Neurological Findings Associated with Neuroimaging in COVID-19 Patients: A Systematic Review

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ABSTRACT
Patients with COVID-19 and other coronavirus infections have been documented as having a variety of neurologic symptoms. In this article, we conducted a thorough evaluation of the imaging results of individuals who had been identified as having neurological symptoms linked to coronavirus infections. The use of CT and MRI has revealed varying radiologic outcomes in the setting of various neurologic presentations. Despite the fact that the majority of patients have normal imaging analyses, certain patients exhibit intra- and extra-axial abnormalities. There have been reports of encephalomyelitis, meningitis, acute disseminated encephalomyelitis (ADEM), ischemic and hemorrhagic strokes, and encephalopathy. Especially during the present COVID-19 pandemic, familiarity with these radiologic patterns can help radiologists and referring doctors investigate coronavirus infections in patients with worsening or progressing neurologic symptoms.
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Introduction

The worldwide unique Coronavirus illness (COVID-19) pandemic, which was initially discovered in Wuhan (China), has drawn much interest. Beyond 605 million cases of COVID-19 and over 6.4 million fatalities had been recorded throughout the world as of August 29, 2022.[1] Although most infected individuals appear with febrility and respiratory manifestations, multiple unusual presentations, including gastrointestinal issues, cardiac problems, renal failure, and neurological abnormalities, have been observed recently.[2,3] A case series that studied 214 COVID-19 hospitalized individuals from hospitals in Wuhan City discovered that 36.4% of them experienced neurological symptoms such as headaches, dizziness, and impairment of consciousness. Additionally, in several additional accounts, neurologic symptoms have been described as the SARS-CoV-2 infection's early presentation.[4-6] COVID-19 provoked a tremendous degree of attraction between researchers, medical professionals, and scientists worldwide.[6]

There has never been anything like the amount of work being done or papers prepared on COVID-19. It was predicted that hundreds of articles would be publicized about the issue starting at the beginning of year.[7] Information about many factors of illness prevention, transmission, pathophysiology, symptoms, and management techniques is beginning to emerge.[8-10] In spite of the fact that COVID-19 positively influences the cardiovascular and respiratory systems, many COVID-19 patients are also susceptible to neurological complications like acute cerebrovascular diseases, encephalopathy, skeletal muscle injury, and impaired consciousness. These complications include neuralgia, hypogeusia, dizziness, and headache.[11,12] Although the literature on COVID-19's characteristic respiratory presentation has received much attention 7, there is a dearth of in-depth research on its cerebral symptoms, particularly the radiological findings. Besides other healthcare professionals, Radiologists must be acquainted with the range of neurological abnormalities connected to this virus despite the current scant evidence. It was because of this that we decided to perform this systematic evaluation of myriad radiological abnormalities with concurrent neurological complaints in patients with COVID-19.

Lessons learned from prior coronavirus epidemics

Similar to the SARS-CoV-2 strains, the previous Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV) and the Middle East Respiratory Syndrome Coronavirus (MERS-CoV) are members of the coronavirus family. Over 10,000 people worldwide have contracted MERS-CoV and SARS-CoV outbreaks over the past 20 years.[8,9] According to several studies, neurologic sequelae, such as seizures, Guillain-Barre syndrome (GBS), encephalopathy, anosmia, encephalitis, neuromuscular abnormalities, as well as demyelinating illnesses, may develop in connection with respiratory coronavirus syndromes.[10–13] The ability to invade and live in neural tissue of additional coronaviruses of humans, such as HCoV-NL63, HCoV-HKU1, HCoV-OC43, and HCoV-229E, as well as the potential links to neurological conditions including multiple sclerosis disease (MS), has also been disputed.[14,15] It has been proposed that the host immunological response, which includes inflammatory cascades including cytokine activation, may be the cause of the said neurological abnormalities.

