Is it time to revisit neuroinflammatory depression? A case of COVID-19 associated. depression and the role of ECT

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Introduction:

1. The neurobiology of depression is heterogeneous with multiple hypotheses including neuroinflammation.
2. Incidence of depression in the community following the COVID-19 pandemic is increasing.
3. Activation of neuroinflammatory pathways by COVID-19 virus might be a contributor to COVID-related depression

Case:

- A 49-year-old male with no past psychiatric history.

ED

- Presented with the new onset of suicidal ideations with plans to cut himself and significant psychomotor features

Symptoms

- He exhibited irritability, anger, anhedonia, negativism, and isolated himself in his room.
- He demonstrated delusional fear about his apartment exploding due to not paying his bills.

Citalopram 20mg, escitalopram 20mg, and bupropion (titrated to 300mg) with the addition of aripiprazole 5 mg were tried without improvement.

ECT was considered and his depression and psychosis

Discharged after completion of 10 ECT treatments on 300 mg of bupropion daily and 5mg olanzapine at night.

Discussion:

1. COVID-19 infection is associated with 'cytokine storm' which may exacerbate neuroinflammation via increases in cytokines and possible activation of mast cells and microglia.
2. Interleukin-6 and CRP are the most strongly linked to depression with a high correlation for anhedonia and psychomotor retardation, prominent features of depression in our case, hinting at a possible role of neuroinflammation.