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Systematic review of EEG findings in 617 patients diagnosed with COVID-19

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ABSTRACT

Objective: We performed a systematic review of the literature to synthesize the data on EEG findings in COVID-19. Frontal EEG patterns are reported to be a characteristic finding in COVID-19 encephalopathy. Although several reports of EEG abnormalities are available, there is lack of clarity about typical findings.

Methods: Research databases were queried with the terms “COVID” OR “coronavirus” OR “SARS” AND “EEG”. Available data was analyzed from 617 patients with EEG findings reported in 84 studies.

Results: The median age was 61.3 years (IQR 45–69, 33.3 % female). Common EEG indications were altered mental status (61.7 %), seizure-like events (31.2 %), and cardiac arrest (3.5 %). Abnormal EEG findings (n = 543, 88.0 %) were sub-classified into three groups: (1) Background abnormalities: diffuse slowing (n = 423, 68.6 %), focal slowing (n = 105, 17.0 %), and absent posterior dominant rhythm (n = 63, 10.2 %). (2) Periodic and rhythmic EEG patterns: generalized periodic discharges (n = 35, 5.7 %), lateralized/multifocal periodic discharges (n = 24, 3.9 %), generalized rhythmic activity (n = 32, 5.2 %). (3) Epileptiform changes: focal (n = 35, 5.7 %), generalized (n = 27, 4.4 %), seizures/status epilepticus (n = 34, 5.5 %). Frontal EEG patterns comprised of approximately a third of all findings. In studies that utilized continuous EEG, 96.8 % (n = 243) of the 251 patients were reported to have abnormalities compared to 85.0 % (n = 311) patients who did not undergo continuous EEG monitoring (χ² = 22.8, p =< 0.001).

Significance: EEG abnormalities are common in COVID-19 related encephalopathy and correlates with disease severity, preexisting neurological conditions including epilepsy and prolonged EEG monitoring. Frontal findings are frequent and have been proposed as a biomarker for COVID-19 encephalopathy.

1. Introduction

The COVID-19 pandemic continues to test the resilience of health systems and the limits of our understanding of viral infections of the nervous system. There has been interest in the EEG findings in COVID-19 related viral encephalitis/encephalopathy.

Neurological complications have been reported in 34.6 % patients with severe COVID-19 and include stroke, headache, and seizures. [1] Over the past few months, a plethora of studies have reported on the EEG profile of patients with COVID-19. The EEG reports in COVID-19 patients range from single case reports [2,3] to larger studies of up to 111 EEGs [4]. Findings from these studies are varied and included mostly normal EEGs [5] to diffuse slowing in all [6–8].

Individual studies stress a specific population demographic or peculiar aspect of the EEG, but together provide a mosaic of EEG findings in varied groups from children to elderly, and asymptomatic patients to those with severe encephalopathy and status epilepticus. Some studies have noted a preponderance of frontal findings, putatively linked to the mode of entry of the virus into the brain. [6,9–12] In summary, there is no common EEG pattern that has evolved in spite of numerous publications in the short period of time since COVID-19 emerged.

Here, we perform a systematic study of the EEG findings in patients with COVID-19 to synthesize the available data and to elucidate common patterns.

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2. Methods

We searched Pubmed, Google Scholar, medRxiv/bioRxiv preprint servers for reports of EEG in patients with COVID-19. The following terms were used in the search: “COVID” OR “coronavirus” OR “SARS” AND “EEG”. Records identified by screening included PubMed (n = 96), Google scholar (n = 29,200), MedRxiv (n = 72), BioRxiv (n = 51), and Research Square (n = 4). Full text of 108 studies were accessed after excluding studies not relevant to the current project and excluding duplicates. A total of 84 manuscripts were selected for analysis after review by two board-certified epilepsy specialists (AA and ZH). We were unable to use part of the data from one study which reported that nine patients had seizures or encephalopathy and three other studies due to lack of specific details. [5,13–15] Findings from two studies with quantitative EEG analysis were included [14,16]. The process of selection of manuscripts for analysis is detailed as per PRISMA recommendations in Fig. 1 [17].

