

• 临床研究 •

COVID-19对前列腺增生患者逼尿肌收缩功能的影响

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[摘要] 目的: 采用超声影像尿动力学检查(sonography video urodynamics studies, SVUDS)分析新型冠状病毒肺炎(coronavirus disease-2019, COVID-19)对前列腺增生(benign prostatic hyperplasia, BPH)患者逼尿肌收缩功能的影响。方法: 纳入桂林医学院第二附属医院及邵阳市中心医院2023年1—6月诊断为BPH伴或不伴COVID-19感染患者124例进行回顾性研究分析。根据病毒核酸检测阳性或阴性情况分为COVID-19(+)组51例和COVID-19(-)组73例。比较两组患者一般情况、炎症指标及SVUDS参数差异, 分析COVID-19(+)组患者炎症指标及无创性SVUDS参数与逼尿肌收缩功能的相关性。结果: COVID-19(+)组患者C反应蛋白(C-reactive protein, CRP)、白介素(interleukin, IL)-6高于COVID-19(-)组患者($P < 0.05$)。两组患者年龄、IL-1 β 、前列腺特异性抗原(prostate specific antigen, PSA)、游离前列腺特异性抗原(free prostate-specific antigen, fPSA)、fPSA/PSA、前列腺体积(prostate volume, PV)、前列腺特异性抗原密度(prostate specific antigen density, PSAD)、前列腺突入膀胱距离(intra-vesical prostatic protrusion, IPP)、最大尿流率(peak flow rate, Qmax)、尿道阻力因子(urethral resistance factor, URA)、膀胱出口梗阻指数(bladder outlet obstruction index, BOOI)、基点(footpoint)、曲率(curvature)差异无统计学意义($P > 0.05$)。相比COVID-19(-)组, COVID-19(+)组最大逼尿肌压(maximum detrusor pressure, Pdet.max)、最大尿流率逼尿肌压(detrusor pressure at peak flow rate, Pdet.Qmax)、膀胱收缩指数(bladder contractility index, BCI)和最大瓦特因子(maximum Watts factor, WF_{max})降低, 膀胱壁厚度(bladder wall thickness, BWT)和残余尿量(post-void residual volume, PVR)增加(P 均 < 0.05)。COVID-19(+)组患者BWT与WF_{max}呈负相关($r = -0.313, P = 0.036$), PVR与BCI($r = -0.471, P = 0.001$)和WF_{max}($r = -0.491, P = 0.001$)均呈负相关。结论: BPH患者若合并COVID-19感染可能进一步加重逼尿肌活动低下, 导致残余尿量增加甚至尿潴留, 逼尿肌活动低下可能是BPH患者“long-COVID”尿动力学表现之一。

[关键词] COVID-19; 前列腺增生; 超声影像尿动力学; 逼尿肌功能; 下尿路症状**[中图分类号]** R697.3**[文献标志码]** A**[文章编号]** 1007-4368(2025)01-22-07**doi:** 10.7655/NYDXBNSN240716

Effect of the COVID - 19 on detrusor contractility in patients with benign prostatic hyperplasia

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[Abstract] **Objective:** The effect of coronavirus disease-2019(COVID-19) on detrusor contractility in patients with benign prostatic hyperplasia(BPH) was investigated using sonography video urodynamics studies(SVUDS). **Methods:** The clinical dataset, including general condition, inflammatory indexes, and SVUDS parameters of 124 BPH patients with or without COVID-19 infection admitted to the Second Affiliated Hospital of Guilin Medical University and Shaoyang Central Hospital between January and June 2023 were retrospectively reviewed. Those patients were divided into the COVID-19(+)($n=51$) and COVID-19(-)($n=73$) groups according to the

