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Editorial

Impact of COVID-19 on Renal Dysfunction

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Coronavirus Disease-2019 (COVID-19) may cause different degrees of renal impairment [1]. One of the studies conducted on hemodialysis patients and infected with COVID-19 revealed that there was no difference between patients regarding the presence or absence of diabetes mellitus and hypertension, but the effect of COVID-19 on the kidney increased with increasing the age [2].

Renal involvement during acute course of COVID-19 is well known, but little is known about it as a consequence of post infection [3]. The virus can enter the kidney by attachment of spike protein that found on the surface of the virus to the angiotensin converting enzyme 2 (ACE2) receptors that found on the surface of the kidney [4]. Later, the virus causes nephronecrosis [5]. On the other hand, cytokine storm develops a result of viral infection. It has been found that IL-6 is associated with renal damage through the effect on endothelial cells which produce pro-inflammatory cytokines, which may cause capillary leak syndrome [1].

Renal injury during acute course of infection may be result from direct viral effect [2]. A significant relation was found between acute kidney injury (AKI) and later impaired renal function in the individuals who were recovered from COVID-19 when followed up for one year [6].

In addition to ACE2 receptors, the virus requires a transmembrane protease serine 2 (TMPRSS2). However both ACE receptors and TMPRSS2 are present in the kidney and seem to be more abundantly the cells of the proximal tubules and collecting ducts [3]. In addition to damage of the tubular apparatus of the kidney, the damage that extend to the glomerular apparatus is characteristic of COVID-19 effect than other causes of renal impairment [5]. The receptor CD147 that found on the epithelial cells of the kidney offer additional way for viral entry [3]. Cytokine storm also differentiate renal damage due to COVID-19 than other viral causes. Different kinds of cytokines are produced during COVID-19 which have adverse effects on the kidney [1].

Infection by SARS-CoV-2 may result in change in the expression of several genes in the cell, the effects may be against the virus or support it to escape the immune response [7]. Regarding human, the large part the genome is non-coding sequences which can be transcript to RNA but not translate to a protein. Long non-coding RNAs (lncRNAs) are a class of transcripts with length more than 200 nucleotides [8]. The relation of lncRNAs with different kidney diseases has been confirmed in the recent years [9]. Micro- RNAs encoded by the virus are called vmiRNAs. The precursor of the vmiRNA is called pre-CvmiR-5 and the mature vmiRNA is called CvmiR-5-5p It was named pre-CvmiR-5 to the precursor, and CvmiR-5-5p to the mature vmiRNA. It has confirmed that the sequence of CvmiR-5-5p is specific to and conserved among SARS-CoV-2 [10]. In humans, miRNAs may have an important role to prevent viral infection during different stages of replication cycle of the virus [9]. Patients with moderate and severe forms of COVID-19 infection showed overexpression of lncRNAs NEAT1 and TUG1, and cytokine storm as well [11].

Long non-coding RNAs may found as biomarkers in serum and urine. They have distinct role in renal pathogenesis through regulation the production of cytokines other immunological factors that involved in fibrosis and development of AKI [8]. Significant correlation was reported between lncRNAs and IL-6 [11].

The distinct role of NEAT1 regarding regulation the expression of IL-6 has been reported. The down-regulation expression of IL-6 and TNF-α, was associated with expression of miR-381 which occurred after inhibition of NEAT1 [11]. However, update studies reported interaction between human and viral ncRNA in patients infected with SARS-CoV-2. The expression of proteins involved in different steps of replication cycle (like ACE2, TMPRSS2, and S) were inhibited by several miRNAs [10]. Non-coding RNAs may offer new field regarding nephrology to understand pathogenesis and development of treatment at the molecular level through modulation different pathways involved in kidney impairment [12].

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