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COVID-19 Impact on Urogenital & Reproductive Systems: A Comprehensive Review

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Abstract

The recent emergence of the COVID-19 in China at the end of 2019 has caused a large global outbreak and a major public health issue. The World Health Organization (WHO) on March 11, 2020, has declared the novel coronavirus (COVID-19) outbreak a global pandemic. As of 12 June 2021, data from the World Health Organization (WHO) have shown that more than 176,052,554 confirmed cases were identified in 220 countries. Notably, coronaviruses was found to invade target cells through the angiotensin-converting enzyme 2 receptor, which can be found in the respiratory, gastrointestinal, cardiovascular, urinary tract, and reproductive organs. Understanding the effects of COVID-19 on the urogenital and reproductive systems is essential to our practice as urologist. This article summarises the latest evidence on COVID-19 effect on genitourinary and reproductive systems.

Keywords: COVID-19, Viral cystitis, Urogenital Urology Urinary tract , Fertility, Reproduction, SARS-CoV-2

Introduction

Since the report of the first cases of pneumonia of unknown cause by the WHO at the end of 2019, the SARS-2 Coronavirus (Sars-CoV-2) and its related disease, CO¬VID-19, has spread rapidly all over the globe. According to the online database of the Johns Hopkins University by June 2021, 176,052,554 infections, 3,800,900 deaths, confirmed worldwide [1]. SARS-CoV-2 is closely related to two bat-derived severe acute respiratory syndrome-like coronaviruses, bat-SL-CoVZC45 and bat-SL-CoVZXC21. It is spread by human-to-human transmission via droplets or direct contact, and infection has been estimated to have mean incubation period of 6.4 days and a basic reproduction number of 2.24-3.58. Among patients with pneumonia caused by SARS-CoV-2 (novel coronavirus pneumonia or Wuhan pneumonia), fever was the most common symptom, followed by cough. Bilateral lung involvement with ground-glass opacity was the most common finding from computed tomography images of the chest [2].

Expression of ACE2 and TMPRSS2 in the Urinary System and COVID-19

SARS-CoV-2 binds to the angiotensin-converting enzyme 2 (ACE2) receptor for entry and the serine protease TMPRSS2 for spike protein priming [3]. ACE2, a negative regulator in the renin–angiotensin system, is highly expressed in the epithelial cells of renal tubules, seminiferous ducts of testis, adult Leydig cells, the adrenal gland and the prostate [4]. TMPRSS2 is highly expressed in the kidney, prostate, seminal vesicles and epididymis [4]. Both of the key factors that mediate SARS-CoV-2 pathogenicity are highly expressed in urogenital organs, suggesting that these organs could be susceptible to damage by this virus.

Impact of Gender in COVID-19

One of the most frequently reported epidemiological data is the gender-related COVID-19 mortality [5].

Chen et al reported COVID-19 disease is more severe and fatal in men, possibly due to the gender-based (androgens) immunological response and additional factors [6]. It has been shown that Angiotensin-Converting Enzyme (ACE) receptors in the lung and the transmembrane serine protease 2 (TMPRSS2) enzyme group are effective in the penetration and spread of the virus into pneumocytes [7,8]. androgen-mediated regulation of the ACE receptors and the TMPRSS2 enzyme group in the host has been implicated in the more frequent occurrence of acute respiratory syndrome coronavirus-2 (SARS-CoV2) infection and higher mortality in men [9,10]. Studies have shown that women wash their hands more frequently than men [11], and men wear masks less than women [12]. These factors may also contribute to the noted differences in COVID-19 rates and mortality between the sexes.

COVID-19 and the Urinary System

[13] pointed out the importance of early recognition of symptoms by urologists for proper triage of patients and to prevent missing possible SARS-CoV-2 infection because of an overlap of COVID-19 and classical urological symptoms. Despite the presence of ACE2 in the urogenital tract, negative urinary PCR results do not support the assumption of strong or even relevant continuous replication in (luminal) tissues of the efferent urinary tract. Since it is unclear whether the receptor is expressed in luminal or basal urothelial cells, the route by which SARS-CoV-2 might cause viral cystitis could be via either viremia from the basal side or urine from the luminal side of urothelial tissue. Furthermore, Kaya et al reported that LUTS might be one of the initial symptoms of COVID-19 [14]. Replication of SARS-CoV-2 RNA in urothelial cells or secondary effects due to local or systemic inflammation, such as endotheliitis, are a hallmark in COVID-19 patients leading to symptoms such as irritative symptoms of the lower urinary tract and urinary frequency [15].

