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Neuropsychiatric symptoms in post-COVID-19 long haulers

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Original Article

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Abstract

Background: Long haulers have been recently reported after contracting coronavirus disease (COVID-19). In the present study, we aimed to screen for the neuropsychiatric signs detected <1 to >6 months after infection by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and to determine whether vaccination has an effect on them. *Methods:* An online survey was conducted among participants who had been diagnosed with laboratory-confirmed SARS-CoV-2 infection. The clinical signs and durations of neuropsychiatric complaints and their correlations to sex, age, severity of COVID-19 signs, and vaccination status were screened. *Results:* A total of 2218 individuals, including 1358 females and 860 males, with an age range of 12–70 years, submitted their responses. The respondents experienced cognitive dysfunction, mood alteration, depression, tinnitus, sleep disorders, and loss of taste and smell, with prevalence rates ranging from 18.9% (tinnitus) to 63.9% (loss of taste and smell). Of the respondents, 2.2–7.7% confirmed the persistence of symptoms for >6 months. Tinnitus was the least common complaint, and only 2.2% of the study participants had tinnitus for >6 months. Meanwhile, mood alteration persisted for >6 months in 7.6% of the study participants. More respondents who received two doses of BNT162b2 vaccine showed persistent symptoms than those in the other groups. Disease severity and female sex were identified as potential determinants of the development and persistency of such symptoms. *Conclusion:* Post-COVID neuropsychiatric symptoms were present in considerable percentages of the study participants with SARS-CoV-2 infection, persisting for >6 months in up to 7.6% of the participants.

Significant outcomes

- Post-COVID neuropsychiatric symptoms, including altered cognitive skills, anosmia and dysgeusia, tinnitus, depression, and sleep disorders, were recorded in 18.9–63.9% of the participants with COVID-19.
- The signs persisted from 2 to >6 months in 5.9–17.9% of the participants, of whom 2.2–7.6% experienced such signs for >6 months.
- Disease severity, female sex, and receiving two doses of the vaccine correlated with the development of the neuropsychiatric signs.

Limitations

- Participants were recruited using social media rather than through primary care, so self-reporting might have influenced the results.
- Differences in interpretation depending on the patient or inaccurate information, including misclassification, were possible.

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the causative agent of the coronavirus disease that emerged in 2019 (COVID-19). Unlike other viral respiratory pathogens, SARS-CoV-2 was not expected to have chronic manifestations (Hu *et al.*, 2021). In the beginning, physicians related post-COVID-19 syndromes to anxiety or stress, which is known as medical gaslighting (Rubin, 2020). Afterwards, post-COVID-19 syndrome or long-term COVID-19, also known as long-haul COVID-19, was used to describe post-COVID signs (Schmidt, 2021). Between 10% and 30% of patients with SARS-CoV-2 infection experienced prolonged health problems, many of whom were never hospitalised or severely ill (Schmidt, 2021). The disease condition can be mild or severe depending on the organ involved. Some patients complained of extreme fatigue, cognitive impairment

(brain fog) or loss of taste and/or smell, chest pain, palpitations, sleep disturbance, and depression (Carfì *et al.*, 2020; Schmidt, 2021). Similar long-term symptoms were reported after the 2002 SASR-CoV infection (Moldofsky & Patcai, 2011). Prolonged mixed complaints of respiratory, cardiac, and/or neurological signs were reported in many patients (Carfì *et al.*, 2020; Ellul *et al.*, 2020; Fraser, 2020).

Growing evidence indicates the neuroinvasive potentiality of SARS-CoV-2. It is currently well established that both SARS-CoV and SARS-CoV-2 use angiotensin-converting enzyme 2 as a cell entry receptor (Hoffmann *et al.*, 2020), which is also expressed in olfactory mucosal cells, the epithelia of the tongue papillae and taste buds (Brann *et al.*, 2020; Park *et al.*, 2022), and human neuronal cells(Khan & Gomes, 2020). These findings could explain the neurological signs detected in patients with COVID-19, including headache, fatigue, sleep disorders, dizziness, ageusia, anosmia, and altered mood (Delavari *et al.*, 2021). On the other hand, psychopathology is among the neglected aspects of COVID-19, especially in patients with pre-existing psychiatric disorders (Goldstein Ferber *et al.*, 2021). Many reports have confirmed that psychopathological syndrome leads healthcare professionals to directions regarding the detection and management of people with long-term COVID (Mair and May, 2014; Colizzi *et al.*, 2020; Greenhalgh *et al.*, 2020; Troyer *et al.*, 2020; Delavari *et al.*, 2021). A study of different populations in China, Denmark, Iran, Italy, Nepal, Spain, Turkey, and the USA revealed a considerably wide range of variation in the prevalence of neuropsychiatric disorders in the general population during the COVID-19 pandemic (Xiong *et al.*, 2020); however, information regarding the Saudi population is inadequate. The aim of the present study was to screen for persistent neuropsychiatric disorders and conditions affecting the peripheral nerves from <1 to >6 months after COVID-19 infection. Possible correlations between initial disease severity and vaccination status were also investigated.

Methods

Study group

Laboratory-confirmed SARS-CoV-2 infection, as evidenced by a positive real-time reverse transcriptase-polymerase chain reaction test result, was the inclusion criterion for answering the questionnaire. The participants (Saudi nationals and residents) were asked to provide their demographic data and COVID-19 status in terms of severity, graded into asymptomatic (laboratory-confirmed cases without clinical signs), mild (mild disease manifestations, 1–3 days), moderate (tolerable signs for up to 1 week), severe (severe signs but did not require hospitalisation), and critical (hospitalised). The questions included items on SARS-CoV-2 vaccination status, the number of vaccine doses, the type of vaccine, and whether they experienced any chronic diseases or immunosuppressive disorders. The participants were then asked about the presence or absence of different neuropsychiatric signs. Those who answered yes were then asked about the signs, including cognitive dysfunction, mood alteration, depression, tinnitus, sleep disorders (insomnia and hypersomnia), and loss of taste (dysgeusia), and smell (anosmia). They were also asked about the duration (<1 to >6 months) of each complaint. Different social media platforms (Twitter, Snapchat, and WhatsApp) were used to distribute the questionnaire among Saudi nationals and residents.

Statistical analysis

Descriptive analysis using Crosstabs procedures was used to display the frequencies and measure the difference between the observed and expected frequencies. Each value was presented as a number with its percentage. Chi-square and Spearman's rho correlation analyses using the SPSS software programme were performed to screen the difference and correlation between the variables.

Results

Cognitive dysfunction

In total, 615 (27.7%) of the 2218 study participants, including 426 females (31.4% of 1358) and 189 males (22.0% of 860), showed cognitive dysfunction after SARS-CoV-2 infection (Table 1, Fig. 1). Most males (671/860, 78.02%) and females (932/1358, 68.63%) did not experience any cognitive dysfunction after SARS-CoV-2 infection, and only 615 (27.7%) of the 2218 participants had cognitive dysfunction (Table 1). A significant difference ($p < 0.001$) was found between the males and females, and a significant correlation ($R = -0.102$) was found between the development of cognitive dysfunction and female sex. Of all the participants, 318 (14.3%), including 220 females (16.2% of 1358) and 98 males (11.4% of 860), experienced cognitive dysfunction from 2 to >6 months. Of the 1358 females and 860 males, 83 (6.1%) and 36 (4.19%) experienced such complaints for >6 months, respectively (Table 1, Fig. 1).

