

# Prevalence of Neurological Symptoms Associated with COVID-19

Syed Hassan Tanvir Ramzi, Ubaidullah Ansari, Sana Manzoor, Namal Ilyas, Nabeel Ahmed

**Abstract**—To better understand the prevalence of neurological symptoms associated with COVID-19, several factors, such as age, gender, and comorbidity, are explored to create a more holistic understanding of the impact of COVID-19. After meeting inclusion and exclusion criteria, 111 patients admitted to Ibne Sina Hospital were recruited between October 2021 and February 2022. A descriptive statistical analysis was conducted to summarize patients most often encountered signs and symptoms concerning the above parameters. Out of 111 patients, a significant proportion of symptoms occurred in patients aged 40-60 years, with Dysgeusia being the most widespread (75.5%), followed by Encephalitis (45.9%), Guillain Barre Syndrome (GBS) (28.8%), Encephalopathy (18.9%), and Ischemic Stroke (6.3%). These were most prevalent in hypertensive individuals (46%) and Diabetes Mellitus (31%). In asthmatic individuals, they are the least prevalent (10.8%). Despite the predominance of neurological manifestations, the present scientific literature cannot demonstrate a definitive causal association between the symptoms and the virus. This study carefully ensures a link between age, gender, and comorbidity, along with the prevalence of neurological manifestations of COVID-19. For a comprehensive treatment plan, a holistic understanding of symptoms is critical.

**Keywords**—COVID-19, neurological associations, GBS, encephalopathy, encephalitis, dysgeusia, stroke.

## I. INTRODUCTION

CORONAVIRUS was first reported in Wuhan, China, in December 2019. It is also referred to as the severe acute respiratory syndrome (SARS) Coronavirus; as of April 24, 2022, over 500 million confirmed cases and over six million deaths have been identified globally [1]. This disease can cause severe pneumonia with a high fatality rate. Every minute, extensive research is being undertaken to determine the best coronavirus management, diagnosis, and treatment methods. At the time of the disease's first outbreak, it was believed to have been spread by animals. Nonetheless, its human-to-human transmission was recognized later, and the World Health Organization (WHO) classified COVID-19 as a global pandemic. To aid in the battle against this pandemic outbreak, numerous lines of study have been expedited to explore all characteristics of the unique COVID-19 virus while maintaining the highest degree of precaution and safety [2].

COVID-19 is most often associated with fever, a dry cough, and lethargy. Nonetheless, several practitioners in impacted regions noticed that some COVID-19 patients did not exhibit usual respiratory symptoms at the time of diagnosis, such as fever and coughing; however, some infected individuals presented with only neurological issues, such as headache,

languidness, unsteady walking, and malaise, which may be caused by non-specific presentations [3].

A recent study of 214 patients affected by COVID-19 showed that 78 (36.4%) had neurological symptoms such as headache, dizziness, acute cerebrovascular problems, and decreased cognition. 40 (18.7%) of the 214 patients needed intensive care unit (ICU) treatments for severe neurological involvement [3]. Despite several incidents of brain hemorrhages in COVID-19 individuals being reported, there is a paucity of comprehensive studies on this link. Consequently, the physiological mechanism through which COVID-19 causes cerebral bleeding is unclear [3], [4].

There has been an increase in reports of neurological impairment of the central and peripheral nervous systems in patients infected with COVID-19. Some patients complain of headaches, and dysgeusia, but a broader range of more serious neurological problems, particularly in hospitalized patients, including stroke, encephalopathy, encephalitis, and polyneuritis have been reported [5].

Coughing, sneezing, or touching unclean objects are how the virus spread swiftly. It is more prevalent in the elderly, males, and those with diabetes, hypertension, cardiovascular disease, or cancer [6]. In order to better understand the disease's biology and transmission, researchers need to know where SARS-CoV-2 is located in the tissues of COVID-19 patients. This study examined samples from eight deceased SARS-CoV-2 patients in the United States using immunohistochemistry and electron microscopy. Upper and lower airway epithelium with extensive alveolar degeneration was the most common respiratory pathology in these individuals. SARS-CoV-2 was found in conducting airways, pneumocytes, alveolar macrophages, and a hilar lymph node, but not in other extrapulmonary tissues [7].

