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New onset neurologic events in people with COVID-19 infection in three regions in China

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The authors report no disclosures relevant to this work.

Abstract

***Objective*:** To investigate new-onset neurologic impairments associated with coronavirus disease 2019 (COVID-19).

***Methods*:** A retrospective multicenter cohort study conducted between 18 January and 20 March 2020 including people with confirmed COVID-19 from 56 hospitals officially designated in three Chinese regions; data were extracted from medical records. New-onset neurologic events as assessed by neurology consultants based on manifestations, clinical examination and investigations, in which critical events included disorders of consciousness, stroke, CNS infection, seizures and status epilepticus.

***Results*:** We enrolled 917 people with average age 48.7 years and 55% were male. The frequency of new onset critical neurologic events was 3.5% (32/917) overall and 9.4% (30/319) among those with severe or critical COVID-19. These were impaired consciousness (n=25) or/and stroke (n=10). The risk of critical neurologic events was highly associated with age above 60 years and previous history of neurological conditions. Non-critical events were seen in less than 1% (7/917), including muscle cramp, unexplained headache, occipital neuralgia, tic and tremor. Brain CT in 28 people led to new findings in nine. Findings from lumbar puncture in three with suspected CNS infection, unexplained headache or severe occipital neuralgia were unremarkable.

***Conclusions*:** People with COVID-19 aged over 60 and neurologic comorbidities were at higher risk of developing critical neurologic impairment, mainly impaired consciousness and cerebrovascular accidents. Brain CT should be considered when new-onset brain injury is suspected, especially in people under sedation or showing an unexplained decline in consciousness. Evidence of direct acute insult of SARS-COV-2 to the CNS is still lacking.

Introduction

A cluster of cases of pneumonia of unknown cause was reported in Wuhan, China in December 2019. The city became the epicenter of the first outbreak of what would become known as coronavirus disease 2019 (COVID-19), caused by a novel type of coronavirus named severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The outbreak quickly evolved into a global pandemic.

People with COVID-19 present with a wide spectrum of symptoms, of which the most frequent, are fever, dry cough and shortness of breath. Many also report non-specific symptoms of fatigue, headache, and myalgia[1, 2], which are presumably due to the systemic disorder and usually resolve without specific treatment.

The presence of new onset neurologic impairment requiring investigation and intervention remains largely unknown in people with COVID-19, apart from two single-center reports and some case reports[3-12]. Studies of another human coronavirus, the SARS-CoV virus, have suggested the possibility that it can directly cause acute or sub-acute neurologic impairment[13-15].

We ascertained new-onset neurologic events during the acute phase of COVID-19. This may help clinicians optimize treatment and management of such individuals, hence improving their prognosis.

Methods

Ethics

The study was approved by the Ethics Board of West China Hospital, Sichuan University (approval 2020[100]). Due to the circumstances and the retrospective nature of the study, the need for informed consent was waived provided data was anonymised.

Participants

We conducted the study in 56 hospitals officially designated as COVID-19 treatment centers from three jurisdictions: Wuhan, Hubei province's capital (and the epicenter); Chongqing municipality, which borders Hubei province and Sichuan province, which borders Chongqing but not Hubei. E-Table 1 in the appendix data provides a list of participating hospitals and the number of people reported by each.

People admitted who met the agreed national guideline (Chinese national guideline – 6th edition) for symptomatic COVID-19 were consecutively enrolled[16]. In Sichuan and Chongqing, enrolment was between 18 January and 3 March 2020 and in Wuhan, between 18 January and 20 March 2020. People (n=304) in this cohort enrolled prior to 18 February were reviewed for seizure-related incidents and this sub-group of the cohort was previously reported elsewhere[17].

Diagnosis was based on the presence of the typical symptoms of fever, cough and /or typical features on chest CT with a positive identification of SARS-CoV-2 RNA by

real-time RT-PCR using the standardized protocol[16]. Those who remained asymptomatic and without CT chest changes were excluded.

Those included were further stratified as having a mild, moderate, severe or critical condition based on the above guideline (see Table 1 for classification criteria)[16].