Meanwhile, postmortem examinations of people who died from SARS illness have shown viral RNA in neurons, suggesting coronaviruses may be capable of infecting neurons 16 by themselves. Cerebrovascular endothelial cells' receptors of angiotensin-converting enzyme (ACE) 2nd may be crucial to this process. We made the decision to review the existing literature on brain imaging results related to COVID-19 in light of the identical viral configurations with similar neurological post-viral consequences. This may help in the fast diagnosis and expeditious therapy for neurological disorders linked to infection with COVID-19, as well as offer insightful information about coronavirus pathogenesis.

Materials and Methods

Search Strategy

"What are the documented results of neuroimaging in cases infected with coronavirus?" was our study question. A thorough literature search was carried out utilizing online databases of Scopus and Medline (evaluated from PubMed) to look for published publications describing pertinent imaging findings. The following keywords were used in the search: "coronavirus" OR "SARS-CoV-2" OR "COVID-19" AND "brain" OR "neurologic" OR "CNS" OR "Central Nervous System" AND "CT-scan" OR "computed tomography" OR "MRI." articles that had the search phrases in the keywords, title, abstract, or body of the paper were chosen for study. This review even looked through the references in the chosen publications to see if there was any further research that would be relevant to the COVID-19 brain imaging findings. A manual inquiry was correspondingly made on Google Scholar and Google Search to include pertinent non-indexed reports. On August 6, 2022, the search was launched, and on August 20th, it was updated. No consideration was given to language. Multiple studies were eliminated. Several publications broadly discussed COVID-19's neurologic symptoms but did not disclose any data from imaging of the brain, which were correspondingly disregarded. This type of reviewing has been used by various researchers in medicine.[43]

Study selection and definitions

Two analysts individually combed through every pertinent publication and summarized them. Included was every paper that discussed MRI or CT of brain results in cases with COVID-19. There were no filters used in the search. The PRISMA declaration explains the article and search selection strategy with a step-by-step diagram shown in Figure. 1.

