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NEUROIMAGING MANIFESTATATIONS IN COVID-19 AND ITS SEQUELAE

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Abstract

BACKGROUND: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) emerged as a novel pathogenic virus in late 2019, infecting millions of people with severe pulmonary infections. It spread worldwide in a short time and became a pandemic, after which the World Health Organization (WHO) declared it a public health emergency. Central nervous system involvement by COVID-19 is already an established entity in the literature. The aim of this study was to evaluate in detail the acute and delayed neurological manifestations of COVID -19.

RESULTS: COVID-19 infection can cause various neurologic manifestations and imaging findings. The most common neuroradiological abnormality is cerebrovascular events, which can also occur as a sequela of the infection. This study found a lower mean age of presentation in patients with cerebrovascular events, but no significant association was found between acute neurologic manifestations and demographic factors (like age, sex) or concomitant diseases. The severity of COVID-19 infection, as measured by the CT severity index (1), did not significantly impact neuroimaging findings, suggesting that they can occur even in patients with milder pulmonary symptoms.

CONCLUSION: Understanding neuroimaging patterns in COVID-19 is crucial for patient care and preventive measures. This study offers a comprehensive insight into the topic.

Keywords

Severe acute respiratory syndrome coronavirus, COVID-19, cerebrovascular accidents.

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INTRODUCTION:

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) emerged as a new pathogenic virus in late 2019 and began infecting millions of people with severe lung infections. It spread worldwide in a short time and became a pandemic, after which the World Health Organisation (WHO) declared it a public health emergency. The SARS-CoV-2 infection that triggered this pandemic has been named Corona virus disease 2019 by the WHO (COVID -19). Because of its uniqueness, contagiousness, and rapid mutation rate, COVID -19 caused worldwide excitement. There is ample evidence that SARS-CoV-2 infection of the brain can lead to a variety of neurological disorders and changes, ranging from mild to severe. The proportion of COVID -19 patients who experience headache, dizziness, fatigue and myalgia ranges from 30 to 45 percent. Anosmia or hyposmia, as well as taste, smell and vision disturbances, are considered moderately severe symptoms. In addition, paralysis, epilepsy and impaired consciousness are also described (2).

While the majority of COVID-19 individuals and those affected by sequelae have normal imaging studies, some patients show extra- and intra-axial abnormalities. There are reports of encephalopathy, meningitis, acute disseminated encephalomyelitis (ADEM), ischemic and hemorrhagic strokes, encephalomyelitis, and meningitis. It will be easier for radiologists and referring physicians to assess coronavirus infections in patients with worsening or progressive neurologic symptoms if they are familiar with these radiologic features (3).

Relative to the number of persons infected with SARS-CoV-2, the incidence of neurologic disease in COVID-19 patients is rather low, although the overall number has increased dramatically because of the pandemic. It is difficult for clinicians to distinguish between direct involvement of the nervous system and neurologic manifestations of systemic causes, such as metabolic disturbances and hypoxia, because many neurologic symptoms and signs are nonspecific, such as confusion, agitation, headache, generalised muscle weakness, delirium, and impaired consciousness.

Considering the above factors, imaging studies have been improved to facilitate the differential diagnosis of various neurological symptoms COVID-19. Impaired mental status, syncope, and localised neurological abnormalities seem to be the most typical reasons for neuroimaging. On the other hand, imaging studies have not been performed in all COVID-19 patients with neurologic symptoms because of the risk of exposing other patients or medical personnel to the virus. To understand the potentially harmful effects and to develop new pharmaceutical therapies and diagnostic methods for the treatment of COVID-19, it is essential to thoroughly understand the neuroinvasive processes and associated interactions. Neurologic symptoms may be caused by immunologic responses against the virus, metabolic abnormalities triggered by the infection, or direct viral effects on the central or peripheral nervous system. (4, 5, 6)

MATERIALS AND METHODS:

This was a retrospective-prospective cross-sectional study of 148 patients. Neuroimaging findings were evaluated over an 18-month period from December 1, 2020, to May 31, 2022, at the Department of Radiology, JSS Hospital, Mysuru. Retrospective data of all cases who met the inclusion criteria were gathered from May 2020 to November 2020 from the PACS and the hospital database. Patients with positive reverse transcription-polymerase chain reaction (PCR) for SARS-CoV-2 and COVID -19 probable case, defined according to the criteria of the WHO definition, who underwent cross-sectional imaging, were included. Patients who had recovered from COVID-19 were also included (All the cases were assessed within the time frame of 3-4 months after their COVID-19 infection). Excluded were subjects with head injury or pre-existing/incidental neoplastic lesions. The brain imaging findings were analysed with the help of 128 slice MDCT scanner and 3-Tesla MRI (Philips Intellispace portal 11.1.3.11608 software). MRI protocols used to evaluate the brain included T2-FSE- Axial, T1-FSE -Sagittal, Diffusion-weighted imaging, SWI, 3D- FLAIR and 3D TOF MRA.

Statistical analysis

Data was analyzed using SPSS 22 and presented as frequencies/proportions for categorical data and mean/standard deviation for continuous data. Chi-square/Fischer's exact test and independent t-test were used for significance tests. Microsoft Excel and Word were used to create graphs. Results with a P-value <0.05 were considered significant.

RESULTS:

Between May 2020 and May 2022, 148 patients who met the study criteria underwent neuroimaging studies. The age range of the study group was from 8 days to over 70 years, with a mean age of 51.65 +/¬- 23.36 years for patients with acute neurologic manifestations. [Fig- I] Young adults (31-50 years) made up 19.6% of cases and primarily had acute demyelinating encephalomyelitis, ANEC, and CVT. Older adults (over 50 years) mainly had cerebrovascular accidents, with a mean age of 56 years. Although strokes were more prevalent in older adults due to comorbidities, a significant number of strokes were also observed in younger subjects. There was no statistically significant difference in mean age between subjects with acute neurological manifestations (51.65 + 23.36 years) and those without (55.2 + 17.42 years). There was also no significant difference between subjects with and without neurological manifestations with respect to gender, with 57 women (38.5%) and 91 men (61.5%) [Tables VI- VII]. Most subjects were referred from the emergency department or intensive care units and had comorbidities. 36 patients had ischemic infarcts, 3 had spontaneous intracranial hemorrhage, 2 had spontaneous subarachnoid hemorrhage and acute on chronic subdural hemorrhage, 2 had acute necrotizing encephalitis and demyelinating encephalitis, 2 neonates had hypoxic ischemic encephalomyelitis, 2 cases had cerebral venous thrombosis with associated venous infarcts, and 1 patient had increased intracranial pressure. One patient in post-COVID status had early cerebritis with proven invasive sinusitis. Old infarcts were the only findings in a few cases that could not be attributed to ongoing or previous COVID-19 viral infection. Two of the subjects had T2W/ FLAIR hyperintensities in the brain parenchyma specific for postictal changes. Of the total 36 cases with ischemic infarcts, 5 had demonstrable thrombotic occlusion of the great vessels, 16 subjects had infarcts in specific vascular territories, 4 had infarcts in external and internal watershed zones, and some were distributed without a specific pattern, which could be due to embolism/vasculitis. A total of 5 subjects had acute infarcts in the status after COVID -19 in contrast to the majority of cases observed during active infection (31 cases). Two of them presented with ANEC in the recovery phase after COVID with altered mental status and decreased responsiveness. SARS-CoV-2-related demyelinating encephalomyelitis was observed in two young patients. In one of them, the manifestation occurred during acute infection and in the other after infection. In our study, two cases of early cerebritis were reported during and after the COVID -19 infection period with concomitant invasive fungal infection of the paranasal sinuses.

