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# A Case Report on Causes of COVID-19 Induced Psychosis and Treatments

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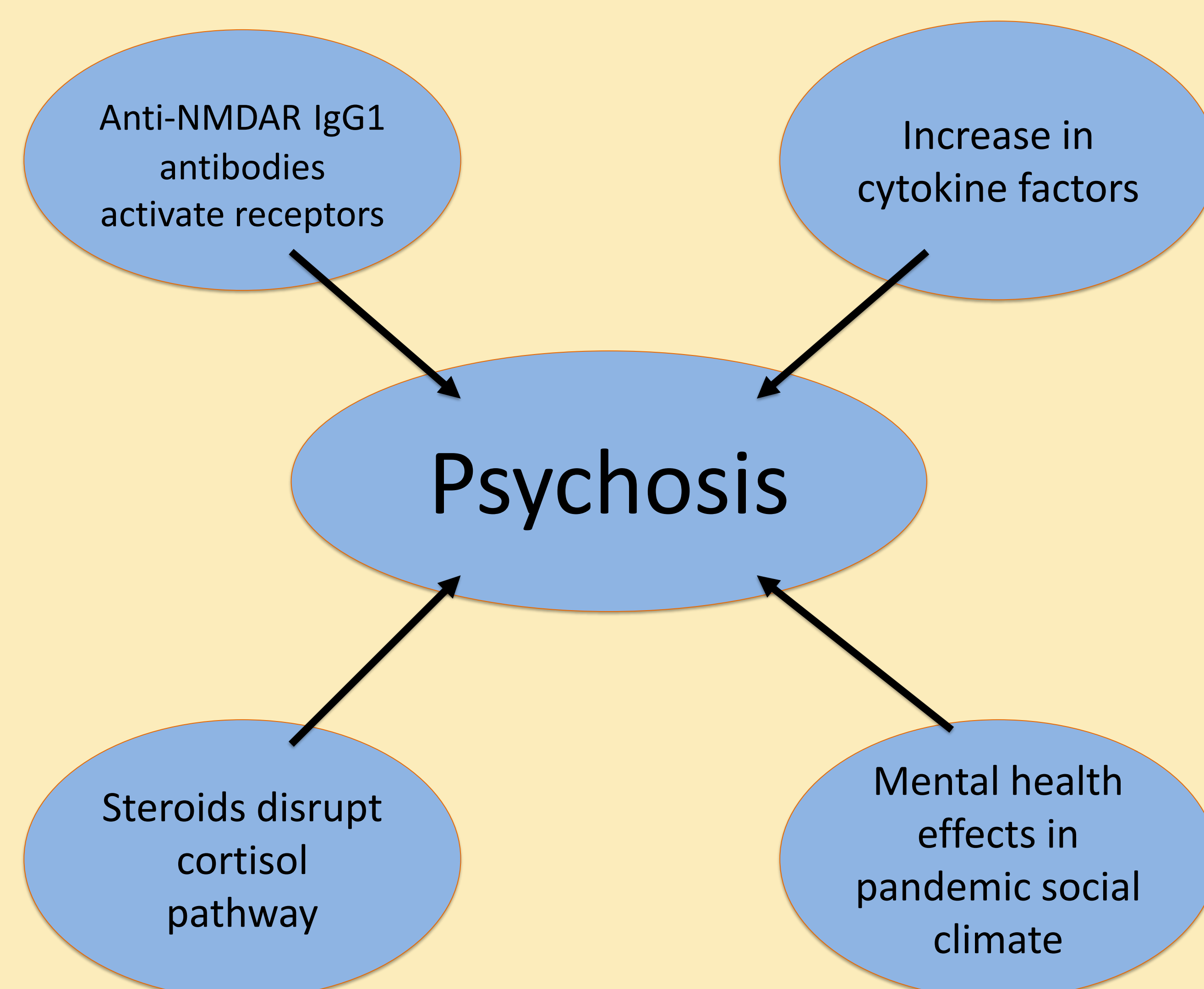
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## Background

- COVID-19 is a viral infection that is caused by an RNA virus in a subfamily of Coronaviridae named severe acute respiratory syndrome (SARS-CoV-2). The family also includes severe acute respiratory syndrome coronavirus (SARS-CoV) and middle east respiratory syndrome coronavirus (MERS-CoV) which have previously been shown to cause respiratory symptoms and psychosis with immunoreactivity to IgG.<sup>1-4</sup>
- Hypothesis 1: Viruses can act on the brain and cause psychosis. Herpes Simplex Virus triggers the formation of anti-NMDAR IgG1 antibodies which activate the NMDA receptor causing neural excitability leading to neuropsychosis.<sup>5</sup>
- Hypothesis 2: Psychosis may be caused by increased cytokine factors including IL-1B, IL-6, TNF and CRP. These cytokines are associated with inflammation and changes in mood, social behavior and cognitive behavior.<sup>5</sup>
- Hypothesis 3: Steroid induced psychosis is the most popular theory currently. Long term synthetic steroids can disrupt the hypothalamo-pituitary adrenal axis including the cortisol pathway leading to mood disorders including depression, anxiety and mania.<sup>6,7</sup>
- Hypothesis 4: The global pandemic is believed to have affected the current mental state of people. The disease precautions such as social distancing and school closings can contribute to increased rates of depression, anxiety and increased substance use which contribute to a person's personal vulnerability to develop psychosis.<sup>8,9</sup>