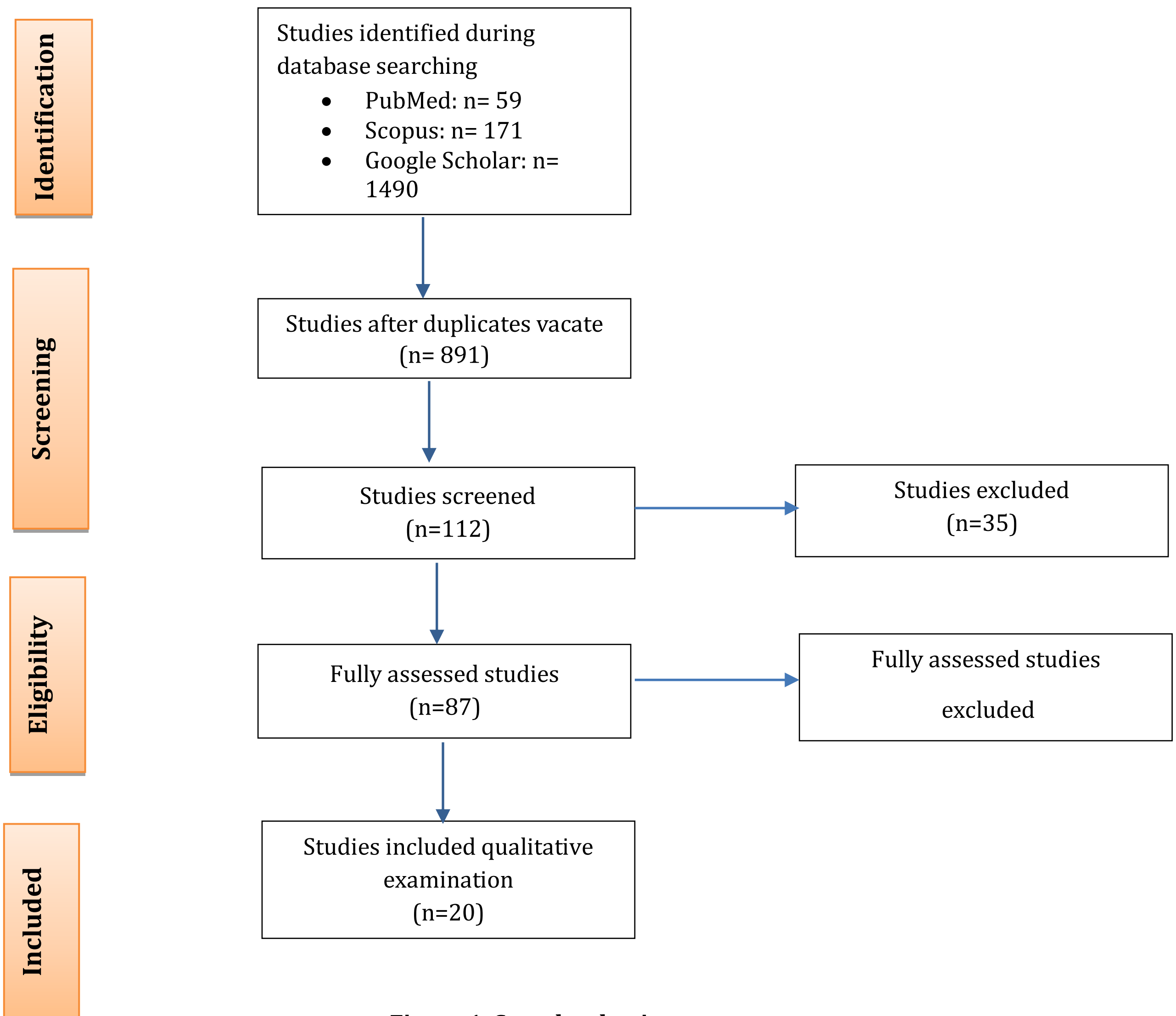

Figure 1. Search selection strategy.

Data extraction, Management, and Quality assessments

Authors separately collected the following information from the forms: research methods, specimen scope, imaging techniques, key findings, lab COVID-19 confirming tests (utilizing CSF or pharyngeal samplings), and significant remarks on various cases. Based on the nine elements contained in the National Institutes of Health Quality Assessment aid in Studies of Case Series 17, the methodological quality of the covered articles was evaluated.

Table 1: Neuroimaging spotting between cases with COVID-19 corresponding neurologic symptoms and signs.

Neuroimaging spottings	Number of patients and explanations (sum of patients = 90)	
No specific spotting (normal)	**37 (41%)**	
Hemorrhage	7	1: Intra-axial/Intra-parenchymal basal ganglia lesion. 1: Giant intra-parenchymal hemorrhage concerning the right hemisphere, with intraventricular elongation and subarachnoid segment. 5: Intra-parenchymal hemorrhage as: 2 frontal, 1 cerebellar, 1 hemispheric, and 1 parietal.
Hemorrhagic PRES	2	
Vascular thrombosis	14	1: PCA thrombosis. 1: Thrombosis in transverse sinus (acute lesion). 1: Right MCA infarction (acute lesion). 1: Right MCA and bilateral ACA regions infarct. 4: Ischemic features (without any additional explanations). 2: Hemorrhagic infarction (due to CVT) 1: AIS (Acute Ischemic Stroke) due to as a complication of gigantic afloat thrombus at common carotid artery site. 3: Two patients with AIS, and the other one with restricted focal DWI and decreased ADC.
Non-specific terms	26	10: Abnormalities in cortical FLAIR signal. 10: two-sided decreased perfusion of frontotemporal territories, and enhancing leptomeningeal regions. 6: Cerebrovascular events (acute strokes).
Others	4	Meningitis/encephalitis (2), ADEM (1), ANE (1).