A significant difference ($p < 0.001$) was found between the degree of disease severity and the development of cognitive dysfunction after SARS-CoV-2 infection. Among those who had asymptomatic, mild, moderate, severe, and critical disease, 9.6% (24/251), 19.0% (115/606), 28.0% (228/813), 45.0% (227/505), and 48.8% (21/43) developed cognitive dysfunction after SARS-CoV-2 infection, respectively. The cognitive dysfunction persisted for 2 to >6 months in 3.2% (8/251), 10.4% (63/606), 14.5% (118/813), 23.6% (119/505), and 25.6% (11/43) of the participants in the severity groups, respectively (Table 1). The higher the disease severity, the higher the percentage of participants with cognitive dysfunction ($R = 0.252$). No significant differences were observed with regard to age, vaccination status, and type of administered vaccine. However, the proportion of participants who developed cognitive dysfunction was higher among those whose ages were within the range of 21–30 years and those who were vaccinated with two doses of the BNT162b2 vaccine (Table 1).

Mood alteration

A total of 951 (42.8%) of the 2218 study participants, including 649 females (47.8% of 1358) and 302 males (35.1% of 860), had mood alteration after SARS-CoV-2 infection (Table 2, Fig. 1). The females were more significantly affected than the males ($p < 0.001$), with a considerably significant correlation ($R = -0.136$) between the development of mood alteration and the female sex. Of all the participants, 398 (17.9%) had mood alteration from 2 to >6 months, including 292 females (21.5% of 1358) and 106 males (12.3% of 860). Meanwhile, 168 (7.6%; 125 [9.2%] of the 1358 females and 43 [5%] of the 860 males) of the 2218 participants had such complaints for >6 months (Fig. 1, Table 2).

A significant difference ($p < 0.001$) was found between the degree of disease severity and the development of mood alteration after SARS-CoV-2 infection. Of the participants who had

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Table 1. The effect of the sex, age, disease severity, and vaccine type on the development of cognitive dysfunction following SARS-CoV-2 infection

Variables		None (n:1603)	Duration of cognitive dysfunction after COVID-19 infection (Months)									Total (n:2218)	Chi-square and correlation
			<1 (n:153)	1(n:144)	2(n:85)	3(n:55)	4(n:25)	5(n:13)	6(n:21)	>6 (n:119)	Cumulative		
Sex	Female	932 (68.6)	104(7.7)	102(7.5)	56(4.1)	37(2.7)	17(1.3)	12(0.9)	15(1.1)	83(6.1)	426(31.4)	1358(61.2)	[a]$p < 0.001$, [b]$R = -0.102$
	Male	671 (78.0)	49 (5.7)	42 (4.9)	29 (3.4)	18(2.1)	8(0.9)	1(0.1)	6(0.7)	36(4.2)	189(22.0)	860(38.8)	
Age (Years)	12–17	100 (65.4)	17(11.1)	9(5.9)	5(3.3)	5(3.3)	2(1.3)	0(0.0)	1(0.65)	14(9.2)	53(34.6)	153(6.9)	[a]$p < 0.428$, [b]$R = -0.005$.
	18–20	284(72.2)	34(8.7)	20(5.1)	16(4.1)	6(1.5)	5(1.3)	2(0.5)	4(1.0)	22(5.6)	109(27.8)	393(17.7)	
	21–30	621(74.2)	45(5.4)	58(6.9)	26(3.1)	21(2.5)	10(1.2)	7(0.8)	5(0.6)	44(5.3)	216(25.8)	837(37.7)	
	31–40	315(72.7)	27(6.2)	27(6.2)	16 (3.7)	12(2.8)	6 (1.4)	3(0.7)	5(1.2)	22(5.1)	118(27.3)	433(19.5)	
	41–50	208(72.0)	17(5.9)	15(5.2)	17(5.9)	9(3.1)	2(0.7)	1(0.3)	5(1.7)	15(5.2)	81(28.0)	289(13.0)	
	51–60	63(67.0)	10(10.6)	12(12.8)	4(4.3)	2(2.1)	0(0.0)	0(0.0)	1(1.1)	2(2.1)	31(33.0)	94 (4.0)	
	61–70	12(63.2)	3(15.8)	3(15.8)	1(5.3)	0(0.0)	0 (0.0)	0 (0.0)	0(0.0)	0(0.0)	7(36.8)	19(0.9)	
Severity	Asymptomatic	227(90.4)	10(4.0)	6(2.4)	1(0.4)	2(0.8)	0(0.0)	0(0.0)	1(0.4)	4(1.6)	24(9.6)	251(11.3)	[a]$p < 0.001$, [b]$R = -0.252$
	Mild	491(81.0)	21(3.5)	31(5.1)	20(3.3)	9(1.5)	6(1.0)	1(0.2)	2(0.3)	25(4.1)	115(19.0)	606(27.3)	
	Moderate	585(72.0)	61(7.5)	50(6.2)	28(3.4)	25(3.1)	8(1.0)	8(1.0)	9(1.1)	39(4.8)	228(28.0)	813(36.7)	
	Severe	278 (55.0)	56(11.1)	52(11.5)	34(6.7)	16(3.2)	11(2.2)	4(0.8)	9(1.8)	45(8.9)	227(45.0)	505(22.8)	
	Critical	22 (51.2)	5(11.6)	5(11.6)	2(4.7)	3(7.0)	0(0.0)	0(0.0)	0(0.0)	6(14.0)	21(48.8)	43(1.9)	
Vaccine	Not vaccinated	118(72.8)	13(8.0)	12(7.4)	6(3.7)	2(1.2)	2(1.2)	2(1.2)	2(1.2)	5(3.1)	44(27.2)	162(7.3)	[a]$p < 0.898$, [b]$R = -0.026$
	Single dose, ChAdOx1	61(64.2)	7(7.4)	11(11.6)	3(3.2)	4(4.2)	2(2.1)	0(0.0)	1(1.1)	6(6.3)	34(35.8)	95(4.3)	
	Two doses, ChAdOx1	166(70.9)	16(6.8)	16(6.8)	11(4.7)	3(1.3)	4(1.7)	0(0.0)	2(0.9)	16(6.8)	68(29.1)	234(10.6)	
	Single dose, BNT162b2	199(75.1)	13(4.9)	16(6.0)	11(4.2)	11(4.2)	1(0.4)	0(0.0)	3(1.1)	11(4.2)	66(24.9)	265(11.9)	
	Two doses, BNT162b2	820 (72.7)	76(6.7)	69(6.1)	40(3.5)	29(2.6)	12(1.1)	9(0.8)	10(0.9)	63(5.6)	308(27.3)	1128(50.9)	
	ChAdOx1/BNT162b2	239(71.6)	28(8.4)	20(6.0)	14(4.2)	6(1.8)	4(1.2)	2(0.6)	3(0.9)	18(5.4)	95(28.4)	334(15.1)	

[a]Chi-square, [b]Spearman's correlation (*R*). The values are listed as numbers, while the percentage was estimated based on the total number of the same row except for the last column that was calculated as the total number of the same column.