According to research, coronaviruses from the SARS family "hijack" the protein angiotensin-converting enzyme-2 (ACE2). It is an aminopeptidase that is membrane-bound and identified in a variety of human cells (respiratory tract, lung, heart, arteries, veins, kidney, brain, and intestines). A protein called SPIKE interacts with the ACE2 receptor, allowing the virus to enter the cell [8]. According to preclinical studies, coronavirus may propagate transneuronally into the brain along olfactory pathways, compromising the integrity of the olfactory neuroepithelium. This is accomplished by the sustentacular cell's synthesis of TMPRSS2 and ACE2 [9]-[11].

Specific populations are more vulnerable to the consequences of COVID-19 than others. Children, the elderly, the disabled, those from rural regions, those with a lower

Syed Hassan Tanvir Ramzi is with Mukhtar A. Shiekh Hospital, Pakistan (e-mail: hassantanvir58@yahoo.com).

socioeconomic standing, and those with pre-existing health problems are just a few examples [12].

## II. MATERIAL/SUBJECTS/PATIENTS AND METHODS

The prevalence of neurological COVID-19-related symptoms was determined using a quantitative research design in patients hospitalized at Ibne Sina Hospital, Multan. Five months of data collection occurred between October 2021 and February 2022. After meeting inclusion and exclusion criteria, a standardized questionnaire was utilized to gather data from a sample of 111 individuals, a descriptive statistical analysis was employed to summarize the findings. Various factors were used to identify participants from outpatient, inpatient, and previous medical records; informed consent was obtained by patients or guardians

- Criteria for inclusion:
  - Age groups over 15 years were employed, with subgroups of 15-30, 31-60, and greater than 60 years.
  - All subjects were diagnosed with a neurological deficit apparent through the history and clinical examination.
  - All participants had a history in which they were either clinically diagnosed with COVID-19 and had symptoms such as fever, cough, homogenous infiltrates bilaterally on lungs with lymphopenia and elevated inflammatory markers as baseline parameters or had a positive PCR.

- Criteria for exclusion:
  - Age less than 15 years.
  - A history of substance abuse
  - Patients suffering from metabolic or septic encephalopathy, such as hepatic or uremic encephalopathy.
  - Neuroimaging revealing the presence of any other disorder.

It is worth mentioning that throughout the inclusion process, patients with clinically confirmed COVID-19 but a negative PCR were also included, suggesting that the PCR performed was less sensitive [13].

## III. RESULTS

Our research involved 111 individuals, 45.9% of whom were female and 54.1% male. The majority of participants (50%) in our research were aged between 40 and 69 years old. Hypertension was the most often reported comorbidity in our participants (46.8).

As seen in Fig. 1, the most frequently reported presenting complaints were fever (87.4%), headache (76.6%), dysgeusia (75.5%), cough (62.2%), drowsiness (57.5%), and irritability (33.3%). On Chest X-rays, 91% had bilateral lung infiltrates. COVID-19 PCR was positive in only 28.8% of cases, 19.8% of patients had lung infiltrates on HRCT. All individuals included had a neurological impairment (Weakness in 5.4%, Paraparesis in 24.3%, Hemiplegia in 8.1%, and Paraplegia in 19.8%).