EEG findings of 617 patients reported across 84 selected retrospective studies were reviewed. A systematic analysis was performed for the following data: age, sex, EEG type (e.g. routine, continuous), indication for EEG, co-morbid neurological conditions, and imaging abnormalities. Detailed data were not available for some parameters in all studies and the analysis details reflect availability-for example, data regarding sex were available in 75 studies and data regarding indications for EEG were available in 423 patients. The EEG type was considered as routine EEG, unless specified as continuous EEG or rapid response 8-channel EEG (Ceribell), which was reported in in three studies. Details of available data are noted in Table 1.

EEG abnormalities were classified into the following three broad categories to highlight similar findings and further sub-analyzed as follows: (1) background abnormalities, (2) Periodic and rhythmic EEG patterns, and (3) other epileptiform changes and seizures/status epilepticus. EEG background abnormalities were sub-categorized as follows: diffuse slowing, focal slowing, slow posterior dominant rhythm, absent posterior dominant rhythm, background attenuation/suppression, and discontinuous EEG/burst suppression, lateralized asymmetry,

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Fig. 1. PRISMA diagram detailing the selection process of studies in the systematic review. * Reasons for exclusion were absence of description of EEG findings (n = 14) and review articles (n = 7). ** Reasons for exclusion are review articles (n = 7), absence of EEG reports (n = 8), articles related to other corona viruses (n = 6) and articles detailing EEG recording techniques and safety during COVID-19 pandemic (n = 3).
and decreased reactivity. Periodic and rhythmic activity [18] were sub-categorized by location into generalized, lateralized/multifocal, and unspecified localization.

Frontal lobe findings were re-analyzed separately since several studies reported EEG findings involving the frontal lobe as being a characteristic finding of COVID-19 encephalopathy. [6,9,12] EEGs performed on children with COVID-19 infection were also assessed separate from adult EEG for unique findings.

Wherever possible, the currently accepted EEG nomenclature was substituted in place of older terminology reported. [18] Some of the older terminology was retained when it was unclear what the most appropriate current terminology would be. For example, with SIRPIDS, it was unclear if the activity was ictal, rhythmic or periodic discharges specifically. EEG under sedation and theta slowing were counted as it was unclear if the activity was ictal, rhythmic or periodic discharges specifically. EEG under sedation and theta slowing were counted as.

EEG findings reported in COVID-19
A. Background Abnormalities
B. Periodic and Rhythmic Patterns
C. Epileptiform Abnormalities

Superscripts indicate the number of studies the data is available from: 1: 73 studies; 2: 75 studies, 3: 423 patients.

Table 1

Demographic features of patients.

| Demographics | Total number (n) | 617 |
|---------------|-----------------|-----|
| Median age (IQR) | 61.3 years (45–69) |
| Gender M:F | 288:144 |
| Children (age<18y) | 14 |
| Routine EEG (n, %) | 60, 71.4 % |
| Continuous EEG (n, %) | 24, 28.6 % |
| Altering mental status (n, %) | 261, 61.7 % |
| Seizure-like events (n, %) | 132, 31.2 % |
| EEG Indications | Cardiac arrest (n, %) | 15, 3.5 % |
| Speech issues (n, %) | 7, 1.7 % |
| Unspecified (n, %) | 8, 1.9 % |

EEG was abnormal in 543 (88.0 %) patients. Nonspecific/ unspecified changes were noted in 31 (5.0 %) patients.

(1) Background abnormalities: The most common background abnormality noted was diffuse slowing (n = 423, 68.6 %). Other background abnormalities included focal slowing (n = 105, 17 %), slow posterior dominant rhythm (n = 14, 2.3 %), absent posterior dominant rhythm (n = 63, 10.2 %), background attenuation/ suppression (n = 8, 1.3 %), and discontinuous EEG/burst suppression (n = 13, 2.1 %), asymmetry (n = 13, 2.1 %), and decreased reactivity (n = 20, 3.2 %).