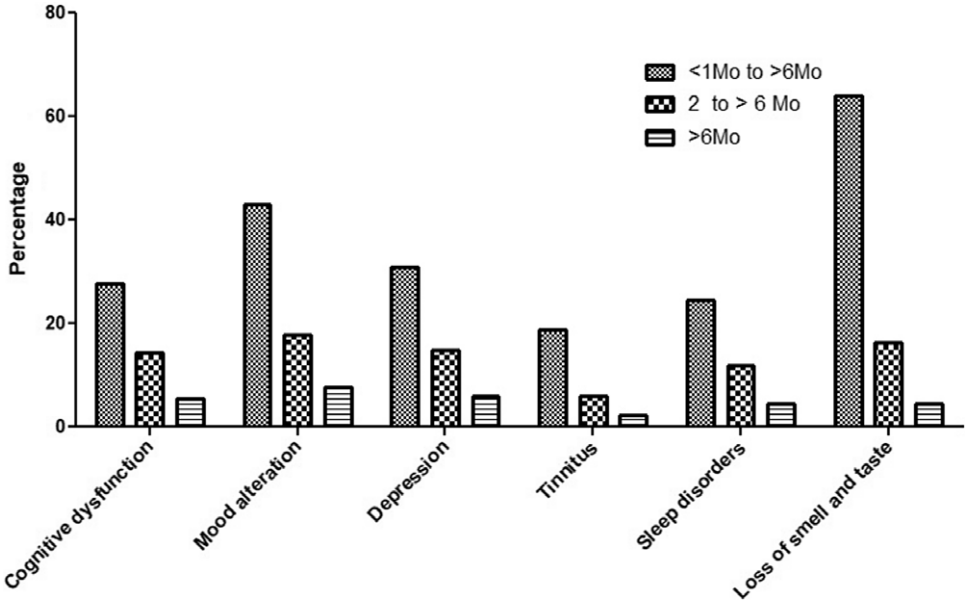

Fig. 1. Neuropsychiatric signs after COVID-19 infections. The percentages of the patients who developed different signs and those who experienced such signs for 2 to >6 months and >6 months are presented.

asymptomatic, mild, moderate, severe, and critical disease, 27.1% (68/251), 30.4% (184/606), 46.1% (375/813), 59.0% (298/505), and 60.5% (26/43) developed mood alteration after SARS-CoV-2 infection, respectively. The mood alteration persisted for 2 to >6 months in 8.0% (20/251), 11.9% (72/606), 18.6% (151/813), 28.3% (143/505), and 27.9% (12/43) of the participants in the severity groups, respectively. The percentage of patients who experienced mood alteration was found to be high in those with mild, moderate, and severe disease ($R = -0.242$; Table 2). No significant differences were observed with regard to age, vaccination status, and type of vaccine. However, the proportion of participants who experienced mood alteration was higher among those whose ages were within the range of 21–30 years and those vaccinated with two doses of the BNT162b2 vaccine (Table 2).

Depression

A total of 686 (30.9%) of the 2218 participants developed depression (Fig. 1, Table 3). A significant difference according to sex was observed, as 34.7% (471/1358) of the females and 25.0% (215/860) of the males developed depression ($p < 0.001$). Meanwhile, of all the participants, 331 (14.9%), including 194 females (14.3% of 1358) and 67 males (7.8% of 860), had depression for 2 to >6 months, of whom 130 (5.9%) had depression for >6 months.

Although the incidence of depression was not significantly different ($p < 0.06$) among the age groups, the participants in the 21- to 30-year-old age group showed the highest incidence of depression, followed by those in the 18- to 20-year-old and 31- to 40-year-old age groups (Table 3).

Disease severity significantly affected the development of depression in the respondents ($p < 0.001$). Of those who had asymptomatic, mild, moderate, severe, and critical disease, 22.7% (57/251), 21.0% (127/606), 31.9% (259/813), 44.0% (222/505), and 48.8% (21/43) developed depression after SARS-CoV-2 infection, respectively. The depression persisted for 2 to >6 months in 10.8% (27/251), 8.3% (50/606), 14.3% (116/813), 25.1% (127/505), and 23.3% (10/43) of the participants in the severity groups, respectively. Only 130 (5.9%) of all the participants developed sleeping disorders for >6 months. A significant correlation was found between disease severity and the development of sleeping disorders ($R = -0.19$; Table 3). A significant increase in the incidence of depression was observed in the group of participants who received two doses of the BNT162b2 vaccine compared with the other groups ($p < 0.044$).

Tinnitus

A total of 420 (18.9%) of the 2218 participants developed tinnitus (Table 4, Fig. 1). A considerable number of respondents developed tinnitus (284/1358 females, 20.9% and 136/860 males, 15.8%; $p < 0.031$), of whom 131 (5.9% of 2218), including 87 females (6.4% of 1358) and 44 males (5.1% of 860), had tinnitus for 2 to >6 months (Table 4, Fig. 1), and most had tinnitus for up to 1 month. A few cases were detected 1 month after COVD-19 infection (Table 4). Only 48 (2.2%) of the 2218 participants had tinnitus for >6 months (Table 4, Fig. 1). Tinnitus occurred among those whose ages were within the range of 18–50 years ($p < 0.007$).

Disease severity significantly influenced the development of tinnitus in the respondents ($p < 0.001$). Tinnitus developed after SARS-CoV-2 infection in 13.5% (34/251), 13.7% (83/606), 19.3% (157/813), 26.3% (133/505), and 30.2% (13/43) and persisted for 2 to >6 months in 1.2% (3/251), 4.8% (29/606), 5.7% (46/813), 9.1% (46/505), and 16.3% (7/43) of the participants who had asymptomatic, mild, moderate, severe, and critical disease, respectively.

No significant differences were observed with regard to vaccination status and type of vaccine. However, more than half of the study participants who reported tinnitus (219/420) received two doses of the BNT162b2 vaccine (Table 4).

Sleep disorders

Of the 2218 participants, 541 (24.4%) developed sleep disorders, including insomnia (341/2218, 15.4%) and hypersomnia (200/2218, 9.0%). Most females ($n = 230$) and males (n = 111) experienced insomnia, and 138 females and 62 males had hypersomnia. A significant difference according to sex was observed, as 368 (27.1%) of the 1358 females and 173 (20.1%) of the 860 males developed sleeping disorders ($p < 0.001$). Meanwhile, 261 (11.8%) of all the participants, including 194 females (14.3% of

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Table 2. The effect of the sex, age, disease severity, and vaccine type on the development of mood alteration following SARS-CoV-2 infection