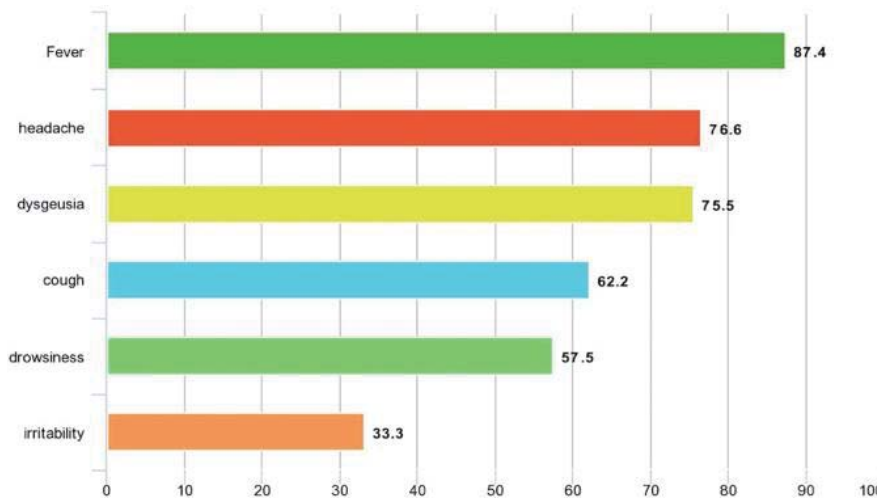


Fig. 1 Prevalence of Clinical Features in the Study Population

Signs of meningeal irritation (Somi) were negative in 64.9% of patients, hyporeflexia was present in 31.5% of patients, and plantars were upgoing in 57.7% of patients. GCS was worsening in 61.3% of patients, and only 13.5% of patients presented with an altered state of consciousness. Additionally, 26.1% exhibited elevated proteins, majority samples had normal glucose levels except for 6.3% whom had reduced glucose, and 29.7% had lymphocytosis on CSF testing. CT and MRI brain scans were available for only 83.8% and 14.4% of patients respectively. There were no abnormal imaging findings on CT Brain or MRI in all patients. As seen in Fig. 2, 75.5% of patients were diagnosed with dysgeusia, 45.9% with

encephalitis, 28.8% with GBS, 18.9% with encephalopathy, and 6.3% with ischemic stroke. Tables I and II summarize the study population's characteristics.

The patients diagnosed with encephalitis and encephalopathy were primarily from the 40-60 years of age group (56.9% and 71.4%, respectively). The diagnosis of ischemic stroke was made in only 6.3% of patients. The majority of the patients were from the 15-40- and 40-60-years age group (42.9% and 42.95%, respectively). The prevalence of neurological diagnosis in various age groups is summarized in Table III.

The prevalence of neurological manifestations with comorbidities (diabetes mellitus, hypertension, asthma) and

steroid abuse is given in Table IV. The neurological manifestation was most prevalent in hypertensive patients

(46%), followed by diabetes mellitus (31%). The neurological diagnosis was least prevalent in asthma patients (10.8%).

