

Inpatient Management of COVID-19 Disease

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COVID-19 Infection I

- Exposure is through contact, droplet nuclei and airborne, but the usual portal of entry is the respiratory tract / mucosal surfaces
- **Up to 40% of infections can be asymptomatic**
- Severity of illness, risk for hospitalization, and death hinges on a number of prognostic factors
- Major symptoms include **fever, SOB and cough**, but fatigue, loss of smell / taste, diarrhea, myalgias, sore throat and headaches are reported
- **Pathophysiology** includes viremia, pneumonic phase, extrapulmonary phase, coagulopathy [endotheliitis], and cytokine storm

COVID-19 Infection II

- **Clinical risk factors** for severe disease and poor outcomes include: Age, Male gender, Blood type, Obesity, Pregnancy, Immunosuppression, Comorbidities [COPD, Asthma, HTN, DM, CAD / MI or CVA, Ca], Medications [PPIs, ?NSAIDs], ?Vitamin D deficiency
- **Socioeconomic risk factors** for severe disease and poor outcomes: NH residents, Minority status [Blacks, non-white Hispanics, Native Americans], Lower income strata, Poverty
- **Immunity** may be short-lived if only from antibody responses, but cell-mediated immune responses are likely to be more durable [? role of prior coronavirus exposures, vaccine efficacy, specificity of antibody tests]

Tissue Targets

- ▶ Zhou et al. (*Nature*) and Hoffmann et al. (*Cell*) identify **ACE2** as a SARS-CoV-2 receptor, and the latter show its entry mechanism depends on cellular serine protease **TMPRSS2**. These results may explain **proinflammatory cytokine release** via the associated angiotensin II pathway and a **possible therapeutic target via the IL-6-STAT3 axis**.

< <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7175868/> >

- ▶ ACE2 is present in most organs: ACE2 is attached to the cell membrane of mainly **lung type II alveolar cells, enterocytes of the small intestine, arterial and venous endothelial cells and arterial smooth muscle cells in most organs**. ACE2 mRNA expression is also found in the cerebral cortex, striatum, hypothalamus, and brainstem.[18] The expression of ACE2 in **cortical neurons and glia** make them susceptible to a SARS-CoV-2 attack, which was the **possible basis of anosmia and incidences of neurological deficits seen in COVID-19**.

< https://en.wikipedia.org/wiki/Angiotensin-converting_enzyme_2 >

COVID-19 Infection III

- Therapy and Management for Inpatients: [multi-system disorder]
 - Oxygen, fluids and electrolytes, vitamin supplementation, antidiarrheals
 - Optimizing treatment of co-morbidities [DM₂, CAD, HTN, COPD, Coll-Vasc Dz]
 - Proning
 - Empiric / Targeted antibacterial therapy
 - Anticoagulants: LMWH, Heparin, DOACs
 - Antiviral agents: < HCQ >, remdesivir, favipiravir
 - Immunomodulators: Dexamethasone
 - Convalescent plasma [?monoclonal Abs]
 - Cytokine storm [IL-6] inhibitors: Tocilizumab [Actemra]
 - HFNC vs BiPAP vs MV/ETT
 - ? Recycled / Re-purposed agents: Famotidine, Ivermectin, Colchicine