Variables		None (n:1603)	Duration of mood alteration after COVID-19 infection (Months) <1 (n:153)	1 (n:144)	2 (n:85)	3 (n:55)	4 (n:25)	5 (n:13)	6 (n:21)	>6 (n:119)	Cumulative	Total (n:2218)	
Sex	Female	709 (52.2)	143(10.5)	214(15.8)	84(6.2)	34(2.5)	21(1.5)	15(1.1)	13(1.0)	125(9.2)	649(47.8)	1358(61.2)	[a]$p < 0.001$, [b]$R = -0.136$
	Male	558 (64.9)	82 (9.5)	114 (13.3)	28(3.3)	17(2.0)	9(1.0)	1(0.1)	8(0.9)	43(5.0)	302(35.1)	860(38.8)	
Age (Years)	12–17	82(53.6)	23 (15.0)	17(11.1)	4(2.6)	5(3.3)	3(2.0)	3(2.0)	1(0.7)	15(9.8)	71(46.4)	153 (6.9)	[a]$p < 0.579$, $R = -0.035$
	18–20	216(55.0)	45(11.5)	58(14.6)	19(4.8)	8(2.0)	6(1.5)	3(0.8)	7(1.8)	31(7.9)	177(45.0)	393(17.7)	
	21–30	481(57.5)	76(9.1)	128(15.3)	45(5.4)	20(2.4)	10(1.2)	9(1.1)	4(0.5)	64(7.6)	356(42.5)	837(37.7)	
	31–40	251(58.0)	38(8.8)	60(13.9)	22(5.1)	8(1.8)	9(2.1)	0(0.0)	6(1.4)	39(9.0)	182(42.0)	433(19.5)	
	41–50	162(56.1)	34(11.8)	47(16.3)	17(5.9)	8(2.8)	2(0.7)	1(0.3)	2(0.7)	16(5.5)	127(43.9)	289(13.0)	
	51–60	62(66.0)	7(0.8)	15(0.2)	5(5.3)	2(2.1)	0(0.0)	0(0.0)	1(1.1)	2(2.1)	32(34.0)	94(4.2)	
	61–70	13(68.4)	2(10.5)	3(15.8)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	1(5.3)	6(31.6)	19(0.9)	
Severity	Asymptomatic	183(72.9)	16(6.4)	32(12.7)	3(1.2)	4(1.6)	2(0.8)	0(0.0)	2(0.8)	9(3.6)	68(27.1)	251(11.3)	[a]$p < 0.001$, [b]$R = -0.242$
	Mild	422(69.6)	49(8.1)	63(10.4)	22(3.6)	9(1.5)	6(1.0)	3(0.5)	1(0.2)	31(5.1)	184(30.4)	606(27.3)	
	Moderate	438(53.9)	93(11.4)	131(16.1)	42(5.2)	21(2.6)	11(1.4)	9(1.1)	11(1.4)	57(7.0)	375(46.1)	813(36.7)	
	Severe	207(41.0)	63(12.5)	92(18.2)	41(8.1)	15(3.0)	11(2.2)	4(0.8)	7(1.4)	65(12.9)	298(59.0)	505(22.8)	
	Critical	17(39.5)	4(9.3)	10(23.3)	4(9.3)	2(4.7)	0(0.0)	0(0.0)	0(0.0)	6(14.0)	26(60.5)	43(1.9)	
Vaccine	Not vaccinated	77(47.5)	30(18.5)	24(14.8)	8(4.9)	4(2.5)	3(1.9)	2(1.2)	2(1.2)	12(7.4)	85(52.5)	162(7.3)	[a]$p < 0.127$, [b]$R = -0.024$
	Single dose, ChAdOx1	52(54.7)	7(7.4)	17(17.9)	8(8.4)	3(3.2)	2(2.1)	0(0.0)	0(0.0)	6(6.3)	43(45.3)	95(4.3)	
	Two doses, ChAdOx1	133 (56.8)	20(8.5)	41(17.5)	8(3.4)	5(2.1)	4(1.7)	0(0.0)	5 2.1)	18(7.7)	101(43.2)	234(10.6)	
	Single dose, BNT162b2	149(56.2)	30(11.3)	31(11.7)	19(7.2)	8(3.0)	7(2.6)	1(0.4)	2(0.8)	18(6.8)	116(43.8)	265(11.9)	
	Two doses, BNT162b2	662(58.7)	105(9.3)	169(15.0)	50(4.4)	27(2.4)	10(0.9)	11(1.0)	7(0.6)	87(7.7)	466(41.3)	1128(50.9)	
	ChAdOx1/BNT162b2	194(58.1)	33(9.9)	46(13.8)	19(5.7)	4(1.2)	4(1.2)	2(0.6)	5(1.5)	27(8.1)	140(41.9)	334(15.1)	

[a]Chi-square, [b]Spearman's correlation (*R*). The values are listed as numbers, while the percentage was estimated based on the total number of the same row except for the last column that was calculated as the total number of the same column.

Table 3. Effect of sex, age, disease severity, and vaccine type on the development of depression post-SARS-CoV-2 infection

Item		None (n:1532)	Duration of depression after COVID-19 infection (Months) <1 (n:156)	1 (n:199)	2 (n:105)	3 (n:44)	4 (n:22)	5 (n:10)	6 (n:20)	>6 (n:130)	Cumulative	Total (n:2218)	Chi-square and correlation
Sex	Female	887 (65.3)	101(7.4)	136(10.0)	70(5.2)	30(2.2)	14(1.0)	10(0.7)	16(1.2)	94(6.9)	471(34.7)	1358(61.2)	[a]$p < 0.001$, [b]$R = 0.08$
	Male	645 (75.0)	55(6.4)	63(7.3)	35(4.1)	14(1.6)	8(0.9)	0(0.0)	4(0.5)	36(4.2)	215(25.0)	860(38.8)	
Age (Years)	12–17	98(64.1)	16(10.5)	12(7.8)	7(4.6)	2(1.3)	5(3.3)	0(0.0)	1(0.7)	12(23.5)	55(35.9)	153(6.9)	[a]$p < 0.06$, [b]$R = -0.017$
	18–20	273 (69.5)	31(7.9)	26(6.6)	18(4.6)	5(1.3)	7(1.8)	0(0.0)	7(1.8)	26(6.6)	120(30.5)	393(17.7)	
	21–30	585 (69.9)	46(5.5)	76(9.1)	38(4.5)	18(2.2)	8(1.0)	7(0.8)	6(0.7)	53(6.3)	252(30.1)	837(37.7)	
	31–40	301(69.5)	30(6.9)	38(8.8)	27(6.2)	9(2.1)	0(0.0)	0(0.0)	3(0.7)	25(5.8)	132(30.5)	433(19.5)	
	41–50	194 (67.1)	22(7.6)	39(13.5)	8(2.8)	8(2.8)	2(0.7)	3(1.0)	3(1.0)	10(3.5)	95(32.9)	289(13.0)	
	51–60	67 (71.3)	8(8.5)	7(7.4)	6(6.4)	2(2.1)	0(0.0)	0(0.0)	0(0.0)	4(4.3)	27(28.7)	94(4.2)	
	61–70	14 (73.7)	3(15.8)	1(5.3)	1(5.3)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	5(26.3)	19(0.9)	
Severity	Asymptomatic	194 (77.3)	10(4.0)	20(8.0)	6(2.4)	3(1.2)	3(1.2)	1(.04)	2(0.7)	12(4.8)	57(22.7)	251(11.3)	[a]$p < 0.001$, [b]$R = -0.190$
	Mild	479 (79.0)	32(5.3)	45(7.4)	15(2.5)	3(0.5)	4(0.7)	1(0,2)	1(0.2)	26(4.3)	127(21.0)	606 (27.3)	
	Moderate	554 (68.1)	70(8.6)	73(9.0)	38(4.7)	17(2.1)	7(0.9)	4(0.5)	7(0.86)	43(5.3)	259(31.9)	813(36.7)	
	Severe	283(56.0)	41(8.1)	54(10.7)	44(8.7)	19(3.8)	8(1.6)	4(0.8)	9(1.8)	43(8.5)	222(44.0)	505(22.8)	
	Critical	22(51.2)	3(7.0)	7(16.3)	2(4.7)	2(4.7)	0(0.0)	0(0.0)	1(2.3)	6(14.0)	21(48.8)	43(1.9)	
Vaccine	Not vaccinated	102(63)	16(9.9)	18(11.1)	6(3.7)	6(3.7)	2(1.2)	1(0.6)	2(1.2)	9(5.6)	60(37.0)	162(7.3)	[a]$p < 0.039$, [b]$R = -0.044$
	Single dose, ChAdOx1	59(62.1)	7(7.4)	8(8.4)	10(10.5)	5(5.3)	0(0.0)	1(1.1)	0(0.0)	5(5.3)	36(37.9)	95(4.3)	
	Two doses, ChAdOx1	150(64.1)	19(8.1)	22(9.4)	14(6.0)	4(1.7)	2(0.9)	0(0.0)	5(2.1)	18(7.7)	84(35.9)	234(10.6)	
	Single dose, BNT162b2	172(64.9)	27(10.2)	32(12.1)	13(4.9)	6(2.3)	1(0.4)	2(0.8)	3(1.1)	9(3.4)	93(35.1)	265(11.9)	
	Two doses, BNT162b2	817(72.4)	65(5.8)	92(8.2)	41(3.6)	20(1.8)	14(1.2)	4(0.4)	8(0.7)	67(5.9)	311(27.6)	1128(50.9)	
	ChAdOx1/BNT162b2	232(69.5)	22(6.6)	27(8.1)	21(6.3)	3(0.9)	3(0.9)	2(0.6)	2(0.6)	22(6.6)	102(30.5)	334(15.1)	