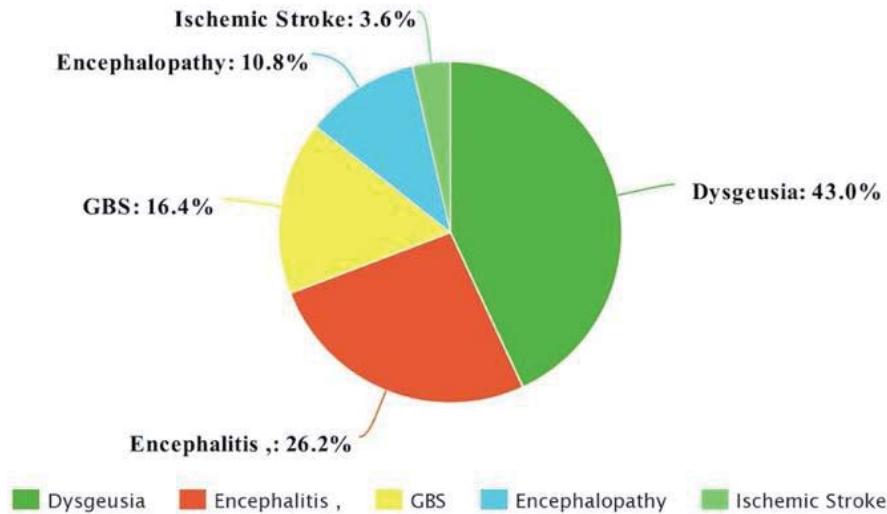


Fig. 2 Prevalence of Neurological Associations among the Study Population

TABLE I  
BASELINE CLINICAL FEATURES OF THE STUDY PARTICIPANTS

Variable	Male	Female
N	60	51
Age, Years n (%)		
15-40	13 (21.7)	4 (7.8)
40-60	25 (41.7)	31 (60.8)
> 60	22 (36.7)	16 (31.4)
Comorbidities		
Diabetes Mellitus, n (%)	6 (10)	29 (56.9)
Hypertension, n (%)	31 (51.7)	21 (41.2)
Asthma n (%)	12 (20)	0 (0)
History of Steroid Abuse (%)	10 (16.6)	4 (7.8)
Clinical Features		
Headache, n(%)	41 (68.3)	44 (86.3)
Fever, n(%)	60 (100)	37 (72.5)
Cough, n (%)	45 (75)	24 (47.1)
Drowsiness, n(%)	28 (46.7)	36 (70.6)
Irritability, n(%)	15 (25)	22 (43.1)
Weakness, n(%)	4 (6.7)	2 (3.9)
Paraparesis, n(%)	15 (25)	12 (23.5)
Paraplegia, n(%)	18 (30)	4 (7.8)
Hemiplegia, n(%)	4 (6.7)	5 (9.8)
Agnosia n(%)	55 (91.6)	27 (52.9)
PCR		
N/A, n(%)	16 (26.7)	16 (31.4)
Positive, n(%)	15 (25)	17 (33.3)
Negative, n(%)	29 (48.3)	18 (35.3)
X-Ray		
N/A, n(%)	3 (5)	2 (3.9)
BL Infiltrates	52 (86.7)	49 (96.1)
Normal	5 (8.3)	0 (0)
HRCT Lungs		
N/A, n(%)	41 (68.3)	43 (84.3)
Show Infiltrates	14 (23.3)	8 (15.7)
Normal	5 (8.3)	0 (0)
Blood Biochemistry		
CBC (Lymphopenia)	47 (78.3)	46 (90.2)
Inflammatory Markers (Raised)	55 (91.7)	51 (100)

The prevalence of neurological manifestations with comorbidities (diabetes mellitus, hypertension, asthma) and steroid abuse is given in Table IV. The neurological manifestation was most prevalent in hypertensive patients

(46%), followed by diabetes mellitus (31%). The neurological diagnosis was least prevalent in asthma patients (10.8%).

TABLE II  
NEUROLOGICAL EVALUATION OF STUDY PARTICIPANTS

Variable	Male	Female
Neurological deficit		
Somi-ve,	37 (61.7)	35 (68.6)
Planters upping,	23 (38.3)	41 (81.4)
Deteriorating GCS	30 (50)	38 (74.5)
Decreased power,	36 (60)	23 (45.1)
Hyporeflexia	27 (45)	8 (15.7)
ASOC	7 (11.7)	8 (15.7)
CSF Findings		
Lymphocytosis		
Increased	14 (23.3)	19 (37.3)
Decreased	0 (0)	0 (0)
Normal	0 (0)	1 (2)
N/A	46 (76.7)	31 (60.8)
Proteins		
Increased	14 (23.3)	15 (29.4)
Decreased	1 (1.7)	0 (0)
Normal	9 (15)	14 (27.5)
N/A	36 (60)	22 (43.1)
Glucose		
Increased	0 (0)	0 (0)
Decreased	3 (5)	4 (7.8)
Normal	17 (28.3)	30 (58.8)
N/A	40 (66.7)	17 (33.3)
MRI Brain		
N/A, n(%)	52 (86.7)	43 (84.3)
Normal	8 (13.3)	8 (15.7)
CT Scan Brain		
N/A, n(%)	11 (18.3)	7 (13.7)
Normal	49 (81.7)	44 (86.3)
Neurological diagnosis		
GBS	27 (45)	5 (9.8)
Encephalopathy	6 (10)	15 (29.4)
Encephalitis	26 (43.3)	25 (49)
Ischemic Stroke	1 (1.7)	6 (11.8)
Dysgeusia	55 (91.6)	27 (52.9)