Features of idiopathic intracranial hypertension were noted in a young woman without known comorbidities during an acute viral infection. Spontaneous SAH and acute on chronic SDH were some of the other manifestations. There was no statistically significant difference between subjects with or without acute neurological manifestations compared to the study variables, including age, sex, COVID -19 status, and comorbidities (p-values of 0.29, 0.73, 0.53, and 0.22, respectively Represented in Table VI- X).The mortality rate in this study group was 11.4%. Thirteen patients underwent follow-up, of who 5 developed invasive fungal sinusitis, 3 developed a new infarct, and one developed hemorrhagic transformation of the previous infarct [Table X].

DISCUSSION:

In this article, we present spectrum of neurological imaging manifestations in COVID-19 patients admitted to JSS Hospital, Mysuru, India with peak during the second pandemic wave. SARS-CoV-2 infected patients most frequently exhibited acute respiratory symptoms, but some patients presented with acute neurologic symptoms at the time of admission or during their hospital stay. They underwent further studies including brain imaging, with CT brain being the first and most of the times the only neuro-diagnostic technique.

Cerebrovascular accidents have a documented incidence that ranges from from 0.9% to 8.0%. (7). Another study found that acute infarction, which affects 31% of patients, is the primary neuroimaging signature, with just 3% of patients experiencing cardioembolic events and 6% experiencing intracranial haemorrhages (8). Acute cerebrovascular accidents in COVID-19 are likely complex in origin, with preexisting diseases such hypertension, diabetes, and cardiovascular disease, as well as the severity of those diseases, being the major contributors. Although the precise function of SARS-CoV-2 is yet unknown, autopsy reports have shown that COVID-19 is linked to an increased frequency of thromboembolic events (9). The etiology of acute cerebrovascular accidents in COVID-19, probably is multifactorial, with preexisting conditions, like hypertension, diabetes, cardiovascular disease, and their severity of disease playing the main part. The exact role of SARS-CoV-2 remains unclear, but it has been documented in autopsy reports that COVID-19 is associated with an increased incidence of thromboembolic events (9).

In a literature review (10) about intracranial hemorrhage in COVID-19 patients, intraparenchymal hemorrhage was the most common variety (62.6%), followed by subarachnoid hemorrhage (15.0%), subdural hemorrhage (11.6%), and intraventricular hemorrhage (1.4%). Fabio Noro et al reported the first case of benign intracranial hypertension in a 35-year-old female COVID-19 patient, without comorbidities (11). Luke Dixon et al reported a 59-year-old COVID-19 patient with acute severe encephalopathy and was considered most likely due to an immune-mediated phenomenon. So the mechanism of neurological involvement in COVID-19 disease is a complex process with varied manifestations. Although no direct association has been found between the manifestations and the demographics/comorbidities in the present study, the interplay between these factors and the effect on neurological manifestations is yet to be studied in depth.

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Conclusions:

It is well known that COVID -19 infection can lead to a variety of symptoms in different organ systems. Understanding the neurologic and imaging findings associated with COVID -19 is critical for proper patient evaluation and therapy. It is well recognised that the disease manifests differently in different age groups. The most common neuroradiological abnormality in COVID -19 patients and in sequelae after COVID -19 was cerebral vascular events. Regarding the incidence of cerebrovascular accidents, a decrease was observed in the mean age group of incidences in the study. It is critical to comprehend long COVID and how they manifest, as many neurological impairments often do not appear until after COVID -19 recovery. In addition, it is important to monitor and follow up patients who have had COVID-19 infection.There was no statistically significant difference between subjects who had neurologic manifestations and those who did not in terms of study variables such as mean age, sex, COVID, or comorbidity status. There was no correlation between the severity of pulmonary COVID -19 infection, as assessed by the CT severity index, and neurologic manifestations, suggesting that neurologic manifestations occur even in the absence of severe pulmonary infection. Routine neuroimaging and follow-up is to be considered in patients with neurological symptoms or deficits, ensuing active COVID-19 infection. High index of suspicion of COVID-19 infection as one of the possible causes, for neurological deficits occurring in the post infective period should be taken into account.

Limitations:

Most of the patients, who underwent NCCT head for initial evaluation, had no significant abnormality, even in the presence of neurological deficits and symptoms. NCCT was not so sensitive, to detect stroke in the window period and few other subtle neurological abnormalities. The assessment of such patients became a tedious task in the study. Follow-up MRI brain was carried out only in few of the instances. The limited study sample size and the lack of follow-up gave inconclusive results in some of the cases.

