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Original Article UROLOGY

Aggravation of lower urinary tract symptoms in patients with benign prostatic hyperplasia after COVID-19

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ABSTRACT

OBJECTIVE: To evaluate the effects of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) on prostate volume, prostate-specific antigen (PSA) values, International Prostate Symptom Score (IPSS), maximum urinary flow rate (Q_{max}) and postvoid residual urine volume (PVR) of patients with BPH.

METHODS: After retrospectively review the hospital database, the study included 104 patients aged 40-75 years, who were detected to have SARS-CoV-2 according to the reverse transcription-polymerase chain reaction (RT-PCR) test of combined oro-nasopharyngeal swab samples between March 2020 and December 2020 who were being followed for BPH and had undergone prostate volume, PSA, IPSS, Q_{max} and PVR measurements within one year prior to their COVID-19 diagnosis. The prostate volume, PSA, IPSS, Q_{max} and PVR parameters were compared before and after COVID-19.

RESULTS: The prostate volume, PSA, IPSS, Q_{max} and PVR values were compared pre-COVID-19 and post-COVID-19. No statistically significant changes were found in prostate volume and PSA levels ($p>0.05$), while a statistically significant difference was observed in IPSS, Q_{max} and PVR values, independent of the severity of the disease ($p<0.001$).

CONCLUSION: COVID-19 appears to have a negative impact on IPSS, Q_{max}, and PVR in BPH patients in the short term.

Keywords: Benign prostatic hyperplasia; COVID-19; lower urinary tract symptoms; SARS-CoV-2; urinary tract.

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Coronavirus disease (COVID-19) first began in Wuhan, China, and affected the whole world and caused the death of hundreds of thousands of people. The first case of COVID-19 is thought to have occurred in consequence of a zoonosis from a livestock market, but subsequent contamination was seen through human-to-human droplets, and symptomatic individuals have become the most important agents in the spread of the disease [1]. The COVID-19 pandemic is one of the worst disasters in human history, having economic impacts on most countries, restricting people's freedom, and posing an increasingly pressing problem on a global scale [2]. In that time according to the World Health Organization (WHO) report, there were approximately 152 million verified cases and three million deaths [3]. Owing to this very important public health problem, most countries have closed their borders, and the disease has been tried to be kept under control with vaccines [4].

COVID-19, caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus, usually

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presents with respiratory symptoms and can progress with a wide range of forms from asymptomatic disease to severe pneumonia and multi-organ failure [5]. It is known that the virus is more likely to bring about acute respiratory distress syndrome (ARDS) and multiple organ failure, especially in the elderly population and people with comorbidities (chronic lung disease, diabetes, hypertension, etc.), compared to the normal and healthy population [6]. SARS-CoV-2 has been shown to use the angiotensin-converting enzyme 2 (ACE-2) receptor and the transmembrane serine protease TMPRSS2 in its pathophysiology [7]. It has been reported that COVID-19 is serious and fatal in men [8], which has been associated with the androgen-mediated regulation of ACE-2 receptors and the TMPRSS2 enzyme group [9].

Benign prostatic hyperplasia (BPH) is a common health problem in elderly men and negatively affects their quality of life [10]. The two most important etiological factors implicated in the development of BPH are aging and androgens [11]. There are also studies showing that prostate tissue expresses ACE-2 and TMPRSS2 [12, 13]. Therefore, considering that both BPH and COVID-19 have a more negative effect on the elderly and male population and androgens play an important role in both processes, it makes sense that the elderly male population with BPH is at greater risk. In the light of this idea, we aimed to investigate whether the prostate volume, prostate-specific antigen (PSA) value, International Prostate Symptom Score (IPSS), maximum urinary flow rate (Q_{max}) and postvoid residual urine volume (PVR) of patients with BPH were affected by COVID-19. For this purpose, in this study, these parameters between pre- and post-disease periods have been evaluated.

Highlight key points

- COVID-19 infection may exacerbate lower urinary tract symptoms (LUTS) in patients with benign prostatic hyperplasia (BPH).
- The urogenital effects of COVID-19, which are often overlooked, should be considered by clinicians.
- COVID-19 was associated with worsening IPSS, decreased Q_{max}, and increased PVR, indicating impaired urinary function after infection.

MATERIALS AND METHODS

Study Population and Design

We retrospectively screened the database of our tertiary care institution and identified 236 male patients aged 40–75 years who were verified to have COVID-19 by a positive reverse transcription-polymerase chain reaction (RT-PCR) test of combined oro-nasopharyngeal swab samples between March and December 2020 while being followed up with a diagnosis of BPH. Eighty-three patients were excluded from the study due to a history of previous transurethral or pelvic surgery or pelvic radiotherapy, a PSA value above 4 ng/ml, a history of anticholinergic drug use, active urinary system infection, signs of prostatitis, bladder stone, urethral stricture, severe neurological disease (cerebrovascular disease, Parkinson's disease, etc.) or neurogenic bladder. In addition, 47 patients who did not have prostate volume, PSA, uroflowmetry, and IPSS records within the last year before COVID-19 and those for whom medical records could not be obtained were excluded from the study. Lastly, of the screened patients, two that received intensive care were excluded due to the insufficient number of cases to form a severely symptomatic group.

