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EXPERIENCE WITH REMDESIVIR FOR THE TREATMENT OF CRITICAL COVID-19 IN A COMMUNITY-BASED HOSPITAL IN EVANSTON, ILLINOIS

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PURPOSE: In late December 2019, a novel coronavirus named SARS-CoV-2 was discovered in Wuhan, China using deep unbiased sequencing in samples from patients with pneumonia. From its discovery, SARS-CoV-2 has caused global public health emergencies, economic crises, and innumerable deaths. To date, only corticosteroids have been proven to be effective in reducing mortality from COVID-19. From antiviral agents, remdesivir has been recently recognized as a promising therapy against COVID-19, but its mortality benefit is still a matter of controversy. In this study, we analyzed the effect of remdesivir on in-hospital death in our community hospital in the Chicago North Shore.

METHODS: We retrospectively reviewed a de-identified dataset of 190 patients with COVID-19 admitted to a community hospital Intensive Care Unit (ICU) in Evanston, Illinois, from March 2020 to December 2020. Only molecularly confirmed COVID-19 cases defined by a positive result on a reverse-transcriptase-polymerase-chain-reaction (RT-PCR) assay or nucleic acid amplification test (NAAT) of a specimen collected on a nasopharyngeal swab were included. We performed a Cox proportional hazards model to analyze the effect of remdesivir on the hazard of in-hospital death in our patient population. To minimize confounders, age, qSOFA score, invasive mechanical ventilation, and other targeted COVID-19 therapies used at any given time (including corticosteroids, tocilizumab, hydroxychloroquine, colchicine, azithromycin, and atorvastatin) were forced as covariables into the model. For sensitivity analysis, we calculated the E value (with the lower confidence limit) for the obtained point estimate. The E value is defined as the minimum strength of association on the risk ratio scale that an unmeasured confounder would need to have with both the exposure and the outcome, conditional on the measured covariates, to explain away a specific exposure-outcome association fully.

RESULTS: Between 190 patients admitted to the ICU, the median age was 69 years (IQR, 59 – 78 years), 125 (65.8%) were male, 62 (32.6%) were White, and 84 (44.2%) were admitted from a long-term care facility. Of those patients, 143 (75.3) received corticosteroids, 67 (35.3%) received remdesivir, and 66 (34.7%) received both. Among survivors, 34/90 (37.8%) received remdesivir compared to 33/100 (33%) nonsurvivors. The Cox regression model showed decreased hazard of in-hospital death associated with the administration of remdesivir (Hazard Ratio [HR] 0.55; 95% CI 0.29 – 0.94, p= .028). The E value for the point estimate was 3.04 and the E value for the lower confidence interval was 1.32, meaning that a confounder not included in the multivariable Cox regression model associated with remdesivir use and in-hospital mortality in patients with critical COVID-19 by a hazard ratio of 1.32-fold each could explain away the lower confidence limit, but weaker confounding could not.

CONCLUSIONS: According to the data presented above, we concluded that in our patient population, the patients who did not receive remdesivir had a 65% chance of dying sooner compared to the ones who did receive remdesivir (when probability = HR/HR + 1). This could indicate a potential mortality benefit of remdesivir in critically ill patients.

CLINICAL IMPLICATIONS: In our patient population, the use of remdesivir was associated with a slower progression to death in critically ill patients with COVID-19.

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