Clinical assessment

A standardized clinical report form was designed to extract data on clinical features, test results and medical history (available from Dryad; eMethods, doi.org/10.5061/dryad.nk98sf7qx), and investigators completed this form using an on-line platform (https://www.wjx.cn/jq/73405304.aspx). We extracted clinical information from medical notes. In case of uncertainty, attending physicians or neurologists contacted. The outcome (discharge, death or still in hospital) was recorded (study-end) for those from Sichuan and Chongqing on 3 March and from Wuhan on 20 March.

An independent neurologist reviewed the notes of people with new-onset neurologic events. We excluded, per protocol, those who only had non-specific symptoms, such as headache, dizziness, fatigue and myalgia, presumably likely due to the systemic condition. We also excluded people if their neurologic symptom, such as impaired consciousness, could be fully accounted for by sedation during ventilation. Records of those identified as having had a critical neurologic event were then reviewed and confirmed by two other neurologists. We defined new-onset specific neurologic

events as those requiring neurologic investigations or interventions.

We further grouped specific new-onset neurologic events into critical and non-critical new neurologic events. We defined critical events as disorders of consciousness, cerebrovascular accidents, CNS infection, seizures or status epilepticus.

Statistical analysis

Age was normally distributed and reported as mean ± SD. We assessed inter-group differences in age for significance using Student's t test. Inter-group differences in the frequencies of categorical variables were assessed using chi-squared tests (or Fisher's exact test if the values were < 5). Variables for multivariable logistic regression on the development of new-onset critical neurologic impairment were selected based on the univariate analysis of the frequency of age over 60, male, non-neurologic/neurologic comorbidities in each group, where P value was < .05. We considered differences associated with 2-tailed $p < .05$ as significant. We performed statistical analyses using Stata 15 for Windows (StataCorp, TX, USA).

Standard Protocol Approvals, Registrations, and Patient Consents

This study was approved by the institutional ethics board of West China Hospital, Sichuan University (approval 2020[100]).

Data availability

We will share anonymized data by reasonable requests from any qualified investigator.

Results

Clinical-demographics

We enrolled 917 people (55% men), comprising 455 from Sichuan, 286 from Wuhan and 176 from Chongqing (Figure 1). Data were complete for all assessed variables and outcomes. The mean age was 48.7 ± 17.1 years (range, 3 months to 91 years). Nearly half (404/917, 44%) had non-neurologic comorbidities, and 28 (3%) had neurologic comorbidities. At study-end, 742 people had been discharged, 145 were still hospitalized (97 in Sichuan and 1 in Chongqing and 47 in Wuhan) and 30 had died. A total case fatality risk of 3.9% (30/772) was seen but was much higher in Wuhan (23/239, 9.6%) than in Sichuan (3/358, 0.8%) or Chongqing (4/175, 2.3%).

After excluding 24 individuals who had depressed levels of consciousness due to sedation, we identified 39 with new-onset neurologic events. Thirty-two of these were critical, and all but two of them had severe or critical COVID-19 (Tables 2 and 3). This corresponded to a prevalence of critical neurologic events of 3% across all 917 people and 9 % among the 319 with severe or critical COVID-19. The prevalence of non-critical events was 0.8%. The equivalent numbers excluding the sub-group (n=304) previously reported were as follows: a prevalence of critical events across

3.4% of the remaining 613 people, and 9.5% among the 211 with severe or critical COVID-19. The prevalence of non-critical events was 0.7%.

New-onset critical neurologic events (Table 3 and eTable 2, available from Dryad: doi.org/10.5061/dryad.nk98sf7qx)

Disorders of consciousness

Fourteen individuals (age 51-85; nine male) had disturbance of consciousness, ranging from drowsiness/stupor to coma, (Glasgow Coma Scale (GCS): 0 – 14, two people died immediately). Four used oxygen masks, and nine used non-invasive positive-pressure ventilation (NIPPV). Impaired consciousness was attributed to brain insult secondary to septic shock in five people, to cardiogenic shock in three, to hypovolemic shock in two; to hyperosmolar hyperglycemia in two and to cardiac arrest in one. One died immediately because of cardiac arrest and another died from hypovolemic shock caused by upper gastrointestinal bleeding. A further eight died between 5 hours and 4 days after deterioration of consciousness. Three who had mild alteration of consciousness (13-14 on GCS) were eventually discharged. The remaining individual with impaired consciousness had cerebral herniation secondary to a pre-existing brain tumor and was still hospitalized (GCS 3-5-6) at study-end.