(2) Periodic and rhythmic patterns: Periodic discharges were classified as generalized (n = 35, 5.7 %), lateralized/multifocal (n = 24, 3.9 %), generalized periodic discharges with triphasic morphology (n = 18, 2.9 %), SIRPIDs (n = 7, 1.1 %), and unspecified (n = 4, 0.6 %). Rhythmic activity was sub-categorized into generalized rhythmic delta activity (GRDA) (n = 32, 5.2 %), lateralized/multifocal rhythmic discharges (n = 16, 2.6 %) and unspecified (n = 2, 0.3 %).

(3) Other epileptiform changes and seizures: Epileptiform discharges were categorized as focal (n = 35, 5.7 %), multifocal (n = 13, 2.1 %), unspecified localization (n = 5, 0.8 %) and generalized (n = 27, 4.4 %).
of the 251 patients were reported to have abnormalities in studies that (n 659) abnormalities were more common in the frontal region (Fig. 3). Of interest, half of all reported focal slowing (52 out of 105) and status epilepticus (10 out of 22) involved the frontal regions. Other EEG features suggesting diffuse encephalopathy included generalized rhythmic delta activity and generalized periodic discharges with triphasic morphology. Lateralized periodic and rhythmic abnormalities were also seen suggesting a co-existent focal dysfunction in some patients. Epileptiform discharges were common (13.0 %) indicating underlying cortical irritability predisposing to seizures. In fact, 5.5 % had seizures or status epilepticus.

Several studies noted characteristic abnormalities in the frontal region hypothesizing that this correlates with the purported entry of COVID-19 into the brain. [6,9–12] Early clinical manifestations of COVID-19 like anosmia and ageusia are thought to be due to viral entry in the nasal and oral mucosa facilitated by ACE-2 receptors [30]. Subsequent spread to the orbitofrontal region [30,31] via different nerves leads to preferential involvement of the olfactory bulb and orbito-frontal/frontal regions and can explain the preponderance of frontal EEG findings. This theory is also corroborated by frontal hypometabolism seen in PET scans in these patients [32]. This viral spread hypothesis and ano-mato-clinico-electrophysiological correlation is analogous to the temporal/frontal EEG findings in Herpes simplex encephalitis corresponding with its pattern of anatomic involvement [30]. Frontal epileptiform discharges have been proposed as biomarkers for COVID-19. [2,3,6,33,34] Examples of frontal monomorphic biphasic slow waves and periodic discharges are shown in Fig. 4 [35]. Approximately half of all reported status epilepticus (10 out of 22) arose in the frontal regions, compared to 18 % (4 out of 22) involving the occipital lobe. In addition to the frontal lobe, the piriform cortex, limbic structures, thalamus, hypothalamus, brainstem and autonomic structures can also be involved by COVID-19. [36] The more extensive EEG finding was not available in most studies. However, a large study of 111 patients reported that EEG studies were performed after a median of 10.8 days after hospitalization. [4] No significant correlation was noted between the timing of EEG and epileptiform abnormalities in relation to the duration of illness. It was noted that EEGs performed early during hospitalization were for cardiac arrest and seizure while the indication for later EEGs was unexplained encephalopathy [4].

In our review, 82.5 % (n = 66) of epileptiform discharges and 67.6 % (n = 23) of seizures/status epilepticus were reported in the 24 studies that utilized continuous EEG monitoring. In addition, 96.8 % (n = 243) of the 251 patients were reported to have abnormalities in studies that utilized continuous EEG, compared to 85.0 % (n = 311) patients who did not undergo continuous EEG monitoring (χ² = 22.8, p = 0.00001).

### 4. Discussion

We present a large systematic review of EEG abnormalities in patients with COVID-19. In our review of 617 patients from 84 reports, we found that EEG abnormalities are common and encompass a wide variety of findings such as background abnormalities, periodic and rhythmic activity and other epileptiform abnormalities. The most common reason for obtaining EEG was altered mentation, and the most common EEG finding was diffuse slowing. Of interest, frontal lobe findings were common and included focal slowing, periodic discharges and rhythmic delta activity. Half of all status epilepticus and focal slowing originated in the frontal lobes.

