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POSSIBLE MECHANISMS OF THE SARS-COV-2-INDUCED AKI PROGRESSION TO CKD: A FORWARD-LOOKING PERSPECTIVEFatemeh Masjedi¹, Jamshid Roozbeh¹, Zeinab Karimi¹¹Shiraz University of Medical Sciences, Shiraz Nephro-Urology Research Center, Shiraz, Iran

BACKGROUND AND AIMS: Coronavirus disease 2019 (COVID 19) was identified in December 2020 and is still growing in most parts of the world. The wide range of affected organs is likely based on the shared expression of the main severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) entry-receptor angiotensin-converting enzyme 2 (ACE2). Therefore, broad distribution of ACE2 receptors in various tissues play a key role in the multi-organ dysfunction and death due to COVID-19.

METHOD: International databases including PubMed, Embase, Web of Science, Scopus, and Cochrane Library Databases were used for search of articles by 30 December 2020. Keywords were nephropathy, COVID-19, coronavirus, renal injury, acute kidney injury, chronic kidney injury, and SARS-CoV-2 or a combination of them in the titles/abstracts.

After the collection of related studies, Mendeley software was used to categorize and eliminate the duplicate titles. Then, studies with inappropriate purposes were removed. The selected studies were done on humans and published in English.

RESULTS: Due to high prevalence of acute kidney injury (AKI) in patients with COVID-19, we summarize the molecular insights into viral infection mechanisms and implications for AKI. Moreover, mechanisms of the AKI to chronic kidney disease (CKD) transition such as relative contribution of immune cell response, fibroblasts activation, endothelial dysfunction and subsequent hypoxia may contribute to association of AKI with worse outcomes during this virus pandemic.

CONCLUSION: We highlight the state of the knowledge on SARS-CoV-2-dependent mechanisms for AKI and list the potential management options for prevention of AKI worsening and the imminent possibility of CKD. Finally, we aim to provide a better understanding of why Coronavirus induce AKI and, subsequently, progression to CKD in the coming years and further discuss the acute as well as long-term renal consequences.

MO469 **Figure:** Potential interrelationship between endothelial dysfunction, tubular epithelial injury, interstitial inflammation, and fibrosis are likely to create a vicious cycle that can lead to the progression of acute kidney injury to chronic kidney disease (AKI to CKD) during COVID-19.