Seven people (30 to 91 years old; five males) had impaired consciousness in the form of nighttime hallucinations, irritability, lack of compliance, inattention, and/or disorganized thinking; two had pre-existing dementia. Delirium occurred in two on

NIPPV and in another two on invasive mechanical ventilation (IMV). One had ischemic stroke before the delirium (case #6), and two had stroke after the delirium (#1 & #5). Six received low-dose dexmedetomidine immediately and two underwent tracheotomy due to progressive hypoxemia. At study-end, one had recovered and was discharged, one died after a possible new-onset stroke (#5), while the remaining five were still hospitalized under sedation, with scores from -2 to -4 on the Richmond Agitation and Sedation Scale (RASS).

Three females between ages 52 to 61 years without a previous history of neurologic or systemic disorders had events resembling typical syncope with no further complications. Electrocardiograms recorded afterwards were normal.

An adult was comatose following traumatic brain injury. On arrival, GCS was 10. Brain CT scan confirmed massive brain injury with a skull fracture. He had recently been in Hubei, was tested and was positive. He was treated conservatively and mild symptoms including fever and dry cough manifested two days later; he made a full recovery and was discharged with only minor neurologic sequelae.

Stroke

Ten people had cerebrovascular accidents (available from Dryad; eTable 2, doi.org/10.5061/dryad.nk98sf7qx). Half had new onset cardiac arrhythmia (n=3) or venous thromboembolism (n=3), and they all had stroke in the late course of

COVID-19. Two individuals (case #5 and #6), who had strokes early in the course of COVID-19 recovered and were discharged with only minor neurologic sequelae. Of the others, two were discharged (#8 and #10), three died (#3-5) and three still hospitalized in a critical condition by study-end. Brain CT scans of some of these people (#1-4,8,9) are shown in Fig. 1. None had investigation for possible stroke etiology such as cerebral angiography or coagulation screen.

Others

No acute symptomatic seizures or epileptic seizures and neither status epilepticus was seen in the cohort. No case of CNS infection was confirmed in the cohort.

Risk factors of critical neurologic events and outcomes

Univariate analysis identified age above 60 years and neurologic comorbidities as significantly associated with new-onset critical neurologic events, of which only age older than 60 emerged as significant in multivariate analysis (Table 4).

Non-critical neurologic events (Table 3)

Occipital neuralgia

Occipital neuralgia was noted in one male (case #11) in his 40s. After a cruise he had new-onset, self-limiting paroxysmal burning-like severe pain in his neck that radiated to his scalp. He was only tested for COVID-19 after a contact from the cruise trip tested positive; he then also tested positive. He later developed fever and a cough, and

the episodes of occipital neuralgia became more frequent and more intense. On examination, only tenderness of the greater occipital nerve area found and cranial CT and lumbar puncture were unremarkable. Neither the virus nor other pathogens were detected in the cerebrospinal fluid. The pain gradually lessened on treatment with pregabalin and he was discharged.

Others

One person with unexplained severe headache, lumbar puncture was normal and no SARS-COV-2 detected.

Neurologic investigations

Brain CT was performed in 28 people and it showed new-onset lesions in nine (Table 5). For those with stroke, detailed findings are provided in eTable 2 (available at doi.org/10.5061/dryad.nk98sf7qx) and Figure 2. Lumbar puncture was performed in one individual (case #1) with suspected CNS infection, one (#11) with occipital neuralgia, and one with unexplained headache. Pressure and routine assays were normal and PCR panel testing for meningitis/encephalitis pathogens and SARS-COV-2 were negative. No EEG recording or brain MRI were performed to decrease potential exposure risk to staff.

Discussion

In this multi-center study, we identified new-onset critical neurologic events including impaired consciousness and cerebrovascular events in fewer than 5% people with COVID-19 and this is overall compatible with a previous report[3, 5].

We report a lower rate of non-critical or overall neurologic events and this is partly explainable. Firstly, we excluded all non-specific neurologic symptoms such as headache and dizziness. Secondly, the previous report was from the epicenter in Wuhan, where higher proportions of people had severe and critical illness[3, 5]. Our cohort included a large number of people from outside Wuhan and only about a third of our sample had severe or critical disease. This may provide a more representative picture of the incidence and spectrum of neurologic manifestations of COVID-19. As we excluded asymptomatic cases, the incidence of neurologic manifestations could have been overestimated. Inclusion of people with pre-existing neurologic conditions, such as brain tumours or dementia might also have overestimated the numbers.

