SARS-CoV-2 and Clinical Urology: There is no Dragon in this Story

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Abstract

Covid-19 disease is caused by the coronavirus of severe acute respiratory syndrome 2. The disease has evolved into a global pandemic that continues to this day. Coronavirus basically causes acute respiratory illness, the symptoms of which may remain milder even three months after the onset of this acute infection. Many patients also experience cardiological, gastrointestinal, and neurological symptoms that last for at least two months. Some patients report worsening of certain urinary symptoms. In this paper, we review the current knowledge about the relationship between SARS-CoV-2 and urinary system. A database and a manual search were conducted in the MEDLINE database of the National Library of Medicine, PubMed, Embase, the Cochrane Library, and other libraries using the keywords "SARS-CoV-2," "COVID-19," and "pandemic," in various combinations with the terms "kidney," "bladder" "prostate," "testicles," "LUTS," "pain," and "infection." A considerable number of articles investigate the possible interaction between SARS-CoV-2 and the urinary system. In addition, to the well-documented involvement of the kidneys, testicle, and penile involvement seems to be possible. There are also studies investigating the development of benign prostatic hypertrophy (BPH) as a complication of SARS-CoV-2 infection and some studies examining the impact of COVID-19 disease on LUTS. In conclusion, the studies published so far do not provide conclusive evidence about a strong association between SARS-CoV-2 and the genitourinary system. Further investigation is warranted to better understand the nature of COVID-19 disease.

Keywords: *COVID-19, severe acute respiratory syndrome 2, urinary system*

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Introduction

COVID-19 disease is caused by the coronavirus of severe acute respiratory syndrome 2 (SARS-CoV-2). The disease has evolved into a global pandemic that continues to this day. Coronavirus basically causes acute respiratory disease, the symptoms of which vary and may remain milder even 3 months after the onset of this acute infection. In addition to systemic and respiratory symptoms, several patients also experience symptoms from the upper respiratory tract, skin, and eyes, as well as cardiological, gastrointestinal, and neurological symptoms, which last for at least 2 months.[1] The most common symptoms are fever, dry cough, and physical exhaustion. Less common symptoms are loss of taste or smell, nasal congestion, sore throat, headache, muscle or joint pain, skin rash, nausea or vomiting, diarrhea, chills, and dizziness.[1] Other less common symptoms include confusion, decreased consciousness, anxiety, depression, and sleep disorders.[2] The increase in cytokines that occurs during infection determines the severity of inflammation from COVID-19 disease. In fact, hypercytokinemia causes acute respiratory distress syndrome, stroke, myocardial infarction, acute renal failure, and vascular damage from vascular disease and serious manifestations due to nervous system malfunction.[3] Symptoms definitely attributed to this disease do not include lower urinary tract symptoms (LUTSs). However, some patients report worsening of some preexisting symptoms. There is currently no literature on a strong association between COVID-19 disease and the urinary tract. In this article, an attempt is made to present current evidence on the relationship between SARS-CoV-2 and the urinary system and to discuss the possible interaction.

Materials and Methods

A database and a manual search were conducted in the MEDLINE database of the National Library of Medicine, PubMed, Embase, the Cochrane Library, and other libraries using the keywords

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How to cite this article: Stamatiou K, Magri V, Perletti G, Trinchieri A. SARS-CoV-2 and clinical urology: There is no dragon in this story. Hellenic Urol 2022;34:8-12.

Submitted: 26-Jun-2022
Revised: 25-Jul-2022
Accepted: 03-Sep-2022
Published: 24-Jul-2023

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Access this article online

Website: www.hellenicurologyjournal.com

DOI: 10.4103/HUAJ.HUAJ_18_22

Quick Response Code:

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Table 1: Impact of severe acute respiratory syndrome coronavirus 2 on bladder function: The most relevant studies and their findings

