Microscopic Colitis Foundation

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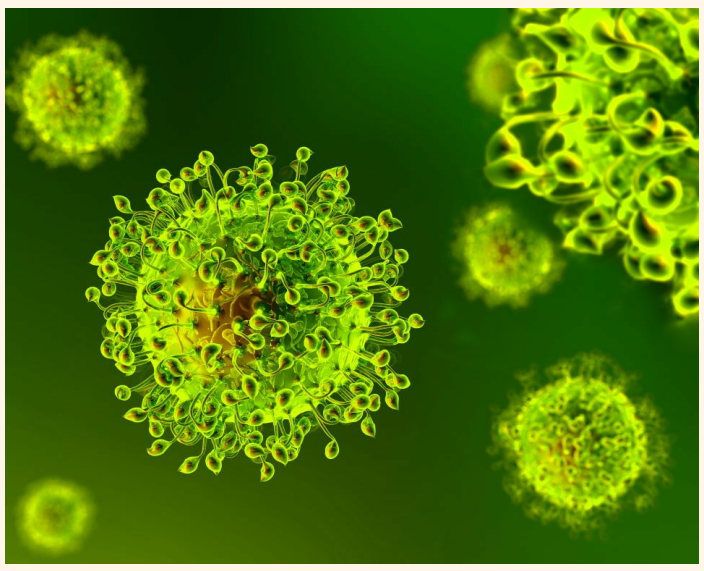
How Does Microscopic Colitis Impact Your Risk of Developing Covid-19?



It's possible that whether or not your odds of becoming infected with Covid-19 are increased by the fact that you have microscopic colitis (MC) may depend on whether or not your MC is in remission. But as we shall see farther down in this article, as we explore the published research, it appears possible (or even likely) that an MC diagnosis may decrease the risk of developing Covid-19. This may be true regardless of whether the disease is in remission, or causing active symptoms.

Common sense tells us that if our body is already severely inflamed, our risk of developing additional diseases is higher, because our immune system is already focusing its full attention (and devoting most of its available energy) toward fighting the inflammation. And this has been well-documented in the medical literature. But if our disease is in remission, then there shouldn't be any preexisting inflammation to distract our immune system and increase the risk. Of course, that's pure speculation, and not proven by any scientific research.

What does the published scientific evidence say about Covid-19 risks for IBD patients?

There's a lot of discussion among researchers about the fact that coronaviruses attach to their target cells in the lungs through angiotensin-converting enzyme 2 (ACE2). ACE2 is present on lung, kidney, and blood vessel epithelial cells. But it's actually the most prevalent on the epithelial cells of the terminal ileum and the colon. And as we (as MC patients) are well aware, the inflammation that causes MC is also usually the most pronounced in the epithelial cells of the terminal ileum and the colon. This is surely just a coincidence, rather than an implication of a direct association between the two diseases, because coronaviruses are primarily respiratory diseases, not digestive system diseases (although they can cause digestive system symptoms).

This may, however, explain why some coronavirus patients have digestive system symptoms in addition to their respiratory symptoms. Some COVID-19 patients even present with the digestive system symptoms of diarrhea and nausea, with no apparent respiratory symptoms. So yes, even though the symptoms may or may not be linked with an IBD, it is certainly possible to develop COVID-19 by way of the digestive system, despite what medical experts may claim.

Statistically, fecal samples from a group of Covid-19 patients in China showed that about half of them tested positive for Covid-19.[1] Although intestinal presence may be a relatively minor characteristic of the disease, it does imply that Covid-19 can also be spread by fecal contamination.

IBDs increase ACE2.

IBDs cause an increase of ACE2 in the intestines of patients when the disease is active.[2] This suggests that IBD patients who have active disease may be at an increased risk of developing Covid-19. But an increased level of ACE2 only occurs when significant inflammation is present. Therefore, IBD patients in remission should not show an increase in ACE2, which implies that they should not be at an increased risk of developing Covid-19.

In a search of published data relevant to Covid-19, one group of researchers concluded:[3]

"These observations suggest that the inflamed gut of IBD patients represents an optimal doorway through which the virus enters human tissues. However, based on a PubMed search on March 17, 2020, we found no evidence to suggest that Covid-19 occurs more frequently in IBD patients than in the general population."

And a group of researchers also pointed out that there are two different forms of ACE2.

There is a full-length form (which is the one discussed above), and there's a soluble form that circulates in the blood.[4] The soluble form of ACE2 was proposed as a possible alternate attachment target of Covid-19.[5] Further studies have indicated that the virus may attach to the soluble form of ACE2 rather than the full-length form. When it does, then attachment to the full-length form will be blocked, so that infection can be prevented. It has also been noted that soluble ACE2 is substantially increased in the blood of patients when their IBD is active.[6] That implies that even patients with active IBDs may have increased protection against coronavirus.

None of this is specific to MC.