[a]Chi-square, [b]Spearman's correlation (*R*). The values are listed as numbers, while the percentage was estimated based on the total number of the same row except for the last column that was calculated as the total number of the same column.

Table 4. The effect of the sex, age, disease severity, and vaccine type on the development of tinnitus following SARS-CoV-2 infection

				Duration of tinnitus after COVID-19 infection (Months)									
Variables		None (n:1798)	<1 (n:175)	1 (n:114)	2 (n:28)	3 (n:22)	4 (n:13)	5 (n:5)	6 (n:15)	>6 (n:48)	Cumulative	Total (n:2218)	Chi-square and correlation
Sex	Female	1074(79.1)	113(8.3)	84(6.2)	20(1.5)	13(1.0)	7(0.5)	5(0.4)	8(0.6)	34(2.5)	284(20.9)	1358(61.2)	[a]$p < 0.031$, [b]$R = -0.064$
	Male	724(84.2)	62(7.2)	30(3.5)	8(0.9)	9(1.0)	6(0.7)	0(0.0)	7(0.8)	14(1.6)	136(15.8)	860(38.8)	
Age	12–17	104(68.0)	23(2.7)	7(4.6)	5(3.3)	1(0.7)	1(0.7)	0(0.0)	4(2.6)	8(5.2)	49(32.0)	153(6.9)	[a]$p < 0.007$, [b]$R = -0.05$
	18–20	316(80.4)	27(6.9)	25(6.4)	5(1.3)	3(0.8)	3(0.8)	2(0.5)	2(0.5)	10(2.5)	77(19.6)	393(17.7)	
	21–30	692(82.7)	60(7.2)	45(5.4)	5(0.6)	7(0.8)	4(0.5)	3(0.4)	4(0.5)	17(2.0)	145(17.3)	837(37.7)	
	31–40	360(83.1)	34(7.9)	14(3.2)	6(1.4)	5(1.2)	1(0.2)	0(0.0)	4(0.9)	9(2.1)	73(16.9)	433(19.5)	
	41–50	232(80.3)	20(6.9)	18(6.2)	6(2.1)	6(2.1)	4(1.4)	0(0.0)	1(0.3)	2(0.7)	57(19.7)	289(13.0)	
	51–60	80(85.1)	6(6.4)	5(5.3)	1(1.1)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	2(2.1)	14(14.9)	94(4.2)	
	61–70	14(73.7)	5(26.3)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	5(26.3)	19(0.9)	
Severity	Asymptomatic	217(86.5)	17(6.8)	14(5.6)	0(0.0)	1(0.4)	0(0.0)	1(0.4)	0(0.0)	1(0.4)	34(13.5)	251(11.3)	[a]$p < 0.001$, [b]$R = -0.129$
	Mild	523(86.3)	35(5.8)	19(3.1)	6(1.0)	3(0.5)	3(0.5)	1(0.2)	6(1.0)	10(1.7)	83(13.7)	606(27.3)	
	Moderate	656(80.7)	64(7.9)	47(5.8)	13(1.6)	9(1.1)	4(0.5)	1(0.1)	3(0.4)	16(2.0)	157(19.3)	813(36.7)	
	Severe	372(73.7)	56(11.1)	31(6.1)	7(1.4)	8(1.6)	5(1.0)	2(0.4)	4(0.8)	20(4.0)	133(26.3)	505(22.8)	
	Critical	30(69.8)	3(7.0)	3(7.0)	2(4.7)	1(4.3)	1(4.3)	0(0.0)	2(4.7)	1(4.3)	13(30.2)	43(1.9)	
Vaccine	Not vaccinated	121(74.7)	17(10.5)	11(6.8)	3(1.9)	5(3.1)	1(0.6)	0(0.0)	0(0.0)	4(2.5)	41(25.3)	162(7.3)	[a]$p < 0.191$, [b]$R = -0.028$
	Single dose, ChAdOx1	82(86.3)	3(3.2)	3(3.2)	2(2.1)	1(1.1)	0(0.0)	0(0.0)	1(1.1)	3(3.2)	13(13.7)	95(4.3)	
	Two doses, ChAdOx1	187(79.9)	18(7.7)	8(3.4)	4(1.7)	4(1.7)	2(0.9)	3(1.3)	2(0.9)	6(2.6)	47(20.1)	234(10.6)	
	Single dose, BNT162b2	223(84.2)	18(6.8)	14(5.3)	2(0.8)	3(1.1)	1(0.4)	0(0.0)	2(0.8)	2(0.8)	42(15.8)	265(11.9)	
	Two doses, BNT162b2	909(80.6)	99(8.8)	57(5.1)	11(1.0)	8(0.7)	8(0.7)	1(0.1)	8(0.7)	27(2.4)	219(19.4)	1128(50.9)	
	ChAdOx1/BNT162b2	276(82.6)	20(6.0)	21(6.3)	6(1.8)	1(0.3)	1(0.3)	1(0.3)	2(0.6)	6(1.8)	58(17.4)	334(15.1)	

[a] Chi-square, [b]Spearman's correlation (*R).* The values are listed as numbers, while the percentage was estimated based on the total number of the same row except for the last column that was calculated as the total number of the same column.