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results of virus nucleic acid detection, and the differences in general conditions, inflammatory indicators, and SVUDS parameters were compared between two groups. The correlations of inflammatory indexes and non-invasive SVUDS parameters with detrusor contractility in BPH patients of the COVID-19(+) group were analyzed. **Results:** The serum level of C-reactive protein (CRP) and interleukin (IL)-6 of BPH patients in the COVID-19(+) group were significantly higher than that of the COVID-19(-) group ($P < 0.05$), whereas no significant differences in age, IL-1 β , prostate specific antigen (PSA), free prostate-specific antigen (fPSA), fPSA/PSA, prostate volume (PV), prostate specific antigen density (PSAD), intravesical prostatic protrusion (IPP), peak flow rate (Q $_{max}$), urethral resistance factor (URA), bladder outlet obstruction index (BOOI), footpoint, and curvature were found between the two groups ($P > 0.05$). Significant thicker bladder wall thickness (BWT), larger post-void residual volume (PVR), and lower value of detrusor contractility indexes, including maximum detrusor pressure (P $_{det,max}$), detrusor pressure at peak flow rate (P $_{det,Qmax}$), bladder contractility index (BCI) and maximum Watts factor (WF $_{max}$), were observed in BPH patients of the COVID(+) group, compared with those of the COVID(-) group ($P < 0.05$). The negative correlations between BWT and WF $_{max}$ ($r = -0.313, P = 0.036$), between PVR and BCI ($r = -0.471, P = 0.001$), and between PVR and WF $_{max}$ ($r = -0.491, P = 0.001$) were found in BPH patients of the COVID-19(+) group. **Conclusion:** COVID-19 may aggravate detrusor underactivity in BPH patients, resulting to the increased PVR even urinary retention, which may be one urodynamics features of long-COVID in those BPH patients.

[Key words] COVID-19; benign prostatic hyperplasia; sonography video urodynamics studies; detrusor function; lower urinary tract symptoms

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良性前列腺增生 (benign prostatic hyperplasia, BPH) 是中老年男性最常见的泌尿系疾病, 其组织学特征常表现为前列腺移行区上皮细胞和间质细胞数量增加^[1]。40岁后发病率逐步升高, 随着年龄增大, 尿频、尿急、夜尿增多、排尿困难、尿不尽等下尿路症状 (lower urinary tract symptoms, LUTS) 逐渐增加^[2]。新型冠状病毒肺炎 (coronavirus disease-2019, COVID-19) 是2019年末出现, 由高致病性严重急性呼吸系统综合征冠状病毒2型 (severe acute respiratory syndrome coronavirus 2, SARS-CoV-2) 引起的全球急性呼吸道感染, 由于当时我国及时实行感染防控措施, 传播得到有效控制^[3]。

SARS-CoV-2致病性累及全身多个系统, 除咳嗽、咳痰、腹泻不适、味觉及嗅觉丧失等症状外, 还出现尿频、尿急、尿潴留等LUTS^[4]。目前研究认为COVID-19导致LUTS涉及多种机制, 包括肾素-血管紧张素系统失调引起全身性炎症反应, 缺血缺氧导致氧化应激等, 但是并无动物及人体实验支持, 而且COVID-19对患者下尿路功能影响的研究有限, 并且多为症状学研究, 极少进行尿动力学 (urodynamics studies, UDS) 研究^[5]。因此, 本研究通过对比合并或未合并COVID-19的BPH患者超声影像尿动力学 (sonography video urodynamics studies, SVUDS) 参数及感染指标差异, 分析COVID-19对BPH患者下尿路功能学及形态学的影响, 期望有助于探讨COVID-19对下尿路功能影响的机制。

1 对象和方法

1.1 对象

回顾性收集桂林医学院第二附属医院及邵阳市中心医院2023年1—6月51例BPH合并COVID-19及73例未合并COVID-19患者的临床资料。本研究经桂林医学第二附属医院伦理委员会批准 (ZLXM-2023012), 所有患者均签署书面知情同意后行SVUDS。结合患者病史、症状及辅助检查, 根据SARS-CoV-2病毒核酸检测阳性或阴性情况分为COVID-19(+)组 (行首次SVUDS前1~2个月内核酸检测阳性, 首次SVUDS检查时已经转阴) 和COVID-19(-)组 (既往核酸检测阴性及自觉无发热咳嗽等COVID-19症状)。