Acknowledgements

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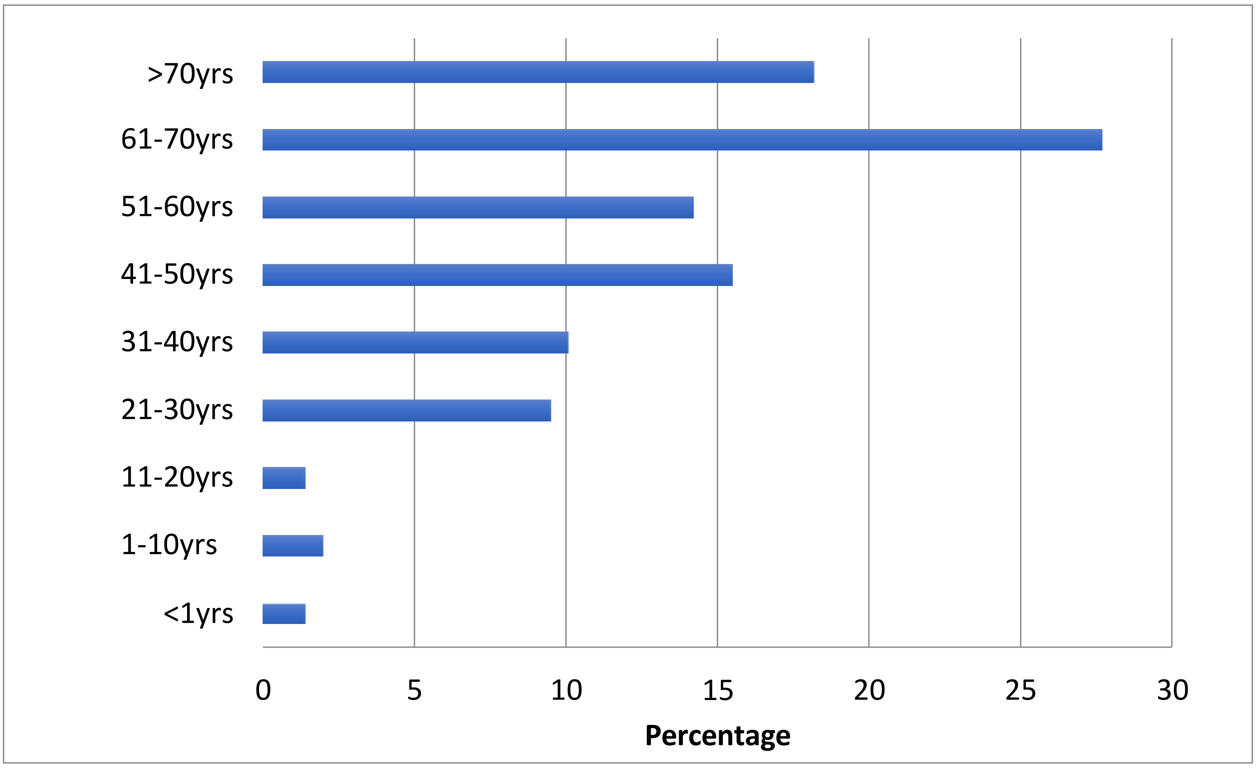
List of abbreviations:

SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2
COVID-19	Corona virus disease of 2019
WHO	World health organization
MRI	Magnetic resonance imaging
CT	Computed tomography
NCCT	Non-contrast CT
CT CA	CT cerebral angiography
3D	3 Dimensional
CVA	Cerebrovascular accident
DWI	Diffusion weighted imaging
SWI	Susceptibility weighted imaging
GRE	Gradient echo sequences
FLAIR	Fluid attenuation inversion recovery
ADC	Apparent diffusion co-efficient
CISS	Constructive Interference Steady State
ADEM	Acute disseminated encephalomyelitis
ANEC	Acute necrotizing encephalomyelitis
GBS	Guillain barre syndrome
CVT	Cerebral venous thrombosis
AIS	Acute ischemic stroke
TIA	Transient ischemic attack
IIH	Idiopathic intracranial hypertension
WHO	World health organization

OBSERVATION AND RESULTS

Figure I: Graph showing distribution of subjects according to age group



The age range of study participants was 8 days to over 70 years, with majority (27.7%) being between 61-70 years old. [Represented in Fig I]

Table I: Distribution of subjects according to gender

	Frequency (n)	Percentage
Female	**57**	**38.5**
Male	**91**	**61.5**
Total	**148**	**100.0**

Majority of the subjects were males (91 cases), among the total 148 cases. [Represented in table I]

Table II: Distribution of subjects according to COVID-19 status

	Frequency(n)	Percentage
COVID	98	66.2
COVID like illness/probable COVID-19.	10	6.8
POST COVID	40	27.0
Total	148	100.0

Of the 148 cases, 66.2% were positive for COVID-19 RT-PCR, 27% were post-COVID-19, and 6.8% had COVID-like illness– Represented in Table II

Table III: Distribution across time period

Time period	Cases
MAY 20-DEC 20	34
JAN 21-JUN 21	68
JUL 21-DEC 21	42
JAN 22-MARCH 22	4
TOTAL	148

Most of the cases were during the year 2021 in the first half as represented in the Table III.

Table IV: Distribution of subjects according to modality

Modality	Frequency(n)	Percentage %
CT(Plain study)	**111**	**75.0**
CT Cerebral angiography	**2**	**1.4**
MRI (Plain study)	**35**	**23.6**
Total	**148**	**100.0**

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The study subjects underwent non contrast CT head (75%), followed by non contrast MRI and CT cerebral angiography as represented by table IV.

Table V: Distribution of subjects according to imaging findings.

	Frequency (n)	Percentage %
No significant abnormality	82	55.4
Acute infarcts	24	16.2
Sub-acute infarcts	12	8.1
Intraparenchymal hematoma	3	2.0
ANEC	2	1.4
Demyelinating encephalomyelitis	2	1.4
Early cerebritis	2	1.4
Idiopathic intracranial hypertension	1	.7
CVT	2	1.4
HIE	2	1.4
SAH	3	2.0
Acute on chronic SDH	2	1.4
Post ictal changes	2	1.4
Chronic infracts	9	6.08
Total	148	100.0

The subjects had varied neuroimaging presentations, among which Cerebrovascular accidents (acute and sub-acute ischemic infarcts) were the majority. Table V represents the spectrum of the neuroimaging presentation of the subjects in the study.

Table VI: Comparison of mean age among subjects with and without acute neurological manifestations

Acute neurological manifestations	Mean	Std. Deviation	P Value
No	55.209	17.4276	0.292
Yes	51.651	23.3608	

Mean age among subjects with acute neurological manifestations was 51.65+23.36yrs and mean age among subjects without acute neurological manifestations was 55.2 +17.42yrs.

According to the individuals' mean ages, there was no statistically significant difference, between the subjects who had neurological manifestations and those who did not. [Represented in table VI]

Table VII: Distribution of subjects according to acute neurological manifestations and gender

	Acute neurological manifestations		Total
	No	Yes	
Female	34	23	57
	59.6%	40.4%	100.0%
Male	57	34	91
	62.6%	37.4%	100.0%
Total	91	57	148
	61.5%	38.5%	100.0%

No statistically significant difference existed between the people who had neurological manifestations and those who did not, when it came to gender of the subjects. (P value: 0.732)- Represented in table VII

Table VIII: Distribution of subjects according to acute neurological manifestations and COVID status

	Acute neurological manifestations		Total
	No	Yes	
COVID	**59**	**39**	**98**
	60.2%	**39.8%**	**100.0%**
COVID LIKE illness.	**5**	**5**	**10**
	50.0%	**50.0%**	**100.0%**
POST COVID	**27**	**13**	**40**
	67.5%	**32.5%**	**100.0%**
Total	**91**	**57**	**148**
	61.5%	**38.5%**	**100.0%**

There was no statistically significant difference between the people with neurological manifestations and those without them, with regards to their COVID status. (P value 0.539) - Represented in table VIII

Table IX: Distribution of subjects according to acute neurological manifestations and comorbidity

	Acute neurological manifestations		Total
	No	Yes	
Absent	74	41	115
	64.3%	35.7%	100.0%
Present	17	16	33
	51.5%	48.5%	100.0%
Total	91	57	148
	61.5%	38.5%	100.0%

When it came to the participants' comorbidity status, there was no statistically significant difference between those who had neurological manifestations and those who did not (P value: 0.224) - Represented in table IX

Table X: Follow up of the cases.