This study was conducted in accordance with the Declaration of Helsinki. Ethics committee approval was obtained on 20.04.2021 with the number 02-2021/05 from Karamanoğlu Mehmetbey University Faculty of Medicine.

Detailed medical histories of all patients in the study were questioned. The patients' age, smoking, body mass index, accompanying comorbid diseases, and the applied COVID-19 treatment were questioned in detail. A total of 104 patients who met the study criteria were mildly symptomatic outpatients and moderately symptomatic patients receiving oxygen therapy in the hospital without the need for intensive care. After confirming recovery from COVID-19 based on RT-PCR negativity in two consecutive tests, the patients were invited to a urological follow-up. During this evaluation, the patients were asked to complete the IPSS questionnaire, their hemogram, C-reactive protein (CRP), kidney function tests (urea and creatinine) and prostate volume, PSA values were measured, and urine analysis was performed. In addition, uroflowmetry and PVR measurement were performed. Patients with active urinary tract infection and prostatitis in the follow-up examination were evaluated again after their appropriate treatment. All patients were compared in point of the prostate volume, PSA, IPSS, Q_{max} and PVR values before and after COVID-19.

Statistical Analysis

All statistical analyses were performed using the Statistical Package for the Social Sciences, version 22.0 (SPSS,

Chicago, IL, USA). It was determined whether the data were normally distributed using Kolmogorov-Smirnov and Shapiro-Wilk tests. Student's t-test and the Mann-Whitney U test were used for continuous data with and without a normal distribution, respectively, while the chi-square test was used for the comparison of categorical variables. The paired t-test or Wilcoxon test was used in dependent groups to compare the parameters before and after COVID-19. P<0.05 was considered statistically significant.

RESULTS

The demographic data of the patients, their comorbidities, and the distribution of treatments applied for COVID-19 are shown in Table 1. Table 2 presents the comparison of the prostate volume, PSA, IPSS, Q_{max}, and PVR parameters before and after COVID-19. When the prostate volume and PSA values before and after COVID-19 were compared, no statistically significant difference was found (p>0.05). In the IPSS evaluation, a statistically significant increase was observed compared to the pre-disease values (p<0.001). In addition, there was a statistically significant decrease in the Q_{max} values and a statistically significant increase in the PVR values compared to their pre-disease evaluation (p<0.001).

Table 1. Distribution of demographic data

Characteristics	n=104 Mean±SD
Age (years)	55.98±0.65
BMI (kg/m²)	28.36±0.38
	%
Smoker	37.5
Hospitalized	21
Comorbidities	
None	46.1
Hypertension	29.8
Diabetes	25
Cardiovascular disease	8.6
Hyperlipidemia	4.9
Respiratory diseases	2.8
BPH medications	
Alpha-blockers	79.8
5-ARI±Alpha-blockers	6.7
Lifestyle modifications	20.2
	Mean±SD
Time from the last negative RT-PCR of naso-oropharyngeal swab to the post-COVID-19 urological examination, (day)	93.12±31.40

SD: Standard deviation; BMI: Body mass index; BPH: Benign prostatic hyperplasia; RT-PCR: Reverse transcription-polymerase chain reaction

Table 2. Comparison of the prostate volume, PSA, IPSS, Q_{max} and PVR parameters before and after COVID-19

Parameters	Before COVID-19	After COVID-19	p
Prostate volume	50.19±14.70	50.73±14.93	0.794[a]
PSA (ng/ml)	1.24±0.82	1.22±0.85	0.561[a]
IPSS	10.69±6.25	15.24±6.58	<0.001[b]
Q_{max} (ml/sec)	17.20±3.79	14.29±4.80	<0.001[b]
PVR (ml)	34.33±32,93	77.31±46.15	<0.001[a]

The results are shown as mean±standard deviation. a: Wilcoxon test: b: Paired t-test. PSA: Prostate-specific antigen; IPSS: International prostate symptom score; Q_{max}: Maximum urinary flow rate; PVR: Postvoid residual urine volume

DISCUSSION

Respiratory system symptoms are common in COVID-19, and the most common clinical presentation is fever, cough and dyspnea. Among severe cases, there is a substantial incidence of ARDS development as a result of cytokine storm caused by COVID-19 [5]. Although the most clinical presentation of COVID-19 is respiratory symptoms, recent studies have shown other organs and systems could be involved [14]. Zou et al. [15] created a risk map by determining the level of ACE2 receptor expression of various tissues and cells to determine which organs are more vulnerable to COVID-19. According to this mapping, it was determined that the cardiovascular, renal and gastrointestinal systems, especially the respiratory system, were at high risk due to the highest expression of ACE2. Varying levels of ACE2 and TMPRSS2 expression in human tissues and organs are considered to be the reason for the wide range of the forms of COVID-19 and variations in the clinical presentation of the disease [16].