纳入标准: ①结合患者病史、症状、辅助检查和SVUDS诊断为BPH; ②所有患者均行SARS-CoV-2病毒核酸检测 (鼻咽拭子)。排除标准: ①有明确神经系统病史及精神失常; ②确诊神经源性膀胱; ③患有糖尿病; ④有脊柱损伤及盆腔神经损伤、盆腔手术史及放疗史; ⑤有尿道损伤及尿道狭窄; ⑥患有泌尿系肿瘤、腹腔积液等其他影响逼尿肌压力的疾病。

1.2 方法

1.2.1 检查方法

采用Laborie (Aquarius XT) 尿动力学分析仪和深圳迈瑞DC-65彩色多普勒超声系统, 按照国际尿控学会 (international continence society, ICS) 操作指

南完成SVUDS检查,并通过Aquarius XT将尿动力学图像和数据与超声影像同步显示及保存^[6-8](图1)。

1.2.2 数据收集

①患者一般情况:年龄、炎症指标,包括超敏C反应蛋白(C-reactive protein, CRP)、白介素(interleukin, IL)-6、IL-1 β ,以及前列腺特异性抗原(prostate specific antigen, PSA)、游离前列腺特异性抗原(free prostate-specific antigen, fPSA)、前列腺特异性抗原密度(prostate specific antigen density, PSAD);②SARS-CoV-2感染情况:患者SARS-CoV-2病毒核酸检查结果(鼻咽拭子);③超声影像参数:前列腺体积(prostate volume, PV)、残余尿量(post-void residual volume, PVR)、前列腺突入膀胱距离(intravesical prostatic protrusion, IPP)、膀胱壁厚度(bladder wall thickness, BWT)等;④尿动力学参数:最大尿流率(peak flow rate, Qmax)、最大逼尿肌压(maximum detrusor pressure, Pdet.max)、最大尿流率逼尿肌压力(detru-

sor pressure at peak flow rate, Pdet.Qmax)、最大瓦特因子(maximum Watts factor, WF_{max})、基点(footpoint)、曲率(curvature)、膀胱收缩指数(bladder contractility index, BCI)、膀胱出口梗阻指数(bladder outlet obstruction index, BOOI)及尿道阻力因子(urethral resistance factor, URA)等。