The major factor associated with neurologic complications was age over 60, which was also a strong risk factor for mortality[18]. When we compared people with COVID-19 infections at the same level of severity, new-onset neurologic critical events increased the risk of death by six-fold. Further studies are warranted to investigate the synergistic effect of other known risk factors such as d-dimer greater than 1 μg/L and cardiac injury in people with critical neurologic events[18-20].

We also found delirium to be present in nearly a tenth of people with critical disease, and it required prompt intervention. It is lower than the previous reports of people with COVID-19 who experienced delirium while in the ICU[4]. Several reasons could explain it: Firstly, early intervention with sedating medications in people on NIPPV or IMV seen in most; secondly, the prevalence may differ between populations being evaluated. Thirdly, as we did not use screening tools we may possibly have under diagnosed of delirium. We did not record EEGs. In some people, especially those with a history of epilepsy (they were none knowingly in the cohort) or findings suggestive of seizures, this could help differentiating delirium from non-convulsive status epilepticus or focal dyscognitive seizures. We administered dexmedetomidine to most people with delirium and to some who under ventilated, as a precaution[21-23]. The use of dexmedetomidine in people with COVID-19, however, requires further assessment as well as the true prevalence of delirium in the people of COVID-19,

A small number of people had stroke during admission, most of which occurred late in the course of COVID-19. Previous studies reported similar incidence of acute ischemic stroke or hemorrhagic stroke in COVID-19[3-5] and in people in ICUs[24]. Incidental findings on brain CT identified three people on sedatives with stroke, which are more difficult to identify clinically. These results highlight the usefulness of CT, as they led to management changes in many of those scanned. Brain CT may be particularly useful given the high prevalence of critical comorbidities such as

coagulopathy; venous thromboembolism and cardiac arrhythmia in people with critical COVID-19[25-27], which all increase the chance of stroke. It also lessens the risk of viral exposure for staff if carried out simultaneously with routine chest CT.

Our results also highlight the importance of a multidisciplinary approach in treating cerebrovascular accidents in people with COVID-19, in whom frequent re-assessments may lead to better management. Brain CT and bedside screening tools, which can detect such events, are key for this purpose, especially for people who are unconscious, have a stroke history, or are on mechanical ventilation. Larger cohorts are warranted to quantify the true prevalence and etiology of cerebrovascular accidents in COVID-19 to optimize their treatment.

We did not find evidence that neurologic impairments were directly caused by the virus. SARS-COV-2 identification was negative in the CSF of all cases tested and systemic condition explained most of them. For those with neurologic complications, the incidence of altered consciousness was not significantly increase, compared to other respiratory illness as chronic obstructive pulmonary disease or asthma[28]. Delirium and strokes are often seen in ICUs[4, 24]. Likewise, mild symptoms such as tics, tremor or muscle cramps, are more likely attributable to acute stress disorder and hypocalcaemia rather than a direct effect of the virus. They have been reports of mostly non-specific neurologic symptoms in COVID-19, including headache, dizziness and myalgia[1, 2] . There also been reports of a wide clinical spectrum of more

severe symptoms such as acute stroke, acute myelitis, pneumonia complicated with tuberculous meningitis, rhabdomyolysis, Guillain-Barré syndrome, Miller Fisher syndrome, polyneuritis cranialis and acute hemorrhagic necrotizing encephalopathy[3-12]. The prevalence of such cases and a causal relationship with the virus is still unknown.

We have not identified any individual with epilepsy but they could have been missed in view of our methodology. As before, we did also not observe seizures of any type in this cohort particularly symptomatic seizure or cases of status epilepticus[17]. This was to some extend surprising particularly as some individuals had clear risk factors for this type of complications.

To date, no evidence of direct impairments by SARS-CoV-2, such as confirmation of RNA in the CSF or neurons on autopsy or post mortem study, has been established. Another human coronavirus, SARS-COV was found in the brain of an individual with encephalopathy in a post-mortem study. Eight others showed that the virus was confined to the hypothalamus and cortex in brain autopsy without neurologic impairment reported[14, 15]. Neurologic symptoms were also reported in four people during or after, Middle East respiratory syndrome which is also caused by a human coronavirus[29]. The pathogenetic role of SARS-COV-2 in neurologic impairment is still unclear and needs more investigation.

