



Cystic Fibrosis Research News

Title:

SARS-CoV-2 and *Burkholderia cenocepacia* infection in a patient with Cystic Fibrosis: an unfavourable conjunction?

Lay Title:

SARS-CoV2 infection in CF patient colonized by Burkholderia cenocepacia

Authors:

Olcese C^{1,2}, Casciaro R¹, Pirlo D³, Debbia C³, Castagnola E⁴, Cresta F¹, Castellani C¹

Affiliations:

1. Cystic Fibrosis Center, IRCCS Istituto Giannina Gaslini, Genoa, Italy

2.Department of Neuroscience, Rehabilitation, Ophthalmology, Genetics, Maternal and Child

Health (DINOGMI), University of Genoa

3. Department of Emergency, IRCCS Istituto Giannina Gaslini, Genoa, Italy

4. Unit of Infectious Diseases, IRCCS Istituto Giannina Gaslini, Genoa, Italy

What was your research question?

We considered whether people with cystic fibrosis (CF) chronically infected by *Burkholderia cenocepacia* (*BC*) might have a greater risk of a more severe clinical outcome of COVID-19 or if, otherwise, COVID-19 might trigger a pulmonary flare-up by *BC* in people with CF who are carrying this germ.

Why is this important?

The effects of the associated infection by *BC* in the CF complex lung environment are not know. Here we present the case of a SARS-CoV2 infection in a 34 year old woman with CF, carrying BC. Understanding the impact of the viral and bacterial component of the infection are important for therapeutic decisions. We consider important to share our experience in managing this case.

What did you do?

The woman was admitted because of high temperature and a positive swab for SARS-CoV2. A few days later, clinical, blood (biochemical) and radiological parameters worsened. A chest CT scan showed images compatible with either COVID-19 or the severe *BC* usually called "cepacia syndrome". Because of the impossibility to understand if the clinical and radiological conditions were mainly caused by a SARS-COV2 infection or by a cepacia syndrome, a broad spectrum antibiotic therapy (a therapy that can be used against many different types of infections) plus

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cfresearchnews@gmail.com



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IntraVenous (IV) steroids, antiviral medication and heparin (to dilute the blood) were used. The woman's long-lasting therapy with lumacaftor/ivacaftor was stopped because of possible interactions.

What did you find?

The combination of therapies resulted in a gradual improvement and the woman was discharged at day 20. We found it problematic to determine if there was an interaction between SARS-CoV-2 and *BC* and to explain the mechanisms of the response to treatments. The relative impacts of the multiple IV antibiotics, of the antiviral and of the steroid therapy are difficult to assess. Possibly, all or most of them to some degree contributed to the positive outcome of the case.

What does this mean and reasons for caution?

CF pulmonary flare-ups may be triggered by viral infections of the airways. Although this relation has not been proved for SARS-CoV2, in this case COVID-19 might have disrupted the balance between immune system response and chronic *Burkholderia* infection and the initial viral dominance of SARS-CoV-2 could have been later outweighed by bacterial infection of BC. Alternatively, the chronic *Burkholderia* infection might have helped the development of a more aggressive COVID-19. Unfortunately, we do not have enough evidence to confirm either of the two theories.

What's next?

Further evidence, including specific markers of inflammation, which were not assessed in this case, must be collected to find if carrying *BC* might be regarded as a risk factor for severe COVID-19 expression in CF or the other way round.

Original manuscript citation in PubMed

https://pubmed.ncbi.nlm.nih.gov/33883098/

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