Hematuria

Haghighi et al reported Hematuria associated with SARS-CoV-2 infection visible hematuria and urinary discomfort in absence of urinary tract infection as the primary presentation of COVID-19 [16,17].

Urgency

Mumm et al reported that urinary frequency as a common symptom of COVID-19. They noticed that hospitalized COVID-19 patients suffered from Low Urinary Tract Symptoms (LUTS). Patients have especially reported increased dysuria symptoms after infection [17].

Acute Kidney Injury in Covid-19

The risk factors and causes of AKI in COVID-19 patients are diverse. The severity of pneumonia is the most important factor in the development of AKI in COVID-19 patients [18]. ACE2 is expressed in kidney cells. Renal functioning needs to be monitored regularly, especially in patients with elevated plasma creatinine levels. In the event of early signs of Acute Kidney Injury (AKI), interventions, such as continuous renal replacement therapy, should be implemented to protect renal functioning as early as possible [19].

Pei et al. performed a research about renal injury in cases with COVID-19 pneumonia and its early prognosis. Pei et al. observed that COVID-19 patients have a high frequency of renal abnormalities, including 75.4% of patients with renal involvement, 65.8% with proteinuria, and 41.7% with hematuria [20]. In a study of 116 COVID-19 hospitalised patients, Wang et al. found that all patients without chronic kidney disease showed no obvious abnormality of renal function, and none of these patients showed AKI [21]. Patients with chronic kidney disease who underwent CRRT were stable, without exacerbation of chronic kidney disease throughout the course of treatment of COVID-19. Those finding indicates that it is necessary to provide in-depth support and careful monitoring of COVID-19 patients with severe pneumonia to improve their complications.

IPSS score correlation with severity of COVID disease

In a prospective study by Karabulut et al using IPPS score relation with severity of covid disease, it was identified that patients with severe IPSS, the length of hospital stay was longer, intensive care requirement was more frequent, and their mortality rates were numerically higher. In the evaluation made regarding the time to intensive care admittance, this was identified to be the shortest in this group [22].

Covid effect on Sex hormones

Sex hormones play a role in regulating the immune system and they can cause differences in immune response between men and women. Generally speaking, testosterone has an immunosuppressive effect, while oestrogen tends to enhance the immune response [23]. Given that estrogen activates the immune system, higher estrogen levels can potentially have a protective effect [24]. Studies have shown that exogenous estrogen therapy can eliminate inflammation and reduce virus titers, thus improving survival [25]. Endogenous testosterone makes men more likely to have more serious complications related to SARS-CoV-2 infection. On the other hand, SARS-CoV-2 infection can lead to hypogonadism in men, and the reduction of androgens may cause serious complications [26].

The British Society of Sexual Medicine BSSM adds that SARS-CoV-2 also damages cells on the inner surface of blood vessels called endothelial cells, a condition which is frequently present in men with erectile dysfunction and testosterone deficiency [27]. A recent study provided insight into impaired male gonadal function after COVID-19 infection [28]. That study showed that the testosterone-to-luteinizing hormone ratio in 81 patients with COVID-19 was dramatically decreased in comparison to 100 age-matched healthy counterparts [29]. The serum testosterone-to-luteinizing hormone ratio could be a potential marker of impairment of reproductive health caused by COVID-19. Another study also confirmed that the serum LH level of patients with CO¬VID-19 was significantly higher than that of healthy men with normal fertility, while the ratio of serum testosterone to luteinizing hormone was significantly decreased, which is indicative of subclinical hypogonadism [30]. A study in Germany found that the majority of men admitted to the hospital with Covid-19 had low testosterone levels and high inflammatory markers. Unfortunately, this study was unable to determine if these low testosterone levels predated their coronavirus infection [31]. A similar study by Rastrelli et al in Italy found low testosterone levels predicted

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worse outcomes in hospitalised patients [32]. A third study from China by Ling et al found similar results [33-35]. A role of gonadal function evaluation among patients recovered from SARS-CoV-2 infection, especially the reproductive-aged men is evaluated.