The severe neurologic complications we have seen are most unlikely directly attributable to the virus but it is still important to acknowledge common neurologic complications so physicians can be prepared, especially when there is no access to neurology. Early detection of impaired consciousness, delirium may help treatment escalation. The use of brain CT should be encouraged to identify strokes in those at high risk. Thrombolytic and coagulants should be used cautiously in this population.

Four of our cases (#6, 11 and two who presented with cerebral herniation and traumatic brain injury) initially manifested typical neurologic symptoms but without typical symptoms of COVID-19. As a detailed travel history was taken and they had investigations including chest CT and virus testing, they were promptly channeled into COVID-19 treatment paths. Clinical staff might have put themselves at risk by continuing to work with them under the assumption that they did not have COVID-19 had they not been tested. Our experience highlights the need to consider the epidemiology of COVID-19 and to implement adequate protective measures even when people are not immediately suspected of having COVID-19.

As well as acute neurologic impairments, one should also be aware of potential long-term sequelae. Neuro-musculo-skeletal disorders following SARS have been reported[13, 30]. For those surviving an acute respiratory distress syndrome, with delirium, mechanical ventilation, and prolonged exposure to sedatives or sepsis, a high prevalence of cognitive impairment, which decreases the quality of life, could be

expected[31].

Our study has several limitations. Firstly, we enrolled people retrospectively; this is unlikely to have introduced bias as the government covers all costs so most would attend hospital. Secondly, we did not include many children as only some the participating hospital could admit children. The numbers of children in our cohort could be underestimated if compared to that in the total population with the infection[32]. Children, however, seem to be spared from the most critical symptoms and in a recent study of critically ill children with COVID-19 no neurologic complication was reported[33]. Thirdly, no brain MRI/CTA/MRA or EEG was performed because of the risk of viral exposure to staff. Lastly, 145 people were still in hospital at study-end, so we were not able to ascertain final outcomes; this may have led to an underestimation of the mortality rate but also of new neurologic events. We do not believe, however, that this would make a major difference.

Despite these limitations, new-onset critical neurologic events were identified in fewer than 5% of people during acute COVID-19 infections and this was highly associated with a poor outcome. Evidence for an acute or direct brain insult by the COVID-19 virus is still lacking. Neurologists should work closely with other specialties via a multidisciplinary approach to protect the nervous system from the short-term and possible long-term impairments. More work, particularly in large cohorts, is warranted to elucidate the full impact of COVID-19 in the CNS

particularly in the medium and long term.

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Appendix 1 Authors

Name	Location	Contribution
Weixi Xiong, MD	Department of Neurology, West China Hospital of Sichuan University, Chengdu, China	Conceptualisation and design of the study; analysis and critical interpretation of data and the results; drafted the manuscript
Jie Mu, MD	Department of Neurology, West China Hospital of Sichuan University, Chengdu, China	Data acquisition and interpretation of the results
Jian Guo, MD	Department of Neurology, West China Hospital of Sichuan University, Chengdu, China	Data acquisition and interpretation of the results
Lu Lu, MSc	Department of Neurology, West China Hospital of Sichuan University, Chengdu, China	Major role in data entry and interpretation of the results
Dan Liu, MD	Department of Pulmonary & Critical Care Medicine, West China Hospital of Sichuan University, Chengdu, China	Data acquisition
Jianfei Luo, MD	Department of Gastrointestinal Surgery, Renmin hospital of Wuhan University	Data acquisition
Nian Li, MD	Department of Medical Affairs, West China Hospital of Sichuan University, Chengdu, China	Data acquisition
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Li He, MD, PhD	Department of Neurology, West China Hospital of Sichuan University, Chengdu, China	Interpretation of the results
Josemir W. Sander, MD, PhD FRCP	UCL Queen Square Institute of Neurology, Queen Square, London WC1N 3BG; United Kingdom	Conceptualisation and design of the study; critical interpretation of data and results; manuscript revisions for critical intellectual content

Dong Zhou, MD, PhD	Department of Neurology, West China Hospital of Sichuan University, Chengdu, China	Conceptualisation and design of the study; critical interpretation of data and results; manuscript revisions for critical intellectual content; supervision of the study and guarantor

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Figure Legends

Figure 1. Flowchart of the study enrollment.