Most studies in the literature have focused on the effects of COVID-19 on many organs and systems, whereas relatively fewer studies have been performed with the urogenital system. Various studies have shown that COVID-19 has effects on the male genital system, reporting that it impairs semen parameters through Ser-

toli and Leydig cells, alters sex hormones through follicle-stimulating hormone, luteinizing hormone, and testosterone levels, and leads to scrotal discomfort and orchitis symptoms [17–19]. In addition, a study reported that 0.32% of ACE 2 and 18.65% of TMPRSS22 were expressed in human prostate epithelial cells [20]. It is also known that the SARS-CoV-2 virus requires the coexistence of both ACE2 and TMPRSS2 in the host cell for its attachment to, penetration into, and replication of a cell [7]. Therefore, it is not unreasonable to consider that the urinary system, which has high expression levels for both ACE2 and TMPRSS2, will be affected by a virus that uses both of these factors in its pathogenesis.

BPH is a common health problem in aging men, affecting approximately 210 million men worldwide. Although 50–75% of men aged 50–80 years have histological findings of BPH, half of these cases also become symptomatic [21]. Although the pathophysiology of BPH is not yet clarified, the two etiological factors that are most implicated in its pathophysiology are aging and androgens [11]. It has been shown that approximately half of existing patients with COVID-19 are over 50 years, and men are more affected by the disease and have a higher mortality rate than women [22]. It is believed that androgens play a role in men being more affected by COVID-19 by aggregating the effects of the virus [9]. Considering the prevalence of BPH in elderly men and its possible etiologies, we aimed to investigate this situation, assuming that COVID-19 would have significant effects on BPH, an elderly male disease. In this cohort evaluating the effect of COVID-19 on BPH, the prostate volume, PSA, IPSS, Q_{max} and PVR parameters were compared before and after the disease. While there was no statistically significant difference in the post-disease prostate volume and PSA values compared to the pre-disease period, the disease was found to have negative effects on the IPSS, Q_{max} and PVR parameters independent of the intensity of the disease.

In a study evaluating the effect of LUTS in predicting the prognosis of COVID-19, it was shown that lower urinary tract symptoms (LUTS) could guide clinicians in predicting the prognosis of COVID-19, and patients with severe LUTS had a longer hospital stay, more frequent intensive care requirement, and a higher mortality rate compared to the mild group [23]. From an opposite perspective, when studies examining the effects of COVID-19 on the urinary system are reviewed, Kashi et al. [24] reported worsening in new-onset LUTS and pre-existing LUTS in patients with COVID-19 and stated that storage symptoms were generally at the forefront. In a study conducted by Kaya et al. [25], 27 female and 19 male patients were evaluated during active COVID-19 infection using IPSS and the Urinary Symptom Profile (USP) scoring system, and it was observed that LUTS became prominent in the early period of the disease. However, that study had the limitations of the questioning of the patients being performed during the active disease, subjective scoring based on the IPSS questionnaire, and the low number of male patients. In comparison, the advantages of our study can be considered a sound comparative evaluation of pre- and post-disease parameters, adequate sample size, evaluation performed after confirming recovery from COVID-19 rather than during active disease that could provide misleading results, and inclusion of objective parameters such as Q_{max} and PVR to support our findings.

SARS-CoV-2 is isolated from nasal, nasopharyngeal and lower respiratory secretions and transmitted by these secretions. Although the virus genome has been rarely detected in other biological fluids such as urine, the general consensus is that it cannot be isolated from urine [26]. In addition, in a study investigating whether SARS-CoV-2 was detected in expressed prostatic secretions (EPS), none of the patients had positive COVID-19 RNA in EPS [27]. Despite the fact that both ACE-2 receptor and TMPRSS2 protein expression has been shown in urinary system, it remains unclear which mechanism a virus that cannot be isolated from urine or prostatic secretion cause LUTS. One of our hypotheses is that the virus reaches the urinary system through the hematogenous route via viremia. Additionally, in a study in the literature, increased levels of interleukin-6 were assessed in the blood of patients with COVID-19 and the resulting immune response was shown to trigger the procoagulant process and cause multi-organ failure, which can be another hypothesis explaining how the urinary system can be affected by this immune response [28]. In a case report that might be related to this pro coagulant condition, it was stated that an elderly patient with BPH who had severe COVID-19 developed prostatic infarction, and the authors recommended that urologists should be alert to this phenomenon [29].

Our study also has limitations. First, it was designed retrospectively, and there was no control group. Second, viral RNA in urine samples was not investigated to prove the invasion of the urinary system by SARS-CoV-2. Third and lastly, the data obtained do not reflect long-term results due to the short duration of follow-up.

CONCLUSION

Based on the results obtained from our study, it was shown that COVID-19 had a negative effect on IPSS, Q_{max}, and PVR values and caused worsening of lower urinary tract symptoms. Although it is known that COVID-19 disease has effects on many organs and systems, we emphasize that its effects on the urogenital system, which may be overlooked, should also be taken into consideration. The data obtained from our study, in which we predict that COVID 19 causes worsening of LUTS, needs to be supported by larger-scale prospective studies.

Ethics Committee Approval: The Karamanoglu Mehmetbey University Faculty of Medicine Ethics Committee granted approval for this study (date: 20.04.2021, number: 02-2021/05).

Informed Consent: Written informed consents were obtained from patients who participated in this study.

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