COVID-19 impact on reproduction and fertility

High levels of cytokines following viral or bacterial infection, illness or injury can cause deterioration in spermatogenesis and steroidogenesis, adversely affecting fertility [36]. Researchers from the Justus-Liebig University in Giessen, Germany published a study in Reproduction, [37] showing that the sperm of men who had been diagnosed with COVID-19 showed increased sperm cell death, inflammation and oxidative stress compared to the sperm of men who had not had the virus [38]. These effects on sperm cells are associated with lower sperm quality and reduced fertility potential said lead researcher Behzad Hajizadeh Maleki. Although these effects tended to improve over time, they remained significantly and abnormally higher in the COVID-19 patients, and the magnitude of these changes were also related to disease severity [27]. Haghpanah et al suggest measuring the sperm DNA Fragmentation Index (DFI) as a determiner of male fertility impairment in patients with COVID-19 along with other options such as sex-related hormones and semen analysis. Invasion of SARS-CoV-2 to the spermatogonia, Leydig cells and Sertoli cells can lead to sex hormonal alteration and impaired gonadal function. Once infected, changes in ACE2 signaling pathways followed by oxidative stress and inflammation could cause spermatogenesis failure, abnormal sperm motility, DNA fragmentation and male infertility [39,40].

Covid Effects on the Testes

This ACE2 enzyme is the primary way SARS-CoV-2 enters cells [34]. The testes are one of the highest sites of ACE2 expression and may be affected by COVID-19. A study reported that 19% of COVID-19 men suffer from scrotal discomfort, suggesting viral orchitis [35]. Orchitis has not been confirmed as a possible complication of SARS-CoV-2 infection. The virus may not directly infect the testes, but it may trigger a secondary autoimmune response that can cause autoimmune orchitis. COVID-19 is related to abnormal blood clotting, so orchitis could also be the result of segmental vasculitis.

Erectile dysfunction and COVID-19

Erectile dysfunction is a complex physiologic and psychologic disorder. Multiple theories describes possible COVID-19 effect on erection . Impaired testosterone secretion from the affected testis, and the direct effects of testosterone in male sexual response [40]. Higher testosterone levels are associated with lower levels of pro-inflammatory cytokines such as tumour necrosis factor alpha (TNF-α), interleukins (IL-6 and IL-1β), and higher levels of anti-inflammatory cytokines (such as IL-10), [41] the immunothrombotic mechanism described for COVID-19 [42] and involving the same cytokines, [43] could be promoted by the hypogonadal state encountered in affected patients. The immunothrombosis could potentially affect the penile vessels, triggering endothelial dysfunction-therefore impairing vascular function and promoting progression to more severe forms of sexual dysfunction [44,45]. Cardiovascular complications of COVID-19, such as cardiomyopathy and myocarditis, [46, 47] could also be involved in the pathogenesis of ED even after the end of the acute phase, potentially becoming long-term cardiovascular sequelae. Other potential factors could contribute to impaired erectile function in COVID-19 patients, such as pulmonary fibrosis [48, 49] causing hypoxia in the penile vascular bed, [50, 51] or anosmia and ageusia, both manifestations of COVID-19 [52, 53] with possible negative effects on sexual health [54-57].

Covid Long term effect

Big Studies Such as C-MORE Capturing MultiOrgan Effects of COVID-19

And PHOSP-COVID: (Post-Hospitalizations COVID-19) study are assessing the long term effect of COVID-19 disease outcomes, impact on multiple organ systems, quality of life and functional capacity and mental health to understand and improve long-term health outcomes of the disease [58].

Conclusion

COVID-19 pandemic led to significant health challenges globally

The key factors that mediate SARS-CoV-2 pathogenicity are highly expressed in urogenital and reproductive organs. Urologist has to play an essential role in managing and treating patients affected by this disease. I summarize all studies and evidence to optimize their management.

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