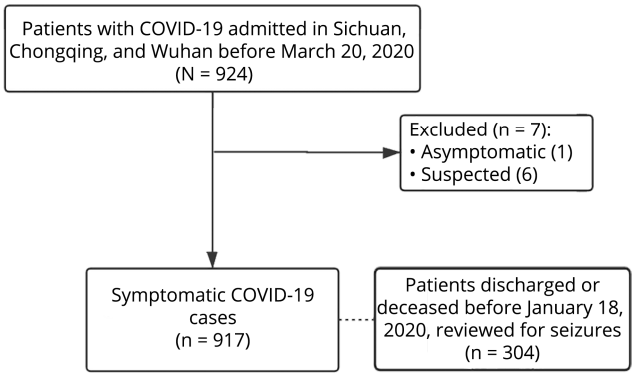

Figure 2. CT brain scans of COVID-19 cases with cerebrovascular accidents.

(a-b) Images of case #1 showing multifocal ischemic stroke in both hemispheres. (c) Image of case #1 taken 10 days after the images in panels a-b, showing the onset of intraventricular bleeding. (d) Image of case #8 showing ischemic stroke of right temporal occipital junction and para hippocampal gyrus. (e-g) Images of case #2 showing multifocal cerebral watershed infarction. (h) Unremarkable image of case #4, taken two days before fatal stroke. (i-k) Scans of case #3 showing bilateral multifocal stroke, hemorrhagic lesions, and large hemispheric infarction on the left side. (l-m) Scans of case #9 showing previous lesions of stroke. (n-p) Images of the new-onset subdural hematoma (arrow) in the follow-up scans 4, 11 and 16 days after images in panel l-m.