## Case Report

Patient is a 63-year-old caucasian female with a history of GERD and hyperlipidemia. MS developed a non-productive cough, nasal congestion, and general myalgia 12 days prior to hospitalization. She was referred for monoclonal antibody treatment. When she arrived at the facility, she was found to have a pulse ox in the mid-80's associated with exertional dyspnea.

ED work up included vital signs: BP: 107/51mmHg, Temperature 99F; Pulse 80BPM, RR: 20/min, Exertional Pulse OX: 90% on RA, Resting Pulse OX: 95% on RA. The chest x-ray showed bilateral patchy interstitial alveolar opacities suggesting pneumonia secondary to COVID-19. EKG showed SR 75BPM with no acute changes. Other significant lab findings included: D-dimer: 1.5; CRP: 176; LDH of 524 and a positive COVID-19 PCR test.

Initial treatment included Decadron 6mg daily for 10 days, Vibramycin 100mg twice a day for 7 days and Eliquis 5mg twice a day. She did not receive Remdesivir as she was outside the therapeutic window. She was discharged after 1 day on observation with oxygen and the above medications.

Five days after the patient was discharged, she returned to the ED via EMS due to "erratic behavior" and tachycardia, 144BPM. The patient presented with tangential, pressured speech, decreased sleep, decreased concentration, irritability and delusions of grandiosity. ED staff discussed the possibility of steroid induced neuropsychiatric symptoms, but she did not improve with the cessation of steroids. Her respiratory symptoms had improved at the time of this evaluation.

She had CT scans and an MRI of her brain which revealed no acute intracranial findings. She experienced 30 seconds of seizure like activity in the MRI prompting an EEG which showed no epileptic activity. The seizures were attributed to 7 days of insomnia, but alcohol withdrawal could not be ruled out. She started on Naltrexone and Quetiapine with minimal improvements prompting a voluntary admission to the psychiatric unit.

After 8 days on the medical floor, the patient spent 11 days on the psychiatric floor. Her medication regime had to adjusted multiple times due to lack of stabilization of psychosis and insomnia. She was finally stabilized and discharged on Depakote 1250mg daily, Quetiapine 300mg daily and Trazadone 100mg daily, with a diagnosis of bipolar affective disorder.

Ten days after she was discharged, she returned to the ED and was re-admitted to the psychiatric unit secondary to a domestic dispute and suspected recurrence of mania. She was stabilized and discharged on the same regime within 4 days.

On follow up six months later, MS had discontinued Trazadone and Quetiapine and was on a low dose of Depakote with no recurrence of symptoms in hopes of complete medication cessation soon.

## Discussion

- Case reports in Madrid suggested two timelines for COVID-19-induced psychosis: subacute onset in which psychotic symptoms lasted for less than one week and quick recovery where symptoms lasted less than two weeks with the assistance of antipsychotics. MS required longer treatment and the addition of a mood stabilizer.<sup>10</sup>
- Another case described a 36-year-old female with no personal or family history who developed similar psychotic symptoms to MS, but this patient developed them 4 days after respiratory symptoms began and they resolved rapidly with an antipsychotic which she discontinued on her own with no recurrence. This difference may be attributed to their age difference.<sup>11</sup>
- Another patient was a 54-year-old female who presented 3 weeks after discharge for COVID treatment with neuropsychiatric symptoms similar to MS. Both patients required a combination therapy of a mood stabilizer and antipsychotic. However, this patient has a personal and family history of psychotic disorders.<sup>12</sup>
- As with most psychiatric conditions, the goal is symptom management with a return to baseline mental status. Various treatments have been discussed such as second-generation antipsychotics, mood stabilizers, IVIG, cessation of steroids and immunotherapies. However more research needs to be conducted so patients can return to their baseline sooner and avoid re-admissions.<sup>5,13,14</sup>

## Conclusion

Overall, MS's prolonged symptoms and requirement of dual treatment of antipsychotic and a mood stabilizer makes her case unique and could help future patients have a shorter timeline of symptoms. It is important to discuss the various causes of COVID-19 induced psychosis to implement preventative measures. However, more importantly the proper management of these patients needs to occur so they can return to baseline sooner, and this area requires more research.

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