	Yes	N (13)	No	N (135)
Follow up imaging	Invasive fungal sinusitis	5		
	Hemorrhagic transformation	1		
	New infarcts	3		
	Nil	4		

Out of the total 148 cases, 13 of them underwent follow up imaging, among which 5 of them had invasive fungal sinusitis, 1 of them had hemorrhagic transformation of the infarcts and 3 of them had new infarcts.

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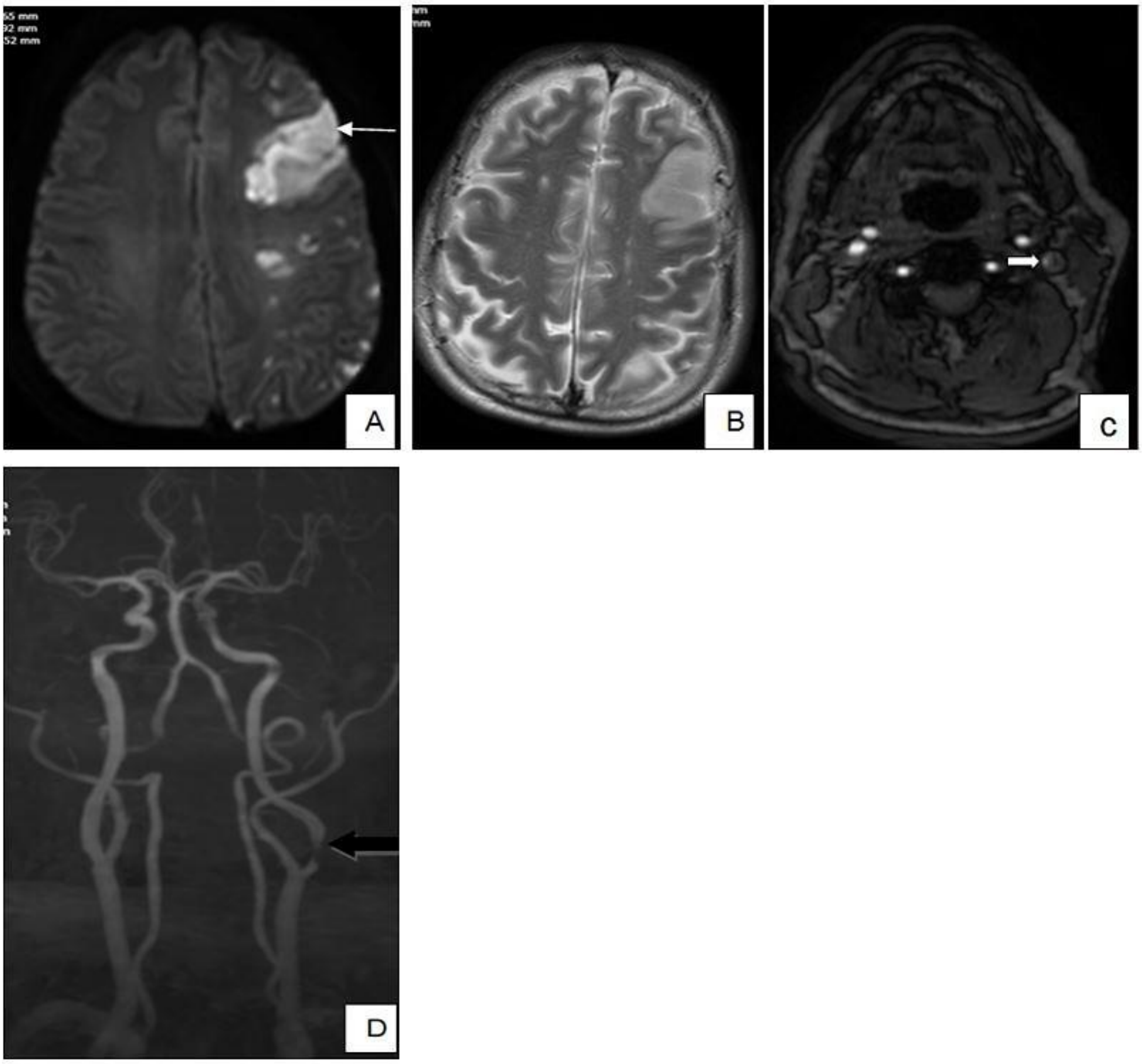

Fig: 1 A 62-year-old male COVID patient, who came with altered sensorium to the ED, had multifocal acute infarcts in left frontoparietal lobes with demonstrable narrowing of the ipsilateral ICA at its origin. Fig X depicts: [A], hyperintensities on DWI images in left front parietal lobes (white arrow) with corresponding T2W hyperintensities in image [B], Absent signal intensity (due to plaques/thrombosis) in ipsilateral left proximal ICA after its bifurcation with ~90% luminal narrowing (thick white and black arrows) resulting in infarcts in left frontoparietal lobes.

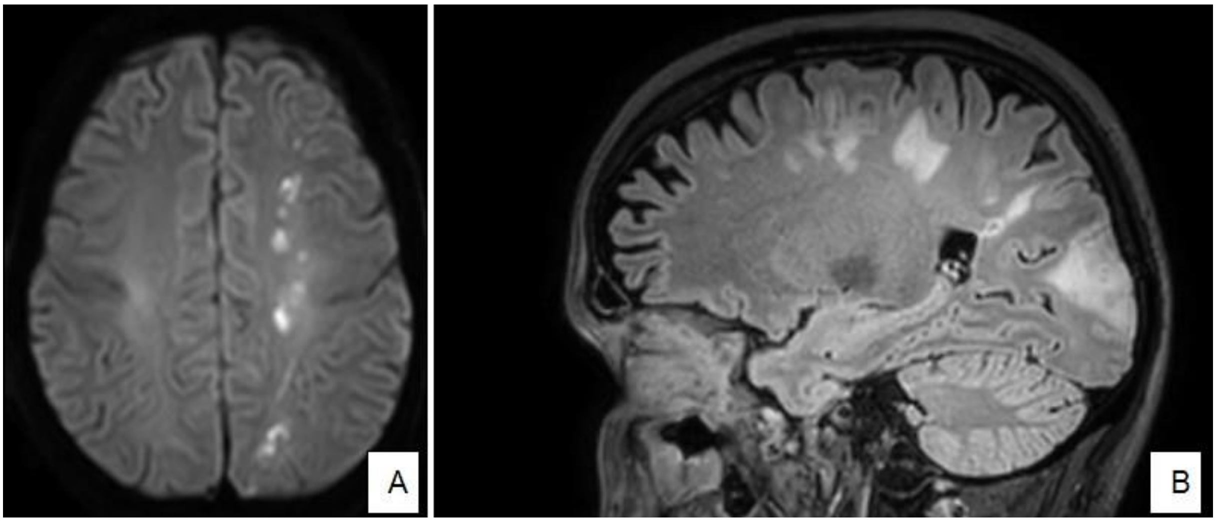

Fig 2: A 38-year-old young male in post-COVID-19 recovery presented with headache and altered sensorium. On MR imaging, [A] Axial DWI images showing hyperintensities in left frontoparietal lobes in watershed territories of ACA-MCA and MCA-PCA with corresponding hyperintensities on 3D FLAIR sagittal section depicted in [B]