Results

A Preview of the Covered Studies

Tables 2 list the papers and pertinent findings that were part of this literature study. The initial results of the electronic literature searches were 850 items. Our systematic review's inclusion criteria were satisfied by 28 studies reporting imaging findings following manual filtering of the papers based on their titles or abstracts. Seven research were presented as case series, while 21 studies were case reports. Twenty research assessed MR imaging, whereas 17 papers employed CT. Due to the infrequently reported occurrences globally, sample sizes in the majority of the included studies were modest. The studies' methodological quality was typically graded as fair, which is an indication of the trim and poor-quality information that there is on findings about viral neurologic phenomena.[18]

MRI and CT Manifestations of COVID-19 Infection

This review included 20 case reports or case series that had been infected with COVID-19. Additionally, there were two more extensive COVID-19 case series of individuals that had neurological symptoms, although they lacked particular radiological evidence.[3,19] Examining 214 patients at Wuhan hospitals served as the first and most extensive investigation of neurologic symptoms in COVID-19 infection.[3] They discovered that 36.4% of those individuals displayed some neurologist symptoms, the most prevalent of which were headache and dizziness. Acute stroke accounted for (5.7%), and awareness deterioration was among those neurological disorders that affected fewer people (15 percent). Although no particular CT findings have been documented, researchers highlighted those acute cerebrovascular illnesses, such as hemorrhagic and ischemic stroke, have been identified by a head CT and the existence of clinical symptoms. The study emphasized how crucial it is to recognize neurologic signs as a critical presentation of COVID-19 indication. Seven individuals had their CSF specimens sampled for infection with SARS-CoV-2, and among the 20 studies that were available,[20–22] only one resulted in a positive result when meningitis/encephalitis signs were present.[21]

The other studies returned negative results. The first instance of meningitis linked to SARS-CoV-2 21 was a male 24 years old presenting with a fever, exhaustion, a headache that was becoming worse, and a sore throat. A couple of days later, the man started having convulsions and lost consciousness. The right lateral ventricle's temporal horn had periventricular diffusion limitation, according to diffusion-weighted imaging (DWI). On Fluid-attenuated Inversion Recovery (FLAIR) imaging, the hippocampus and right mesial temporal lobe showed hyperintense signal irregularities associated with mild hippocampal shrinkage. Imaging with contrast did not clearly reveal any aberrant intracranial enhancement. These results suggested encephalitis and right lateral ventriculitis. SARS-CoV-2 RNA was not found inside the swab of the patient's nasopharynx, but the virus was found in the CSF sample. According to the authors, there is a slight chance that the COVID-19 virus might occasionally directly infect the brain. Thirty-seven cases (41%) in which their COVID-19 infection was confirmed by laboratory exhibited no critical irregularities on brain MRI or CT between the 20 case reports (90 patients) with accompanying neurological symptoms.[4,6,20,22–26] These individuals' symptoms included hemisensory paresthesia, frequent seizures, epileptics, changed mental state, headache, and so on. The rest of the 12 articles (53 patients) that had COVID-19-related neurological symptoms have reported atypical findings on their brain radiology, including hemorrhage[5,26,27] in seven patients, acute hemorrhagic necrotizing encephalopathy 33 (ANE; one patient), vascular thrombosis in 14 patients[22,26,29-32,35] hemorrhagic posterior reversible encephalopathy syndrome 28, acute disease. In a case series implemented by Helms and others 35, 13 individuals who had undergone brain MRI were shown to have leptomeningeal enhancement, bilateral frontotemporal hypoperfusion, and abnormalities related to stroke. Tables 1 and 2 detail all of the findings mentioned earlier.

Table 2: Neuroimaging discoveries of cases with COVID-19.

Study topic	Study type	Number of cases	Imaging method	Neurologic findings	Radiologic discoveries
Vollono C, et al. [4]	Case report	1	MRI	When SARS-CoV-2 infection is present alongside well-controlled post-encephalitic epilepsy, the initial presentation of the condition is focal status epilepticus.	For acute lesions, negative. Absent fresh brain lesions, the left temporo-parietal lobe exhibits gliosis and atrophy.
Poyiadji N, et	Case report	1	CT, MRI	Altered mental status.	Bilateral medial thalami