Table 5. The effect of sex, age, disease severity, and vaccine type on the development of sleeping disorders following SARS-CoV-2 infection

Item		None (n:1677)	Insomnia (n:341)	Hypersomnia (n:200)	Duration of sleeping disorders after COVID-19 infection (Months) <1 (n:174)	1 (n:106)	2 (n:57)	3 (n:34)	4 (n:16)	5 (n:11)	6 (n:42)	>6 (n:101)	Cumulative	Total (n:2218)	Chi-square and correlation
Sex	Female	990 (72.9)	230(16.9)	138(10.2)	109(8.0)	65(4.8)	34(2.5)	26(1.9)	11(0.7)	7(0.5)	40(2.9)	76(5.6)	368(27.1)	1358(61.2)	[a]$p < 0.001$, [b]$R = 0.08$
	Male	687(79.9)	111(12.9)	62(7.3)	65(7.6)	41(4.8)	23(2.7)	8(0.9)	5(0.6)	4(0.5)	2(0.2)	25(2.9)	173(20.1)	860(38.8)	
Age (Years)	12–17	99 (64.7)	35(22.9)	19(12.4)	25(16.3)	9(5.9)	5(3.3)	2(1.3)	2(1.3)	2(1.3)	2(1.3)	8(5.2)	54(35.3)	153(6.9)	[a]$p < 0.003$, [b]$R = -0.09$
	18–20	300(76.3)	53(13.5)	40(10.2)	16(4.1)	24(6.1)	12(3.1)	8(2.0)	3(0.8)	2(0.5)	10(2.5)	18(4.6)	93(23.7)	393(17.7)	
	21–30	655(78.3)	103(12.3)	79(9.4)	55(6.6)	27(3.2)	17(2.0)	12(1.4)	6(0.7)	4(0.5)	17(2.0)	43(5.1)	182(37.7)	837(37.7)	
	31–40	336 (77.6)	68(15.7)	29(6.7)	35(8.1)	18(4.2)	8(1.8)	3(0.7)	2(0.5)	2(0.5)	9(2.1)	21(4.8)	97(22.4)	433(19.5)	
	41–50	203 (70.2)	60(20.8)	26 (9.0)	33(11.4)	21(7.3)	10(3.5)	7(2.4)	3(1.0)	1(0.3)	4(1.4)	7(2.4)	86(29.8)	289 (13.0)	
	51–60	71(75.5)	17(18.1)	6(6.4)	7(7.4)	6(6.4)	3(3.2)	2(2.1)	0(0.0)	0(0.0)	0(0.0)	4(4.3)	23(24.5)	94(4.2)	
	61–70	13(64.4)	5(26.3)	1(5.3)	3(15.8)	1(5.3)	2(10.5)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	6(31.6)	19(0.9)	
Severity	Asymptomatic	216 (86.1)	24(9.6)	11(4.4)	13(5.2)	10(4.0)	0(0.0)	0(0.0)	0(0.0)	4(1.6)	2(0.8)	6(2.4)	35(13.9)	251(11.3)	[a]$p < 0.001$, [b]$R = -0.214$
	Mild	512 (84.5)	56(9.2)	38(6.3)	35(5.8)	14(2.3)	12(2.0)	5(0.8)	2(0.3)	2(0.3)	5(0.8)	18(3.0)	94(15.5)	606(27.3)	
	Moderate	621 (76.4)	117(14.4)	75(9.2)	64(7.9)	37(4.6)	20(2.5)	14(1.7)	6(0.7)	2(0.2)	14(1.7)	36(4.4)	192(23.6)	813(36.7)	
	Severe	304(60.2)	129(25.5)	72(14.3)	59(11.7)	41(8.1)	22(3.4)	13(2.6)	8(1.6)	2(0.4)	18(3.6)	38(7.5)	201(39.8)	505(22.8)	
	Critical	24 (55.8)	15(34.9)	4(9.3)	3(7.0)	4(9.3)	3(7.0)	2(4.7)	0(0.0)	1(2.3)	3(7.0)	3(7.0)	19(44.2)	43(1.9)	
Vaccine	Not vaccinated	114 (70.4)	38(23.5)	10(6.2)	18(11.1)	7(4.3)	7(4.3)	4(2.5)	1(0.6)	1(0.6)	3(1.9)	8(4.9)	48(29.6)	162(7.3)	[a]$p < 0.263$, [b]$R = -0.017$
	Single dose, ChAdOx1	71(74.7)	15(15.8)	9(9.5)	9(9.5)	4(4.2)	2(2.1)	1(1.1)	0(0.0)	1(1.1)	4(4.2)	3(33.3)	24(25.3)	95(4.3)	
	Two doses, ChAdOx1	183(78.2)	33(14.1)	18(7.7)	20(8.5)	6(2.6)	6(2.6)	6(2.6)	2(0.9)	2(0.9)	2(0.9)	8(3.4)	51(21.8)	234(10.6)	
	Single dose, BNT162b2	202 (76.2)	43(16.2)	20(7.5)	22(8.3)	16(6.0)	4(1.5)	3(1.3)	3(1.3)	1(0.4)	4(1.5)	9(3.4)	63(23.8)	265(11.9)	
	Two doses, BNT162b2	855(75.8)	163(14.5)	110(9.8)	78 (6.9)	57(5.1)	30(2.7)	13(1.2)	9(0.8)	6(0.5)	24(2.1)	55(4.9)	273(24.2)	1128(50.9)	
	ChAdOx1/ BNT162b2	252 (75.4)	49(14.7)	33(9.9)	27(8.1)	16(48.5)	8(2.4)	7(2.1)	1(0.3)	0(0.0)	5(1.5)	18(5.4)	82(24.6)	334(15.1)	

[a] Chi-square, [b]Spearman's correlation (*R*). The values are listed as numbers, while the percentage was estimated based on the total number of the same row except for the last column that was calculated as the total number of the same column.

1358) and 67 males (7.8% of 860), had sleeping disorders for 2 to >6 months (Table 5, Fig. 1).

The incidence of sleeping disorders significantly differed ($p < 0.001$) among the age groups. It was highest in the 21- to 30-year-old age group, in which 837 participants had sleeping disorders, of whom 103 had insomnia and 79 had hypersomnia, followed by the 18- to 20-year-old and 31- to 40-year-old age groups.

Disease severity significantly affected the development of sleeping disorders in the respondents ($p < 0.001$). Of those who had asymptomatic, mild, moderate, severe, and critical disease, 13.9% (35/251), 15.5% (94/606), 23.6% (192/813), 39.8% (201/505), and 44.2% (19/43) developed sleeping disorders after SARS-CoV-2 infection, respectively. The sleeping disorders persisted for 2 to >6 months in 4.8% (12/251), 7.5% (45/606), 11.2% (91/813), 20.0% (101/505), and 27.9% (12/43) of the participants in the severity groups, respectively. Only 101 (4.6%) of all the participants had sleeping disorders for >6 months (Fig. 1). A significant correlation was observed between disease severity and the developing of sleeping disorders ($R = -0.214$; Table 5). No significant differences were observed with regard to the vaccination status and type of vaccine. However, 273 (50.5%) of the 541 participants who reported sleeping disorders received two doses of the BNT162b2 vaccine (Table 5).