#### IV. DISCUSSION

SARS-CoV2 has neurological repercussions comparable to

previous coronavirus outbreaks, notably in 2003, SARS and the 2012 Middle East acute respiratory syndrome. Encephalopathy, encephalitis, GBS, ischemic stroke, and hemorrhagic stroke were all recorded in those papers due to hypercoagulability, sepsis, and vasculitis [14].

A study from a tertiary care hospital on the frontline sampled 50 patients. Encephalopathy, cerebrovascular illness, cognitive impairment, seizures, hypoxic brain damage, dysgeusia, and aberrant extraocular movement were neurological signs [15].

In research conducted in Wuhan, 78 of 214 COVID-19 participants had neurological symptoms for four weeks. These patients were more seriously ill, older, and had a higher prevalence of comorbidities, particularly hypertension, and for some, the neurological symptom was the first indicator of COVID-19 infection. Apart from six patients (2.8%) who had a stroke, neurological symptoms might be caused by a viral infection (loss of smell and taste) or by the consequences of severe systemic illness in an intensive care unit, such as infection and hypoxia [16].

TABLE III  
PREVALENCE OF NEUROLOGICAL MANIFESTATION IN VARIOUS AGE GROUPS

Neurological Manifestations	Age Groups	N	%
Encephalitis	15-40	5	9.8
	40-60	29	56.9
	>60	17	33.3
Encephalopathy	15-40	1	4.8
	40-60	15	71.4
	>60	5	23.8
GBS	15-40	8	25.0
	40-60	9	28.1
	>60	15	46.9
Ischemic Stroke	15-40	3	42.9
	40-60	3	42.9
	>60	1	14.3
Dysgeusia	15-40	28	34.1
	40-60	41	50
	>60	13	15.8

TABLE IV  
PREVALENCE OF NEUROLOGICAL MANIFESTATIONS IN PATIENTS WITH VARIOUS COMORBIDITIES

Comorbidities	Encephalitis	Encephalopathy	GBS	Ischemic Stroke	Agnosia
Diabetes	16 (31.4)	15 (71.4)	4 (12.5)	0 (0)	26 (23.4)
Hypertension	25 (49)	8 (38.1)	18 (56.3)	1 (14.3)	56 (68.2)
Asthma	5 (9.8)	1 (4.8)	6 (18.8)	0 (0)	0 (0)
Steroid Abuse	5 (9.8)	3 (14.3)	6 (18.8)	0 (0)	0 (0)

According to the Strasbourg group, 40/58 patients (69%) had agitation, whereas 26/40 (65%) had disorientation, and 39/59 had corticospinal tract symptoms (67%). MRI revealed meningeal enhancement, ischemic stroke, and perfusion abnormalities in 22 patients. Myoclonus and demyelination have been reported [17].

Coronavirus infections in the brain have previously been reported in patients with the SARS, caused by the SARS-CoV virus, and Middle East Acute Respiratory Syndrome, caused by the MERS-CoV virus. The severity and long-term consequences of these disorders vary significantly amongst people. SARS-CoV-2 infection of the cerebrospinal fluid (CSF) has been assessed only in a small number of cases, and positive findings are unusual.

As per Lewis and colleagues' systematic review, 6% of patients who underwent CSF testing was positive for SARS-CoV-2. The CSF cell count was increased in 43% of fatal cases, 25.7% of severe cases, and 29.4% of non-severe cases, with lymphocytosis being the most prevalent. The great majority of those individuals suffer from neurological issues involving the central nervous system (CNS) [18].