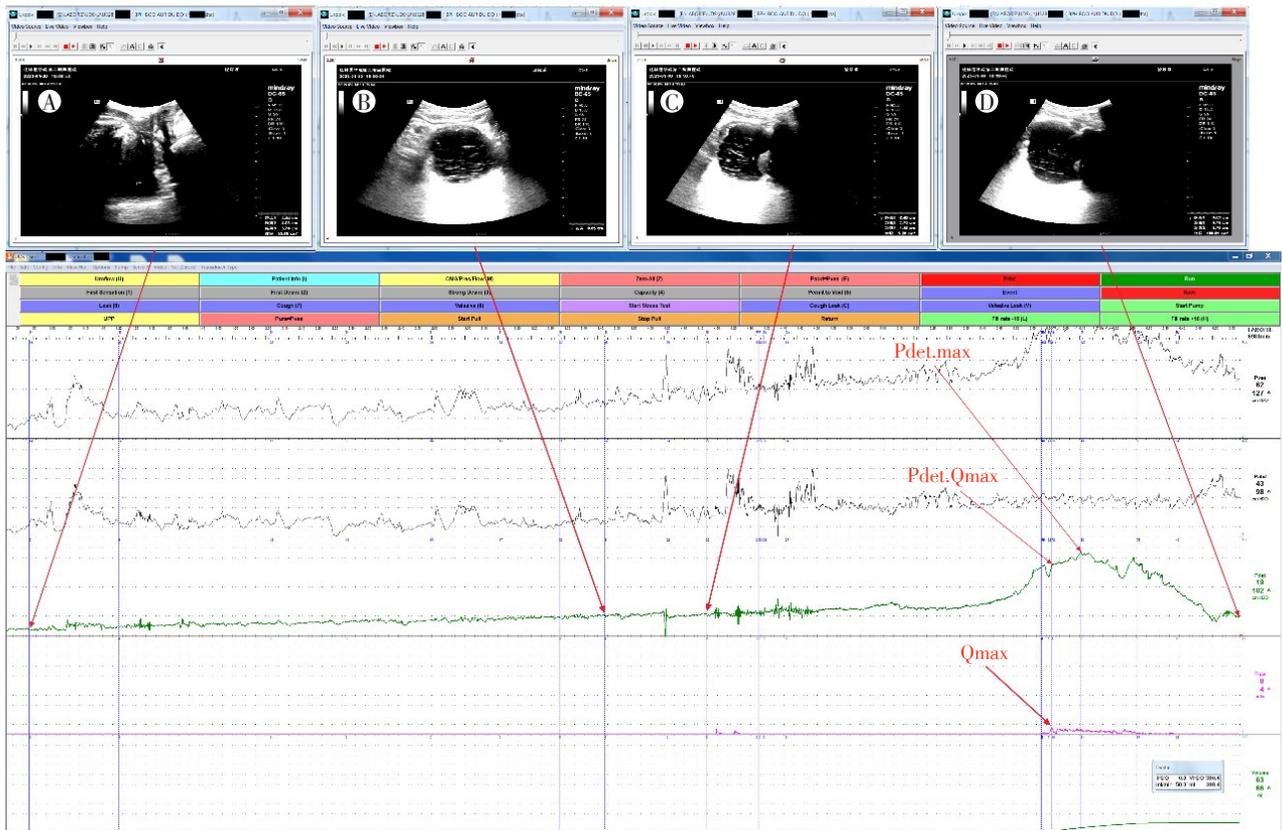
1.3 统计学方法

采用SPSS25.0软件进行数据统计分析,对数据进行正态性分布检验。符合正态分布的数据采用均数 \pm 标准差($\bar{x} \pm s$)表示,采用t检验和Pearson相关分析;不符合正态分布的数据使用中位数(四分位数) $[M(P_{25}, P_{75})]$ 表示,采用Mann-Whitney U检验和Spearman相关分析。 $P < 0.05$ 为差异有统计学意义。

2 结果

2.1 两组患者一般情况及炎症指标对比

根据纳入和排除标准,共纳入124例BPH患者,其中COVID-19(+)组51例,COVID-19(-)组73例。



A: Prostate volume at bladder volume of 50–100 mL. B, C: Bladder wall thick (B) and intravesical prostate protrusion (C) at bladder volume of 150–200 mL. D: Post-voiding residual volume. Maximum watts factor (WF_{max}), footpoint, curvature, bladder contractility index (BCI), bladder outlet obstruction index (BOOI) and urethral resistance factor (URA) were automatically generated by software of urodynamics analysis. E: The image of sonography video urodynamic studies of a patient with benign prostate hyperphasia and detrusor underactivity combined with COVID-19.

图1 SVUDS检测方法

Figure 1 Detection method of SVUDS

COVID-19(+)组患者CRP、IL-6高于COVID(-)组患者($P < 0.05$)。两组患者年龄、IL-1 β 、PSA、fPSA、fPSA/PSA、PSAD差异均无统计学意义($P > 0.05$,表1)。

2.2 两组患者SVUDS参数对比

两组患者PV、IPP、Qmax、URA、BOOI、footpoint、curvature差异均无统计学意义($P > 0.05$)。相比COVID-19(-)组,COVID-19(+)BPH患者Pdet.max、Pdet.Qmax、BCI及WF_{max}降低($P < 0.05$),BWT和

PVR均增加($P < 0.05$,表2)。

2.3 COVID-19(+)组患者炎症指标及无创性SVUDS参数与逼尿肌收缩能力的相关性分析

根据2.1和2.2选取两组间有差异的炎症指标CRP、IL-6及无创性SVUDS参数BWT和PVR,与逼尿肌收缩功能指标BCI和WF_{max}进行相关性分析。选择的4个指标及参数均为非正态分布,故进行Spearman相关性分析。结果显示,BWT与WF_{max}呈

表1 两组患者一般情况及炎症指标比较

Table 1 Comparison of patients' characteristics and inflammation indexes between two groups