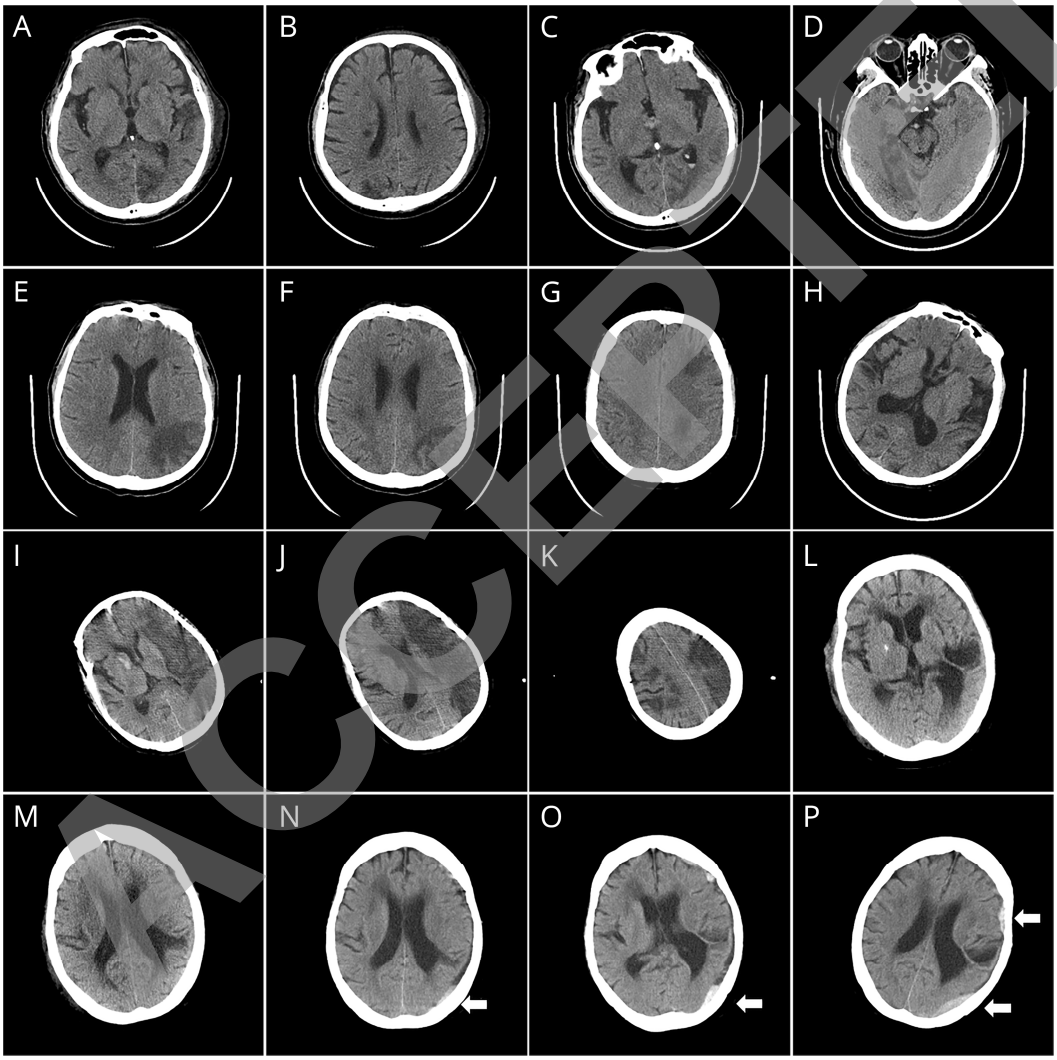

Table 1. Diagnosis and Treatment Protocol for COVID-19 (Trial Version 6) [16]

Classification	Criteria
Mild	Light clinical symptoms and no sign of pneumonia on lung imaging
Moderate	Fever, respiratory tract symptoms and other symptoms; Imaging suggests pneumonia
Severe	Any of the following: (1) respiratory distress, respiration rate ≥ 30 times / min; (2) oxygen saturation ≤ 93% at rest; (3) PaO2 / FiO2 ≤ 300 mmHg (1mmHg = 0.133 kPa)
Critical	As in severe + any of the following: (1) respiratory failure occurs and mechanical ventilation is required; (2) shock; (3) complicated with other organ failure and need of intensive care unit (ICU) monitoring and treatment

Table 2 Demographic features of 917 people with COVID-19

Features	Critical neurological events n=32	No critical neurological events n=885	p
Age (years, mean ± SD)	67.6 ± 15.4	48.1 ± 16.8	5.5×10^{-10}
Sex (M/F)	19/13	487/398	0.63
Clinical classification (n)			
Mild	0	65	
Moderate	2	531	1.34×10^{-22}
Severe	5	233	
Critical	25	56	
History of non-neurological conditions (n)	17	387	0.29
History of neurological conditions (n)	5	23	2.6×10^{-5}
New-onset non-neurological complications (n)	24	418	2.0×10^{-3}

Table 3 Thirty-nine people with new-onset neurologic events

Type of events	Neurological events		N	Timing of the events (days from the initial symptoms)	case #
Critical N=32	Conscious disturbance N=25	Delirium*	7	Mean 12 days (range, 4 to 23 days)	
		Drowsiness/stupor/coma	14	Mean 12 days (range, 1 to 24 days)	
		Syncope	3	Day 2, 22 and 27	
		TBI	1	Day1	
	stroke* N=10	Early onset	2	Day 1 and 6	6,7
		Late onset	8	Mean 31 days (range, 18 to 48 days)	1-5,8-10
Non-critical N=7	Muscle cramp		2	Day 8 and 16	
	Unexplained headache		2	Day 14 and 19	
	Occipital neuralgia		1	Day 1	11
	Functional? Tic/tremor		2	Day 8 and 41	

TBI: traumatic brain injury;

* Three had delirium and stroke (case #2, 6 and 7).

Table 4 Multivariate logistic regression analysis of new onset critical neurological events

Independent variables	Odds ratio	P value	95% CI
Age above 60 years	6.75	0.000	3.01 to 15.14
Neurological comorbidities	2.93	0.051	0.99 to 8.67

Table 5 Findings of brain CT in the 28 people

CT indications	N	New lesions
Headache	2 unexplained and 8 non-specific	none
Under sedation	6	3 new onset stroke (case #1-3)
stroke	4 (case #6-8,10)	3 new onset stroke
Previous history of stroke	4	1(+)(case #9)
Occipital neuralgia	1 (case #11)	none
Declined consciousness due to brain tumor	1	Brain tumor
TBI	1	frontal epidural hematoma, bilateral frontal lobe contusion and laceration, subarachnoid haemorrhage, multiple fractures of the skull base, frontal and parietal bone
Syncope	1	none

TBI: traumatic brain injury

New onset neurologic events in people with COVID-19 infection in three regions in China

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