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Before Urinary Incontinence can be attributed to COVID-19, All Differential Causes Must Be Ruled Out

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LETTER TO THE EDITOR

We read with interest Sousa *et al*'s article about a case series of three female patients with new-onset urinary incontinence (UI) shortly after SARS-CoV-2 infection [Sousa, F. C. *et al*., 2023]. UI in patient-1 (66yo female) was classified as mixed due to leakage during the filling phase and leakage during exertion, and as stress incontinence in patient-2 (61yo female) and in patient-3 (63yo female) [Sousa, F. C. *et al*., 2023]. It was concluded that SARS-CoV-2 infections may be complicated by UI, possibly due to pelvic floor muscle (PFM) weakness [Sousa, F. C. *et al*., 2023]. The study is impressive, but some limitations should be discussed.

The major limitation of the study is that the pathophysiology of UI was not adequately discussed and explained [Sousa, F. C. *et al*., 2023]. In general, UI can be due to a neurological, psychiatric, urogenital, renal, or functional problem. The neurological problem may be at the level of the brain, spine, peripheral autonomous nervous system, or smooth or striated muscles of the bladder (sphincter, detrusor) or the PFM. Accordingly, several UI subtypes are distinguished, such as stress incontinence, urge incontinence, giggling incontinence (subtype of urge incontinence), mixed incontinence (stress plus urge incontinence), reflex incontinence (neurogenic bladder), overflow incontinence (in case of chronic urine retention), extra-urethral incontinence, nocturia, or enuresis.

Since SARS-CoV-2 infections often manifest in the central nervous system (CNS) or peripheral nervous system (PNS), we should know whether the three patients were examined by a neurologist, and whether any of the three had evidence of CNS or PNS complications of a SARS-CoV-2 infection. Even if the neurological examination was normal, it would have been mandatory to perform cerebral and spinal imaging to rule out stroke, bleeding, infectious, or immunological disease. If an infectious or immunologic CNS was suspected, analysis of the cerebrospinal fluid (CSF) for infectious agents and immunological biomarkers, such as cytokines, chemokines, glial factors, 14-3-3, neurofilament, anti-cytokine autoantibodies, sphinganine, dihydro-neopterine, and tau protein, would also have been required.

To rule out radiculitis, the patient should have undergone nerve conduction studies (NCSs) and CSF analysis. This is because Guillain-Barre syndrome (GBS) is a common complication of SARS-CoV-2 infections [Finsterer, J. *et al*., 2021]. Since patient-3 was diabetic and since diabetes is the most common cause of plexopathy, we should know whether diabetes in patient-3 was well or poorly controlled and whether worsening diabetes control during the SARS-CoV-2 infection could explain the development of UI.

Since UI can also be a symptom of depression and anxiety syndrome [Cheng, S. *et al*., 2020], it should be discussed whether UI, especially in patient-1, could be due to the psychiatric disorder.

Several medications can cause UI by decreasing bladder outlet resistance or increasing intra-vesical pressure, disrupting the physiologic pressure relationship between bladder and urethra [Salazar, D. M. *et al*., 2008]. Drugs can also interfere with CNS control of voiding, thereby leading to UI. Drugs (e.g. diuretics) can also lead to overproduction of urine and thus UI [Salazar, D. M. *et al*., 2008]. Drugs that can theoretically cause UI include alpha-1 receptor antagonists, antipsychotics, benzodiazepines antidepressants, and hormone replacement therapy [Salazar, D. M. *et al*., 2008]. Since patient-1 suffered from depression for 10 years and was regularly taking desvenlafaxine (50mg/d) and bromazepam (3mg/d), we should know whether impaired metabolization and excretion of these drugs occurred during the SARS-CoV-2 infection and could be made responsible for UI in the index

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case. Overflow incontinence has been reported with carbamazepine. There is also a report of a patient having stress incontinence due to phenobarbital, one of the regular medications of patient-2 [Hoyt, H. S, 1949].

Since patient-2 had a history of incontinence, we should know how to differentiate UI due to SARS-CoV-2 from exacerbation of previous incontinence.

In summary, the excellent study has limitations that should be addressed before drawing final conclusions. Clarifying the weaknesses would strengthen the conclusions and could improve the study. Before UI in females can be attributed to SARS-CoV-2 infections, the wide range of differential causes must be ruled out.

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