al. [33]					show symmetric hypoattenuation on the CT scan. standard CTA and CTV. MRI: Bilateral thalami, medial temporal lobes, and subinsular areas with hemorrhagic rim enhancing lesions. Dx: ANE.
Vu D, et al. [5]	Case report	1	CT. MRI	Right facial droop, dysarthria, and hemiparesis to the right	acute left basal ganglia hemorrhage measuring 1.7 cm
Singhania N, et al. [6]	Case report	1	CT	altered state of mind, syncope	whiteout any acute lesion
Karimi N, et al. [20]	Case report	1	MRI	Generalized tonic-clonic, seizure	whiteout any acute lesion
Ye M, et al. [25]	Case report	1	CT	Confusion, Myalgia, Extensor plantar response, Meningeal irritation signs	whiteout any acute lesion
Sharifi-Razavi A, et al. [27]	Case report	1	CT	abrupt unconsciousness	a significant intraventricular and subarachnoid hemorrhage, along with a major ICH in the right brain.
Moriguchi T, et al. [21]	Case report	1	MRI	Generalized tiredness, a headache, a brief generalized seizure consciousness loss, Neck discomfort	DWI: Hyperintensity along the right lateral ventricle's temporal horn wall. FLAIR: Right mesial temporal lobe and hippocampus exhibit hyperintense signal alterations with mild hippocampal atrophy. There was no discernible dural augmentation. Dx: Right lateral ventriculitis with encephalitis, mostly affecting the hippocampus and right mesial lobe. On T2-weighted images, pan-sinusitis was further detected.
Filatov A, et	Case report	1	CT	altered mental	There are no obvious

al. [23]				condition and a headache.	anomalies. Left temporal encephalomalacia, which is indicative of a distant embolic stroke.
Mao L, et al. [3]	Case series	6	CT	Not described	Acute Cerebrovascular disease
Herrnberger M, et al. [24]	Case report	1	MRI	Paresthesia in the left face, left arm, and left leg. Refractory headache.	standard immediate MRI
Fields B, et al. [29]	Case report	1	CT	Loss of eyesight on the right side, clumsiness Right homonymous hemianopsia with macular sparing and dysmetria in the right extremities were found during the examination.	CT: Reduced parenchymal hypoattenuation in the right cerebellar hemisphere, with loss of gray-white matter distinction in the left occipital and temporal lobes. CTA: A thrombus in the left PCA was confirmed.
Zhang T, et al. [34]	Case report	1	CT, MRI	Encephalopathy, dysarthria, and dysphagia	There was no sign of ICH on the CT scan, although there were many patches of hypoattenuation. MRI: Bilateral frontoparietal white matter, anterior temporal lobes, basal ganglia, external capsules, and thalami show extensive patchy regions of aberrant signal. Some displayed dubious little improvement together with DWI modifications and matching ADC alterations. MRA: Regular. ADEM: DDX
Kandemirli SG, et al. [22]	Case series	27	MRI	ICU patients with COVID-19 who experience neurological symptoms (Not precisely described).	15 of 27 (56%) had a normal MRI. Acute MRI findings on 12/27 (44%), including: 10/27 (37%) patients had aberrant cortical FLAIR signals, with the frontal lobe accounting for 4

					instances, the parietal lobe for 3, the occipital lobe for 4, the temporal lobe for 1, the insular cortex for 3, and the cingulate gyrus for 3 cases (accompanied by subcortical and deep white matter signal abnormality on FLAIR images). Acute right MCA infarction and transverse sinus thrombosis (1 case each) (1 case).
Fields B, et al. [29]	Case report	2	CT, MRI	Hemorrhagic PRES: Improved prognosis, labile blood pressure, and altered mental condition	First case: On CT and MRI, there was a minor right-sided bleeding along with bilateral posterior parietooccipital lobe focal vasogenic/cytotoxic edema. CTV: Ordinary. The corpus callosum has significant petechial hemorrhages throughout. Second case: MRI revealed many regions of limited diffusion and accompanying edema, mostly in the right frontal lobe, basal ganglia, and cerebellar hemispheres but also mostly in the posterior parietooccipital lobes. SWI: Abnormal enhancement and large superimposed hemorrhages in the parietooccipital area. Diagnosis: syndrome of hemorrhagic posterior reversible encephalopathy (PRES).
Goldberg MF, et al. [30]	Case report	1	CT, CTA	stroke with left hemiparesis and	CT: In the right MCA region and bilateral