Loss of taste and smell

Of all the participants, 1412 (63.9%) lost their senses of taste and smell (Fig. 1). Most respondents lost both their senses of taste and smell (906/1358 females, 66.7% and 506/860 males, 62.8%; $p < 0.001$; Table 6). A considerable number (363/2218, 16.4%) of participants, including 244 females and 119 males lost their senses of taste and smell for 2 to >6 months. Meanwhile, 103 participants (4.6%), including 63 females (4.1% of 1358) and 43 males (5.0% of 860), lost their senses of taste and smell for >6 months (Table 6, Fig. 1).

Disease severity highly correlated with and significantly affected the development of loss of the senses of taste and smell in the respondents ($p < 0.001$, $R = -0.266$). Of those who had asymptomatic, mild, moderate, severe, and critical disease, 25.9% (65/251), 59.4% (360/606), 70.0% (569/813), 77.4% (391/505), and 62.8% (27/43) lost their senses of taste and smell after SARS-CoV-2 infection, respectively. In these severity groups, 6.0% (15/251), 13.4% (81/606), 16.7% (136/813), 24.0% (121/505), and 23.3% (10/43) of the participants lost their senses of taste and smell for 2 to >six months, respectively (Table 6).

Although no significant differences were detected among the age groups, significant numbers of participants who belonged to the 21- to 30-year-old and 31- to 40-year-old age groups showed loss of taste compared with the other age groups (Table 6). Although no significant differences were observed with regard to vaccination status and type of vaccine, the participants who received two doses of the BNT162b2 vaccine showed the highest incidence of loss of taste and smell after SARS-CoV-2 infection (Table 6).

Discussion

The results from the present study, which included 2218 individuals who were diagnosed with laboratory-confirmed COVID-19 illness, suggest that long-term COVID-19 infection can lead to many neuropsychiatric sequelae, concurring with previous studies (Xiong *et al.*, 2020; Rank *et al.*, 2021; Seeßle *et al.*, 2021). Such neurological disorders have possible direct and indirect causes such as hypoxia, which directly affects the cerebral blood vessels, and hypoxemia or psychological aspects, respectively (Lopez-Leon *et al.*, 2021). The latter could be related to the panic caused by the continuous follow-up of toll death numbers from COVID-19 and the daily huge numbers of cases worldwide in various news portals. Accordingly, the present study focused on the common neuropsychiatric signs experienced by patients with COVID-19 that extended for >6 months in the Saudi population. Previous studies have identified evidence of cognitive dysfunction induced by COVID-19 illness, with few studies conducted in the non-hospitalised population (Ding *et al.*, 2020).

In the present study, the incidence of decreased cognitive skills (slow thinking and difficulty concentrating), which is termed as brain fog, was found to be lower than detected in an international study that included participants from the USA, Canada, and Europe (the UK, Spain, the Netherlands, Ireland, Sweden, and others) (Davis *et al.*, 2021). In the international study, decreased cognitive skills (>3 months post-infection) were reported in 66.7% of their participants, which decreased to 55.5% (seventh month post-infection). In another study, 21.2% of respondents complained of persistently decreased cognitive activities for up to 12 months post-infection (Kim *et al.*, 2022). By contrast, decreased cognitive skills were detected in only 7.2% of the patients admitted to healthcare facilities in Iran (Asadi-Pooya *et al.*, 2021). In our study, we observed a rapid improvement in brain fog, from 27.7% (during the first month post-infection) to 17.9 (at 2 to >6 months post-infection), including 4.7% of the participants showing persistent brain fog for >6 months post-infection.

The development of brain fog correlated with disease severity in non-hospitalised and some hospitalised participants, as detected in the present study. Other studies found this correlation in non-hospitalised COVID-19 long haulers (Graham *et al.*, 2021; Hampshire *et al.*, 2021). We also found small percentages of respondents who experienced asymptomatic and mild disease and had brain fog.

In the present study, the incidence rates of depression and mood alteration were 30.9% and 42.9%, respectively. The percentages of affected participants decreased over time, and only 5.9% and 7.6% still had these complaints for >6 months. These percentages were lower than that recorded for Korean patients who had depression for >12 months (17.8% of patients) (Kim *et al.*, 2022). This confirms the finding of Ekn, who reported a significant variation among individuals of different races (Eken *et al.*, 2021). Other studies confirmed the link of depression to female sex (Sønderskov *et al.*, 2020) and age <40 years (Ahmed *et al.*, 2020; Gao *et al.*, 2020).

Tinnitus is a common chronic disorder that affects 12–30% of the adult population (McCormack *et al.*, 2016). Stress is a potential triggering factor of tinnitus (Mazurek *et al.*, 2012). The panic caused by the COVID-19 pandemic increased the stressful conditions in the general population, which led to a subsequent increase in the risk of developing tinnitus (Mazurek *et al.*, 2019). Few studies discussed the duration and persistence of tinnitus (Lamounier *et al.*, 2020; Chirakkal *et al.*, 2021; Jafari *et al.*, 2021). The mean incidence of post-COVID-19 tinnitus was estimated to be 8%, ranging from 4.5% to 14.8% (Almufarrij & Munro, 2021; Beukes *et al.*, 2021; Jafari *et al.*, 2021), showing a low improvement rate (Beukes *et al.*, 2020). However, our study revealed a higher percentage of participants who developed tinnitus (18.9%), of whom 5.9% had tinnitus for 2 to >6 months and only 2.2% had tinnitus for >6 months. In most of these participants, tinnitus persisted for up to 1 month only.

Patients with COVID-19 experience depression and sleep disturbance during hospitalisation (Franceschini *et al.*, 2020) and after infection, as indicated by a 2-month follow-up study (Islam *et al.*, 2021). In accordance with previous studies, we found

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Table 6. The effect of sex, age, disease severity, and vaccine type on development of loss of taste and smell following SARS-CoV-2 infection

