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Commentary

Antibodies Targeting Viruses during Mild Cases of COVID-19

Conger Patrick*

Department of Pediatrics, Ludwig Maximilian University of Munich, Munich, Germany

DESCRIPTION

Antibodies are proteins generated by the body's immune system to fight disease. Antibodies to SARS-CoV-2, the virus that causes COVID-19, can be detected in the blood of people who have recovered from COVID-19 or people who have been vaccinated against COVID-19. Vaccination against COVID-19 increases their body's antibody response, which improves their protection.

COVID-19 antibodies preferentially target a different part of the virus in mild cases of COVID-19 than they do in severe cases, and wane significantly within several months of infection, according to a new study by researchers at Stanford Medicine.

The findings reveal novel connections between the progression of the disease and the immunological response of the patient. They also raise questions about whether people can re-infect themselves, if antibody testing to detect earlier infection may underestimate the pandemic's scope, and whether vaccinations should be repeated at regular intervals to maintain a protective immune response.

This is one of the most extensive studies of the antibody immune response to SARS-CoV-2 in persons with disease severity ranging from asymptomatic to deadly and looked at a variety of points and sample types, as well as the quantities of viral RNA in nasopharyngeal swabs and blood samples from patients.

When compared to the number of antibodies targeting proteins of the virus's inner shell, patients with severe COVID-19 have a low proportion of antibodies targeting the spike protein used by the virus to enter human cells.

Virus binds to ACE2 receptor

The researchers looked at 254 persons with asymptomatic, mild, or severe COVID-19 who were diagnosed with signs of COVID-19 by routine testing or occupational health screening at Stanford Health Care. 25 people with symptoms were treated as outpatients, 42 were admitted to the hospital outside the intensive care unit, and 37 were admitted to the intensive care unit. The sickness claimed the lives of twenty-five people in the research. SARS-CoV-2 attaches to human cells by a spike protein structure on its surface. This protein interacts to the ACE2 receptor on human cells. The virus is able to enter and infect the cell because of the binding. The virus sheds it's outer coat once enter inside, revealing an inner shell that houses its genetic information.

The virus quickly takes over the cell's protein-making machinery and uses it to produce new viral particles, which are subsequently discharged to infect neighbouring cells.

Antibodies that recognise and bind to the spike protein prevent the virus from infecting cells by blocking its capacity to bind to ACE2, whereas antibodies that recognise other viral components are unlikely to stop viral propagation. Parts of the spike protein are used in current vaccine candidates to trigger an immunological response. As the sickness proceeded and individuals either healed or became sicker, researchers looked at three types of antibodies: IgG, IgM, and IgA, and the proportions of each that targeted the viral spike protein or the virus's inner shell. And also looked at how much viral genetic material was present in the patients' nasopharyngeal swabs and blood. Finally, they tested the antibodies' ability to stop the spike protein from binding to ACE2 in a lab dish. Although earlier research evaluated the viral proteins targeted by these antibodies, and analyzed the entire antibody response after infection.

The ratio of antibodies recognising domains of the spike protein relative to other non-protective viral targets correlates with the severity of the sickness. Anti-spike antibodies were more common in those with moderate sickness, while those who died from their disease had more antibodies that recognized other components of the virus.

Variations in immune response

Although the study discovered commonalities among a group of patients, individual patients' immune responses, particularly those with severe disease, still vary significantly. Antibody reactions are unlikely to be the solitary factor that determines a person's fate. Some patients with serious sickness die, while others recover.

Some of these patients have a strong immunological response, while others have a less one. There are a lot of other things going on, as a result. Other aspects of the immune system are also involved. It's vital to remember that our findings show correlations but not causation.

Correspondence to: Dr. Conger Patrick, Department of Pediatrics, Ludwig Maximilian University of Munich, Munich, Germany, E-mail: patrickconger@gmail.com

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Antibodies were found to be lower in people with asymptomatic and moderate sickness than in individuals with severe disease.

IgM and IgA levels in most patients dropped progressively to low or undetectable levels after recovery, about one to four months following illness onset or estimated infection date, whereas IgG levels dropped dramatically.

It's still unclear whether the immune response induced by SARS-CoV-2 vaccination is greater or lasts longer than the one induced by spontaneous illness.