				breathlessness	ACAs, there are significant areas of moderate hypoattenuation and lack of gray-white matter distinction. High-grade stenosis at the proximal ICA on extracranial CTA. Intracranial CTA: Significantly reduced flow at the right MCA branch Follow-up CT: Increasing cerebral edema and mass impact brought on by the bilateral ACA infarcts and right MCA, as well as the progression of acute ischemia.
Viguier A, et al. [31]	Case report	1	CT, CTA, MRI, DS	Right hemiparesis, acute aphasia. Examination: NIH stroke scale of 10 with left MCA syndrome.	Subtle left frontal cortical hypoattenuation on CT and CTA, along with distal branch blockage and surrounding hypoperfusion. Cervical CTA: The left CCA's hypoattenuated, non-stenosing plaque has a sizable intraluminal floating thrombus attached to it. The diagnosis of a significant thrombus attached to a thin atheromatous plaque was confirmed by dedicated wall imaging using 3 T MRI and DS. DWI: AIS across the left carotid region with sporadic foci of hyperintensity. Dx: A big floating thrombus at CCA is complicated by an AIS.
Poillon G, et al. [32]	Case report	2	CT, MRI	Headache and visual changes in the first patient are swiftly	Large hemorrhagic infarction in the left temporal lobe and large

				followed by a right hemicorporeal deficit and disturbed consciousness. Second patient: excruciating pain	confluent ICH at the left frontotemporal lobes were seen on CT and MRI scans, respectively (2nd case). CTV and MRA: Left transverse sinus, internal cerebral veins, straight vein, vein of Galen, and CVT of the left transverse sinus (1st instance) (2nd case).
Andrea G, et al. [26]	Case report	26	CT, MRI	Dizziness, headache, paresthesia, and others	Non-acute incidents made up 16/26 (61.6%). 5/26 (19.2%): Hemorrhage in the parenchyma. Ischemic alterations on 4/26 (15.4%). Encephalitis on 1/26 (3.8%).
Helms J, et al. [35]	Case report	13	MRI	Unaccounted-for encephalopathic symptoms	Leptomeningeal augmentation in 8 instances 11/11 cases: All 11 patients who underwent perfusion imaging had bilateral frontotemporal hypoperfusion. Two cases: in each, there was a mild acute ischemic stroke accompanied with overlapping reduced ADC and localized hyperintensity on DWI. 1 case: superimposed enhanced DWI and ADC signals from a subacute ischemic stroke.

CT: Computer Tomography; CTA: Computer Tomography Angiogram; MRI: Magnetic Resonance Imaging; CTV: Computer Tomography Venogram; DS: Doppler Sonography; MRA: Magnetic Resonance Angiogram; DWI: diffusion weighted imaging; ADEM: Acute Disseminated Encephalomyelitis; ICH: Intracranial Hemorrhage; ADC: Apparent Diffusion Coefficient; CCA: Common Carotid Artery; MCA: Middle Cerebral Artery; CVT: Cerebral Venous Thrombosis; ACAs: Anterior Cerebral Arteries; AIS: Acute Ischemic Stroke; ANE: Acute Necrotizing Encephalopathy