			Duration of loss of taste and smell after COVID-19 infection (Months)										
Variables		None (n:806)	<1 (n:622)	1 (n:427)	2 (n:118)	3 (n:63)	4 (n:31)	5 (n:25)	6 (n:23)	>6 (n:103)	Cumulative	Total (n:2218)	Chi-square and correlation
Sex	Female	452(33.3)	389(28.6)	273(20.1)	80(5.9)	48(3.5)	23(1.7)	18(1.3)	12(0.9)	63(4.6)	906(66.7)	1358(61.2)	[a]$p < 0.003$, [b]$R = -0.003$
	Male	354(41.2)	233(27.1)	154(17.9)	38(4.4)	151.7)	8(0.9)	7(0.8)	11(1.3)	40(4.7)	506(58.8)	860(38.8)	
Age	12–17	62(40.5)	45(29.4)	18(11.8)	6(3.9)	6(3.9)	5(3.3)	2(1.3)	0(0.0)	9(5.9)	91(59.5)	153(6.9)	[a]$p < 0.125$, [b]$R = -0.006$.
	18–20	152(38.7)	100(25.4)	71(18.1)	22(5.6)	9(2.3)	8(2.0)	7(1.8)	7(1.8)	17(4.3)	241(61.3)	393(17.7)	
	21–30	286(34.2)	238(28.4)	175(20.9)	45(5.4)	26(3.1)	8(1.0)	10(1.2)	8(1.0)	41(4.9)	551(65.8)	837(37.7)	
	31–40	147(33.9)	138(31.9)	79(18.2)	27(6.2)	18(4.2)	3(0.7)	4(0.9)	1(0.2)	16(3.7)	286(66.1)	43319.5)	
	41–50	107(37.0)	74(25.6)	59(20.4)	15(5.2)	3(1.0)	6(2.1)	2(0.7)	5(1.7)	18(6.2)	182(63.0)	289(13.0)	
	51–60	45(47.9)	23(24.5)	18(19.1)	2(2.1)	1(1.0)	1(1.0)	0 (0.0)	2(2.1)	2(2.1)	49(52.1)	94(4.2)	
	61–70	7(36.8)	4(21.1)	7(36.8)	1(5.3)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	12(63.2)	19(0.9)	
Severity	Asymptomatic	186 (74.1)	30(12.0)	20(8.0)	6(2.4)	1(0.4)	2(0.8)	1(0.4)	0(0.0)	5(2.0)	65(25.9)	251(11.3)	[a]$p < 0.001$, [b]$R = -0.266$
	Mild	246(40.6)	185(30.5)	94(15.5)	30(5.0)	11(1.8)	8(1.3)	7(1.2)	4(0.7)	21(3.5)	360(59.4)	606(27.3)	
	Moderate	244(30.0)	252(31.0)	181(22.3)	43(5.3)	23(2.8)	7(0.9)	8(1.0)	11(1.4)	44(5.4)	569(70.0)	813(36.7)	
	Severe	114(22.6)	146(28.9)	124(24.6)	36(7.1)	25(5.0)	13(2.6)	8(1.6)	6(1.2)	33(6.5)	391(77.4)	505(22.8)	
	Critical	16(37.2)	9(20.9)	8(18.6)	3(7.0)	3(7.0)	1(2.3)	1(2.3)	2(4.7)	0(0.0)	27(62.8)	43(1.9)	
Vaccine	Not vaccinated	48(29.6)	51(31.5)	39(24.1)	9(5.6)	5(3.1)	3(1.9)	1(0.6)	0(0.0)	6(3.7)	114(70.4)	162 (7.3)	[a]$p < 0.385$, [b]$R = -0.011$
	Single dose, ChAdOx1	23(24.2)	31(32.6)	23(24.2)	6(6.3)	4(4.2)	0(0.0)	0(0.0)	1(1.1)	7(7.4)	72(75.8)	95(4.3)	
	Two doses, ChAdOx1	88(37.6)	66(28.2)	34(14.5)	17(7.3)	11(4.7)	5(2.1)	2(0.9)	2(0.9)	9(3.8)	146(62.4)	234(10.6)	
	Single dose, BNT162b2	87(32.8)	68(25.7)	62(23.4)	17(6.4)	8(3.0)	5(1.9)	1(0.4)	5(1.9)	12(4.5)	178(67.2)	265(11.9)	
	Two doses, BNT162b2	435(38.6)	311(27.6)	205(18.2)	53(4.7)	27(2.4)	16(1.4)	16(1.4)	12 (1.1)	53(4.7)	693(61.4)	1128(50.9)	
	ChAdOx1/BNT162b2	125(37.4)	95(28.4)	64(19.2)	16(4.8)	8(2.4)	2(0.6)	5(1.5)	3(0.9)	16(4.8)	209(62.6)	334(15.1)	

[a]Chi-square, [b]Spearman's correlation (*R*). The values are listed as numbers, while the percentage was estimated based on the total number of the same row except for the last column that was calculated as the total number of the same column.

that a significant proportion of patients had poor sleep quality post-COVID-19. Accordingly, in the present study, 24.4% of the participants developed sleep disorders, including insomnia (15.4%) and hypersomnia (9.0%). Sleep disorders are known to occur most commonly in young age groups (McArdle *et al.*, 2020), which matches with our finding that the highest incidence of sleep disorders was in the 21- to 30-year-old age group.

In the present study, anosmia and dysgeusia were found to be the most dominant complaints associated with SARS-CoV-2 infection. As the study was conducted after complete recovery from COVID-19, it denotes long-term COVID signs. We found wide discrepancies among previous reports of the prevalence of anosmia (5.14–98.33%) and dysgeusia (5.61–92.65%). These differences could be related to the race of the study population and disease severity (Tong *et al.*, 2020). In the present study, 63% of the respondents reported loss of taste and smell, which decreased over time. Of these respondents, 16.4% developed both anosmia and dysgeusia for 2 to >6 months, while 4.6% showed persistent complaints for >6 months. Our findings agree with other reports that confirmed recovery from both gustatory and olfactory dysfunctions (Vaira *et al.*, 2020a). Virus replication was assumed to occur in the sensory receptors of the taste bud and olfactory epithelia taste and smell receptors rather than in the central nervous system (Vaira *et al.*, 2020b). Yong assumed that the involvement of the brain stem could be responsible for such neurological signs (Yong, 2021); however, brain stem involvement leads to long-lasting changes that have not been reported to date.

The present study agrees with previous findings that female sex and disease severity correlated with the development of persistent long-term adverse effects of COVID-19, which matched the results of previous studies (Sudre *et al.*, 2020; Asadi-Pooya *et al.*, 2021). We found that receiving two vaccine doses could inhibit the appearance of post-COVID-19 long-term side effects. This finding is in accordance with previous findings that confirmed the appearance of post-vaccination side effects, including neurological signs (Alghamdi *et al.*, 2021; Patone *et al.*, 2021).

Long-COVID constitutes a challenge to healthcare settings. Although clinicians in some countries managed persistent symptoms in patients affected by MERS-CoV (in Saudi Arabia and other Middle East countries) and SARS-CoV (mainly in China), however, the number of cases in such two outbreaks were exceptionally low and such cases were clustered in certain countries. By contrast, millions more people have had COVID-19 in comparison to the diseases caused by MERS-CoV and SARS-CoV. Accordingly, the potential of having prolonged health problems could be ridiculously huge and long-term COVID-19 care may constitute a complicated issue. Multidisciplinary research about long-term COVID-19 that investigates the full spectrum of long-COVID term effects is highly recommended.

Conclusion

Post-COVID neurological symptoms were reported in 18.9–63.9% of the participants with COVID-19, including decreased cognitive skills, anosmia and dysgeusia, tinnitus, depression, and sleep disorders. Such signs persisted in 2.2–7.6% of the patients for >6 months. Disease severity and female sex were identified as potential determinants of the development and persistency of such symptoms.

Author contributions. HYA, AMA and AMJ analysed and interpreted the data. HYA, AMA and AMJ processed the results and drafted the manuscript. ASA conceived the study critically and revised the manuscript. All authors read and worked on the manuscript.

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