Inpatient Management of COVID-19 Disease

NOE B. MATEO, MD
SANFORD BISMARCK HOSPITAL
090220
COVID-19 Infection

- 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]
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.

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.
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