Characteristic	COVID-19(-)(n=73)	COVID-19(+)(n=51)	t/Z	P
Age(years, $\bar{x} \pm s$)	70.68 \pm 8.85	70.45 \pm 9.56	0.140	0.889
PSA(ng/L, $\bar{x} \pm s$)	4.42 \pm 3.48	4.51 \pm 3.51	0.344	0.732
fPSA[ng/L, $M(P_{25}, P_{75})$]	0.96(0.41, 2.35)	1.34(0.79, 1.58)	1.510	0.137
fPSA/PSA [$M(P_{25}, P_{75})$]	0.37(0.20, 0.57)	0.32(0.18, 0.36)	0.894	0.371
PSAD [$M(P_{25}, P_{75})$]	0.06(0.03, 0.12)	0.10(0.04, 0.14)	1.777	0.076
CRP[mg/L, $M(P_{25}, P_{75})$]	2.07(0.83, 5.74)	7.44(2.47, 15.00)	-3.106	0.003
IL-6[pg/mL, $M(P_{25}, P_{75})$]	9.22(3.42, 13.82)	20.23(9.71, 48.76)	-3.062	0.007
IL-1 β [pg/mL, $M(P_{25}, P_{75})$]	5.20(3.42, 13.82)	2.92(1.40, 9.61)	-0.437	0.662

表2 两组患者SVUDS参数对比

Table 2 Comparison of SVUDS parameters between two groups

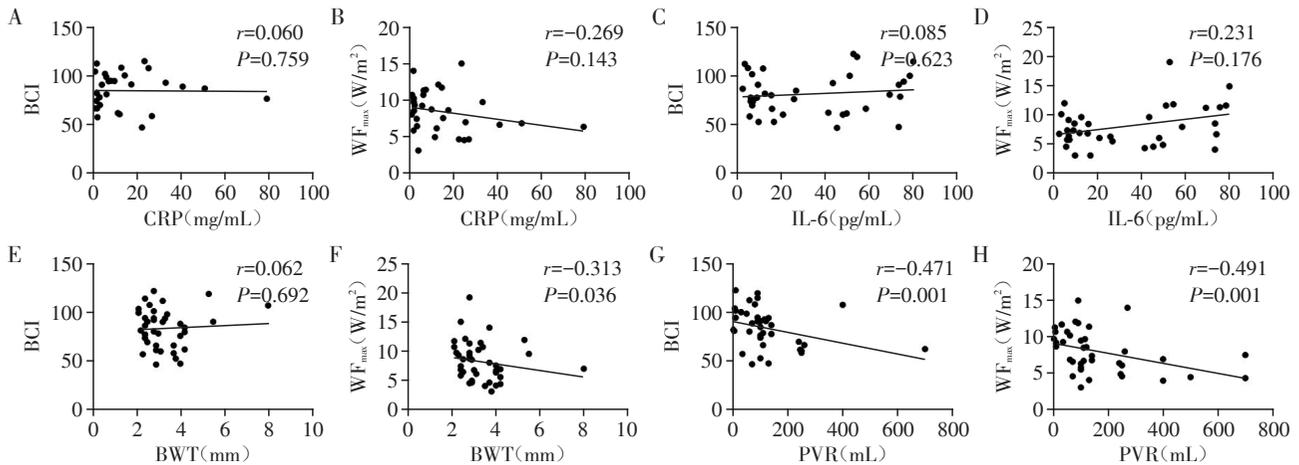
Parameter	COVID-19(-)(n=73)	COVID-19(+)(n=51)	t/Z	P
PV [mL, $M(P_{25}, P_{75})$]	47.00(30.00, 74.00)	42.00(28.00, 55.00)	1.192	0.233
IPP[cm, $M(P_{25}, P_{75})$]	1.80(1.10, 2.50)	1.40(1.07, 1.86)	1.305	0.192
PVR[mL, $M(P_{25}, P_{75})$]	70.00(10.00, 137.50)	100.00(55.00, 230.00)	2.527	0.014
BWT[mm, $M(P_{25}, P_{75})$]	2.50(2.35, 2.95)	2.90(2.50, 3.70)	3.053	0.003
Pdet.max [cmH ₂ O, $M(P_{25}, P_{75})$]	76.20(52.50, 92.30)	58.80(38.85, 87.35)	2.907	0.036
Pdet.Qmax[cmH ₂ O, $\bar{x} \pm s$]	60.01 \pm 21.31	48.32 \pm 21.55	2.752	0.007
Qmax [mL/s, $M(P_{25}, P_{75})$]	5.90(4.10, 8.70)	5.50(4.55, 9.03)	0.101	0.919
BCI($\bar{x} \pm s$)	96.54 \pm 24.38	83.07 \pm 20.76	2.917	0.004
WFmax(W/m ² , $\bar{x} \pm s$)	10.76 \pm 5.34	8.26 \pm 3.35	2.699	0.008
Footpoint($\bar{x} \pm s$)	33.83 \pm 20.48	29.91 \pm 19.96	0.981	0.329
Curvature[$M(P_{25}, P_{75})$]	0.62(0.28, 1.40)	0.56(0.23, 1.65)	0.051	0.959
URA[cmH ₂ O, $\bar{x} \pm s$]	37.11 \pm 15.41	38.02 \pm 17.69	0.261	0.794
BOOI($\bar{x} \pm s$)	45.83 \pm 24.08	39.67 \pm 23.25	1.233	0.221