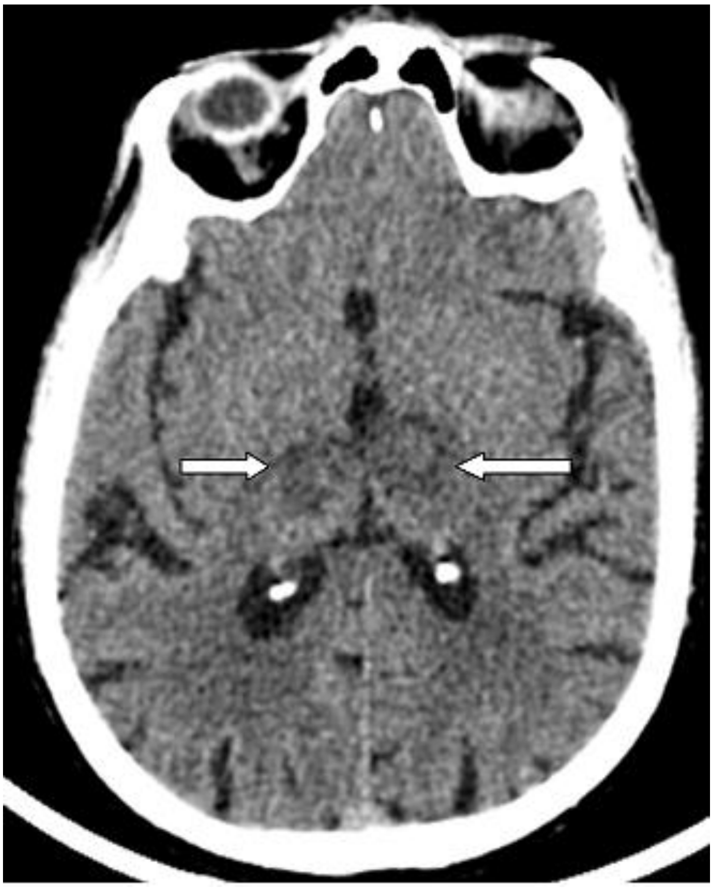

Fig 3: [A] 67-year-old male with decreased responsiveness and coexisting COVID-19 infection, underwent head CT plain where NCCT axial section shows bilateral thalamic infarcts with hemorrhagic transformation

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Fig 4: A 38 year old female patient with no known comorbidities came with hemiparesis and altered sensorium, NCCT plain axial sections showed large intra-parenchymal bleed in the right frontal lobe, capsulo-ganglionic regions with intra-ventricular extension, leftward midline shift and subfalcine herniation

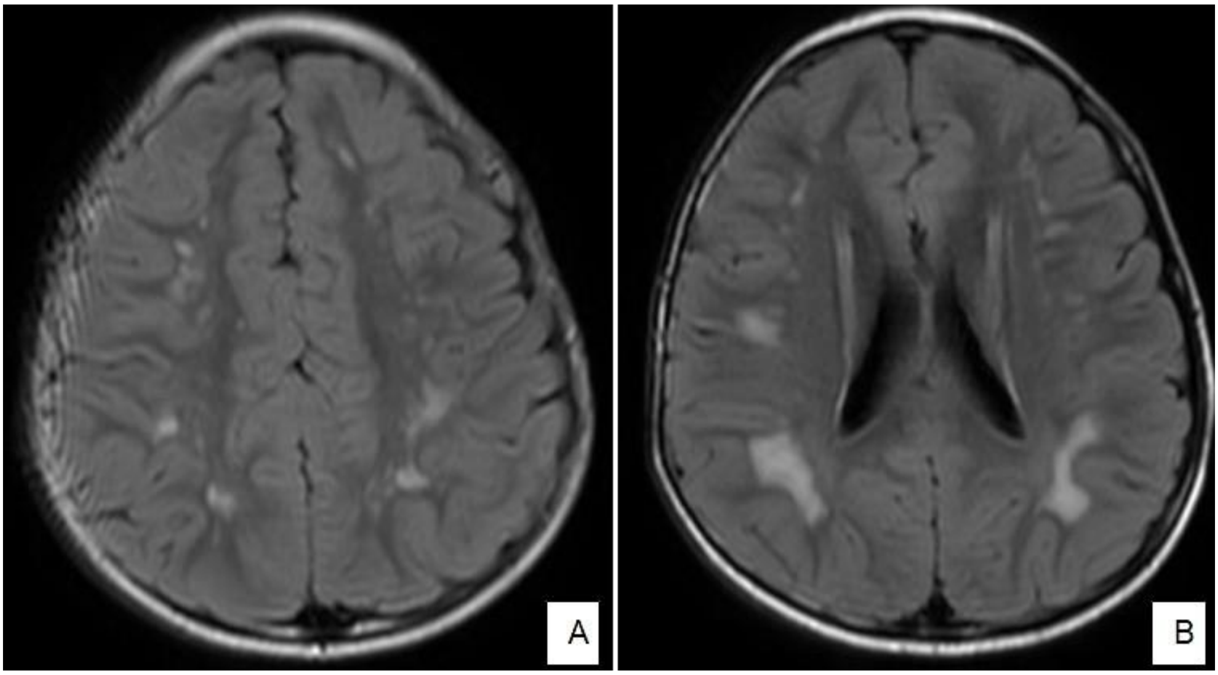

Fig 5: 5-year-old male patient during the post-COVID recovery period, presenting with few episodes of seizures had symmetrical FLAIR and T2W hyperintensities in bilateral fronto-parietal-temporal subcortical white matter. Fig [A] and [B] show the symmetrical FLAIR hyperintensities in bilateral fronto-parietal-temporal subcortical white matter with the sparing of the U fibres. There was no corresponding restricted diffusion/ blooming in these areas. A diagnosis of post-COVID demyelinating encephalomyelitis was made based on the history and imaging findings.

