LETTERS: NEW OBSERVATION

Seroprevalence of SARS-CoV-2 in Parkinson's Disease Patients: A Case–Control Study

A novel coronavirus, the so-called "Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV-2)," caused the ongoing pandemic, which was initially identified in Wuhan, China.¹ Undefined rates of asymptomatic infections have raised concerns about a possibly high frequency of undiagnosed infections of SARS-CoV-2.² The impact of the COVID-19 pandemic on patients with Parkinson's disease (PD) is yet to be determined.³ Studies showed diverse prevalence and outcomes among PD patients.^{4,5}

Assessing a precise approximation of the prevalence of COVID-19 necessitates testing antibodies in people who are not symptomatic.⁶ Therefore, the aim of this study is to evaluate the seroprevalence of SARS-CoV-2 among PD patients who did not have the symptomatic infection.

The Iran National Committee for Ethics in Biomedical Researches approved this cross-sectional, case–control study (IR.SBMU.RETECH.REC.1399.1228) [Correction added on

	PD patients	PD patients mean $IgG \pm SD$	Healthy controls	Controls mean $IgG \pm SD$	IgG ratio comparison
Patients n	90	5 15 + 5 21	97	4 97 + 4 44	$P \approx 0.00086$
Men n (%)	66(73,33)	1.56 ± 3.14	48(49.5)	0.91 ± 2.58	$P \approx 0.003$
Women, n (%)	24(26.67)	1.45 ± 3.96	49(50.5)	0.71 ± 1.71	$P \approx 0.19$
Mean age, years $+$ SD	57.68 ± 13.99		58.97 ± 19.65	• ±	
Age classification, n (%)					$P > 0.05^{a}$
<50 years	26(28,88)	1.34 ± 3.74	35(36.08)	0.67 ± 1.94	1 / 0100
50–70 years	46(51,11)	1.74 ± 3.48	31(31.95)	0.87 ± 2.79	
>70 years	18(20)	1.28 ± 2.46	31(31.95)	0.89 ± 1.74	
Mean disease duration, n (%)			01(01100)		$P > 0.05^{a}$
<3 vears	27(30)	1.82 ± 4.02			
3–10 years	34(37,77)	1.41 ± 2.47			
>10 years	29(32.22)	1.41 ± 3.68			
Underlying disease, n (%)	()				$P \approx 0.18$
Hypertension	20(22.22)	1.55 ± 2.58	51(52.57)	0.93 ± 2.06	
Medication. n (%)	()				<i>P</i> > 0.05
Amantadine	25(27.77)	1.99 ± 4.55			
Levodopa	80(88.88)	1.28 ± 2.83			<i>P</i> > 0.05
Direct contact with Covid-19-confirmed patients, n (%)	24(26.66)	0.97 ± 1.67	11(11.34)	0.37 ± 0.32	$P \approx 0.78$
No direct contact with Covid-19-confirmed patients, n (%)	66(73.33)	1.74 ± 3.78	86(88.65)	$\textbf{0.86} \pm \textbf{2.30}$	<i>P</i> < 0.00001
No direct contact with Covid-19-confirmed patients and positive IgG test, n (%)	19(28.7)	5.30 ± 5.72	11(12.7)	5.30 ± 4.50	$P \approx 0.067$ (proportion comparison) $P \approx 0.91$ (positive IgG comparison)
Duration of contact, n (%)					<i>P</i> > 0.05 ^a
<1 week	6(25)	1.64 ± 2.45	6(54.55)	0.44 ± 0.42	
1 week	5(20.83)	0.89 ± 1.46	0(0)		
>1 week	13(54.16)	0.69 ± 1.35	5(45.45)	0.28 ± 0.13	

TABLE 1. Characteristics of PD patients and control group

^aComparison between PD and control subgroups. IgG: negative, <0.9; borderline, 0.9–1.1; and positive, >1.1.

PD, Parkinson's disease; SD, standard deviation.

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Key Words: Parkinson's disease; SARS-CoV-2; seroprevalence

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7 June, 2021 after first online publication: In the preceding sentence, the approval number was updated.]. All PD patients who had visited Shohada-e-Tajrish University Hospital, a referral Movements Disorders center, in December 2020, when Iran had experienced the third wave of COVID-19 pandemic, were enrolled in the study. Patients who had the symptomatic infection with SARS-CoV-2 were excluded, and after signing the informed consent forms, patients completed a questionnaire that included demographics data; PD-related information; comorbid conditions; details regarding having had close contact with SARS-CoV-2-infected individuals; and symptoms such as anosmia, cough, coryza, fever, malaise, and musculoskeletal pain. A blood sample was taken to check IgG antibodies for SARS-CoV-2 IgG. Also, blood serum of healthy controls who did not have the symptomatic infection during the same time was checked for SARS-CoV-2 IgG.

All blood samples were tested using the enzyme-linked immunosorbent assay (ELISA) technique, using commercially accessible kits (SARS-CoV-2. IgG 96 Elisa Kit. Ideal, Tehran, Iran), with a sensitivity of 81.82% and a specificity of 94.83%.

A total of 90 subjects who were identified with PD and 97 healthy controls were included in the study. Table 1 presents the demographic, disease-associated, and COVID-19-related data. The difference in the proportion of variables is compared using Fisher's exact test, and the difference in mean IgG ratio in different groups is compared using the *z* score and Mann-Whitney *U* test depending on the distribution of data. About 25.56% of PD patients and 12.37% of controls tested positive for SARS-CoV-2 IgG antibody, and these proportions were significantly different (P < 0.05). The mean total IgG ratio was 1.53 ± 3.36 and 0.80 ± 2.17 in PD and control groups, respectively, and the difference was statistically significant (P < 0.01).

There was no statistical difference between the IgG ratio of PD patients and the control group who had direct contact with SARS-CoV-2-positive individuals (P > 0.05). Nevertheless, we found a statistically significant difference between the IgG ratio of PD patients and control group who had not direct contact with SARS-CoV-2-positive individuals (P < 0.00001). Moreover, the proportion of PD patients with positive IgG test who had no direct contact with Covid-19 patients was significantly higher than that of the same individuals in the control group (P < 0.05).

A study conducted in the United Kingdom supports our outcome⁴; nevertheless, another study conducted in Italy showed that PD patients do not pose a higher risk of SARS-CoV2 infection,⁵ but this study evaluated patients based on having an asymptomatic infection.

Therefore, the result of the current study indicates that PD patients can be more susceptible to Covid-19 infection. But more studies with higher sample sizes should be performed to confirm these results.

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