According to another study by Tandon and colleagues that primarily focused on CSF protein levels, The most frequently encountered CSF finding was increased CSF proteins. They observed that patients who died with COVID-19 had significantly higher protein levels in their CSF (100%) and an average of 61.28 mg/dl than those who survived (65%) and had an average of 56.73 mg/dl. Similar increases in CSF protein levels were observed in 74.5% of patients with mild COVID-19 infection and 68.6% of patients with severe COVID-19

illness [19].

The US Food and Drug Administration (USFDA) employs a methodical approach in combating COVID-19 and has approved only remdesivir medications for use in COVID-19 hospitalized patients. The Food and Drug Administration (FDA) of the United States granted an emergency use authorization for antibodies neutralization (bamlanivimab + etesevimab and casirivimab/imdevimab), antiviral combination treatment (remdesivir + baricitinib), and COVID-19 convalescent plasma [20].

It is reasonable to expect that 80% of patients who recovered from COVID-19 with minor symptoms will have no long-term consequences and will eventually make a full recovery. Patients with moderately severe symptoms who needed hospitalization but not mechanical ventilation had no long-term repercussions. Patients who need mechanical ventilation due to severe symptomatology are more likely to suffer long-term complications and delayed recovery as they age. Changes in SARS-CoV-2 pathophysiology, inflammatory damage, and immunologic abnormalities in COVID-19 might lead to post-COVID-19 sequelae. Numerous multiorgan systems may be compromised in severe COVID-19 survivors [21].

One of the purposes of this research was to investigate the association between age and neurological symptoms. However, the outcomes differed depending on the category. Those aged 40-60 years had the most significant rates of encephalitis and encephalopathy (56.9% and 71.4, respectively). In the case of ischemic stroke, both the age categories of 15-40 years and 40-60 years tied at 42.9%. The research also looked at comorbidity. Hypertensive people (46%) had the most significant

## REFERENCES

neurological manifestations, followed by those with diabetes mellitus (31%). Asthmatic individuals had the lowest prevalence of neurological disorders (10.8%). While some patients may complain of headaches, anosmia, and dysgeusia, studies have found that a broader range of more significant neurological issues, especially in hospitalized patients, may occur, including stroke, encephalopathy, encephalitis, and polyneuritis [21]-[23]. These results correspond to the study's findings and the incidence of neurological complaints reported by patients. As the findings show, the research has clinical implications by emphasizing the role of comorbidity for better prognosis.

A study revealed that COVID-19-related deaths, cardiovascular diseases such as hypertension, and diabetes are highly significant ( $p < 0.0001$ ). Similarly, deaths resulting from kidney diseases and neurological issues are also significantly higher than the total number of hospitalized patients for that particular health concern [24].

As a result, if comorbidities are not considered while developing a treatment plan, the illness prognosis may deteriorate dramatically. Contrary to common opinion, ACEI such as Valsartan has been shown in multiple studies to benefit and protect patients with hypertension thus, treatment should not be altered [25].

## V.CONCLUSION

Evidence suggests the likelihood of COVID-19-related CNS pathologies. Consequently, healthcare providers should be aware of the wide variety of neurological signs and symptoms associated with COVID-19 in order to establish an early diagnosis and isolate patients. Additionally, further research is required to improve the identification and treatment of COVID-19 patients who develop neurological or mental health disorders, as well as associated comorbidities, following SARS-CoV-2 infection.

This study explored the incidence of neurological symptoms related to COVID-19. Despite the prevalence of neurological symptoms, there is limited research on their association with COVID-19 patients in Pakistan. To emphasize the significance of this occurrence, statistics revealed that all subjects had some neurological abnormality. This research relied heavily on many critical characteristics, including gender, comorbidity, and age. Between the ages of 40 and 60 years, neurological symptoms such as encephalitis and encephalopathy were most prevalent. In terms of gender and comorbidity, women had a significantly greater prevalence of diabetes (56.9%), but males had a significantly higher prevalence of hypertension (51.7%). Men were more likely to have fever, cough, paraparesis, and paraplegia, while women were more likely to experience headaches, sleepiness, irritability, and hemiplegia. This study provides essential insights into the link between gender, comorbidity, age, and neurological symptoms for future research. Its objective is to establish a framework for more comprehensive treatment programs that include all of the aspects mentioned above in order to build a holistic solution.