Authors (study)	Patients	Instruments	Findings
Dhar *et al*. (observational)	39	OAB assessment tool	Frequency ≥13 episodes/24 h (85%) Nocturia ≥4 episodes/night (87%)
Can *et al*. (prospective)	94 (62 >50)	IPSS	IPSS score before COVID-19 (1.3±1.6) IPSS score during COVID-19 (5.1±4.1)
Kaya *et al*. (retrospective)	46	IPSS Urinary symptom profile	Male patients: Difference in storage IPSS Female patients: Difference in SI/OAB incidence
Chen *et al*. (prospective)	889	OABSS	Worse storage LUTS after vaccination (13.4%)
Mumm (retrospective)	57	Patients history	Onset of urinary frequency (12.5%)

OAB: Overactive bladder, IPSS: International Prostate Symptom Score, OABSS: OAB symptom score, LUTS: Lower urinary tract symptoms, SI: Symptom index

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"«SARS-CoV-2," "COVID-19," and "pandemic," in various combinations with the terms "kidney," "bladder" "prostate," "testicles," "LUTS," "pain," and "infection." Two independent reviewers performed data extraction using identical extraction tables. We included all clinical studies with available information. We considered full-text written articles. We also included reviews and case reports. Bibliographic information in the selected publications was checked for relevant records not included in the initial search.

Results

SARS-CoV-2 has a specific three-dimensional spike protein structure characterized by strong binding affinity to the angiotensin-converting enzyme 2 (ACE-2). Given the mode of transmission through the respiratory tract and as ACE-2 is abundant in Type 2 alveolar cells of the lungs, the late are affected by the disease more easily than other organs.[4] However, ACE-2 main function is taking place in the renal vascular endothelium, therefore, kidneys can be easily affected by SARS-CoV-2. In fact, immunohistochemical studies in renal tissues obtained from infected individuals have confirmed the accumulation of SARS-CoV-2 antigen in the renal tubules.[5] Given that, the ACE-2 pathway is present in other organs of the genitourinary system, their involvement in COVID-19 disease has been investigated in a considerable number of articles. In addition, to the well-documented involvement of the kidneys, testicle and penile involvement were also reported. There are also studies investigating the development of benign prostatic hypertrophy (BPH) as a complication of SARS-CoV-2 infection and studies examining the impact of COVID-19 disease on LUTS and studies examining the impact of COVID-19 disease on LUTS [Table 1].

Discussion

Impact of severe acute respiratory syndrome 2 on the kidneys and renal drainage system

Up to the present day, approximately 30% of COVID-19 treated patients were found to have moderate renal impairment.[5] Although the exact mechanism by which SARS-CoV-2 causes renal impairment is currently unknown, glomerulopathy, damage of the proximal tubules, and accumulation of protein in Bowman's capsule related to the ACE-2 pathway activation are common findings in COVID-19 patients.[6,7]

Currently, there is no evidence of a pathogenic effect of SARS-CoV-2 on the renal drainage system. To our best knowledge, viral RNA was mostly detected in the urine of patients with moderate-to-severe disease; however, the detection of viral RNA in the urine of patients appears to be low to nonexistent and the presence of the virus in the urine is not related to the course of the disease.[8]

Impact of severe acute respiratory syndrome 2 on the bladder function

With respect to bladder function following COVID-19 disease, available information is somehow confounding. While Dhar *et al*. reported increased frequency and nocturia in more than 85% of patients with a history of overactive bladder (OAB), Kaya *et al*. and Can *et al*. did not detect significant differences in International Prostate Symptom Score (IPSS) comparing LUTS before or during hospitalization due to COVID-19. However, in the latter study, a slight increase of IPSS during hospitalization was assessed in the subgroup of patients >50 years old.[9-11] Selvi *et al*. recorded urodynamically proven lower urinary tract dysfunction following COVID-19 in three young patients. Mumm *et al*., in a small series of 57 cases, reported a significant increase of urinary frequency in 12.5% of the patients. The remaining studies did not find significant differences in LUTS severity before and during SARS-CoV-2 infection.[12-16] Interestingly, Chen *et al*. showed that COVID-19 vaccination worsened storage LUTS in up to 13.4% of patients with preexisting OAB. The mechanism by which SARS-CoV-2 infection could impact of bladder function remains unknown. Recently, Lamb *et al*. documented an elevation of proinflammatory cytokines in the urine of COVID-19 patients, that is possibly related to urgency and urinary incontinence.[14]