负相关($r = -0.313, P = 0.036$),PVR与BCI($r = -0.471, P = 0.001$)和WF_{max}($r = -0.491, P = 0.001$)均呈负相关,其余指标和参数与BWT和WF_{max}无相关性($P > 0.05$,图2)。

3 讨论

SARS-CoV-2是一种囊膜单链RNA病毒,其携带基因编码蛋白分为非结构蛋白和结构蛋白^[9]。

SARS-CoV-2首先侵犯肺泡上皮细胞引起肺损伤,还可能出现心脏、脑、肝脏、肾、前列腺等器官损伤^[10]。文献报告,SARS-CoV-2感染后LUTS症状以储尿期为主,尿频、尿急最为多见,认为膀胱黏膜也有血管紧张素转换酶2(angiotensin-converting enzyme 2, ACE2)受体表达,SARS-CoV-2可以通过刺突蛋白(S蛋白)诱导COVID-19相关膀胱炎(COVID-associated cystitis, CAC),前列腺受累可能也参与LUTS^[11]。虽



A: The correlation between BCI and CRP. B: The correlation between WF_{max} and CRP. C: The correlation between BCI and IL-6. D: The correlation between WF_{max} and IL-6. E: The correlation between BCI and BWT. F: The correlation between WF_{max} and BWT. G: The correlation between BCI and PVR. H: The correlation between WF_{max} and PVR ($n=51$).

图2 COVID-19(+)组患者炎症指标及无创性SVUDS参数与逼尿肌收缩能力的相关性分析

Figure 2 Correlation analysis of inflammatory markers and non-invasive SVUDS parameters with detrusor muscle contractility in the COVID-19(+) group

然本研究发现炎症因子CRP和IL-6在COVID-19(+)BPH患者中增高,但是与逼尿肌收缩功能受损程度并不相关,提示COVID-19导致的逼尿肌活动低下(detrusor underactivity, DU)可能还有CAC以外的机制参与。

BPH可以导致膀胱出口梗阻(bladder outlet obstruction, BOO),早期逼尿肌代偿性改变,以尿频、尿急等LUTS为主^[12]。为克服BOO,逼尿肌平滑肌代偿性肥大,随着梗阻时间延长,梗阻程度加重,逼尿肌超微结构和间隙连接发生病变,导致不可逆性损伤,可出现排尿困难、尿不尽甚至尿潴留等DU症状^[13]。研究发现,BPH患者合并COVID-19后,LUTS、国际前列腺症状评分、生活质量评分及PVR等恶化,然而症状加重及相关评分并不能评估膀胱逼尿肌收缩功能^[14-15]。因此,本研究通过UDS压力流率实验发现,在膀胱出口梗阻程度相似的前提下,相比COVID-19(-)BPH患者,COVID-19(+)BPH患者逼尿肌收缩功能参数Pdet.max、Pdet.Qmax、BCI及 WF_{max} 均降低,提示COVID-19可能进一步损伤BPH患者逼尿肌收缩功能,增加DU发生率,恶化DU程度。