- [1] Weekly epidemiological update on COVID-19 - 27 April 2022. www.who.int.
- [2] Niazkar HR, Zibae B, Nasimi A, Bahri N. The neurological manifestations of COVID-19: a review article. *Neurological Sciences*. 2020 Jun 1.
- [3] Wang H-Y, Li X-L, Yan Z-R, Sun X-P, Han J, Zhang B-W. Potential neurological symptoms of COVID-19. *Therapeutic Advances in Neurological Disorders*. 2020 Jan;13:175628642091783.
- [4] Mao L, Wang M, Chen S, He Q, Chang J, Hong C, et al. Neurological Manifestations of Hospitalized Patients with COVID-19 in Wuhan, China: a retrospective case series study. 2020 Feb 25.
- [5] Wu Y, Xu X, Chen Z, Duan J, Hashimoto K, Yang L, et al. Nervous system involvement after infection with COVID-19 and other coronaviruses. *Brain, Behavior, and Immunity*. 2020 Mar 30;87:18–22.
- [6] Rashedi J, Mahdavi Poor B, Asgharzadeh V, Pourostadi M, Samadi Kafil H, Vegari A, et al. Risk Factors for COVID-19. *Le Infezioni in Medicina*. 2020 Dec 1;28(4):469–74.
- [7] Martines RB, Ritter JM, Matkovic E, Gary J, Bollweg BC, Bullock H, et al. Pathology and Pathogenesis of SARS-CoV-2 Associated with Fatal Coronavirus Disease, United States. *Emerging Infectious Diseases*. 2020 Sep;26(9).
- [8] Allado E, Poussel M, Valentin S, Kimmoun A, Levy B, Nguyen DT, et al. The Fundamentals of Respiratory Physiology to Manage the COVID-19 Pandemic: An Overview. *Frontiers in Physiology*. 2021 Feb 18;11.
- [9] Xydakis MS, Dehgani-Mobaraki P, Holbrook EH, Geisthoff UW, Bauer C, Hautefort C, et al. Smell and taste dysfunction in patients with COVID-19. *The Lancet Infectious Diseases*. 2020 Apr.
- [10] Li YC, Bai WZ, Hashikawa T. The neuroinvasive potential of SARS-CoV2 may play a role in the respiratory failure of COVID-19 patients. *Journal of medical virology*. 2020 Jun;92(6):552-5.
- [11] Desforgues M, Le Coupancec A, Brison É, Meessen-Pinard M, Talbot PJ. Neuroinvasive and Neurotropic Human Respiratory Coronaviruses: Potential Neurovirulent Agents in Humans. *Infectious Diseases and Nanomedicine I*. 2014 Mar 12;807:75–96.
- [12] Kuy S, Tsai R, Bhatt J, Chu QD, Gandhi P, Gupta R, et al. Focusing on Vulnerable Populations During COVID-19. *Academic Medicine*. 2020 Jul 14;95(11).
- [13] Kortela E, Kirjavainen V, Ahava MJ, Jokiranta ST, But A, Lindahl A, et al. Real-life clinical sensitivity of SARS-CoV-2 RT-PCR test in symptomatic patients. Ricci S, editor. *PLOS ONE*. 2021 May 21;16(5):e0251661.
- [14] Paterson RW, Brown RL, Benjamin L, Nortley R, Wiethoff S, Bharucha T, et al. The emerging spectrum of COVID-19 neurology: clinical, radiological and laboratory findings. *Brain*. 2020 Jul 8;143(10).
- [15] Neurological manifestations and COVID-19: Experiences from a tertiary care center at the Frontline. *Journal of the Neurological Sciences*. 2020 Aug 15 (cited 2020 Nov 19);415:116969.
- [16] Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic Manifestations of Hospitalized Patients With Coronavirus Disease 2019 in Wuhan, China. *JAMA Neurology*. 2020 Apr 10;
- [17] Helms J, Kremer S, Merdji H, Clere-Jehl R, Schenck M, Kummerlen C, et al. Neurologic Features in Severe SARS-CoV-2 Infection. *New England Journal of Medicine*. 2020 Apr 15;
- [18] Lewis A, Frontera J, Placantonakis DG, Lighter J, Galetta S, Balcer L, et al. Cerebrospinal fluid in COVID-19: A systematic review of the literature. *Journal of the Neurological Sciences*. 2021 Feb;421:117316.
- [19] Tandon M, Kataria S, Patel J, Mehta TR, Daimee M, Patel V, et al. A Comprehensive Systematic Review of CSF analysis that defines Neurological Manifestations of COVID-19. *International Journal of Infectious Diseases*. 2021 Mar;104:390–7.
- [20] Basu D, Chavda VP, Mehta AA. Therapeutics for COVID-19 and post COVID-19 complications: An update. *Current Research in Pharmacology and Drug Discovery*. 2022;3:100086.
- [21] Karadaş Ö, Öztürk B, Sonkaya AR. A prospective clinical study of detailed neurological manifestations in patients with COVID-19. *Neurological Sciences*. 2020 Jun 25.
- [22] Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic Manifestations of Hospitalized Patients with Coronavirus Disease 2019 in Wuhan, China. *JAMA Neurology*. 2020 Apr 10.
- [23] Koh JS, De Silva DA, Quek AML, Chiew HJ, Tu TM, Seet CYH, et al. Neurology of COVID-19 in Singapore. *Journal of the Neurological Sciences*. 2020 Nov;418:117118.
- [24] Jakhmola S, Indari O, Baral B, Kashyap D, Varshney N, Das A, et al.