Discussion

Cases infected with COVID-19 or different coronaviruses have been identified in the company of a variety of clinical neurologic symptoms. On this subject, there is, however, little information. Since the majority of the articles do not contain the matching results from imaging of the brain, there is even less information accessible concerning connected neuroimaging discoveries. Still, in the absence of pulmonary symptoms, coronaviruses may exhibit neurotropic and neuroinvasive characteristics, according to earlier investigations. Similar to this, multiple fresh case reports during the current COVID-19 epidemic have once more raised the possibility of a connection linking neurological signs and infection with COVID-19. Headache, anosmia, myalgia, and cerebrovascular illness, besides encephalopathy, are some of the uttermost prevalent neurologic signs connected to infection with SARS-CoV-2, according to a reasonably recent analysis.[42] Several theories have been put up to explain these anomalies. While some writers have postulated neuronal retrograde dissemination, transcribrial, and hematogenous, routes as a straightforward way of a viral attack on cells of the human brain, others have proposed a hyperimmune reaction related to storms of the cytokine to explain the said neurological manifestations. Additionally, the receptors of angiotensin-converting enzyme 2 (ACE2), which endothelial cells from brain capillary express, may act as a mediator in the CoV neurotropism, particularly SARS-CoV-2. Brain injury that is permanent as a result of cerebral endothelial rupture has a part in the pathogenesis of SARS-CoV-2 neurologic symptoms.[36-40] In addition, coronavirus patients may experience cerebrovascular events as a result of raised amounts of D-dimer and CRP brought on by activation of hypercoagulation cascade and a state of high inflammation. As a result, although it is still too early to be confident, a mix of immunological, vascular, and neural variables might be the likely pathways. Beyond one-third of the cases that were hospitalized are said to have CNS involvement from coronavirus infection[42], with severity ranging from minor to life-threatening diseases. Therefore, particularly during the ongoing pandemic, it should be taken into account in the differential diagnosis of each individual presenting with indefinable increasing neurological symptoms. In these situations, neurologic imaging could be useful since a prompt diagnosis is crucial for preventing additional brain injury. Here, the article evaluated the radiologic symptoms in coronavirus infection of the CNS caused by several coronavirus strains (COVID-19, MERS-CoV, SARS-CoV, and different strains of CoV). According to our information, this is the first systematic study that describes the outcomes of neuroimaging in cases infected with coronavirus, especially COVID-19. Like previously indicated, several results have been made using neuroimaging modalities, including CT and MRI, in the context of diverse clinical settings. In contrast to the other patients who showed imaging irregularities of various regions in the brain, such as acute cerebrovascular occurrences (hemorrhagic and ischemic classifications), demyelinating disorders (ADEM), encephalopathy, meningitis, myelitis, and only a tiny percentage of COVID-19 patients (40 percent) had expected results. The most prevalent neuroradiological anomaly observed in COVID-19 individuals is reported to be cerebrovascular occurrences, including both hemorrhagic and ischemic episodes (27 percent, Table 1).

Individuals with coronavirus infection are thought to be more susceptible to cerebrovascular illness and incredibly sick elderly patients with a variety of vascular risk factors. As was already indicated, the etiology of cerebrovascular disease in these individuals may be significantly influenced by coagulation malfunction and a hyperinflammatory response. According to Munhoz RP and others 42, between 2.8 and 5.7% of individuals infected with the virus had an acute cerebrovascular illness (primarily ischemic, seldom venous thrombosis or hemorrhagic). According to Asadi-Pooya AA et al.[18] and also Mao L.3, 5-5.[7] percent of COVID-19-related neurologic symptoms are caused by ischemic or hemorrhagic CVD. Due to their augmented danger of acute cerebrovascular events, individuals having robust coronavirus infection and vascular risk factors should be treated with particular caution. In a few cases, myelitis and encephalopathy-related alterations that appear like a parenchymal sign irregularity of various areas in the brain were documented as additional neuroimaging abnormalities (Tables 1,2). Generally speaking, the neurological symptoms linked to infections with coronavirus, particularly SARS-CoV-2, point to a potential causative or synergistic connection between the infection and brain ischemic/hemorrhagic/inflammatory symptoms. It should be emphasized that some of these results could be explained by coincidental events rather than random association. Prolonged neurological consequences have also not been yet researched. Therefore, additional research is necessary to resolve these unanswered problems.

Conclusion

Numerous case reports from the present worldwide COVID-19 pandemic have raised the possibility that SARS-CoV-2 viral infection and neurological manifestations are related, comparable to findings in CNS at the time of and following the earlier SARS and MERS outbreaks. Especially during the present COVID-19 pandemic, being aware of these features can ensure doctors evaluate COVID-19 infection when observing unexplained neurologic symptoms. There is a need for more study on this subject, especially on its long-term neurologic effects, because there is a dearth of information.

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