研究发现,COVID-19后LUTS加重的BPH患者预后较差,而且认为COVID-19是BPH患者尿潴留的危险因素之一^[16]。因此,DU可能是一个潜在的评估BPH患者合并COVID-19的预后因子。目前,COVID-19相关LUTS研究多通过症状学评分,由于DU需要通过侵袭性UDS诊断,除少数个案报道外,

尚无采用UDS研究的系统报道^[17]。值得注意的是,本研究COVID-19(+)BPH患者行SVUDS时核酸检测已转阴性,而且距离首次检测阳性时间平均约1个月,说明BPH患者在COVID-19发生1个月后仍有DU。另外,3例BPH患者(感染前无尿潴留病史)合并COVID-19后尿潴留,反复试拔管失败后行清洁间歇性导尿,随访至感染后6个月才逐渐恢复逼尿肌收缩功能。因此,DU可能是老年BPH/BOO患者“long-COVID”UDS表现之一。

PVR由于变异较大,难以作为评估逼尿肌收缩功能的无创性指标^[18-20]。但是,本研究发现不仅COVID-19(+)BPH患者PVR较阴性患者增加,而且随着PVR增加,COVID-19(+)患者BCI和 WF_{max} 均有降低趋势,与逼尿肌收缩功能呈负相关,提示PVR具备无创性评估BPH合并COVID-19患者逼尿肌收缩功能的潜在价值。BWT作为另一个常用的无创性指标,研究发现其具备鉴别女性DU及逼尿肌过度活动的潜力^[21]。与此类似,本研究也发现逼尿肌收缩功能更差的COVID(+)BPH患者BWT更厚,可能与COVID-19引起的炎症反应导致逼尿肌平滑肌、膀胱黏膜及浆膜等膀胱壁组织增生性改变有关。虽然,BWT与阳性患者 WF_{max} 呈负相关,而与BCI之间却无类似发现。 WF_{max} 通过复杂公式计算所得,体现了膀胱表面单位面积逼尿肌收缩产生的功率,相比BCI,应该更能准确反映真实逼尿肌收缩功能,但是尚无两者在特定人群中评估逼尿肌收缩

功能的“head-to-head”研究^[22]。

本研究存在一定的局限性。首先,本研究虽然纳入不同地区2个单位的病例,但是纳入样本数量仍较少,尚需开展大样本、多中心研究,研究结论供临床参考。另外,本研究为回顾性研究,而且选择的BPH患者年龄较大,普遍存在BOO,可能已有潜在的DU,存在样本选择偏倚风险。最后,本研究缺乏逼尿肌功能正常人群的对比研究,而且不能排除COVID(-)组存在隐性感染可能,亟待日后更广泛人群的前瞻性研究取得更确实的结论。

综上所述,结合COVID-19导致长期多器官功能障碍的可能,“long-COVID”的诊治受到广泛重视^[23-25]。笔者认为,BPH患者若合并COVID-19可能进一步加重DU,导致PVR增加甚至尿潴留,DU可能是老年BPH/BOO患者“long-COVID”UDS表现之一。因此,BPH患者若合并COVID-19,需警惕逼尿肌收缩功能受损风险,及时评估PVR等指标,早期处理并发症及保护膀胱功能。

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所有作者声明无利益冲突。

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Author's Contributions:

XIAO Ning, XIAO Jinhua, HUANG Hai, TANG Qi, ZHAO Huashen, and WANG Jianfeng designed the research; XIAO Ning, XIAO Jinhua, TANG Qi, CHEN Dan, HUANG Qiuxia, ZHAO Huashen, and WANG Jianfeng performed research; XIAO Ning, XIAO Jinhua, and TANG Qi analyzed data; XIAO Ning and XIAO Jinhua wrote and revised the manuscript.

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