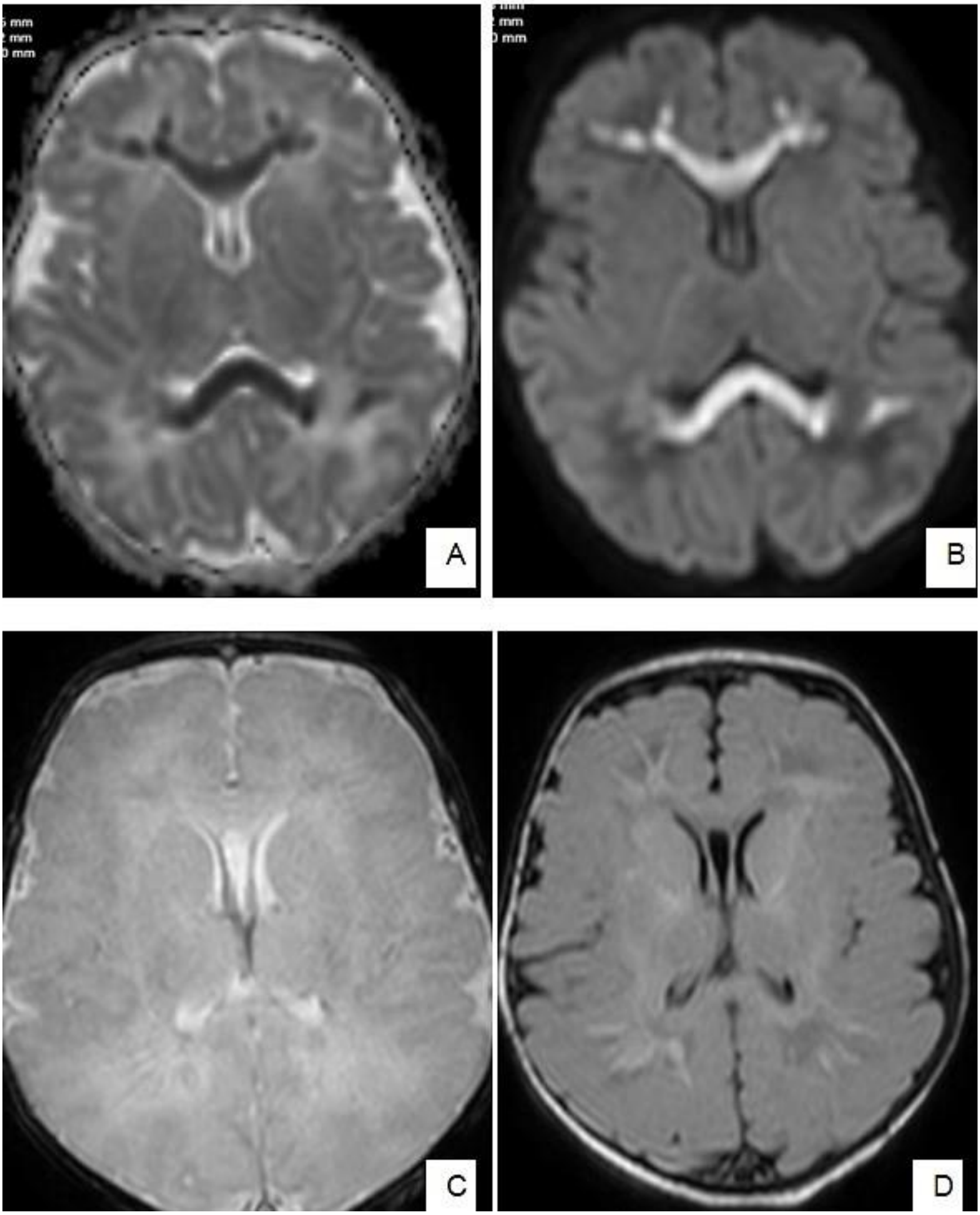

Fig 6: An eight day old male neonate presented with decreased responsiveness and had to be intubated because of low oxygen saturation in the ICU, MR imaging of the brain revealed altered signal intensities in bilateral corona radiata, genu and splenium of corpus callosum, subcortical white matter of bilateral fronto-temporo-parietal lobes with true restricted diffusion depicted in Figs [A] and [B] and appeared hyperintense on T2/ FLAIR depicted in Fig: [C] and [D].