However, as we often have to do because of the lack of MC-specific research, based on our knowledge of how MC compares with other IBDs, we can certainly extrapolate these findings to gain insight into how an MC diagnosis might affect our relative Covid-19 risk. Back in 1990 researchers determined that Crohn's disease patients experience a significantly greater increase in ACE2 than ulcerative colitis patients.[7] We have for a long time been aware that MC has more characteristics in common with Crohn's disease, than with ulcerative colitis, particularly the fact that it can affect virtually any part of the digestive tract, whereas ulcerative colitis is usually limited to the lower section of the colon. Based on that observation, it's likely that MC patients experience an increase in ACE2 also, when their disease is active.

Can we reach any conclusions from all this evidence?

Despite the lack of MC-specific research data, when we consider the best evidence available, it appears very likely that a diagnosis of MC probably has no significant effect on the risk of developing Covid-19. There are almost surely some protective attributes, and there are likely some negative features. But in most cases, these effects probably cancel each other out to various degrees so that there is no significant increase or decrease in the Covid-19 risk for most MC patients. Because we tend to be so different, as individuals, with different food sensitivities, clinical symptoms, and so forth, it's likely that we will each have our own degree of risk, relative to Covid-19.

Based on the best information currently available, it's unlikely that our risk is significantly different from others in the general public.

Noteworthy News about Covid-19

Underlying conditions known to significantly increase Covid-19 risks are the predominant threats associated with coronaviruses.

And as we are all very aware, vitamin and mineral deficiencies appear to play a very, very important role in the development of those underlying conditions, specifically Vitamin D and magnesium deficiencies.

Severity of COVID-19 patients appears to be predicted by their cortisol levels.

A study of 535 patients in London showed that doubled cortisol levels were associated with a 42 % increase in the risk of death.[8] Patients who had a baseline cortisol concentration of 744 nmol/L or less (when admitted to a hospital), survived for at least 24 days (or longer). Patients whose cortisol level was more than 744 nmol/L survived for10 to 36 days.

Note that patients who were undergoing glucocorticoid treatments for an unrelated issue (such as IBD patients) were excluded from the study. However, as the researchers pointed out, these study results suggest that it might be risky for COVID-19 patients to attempt to self-medicate using steroids, due to the risk of increasing their cortisol levels.

And, as we (the Microscopic Colitis Foundation) have pointed out numerous times in the past, it's never a good idea to risk suppressing your immune system when facing a COVID-19 risk. Your immune system is your best weapon when fighting any virus-based risk.

Metformin may reduce the fatality risk for women who have COVID-19.

Metformin is a popular oral treatment for diabetes. Women who had filled their 90-day metformin prescriptions in the study of more than 6,200 adults who were hospitalized because of COVID-19 had at least a 20 % reduced risk of death.[9] The study showed no similar benefit for men.

The CDC classifies obesity as a greater risk (among underlying conditions) than hypertension.

The Centers for Disease Control and Prevention (CDC) pointed out that individuals with a body mass index (BMI) over 40 had the greatest risk, whereas a BMI above 30 had a lower level (but still increased) risk.[10] Heart conditions, sickle cell disease, type 2 diabetes, and pregnancy are also listed as underlying conditions that can lead to more serious outcomes for COVID-19 patients.

Recovery from COVID-19 may not convey immunity.

The same Medscape article referenced above stated that preliminary data show that antibody protection of recovered COVID-19 patients may be inconsistent. 33% of patients in one study showed no immunity to repeat

infection risk. Hospitalized patients may have some degree of immunity after recovery, but more data is needed to verify the extent of immunity.

COVID-19 may cause brain damage.

Neurologists have found that COVID-19 patients who have shortness of breath, headache, or dizziness may have neurological issues that may not be discovered until after the patient is discharged from the hospital.[11] Possible issues include stroke, psychosis, an altered mental state, and a dementia-like syndrome. Because of the risks, they suggest that hospitalized patients should have a neurological evaluation and an MRI scan of their brain before they are discharged. Longer-term studies of COVID-19 survivors who were either hospitalized or spent time in an intensive care unit show that respiratory dysfunction and reduced exercise capacity are common problems, in addition to the psychological problems of post-traumatic stress disorder (PTSD), depression, anxiety, and reduced quality of life in general.[12]

There's disagreement about the risk carried by airborne particles.

240 scientists have written a letter to the World Health Organization (WHO) asking the organization to revise its recommendations. They describe why they feel there is plenty of evidence that COVID-19 can be spread by tiny airborne droplets. The WHO has claimed that there is no documented evidence to support this position. This appears to be another case of failure to take action because of a lack of medical research. Apparently, the WHO does not believe in erring on the side of caution.

Preliminary evidence suggests that PPIs may increase COVID-19 risks.

Although the validity of the study has been questioned by a number of medical authorities because it contains some inconsistencies, it showed that people taking a single PPI daily had twice the risk of those not taking a PPI.[13] The study also showed that taking two PPIs each day quadrupled the risk.

But this would appear to be a very logical effect, since PPIs are notorious for depleting magnesium. And as we have maintained all along, magnesium is critical for assuring a robust immune system.

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