Comorbidity Assessment Is Essential During COVID-19 Treatment.  
Frontiers in Physiology. 2020 Aug 4;11.

- [25] Kumar M, Thakur AK. Neurological manifestations and comorbidity associated with COVID-19: an overview. Neurological Sciences. 2020 Oct 14.

**Dr. Syed Hassan Tanvir Ramzi** was born in Multan, Pakistan in 1997 and completed his matriculation from high school, Multan in 2013 and his undergraduate medical degree (MBBS) from the University of Health Science, Lahore, Pakistan in 2021.

He worked in neurology during his internship years in Multan, alongside teaching further experience as a medical officer in internal medicine under the supervision of a senior consultant in Multan. He is also actively involved in clinical research with eight first-author PubMed indexed papers, meta-analyses, and case reports. Dr. Ramzi was ranked in the top ten during his undergraduate years and has presented his work on public awareness events.

**Dr. Ubaid Ullah Ansari** was born in Multan, Pakistan in 1989 and completed his undergraduate in 2012. He completed his postgraduate training in the field of Neurology at Punjab University Lahore, Pakistan in 2016, and joined as an associate professor in Neurology at Ibn-e-Sina Hospital and still to date.

Dr. Ansari is an active member of the Pakistan Society of Neurology.

**Dr. Sana Manzoor** was born in Multan, Pakistan in 1996 and completed her matriculation in high school, Multan in 2011. She completed her undergraduate medical degree from the University of Lahore, Pakistan in 2020 and completed an internship at Nishtar hospital Multan in 2020-2021. She is currently working as a medical officer at Ibn-e-Sina Hospital.

**Dr. Namal** was born in Faisalabad in 1996 and completed high school at DPS, an undergraduate from the University of Health Sciences with state honors. Currently working in a Social Security hospital in the Department of Medicine and High dependency unit since November till date.

**Dr. Nabeel Ahmed** was born in Multan, Pakistan in 1996 and completed her matriculation at high school, Multan in 2011. She completed her undergraduate medical degree from Lahore, Pakistan in 2020 and completed an internship from Nishtar Hospital, Multan in 2020-2021. She is currently working as a Postgraduate resident trainee in internal medicine at Ibn-e-Sina Hospital.