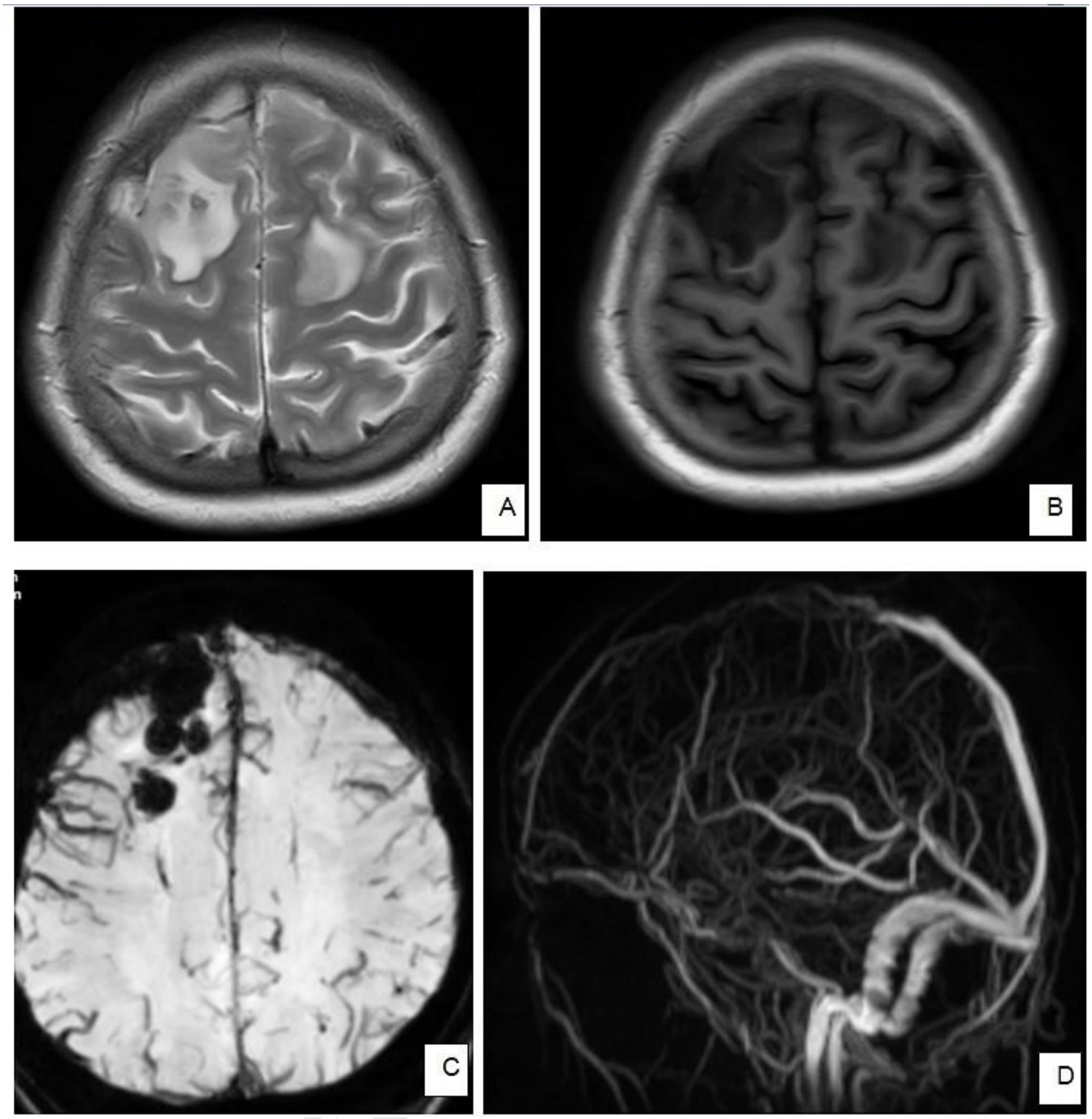

Fig 7: A 21 year old female with COVID-19 infection presented with seizures and fever, underwent MR imaging. Bilateral frontal lobe infarcts with hemorrhagic transformation on the right side was noted secondary to thrombosis of anterior half of superior sagittal sinus. (A) Altered signal intensities in bilateral frontal lobes in the parasagittal location appearing hyperintense on T2,(B) hypointense on T1 with blooming on GRE sequence in the right frontal lobe (C) and absent signal intensity in anterior half of superior sagittal sinus in (D) on 3D TOF-MRV.

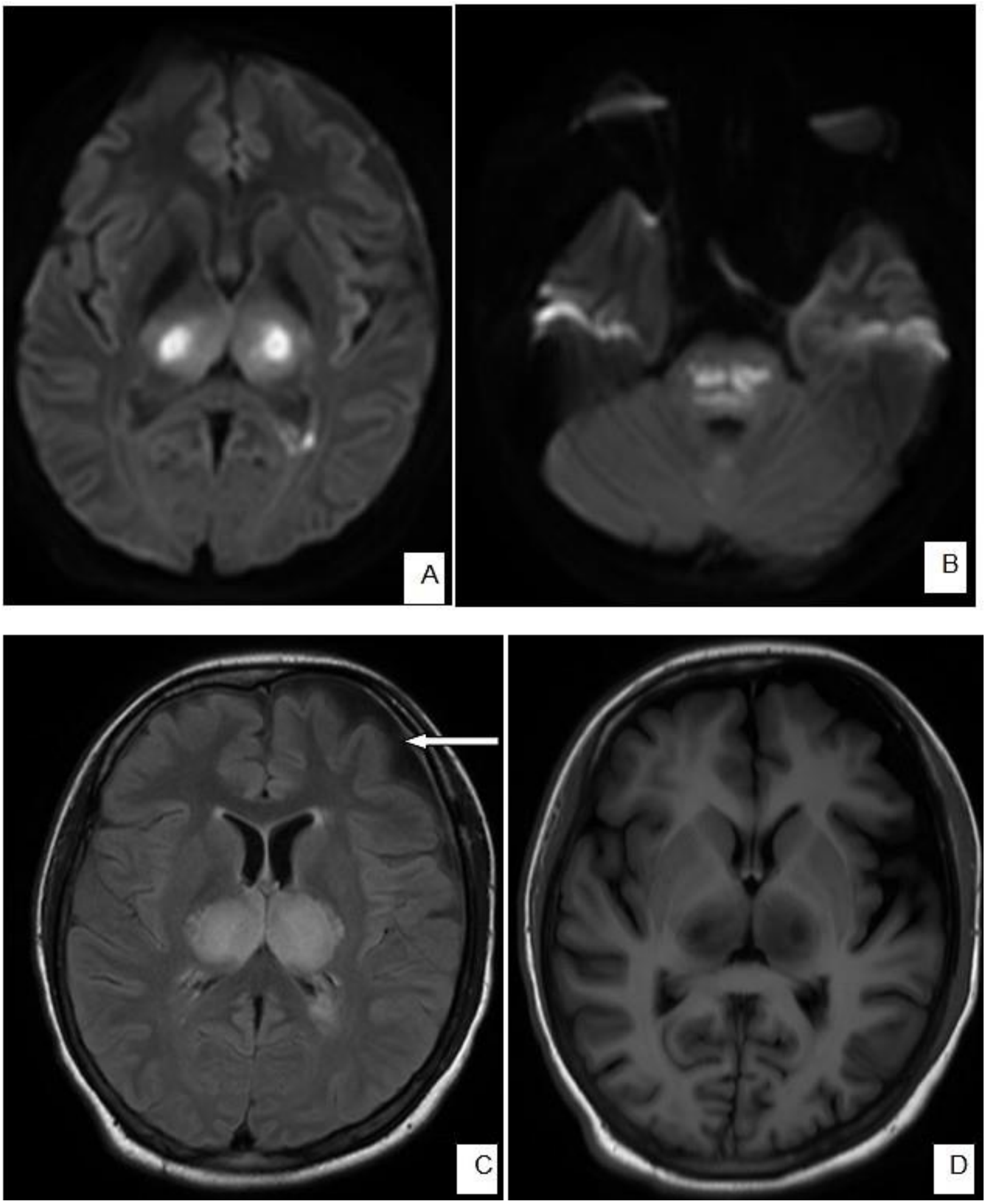

Fig 8: A 22-year-old female patient in post COVID-19 status with suspected meningoencephalitis, underwent MR imaging and multifocal near-symmetrical acute infarcts were noted in the supra and infratentorial brain parenchyma, a diagnosis of acute necrotizing encephalitis was made. [A and B] shows acute infarcts in bilateral thalami and pons with high DWI intensities, [C] shows corresponding T2 hyperintensities in bilateral thalami. Incidental left frontal subdural hygroma was also noted (white arrow). [D] shows central intense T1 hypointensity, representing necrosis.