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Impact of severe acute respiratory syndrome 2 on prostate enlargement

There is currently no evidence on a direct association between COVID-19 disease and prostate enlargement. Nevertheless, the worsening of obstructive LUTS shown in some of the aforementioned studies indicates a possible impact of SARS-CoV-2 on the prostate gland. According to the literature, various mechanisms such as alteration of ACE-2 signaling, alteration of androgen receptor-related mechanisms, inflammation, and metabolic disorders during or after the course of SARS-CoV-2 infection may lead to worsening of LUTS related to BPH.[17] Although the exact cause of BPH development is unknown, changes in male sex hormones occurring during aging are reputed to be the most probable causative factor. Remarkably, studies have shown that men are more prone to SARS-CoV-2 infection and the elderly population appears to develop more severe COVID-19 disease.[18,19] The most likely pathogenic mechanism that indirectly associates COVID-19 with BPH has already been described: the co-expression of ACE-2 and TMPRSS2 in an organ is vital for the virus to infect it. Co-expression of ACE-2 and TMPRSS2 occurs not only in the lungs but also in the prostate.[19,20] However, to date, no presence of SARS-CoV-2 RNA has been reported in the prostate secretion of patients with COVID-19.[21] Given the age-dependent increase in the prevalence of BPH, one can assume that a significant group of elderly male COVID-19 patients may have BPH as a comorbid condition and that this condition may be exacerbated by COVID-19. In confirmation to the above Luciani *et al*. reported a worsening of BPH-induced hematuria greatly after contracting symptomatic COVID-19 infection during hospitalization in two patients. On the other hand, studies that have investigated trends in urological emergencies during the first wave of the epidemic have shown a marked reduction of chronic kidney disease-related urinary retention cases in emergency departments.[22-26]

Impact of severe acute respiratory syndrome 2 on the genital system

With respect to testicle involvement, Chen *et al*. studied 142 COVID-19 patients and found orchitis in 4.2% of cases, epididymitis in 4.9%, combined orchitis–epididymitis in 13.3%, and scrotal infections in 19.8%. The last two were more common in severely ill patients. However, this association was not statistically confirmed.[27] The studies of Ning *et al*. (112 patients) and Alkhatatbeh *et al*. (253 patients) reported neither testicular edema nor orchitis.[28,29] Ediz *et al*. reported orchitis–epididymitis/testicular pain in 10.9% of cases and testicular edema in 9.9% of cases in a cohort of 91 COVID-19 patients, while Pan *et al*. and Holtmann *et al*. in two smaller studies reported testicular discomfort in 17.6% and 5,5% of cases, respectively.[30-32] Individual reports of testicular pain and orchitis[33-36] do not add more evidence on the association between SARS-CoV-2 infection and testicular involvement.

Nineteen studies investigated the presence of SARS-CoV-2 in semen.[21,28,31,32,37-50] The sample size was very small in most of these studies. The severity of COVID-19 between cohorts varied and semen samples was collected in different periods of the disease. Only 3 out of 19 studies provided positive results. However, the possibility that the virus found in semen was actually originated in the urine could not be ruled out. Of note, the fact that the sperm quality of patients with moderate COVID-19 infection was lower than that of both patients with mild infection and healthy controls may be associated with fever and inflammation.[51,52] Furthermore, no viral RNA was detected in testicular biopsy material from dead patients.[53]

Penile involvement has been also documented through case reports describing priapism development in seriously ill patients.[54-58] Although priapism is the result of the hypercoagulable state of all these patients, given the rarity of these cases, priapism could not be recognized as a systematic side effect of COVID-19 disease.

Conclusions

The studies published so far do not provide conclusive evidence about a strong association between SARS-CoV-2 and the genitourinary system. Further investigation is warranted to better understand the nature of COVID-19 disease.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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