaviruses, SARS-Cov-2 infiltrates the body by attaching to a receptor, ACE2, found on some cells. But the makeup of the spikes that protrude from this virus is somewhat different, allowing the virus to bind more tightly. As a result, fewer virus particles are required to infect the host. This also may help explain why this virus is so much more infectious than SARS, Rasmussen said.

Other factors can't be ruled out in transmission, she said, including the amount of virus people shed and how strictly they observe social distancing rules.

Once inside a cell, the virus replicates, causing chaos. ACE2 receptors, which help regulate blood pressure, are plentiful in the lungs, kidneys and intestines — organs hit hard by the pathogen in many patients. That also may be why high blood pressure has emerged as one of the most common preexisting conditions in people who become severely ill with covid-19.



A colorized scan of a cell (shown in red) infected with SARS-COV-2 virus particles (shown in yellow), isolated from a patient sample. (National Institute of Allergy and Infectious Diseases)

The receptors differ from person to person, leading to speculation that genetics may explain some of the variability in symptoms and how sick some people become.

Those cells "are almost everywhere, so it makes sense that the virus would cause damage throughout the body," said Mitchell Elkind, a professor of neurology at Columbia University's College of Physicians and Surgeons and president-elect of the American Heart Association.

Inflammation spurs clotting as white blood cells fight off infection. They interact with platelets and activate them in a way that increases the likelihood of clotting, Elkind said.

Such reactions have been seen in severe infections, such as sepsis. But for covid-19, he said, "we are seeing this in a large number of people in a very short time, so it really stands out."

"The virus can attack a lot of different parts of the body, and we don't understand why it causes some problems for some people, different problems for others — and no problems at all for a large proportion," Elkind said.

Coughlin, in critical condition at a hospital in Connecticut, deteriorated quickly after she reached the emergency room. Her fever shot up to 105, and pneumonia developed in her lungs.

On Wednesday, she called her six daughters on FaceTime, telling them doctors advised she go on a ventilator.

"If something happens to me, and I don't make it, I'm at peace with it," she told them.

The conversation broke daughter Coleman's heart.

"I am deciding to help her go on a ventilator, and she may never come off," she said. "That could have been my last phone conversation with her."

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Pablo%20AS il y a 3 mois

Isnt all this more than enough proof to realize that this "CCP virus" is completely specially man made to kill humans?? Researches on this "CCP VIRUS" must test thinking it was manmade and not rely on previous information from natural coronavirus. What I have understood from this virus is that it will kill about 20% of the infected when no medical help is received, which is the case when the health system is completely overwhelmed, people will die where they are, many people die in their homes and with the widespread range of symptoms i bet a enormous part of deaths are cataloged with another cause.
Things that a man-made virus need to be extremely efficient.
1-Long incubation period
2-be extremely contagious
3-Made to attack an specific part of the body, in this case ACE2 receptors, which leads the virus to basically attack every part of the body
4-Regular Medicine being completely ineffective
5-Kill only the amount you want to kill (this case about 10-20%). Just to notice that its only about 3-6% when medical help is received, but it goes up to 15-20% when you cannot get medical assistance. So you make it that only those with weaker health or weaker immune system get killed.

And there you have it. Covid-19 is the "Perfect" virus made to cut down population. But leaders all over the world are more worried about their next election, how much money they can get before things get completely out of hand like in my country chile where all politics refused to reduce their "payment" which is over 100 times the minimum wage.

Like 1 Link Signaler

Boogie Knights il y a 3 mois

Am I reading this right? This has to be worded incorrectly. this was in a study published in the Lancet:

> The RCGP surveillance system, set up in 1957, monitors consultations for communicable diseases using a network of 500 general practitioner practices across England, which are broadly representative of the population. Twice-weekly automatic data downloads provide a real-time warning of impending epidemics. In January, 2020, the network expanded to include the testing for SARS-CoV-2 among individuals presenting with symptoms of influenza or respiratory infection. COVID-19 surveillance data, supplemented with data from contact tracing or routine National Health Service facilities, were linked with electronic health records. Of 3802 tests, 587 (15·4%) were positive for SARS-CoV-2. Prevalence of infection was less than 5% in patients younger than 18 years (23 patients were positive [4·6%] of 499 tested) but almost four times as high in people aged 40 years or older (480 [18·2%] of 2637). After adjustment for other factors, infection risk was higher among men than women (odds ratio [OR] 1·55 [95% CI 1·27–1·89]), in black people than white people (OR 4·75 [2·65–8·51]), and in people with obesity than normal-weight people (1·41 [1·04–1·91]). Infection risk was also higher in those living in more deprived or in urban versus rural locations. Surprisingly, household size did not significantly affect infection risk. Among chronic comorbidities examined, only those with chronic kidney disease had an increased risk of infection, whereas the risk in active smokers was around half that observed in never smokers.

Does that really say that active smokers have half the risk of becoming infected as people who have never smoked? Huh??

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Boogie Knights il y a 3 mois

https://www.thelancet.com/journals/laninf/article/PIIS1473-3099(20)30395-9/fulltext

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MarcusAlwaysVotes il y a 3 mois

The key phrase is "after adjustment for other factors." They adjust for respiratory disease, which eliminates a lot of the smokers, since many of them have respiratory ailments. In general, smokers have a higher risk, according to one expert commenting on this paper on TV.