The Role of Hypertension in Incidence and Morbidity of COVID-19: A One-Year Review in US Veterans

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Background: The discovery that ACE2 was a co-receptor of COVID-19 as well as early clinical findings induced interest in the role of hypertension (HTN) and its treatment with angiotensin converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARB) with regard to COVID-19 incidence and morbidity. We examined the effect of demographic and common risk factors of HTN and treatment with ACEI, ARBs, calcium channel blockers (CCB) and beta blockers (BB) in patients with COVID-19.

Methods: The VA COVID resource data combines hospital data, administrative and clinical record search results. The prevalence of HTN was defined by its presence in the last 2 years prior to COVID-19 testing. New event (incidence) was determined as occurrence within 60 days thereafter. ACEI and ARB, and CCB and BB were combined, basic demographic and risk factors were categorized for comparisons. Data sets were propensity matched, statistical analysis (SAS enterprise guide 7.1) used frequency distributions (chi square). The data was limited to the first year of collection.

Results: Of 1,305,466 veterans, we found positive tests (18.1%), HTN (56.9%), ACE or ARB (33.7%), and CCB or BB (15.4%). HTN and treatment had no effect on COVID-19 incidence (OR HTN 1.08, ACE/ARB 1.01, CCB/BB 0.94). Male, white patients aged over 60 years predominated, Age, race, and smoking had no effect on incidence, but DM2 (OR 1.2) and higher BMI (OR 1.4) did. We then examined demographics and risk factors in the first COVID-19 positive population. Male gender (5.4), age > 60 years (7.5), race non-white (1.6), BMI >30 (2), smoker (2.8), and DM2 (11.8). In turn, these factors at most affected outcomes (OR) such as all-cause mortality (7.9), admissions (2.1), ICU admissions (2.5), and ventilator use (2.7) with the exception of BMI which was associated with improved outcomes (0.6). ACE or ARB had no effect (<1.1) while CCB or BB had a small effect (1.26) on outcome.

Conclusions: In conclusion, HTN and anti-hypertensive treatment had no effect on COVID-19 incidence. HTN is associated with age, race, smoking and a diagnosis of DM2. Treatment with ACE or ARB has no effect on morbidity while CCB or BB had a small effect that deserves further evaluation.

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Prevalence and Outcomes Associated with Hyperuricemia in Hospitalized Patients with COVID-19

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Background: COVID-19 can increase catabolism and result in hyperuricemia. Uric acid (UA) potentially causes kidney damage by alteration of renal autoregulation, inhibition of endothelial cell proliferation, cell apoptosis, activation of the pro-inflammatory cascade, and crystal deposition. Hyperuricemia in patients with COVID-19 may contribute to acute kidney injury and poor outcomes.

Methods: We included 834 patients with COVID-19 who were >18 years old and hospitalized for >24 hours in the Mount Sinai Health System and had at least one measurement of serum UA. We examined the association between the first UA level and major adverse kidney events (MAKE, defined by a composite of all-cause in-hospital mortality or RRT or 100% increase in serum creatinine from baseline), as well as markers of inflammation and cardiac injury.

Results: Among the 834 patients, the median age was 66 years, 42% were women, and the median first UA was 5.9 mg/dl (IQR 4.5-8.8). Overall, 52% experienced MAKE, and 32% died during hospitalization. After adjusting for demographics, comorbidities, and laboratory values, a doubling in serum UA was associated with increased MAKE (OR 2.5 per doubling, 95% CI 1.7-3.5) and in-hospital mortality (OR 1.7 per doubling, 95% CI 1.3-2.3) (Figure A & B). Serum UA levels were independently associated with a higher level of procalcitonin (p<0.6; SE 0.2) and troponin (p<1.2; SE 0.2) but was not associated with the serum ferritin, CRP or IL-6 (Figure C).

Conclusions: In patients admitted to the hospital for COVID-19, higher UA levels were independently associated with MAKE and mortality in a dose-dependent manner. In addition, hyperuricemia was associated with higher procalcitonin and troponin levels.

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A) & B) Association between serum UA and outcomes, MAKE (A) and in-hospital mortality (B) using restricted cubic spline models and mean serum UA (6.9 mg/dL) as a reference C) Association between serum UA and log, transformed markers of inflammation and cardiac injury.