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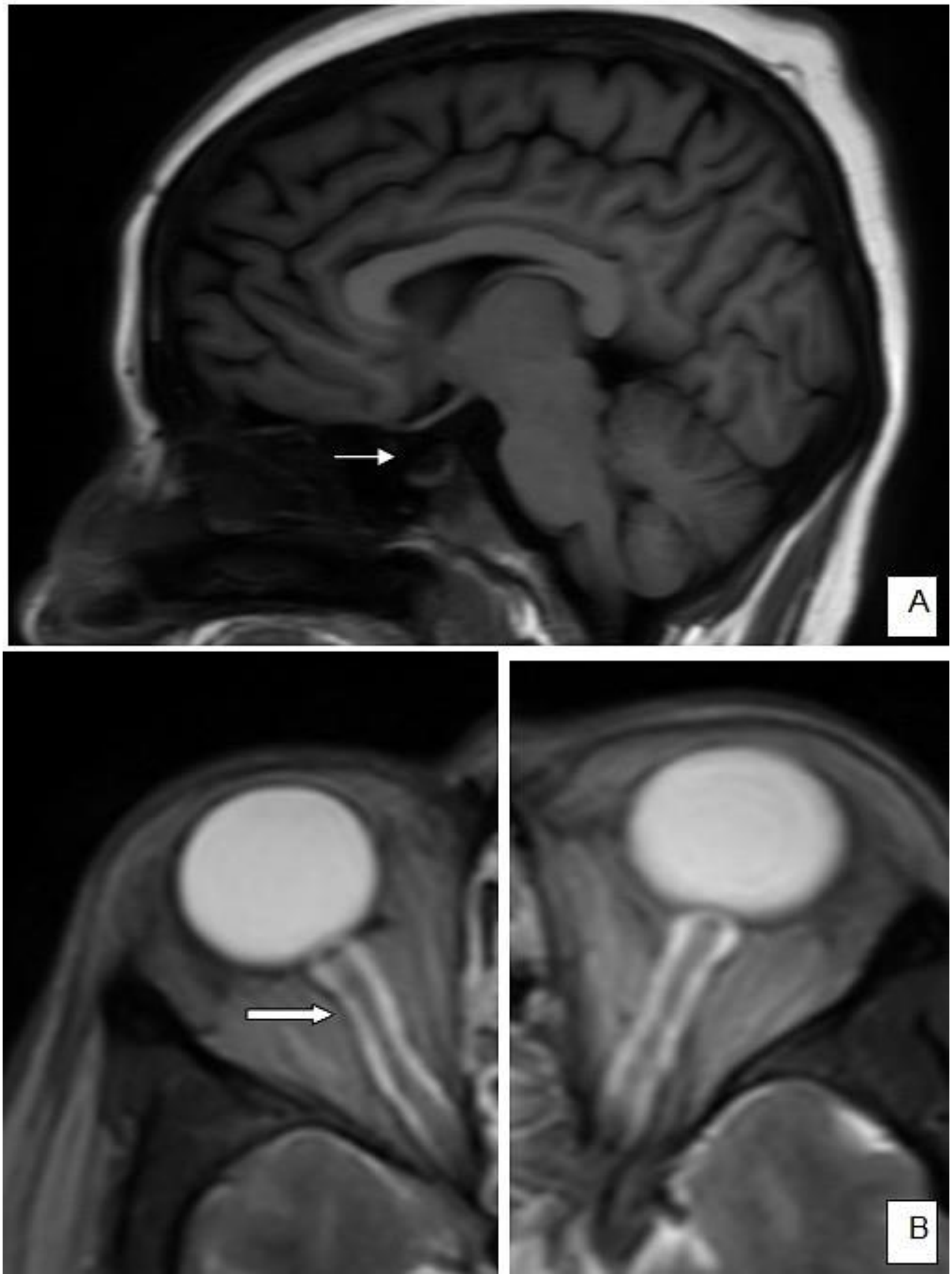

Fig 9: A 33-year-old female patient with COVID-19 bronchopneumonia presented with features of idiopathic intracranial hypertension. Fig 13A shows empty sella with flattening of the pituitary gland, 13B shows prominent subarachnoid spaces around bilateral optic nerves with posterior scleral indentation (indicating papilledema)

References:

1. Yang R, Li X, Liu H, Zhen Y, Zhang X, Xiong Q, Luo Y, Gao C, Zeng W. Chest CT severity score: an imaging tool for assessing severe COVID-19. Radiology: Cardiothoracic Imaging. 2020 Mar 30;2(2):e200047.
2. Singh V, Allawadhi P, Khurana A, Banothu AK, Bharani KK. Critical neurological features of COVID-19: Role of imaging methods and biosensors for effective diagnosis. Sensors international. 2021 Jan 1;2:100098.
3. Katal S, Balakrishnan S, Gholamrezanezhad A. Neuroimaging and neurologic findings in COVID-19 and other coronavirus infections: a systematic review in 116 patients. Journal of Neuroradiology. 2021 Feb 1;48(1):43-50.
4. C.-k. Chang, et al, Recent insights into the development of therapeutics against coronavirus diseases by targeting N protein, Drug Discov. Today 21 (4) (2016) 562–572.
5. Y. Guo, et al., Pathogenetic mechanisms of severe acute respiratory syndrome, Virus Res. 133 (1) (2008) 4–12.
6. Ladopoulos T, Zand R, Shahjouei S, Chang JJ, Motte J, Charles James J, Katsanos AH, Kerro A, Farahmand G, Vaghefi Far A, Rahimian N. COVID-19: Neuroimaging features of a pandemic. Journal of Neuroimaging. 2021 Mar;31(2):228-43.
7. Akhtar N, Abid F, Singh R, Kamran S, Imam Y, Al-Jerdi S, Salamah S, Al Attar R, Yasir M, Shabir H, Morgan D. Ischemic stroke in patients that recover from COVID-19: Comparisons to historical stroke prior to COVID-19 or stroke in patients with active COVID-19 infection. Plos one. 2022 Jun 24;17(6):e0270413.
8. Radmanesh A, Derman A, Lui YW, Raz E, Loh JP, Hagiwara M, Borja MJ, Zan E, Fatterpekar GM. COVID-19–associated diffuse leukoencephalopathy and microhemorrhages. Radiology. 2020 Oct;297(1):E223-7.
9. Wichmann D, Sperhake JP, Lütgehetmann M, Steurer S, Edler C, Heinemann A, Heinrich F, Mushumba H, Kniep I, Schröder AS, Burdelski C. Autopsy findings and venous thromboembolism in patients with COVID-19: a prospective cohort study. Annals of internal medicine. 2020 Aug 18;173(4):268-77.
10. Cheruiyot I, Kipkorir V, Ngure B, Misiani M, Munguti J, Ogeng'o J. Arterial thrombosis in coronavirus disease 2019 patients: a rapid systematic review. Annals of vascular surgery. 2021 Jan 1;70:273-81.
11. Noro F, Cardoso FD, Marchiori E. COVID-19 and benign intracranial hypertension: A case report. Revista da Sociedade Brasileira de Medicina Tropical. 2020 Jun 8;53.