Diffuse background slowing was the most common EEG finding reported in two-thirds (68.6 %) of patients indicating that a diffuse non-specific encephalopathy was the most common brain abnormality in this condition. Other EEG features suggesting diffuse encephalopathy included generalized rhythmic delta activity and generalized periodic discharges with triphasic morphology. Lateralized periodic and rhythmic abnormalities were also seen suggesting a co-existent focal dysfunction in some patients. Epileptiform discharges were common (13.0 %) indicating underlying cortical irritability predisposing to seizures. In fact, 5.5 % had seizures or status epilepticus.

Several studies noted characteristic abnormalities in the frontal region hypothesizing that this correlates with the purported entry of COVID-19 into the brain. [6,9–12] Early clinical manifestations of COVID-19 like anosmia and ageusia are thought to be due to viral entry in the nasal and oral mucosa facilitated by ACE-2 receptors [30]. Subsequent spread to the orbitofrontal region [30,31] via different nerves leads to preferential involvement of the olfactory bulb and orbito-frontal/frontal regions and can explain the preponderance of frontal EEG findings. This theory is also corroborated by frontal hypometabolism seen in PET scans in these patients [32]. This viral spread hypothesis and anatomic-clinico-electrophysiological correlation is analogous to the temporal/frontal EEG findings in Herpes simplex encephalitis corresponding with its pattern of anatomic involvement [30].

Frontal epileptiform discharges have been proposed as biomarkers for COVID-19. [2,3,6,33,34] Examples of frontal monomorphic biphasic slow waves and periodic discharges are shown in Fig. 4 [35]. Approximately half of all reported status epilepticus (10 out of 22) arose in the frontal regions, compared to 18 % (4 out of 22) involving the occipital lobe. In addition to the frontal lobe, the piriform cortex, limbic structures, thalamus, hypothalamus, brainstem and autonomic structures can also be involved by COVID-19. [36] The more extensive EEG finding seen in other patients could be due to greater viral spread or other factors like pulmonary involvement/hypoxia, cardiac arrest, other metabolic changes, sedation, systemic inflammatory response syndrome, hypercoagulability, vasculitis, and strokes. In support of systemic causes, one report found the severity of EEG abnormalities to correlate with oxygen saturation at admission, but not with neuroimaging [37]. EEG changes could also be due to pre-existing neurological diseases like epilepsy [4,9].

In our review, approximately one-third of the patients with EEGs had abnormal neuroimaging. The higher incidence of abnormal neuroimaging in our review could be due to the older age group, larger proportion of pre-existing neurological conditions and the possibility of sicker patients being triaged for neuroimaging. Several studies have shown a higher sensitivity for EEG compared to brain CT or MRI for COVID-19 related encephalopathy. Background abnormalities, with or without epileptiform features, were frequently seen in patients with neurological symptoms when neuroimaging was unremarkable. [8,37,38] In one study, 59 % of patients with neurological symptoms had an abnormal MRI, while EEG was abnormal in 83 % [13]. In another study, 13 patients with abnormal EEGs suggesting encephalopathy had normal neuroimaging [39].

In general, the extent of EEG abnormalities correlated with the clinical status of patients and pre-existing neurological diseases. [6,8,9]
We included only COVID-19 positive patients in our analysis; however, when those who tested negative were reported, it was found that a higher proportion of positive patients had epileptiform abnormalities (40.9%) compared to those who were negative (16.7%) [6]. EEG abnormalities may be correlated with the severity of COVID-19 infection. [12,38] A history of CNS pathology was reported in 43% of patients with epileptiform findings compared to 9% in those without epileptiform findings [9]. Interestingly, in another study the absence of the posterior dominant rhythm was more common in patients who were on sedation on the day of EEG or the prior to the test compared those who were not on it [9]. A significantly higher proportion of EEG abnormalities - diffuse slowing in 100%, generalized periodic discharges in 31.8%, sharp waves in 13.6% and seizures in 9.1% were reported in one study, possibly due to greater use of continuous EEG monitoring [8].

In our systematic review, continuous EEG studies reported significantly more abnormalities compared to routine EEGs suggesting higher sensitivity of prolonged EEG monitoring.

In our review, 56.8% of the follow up EEG studies reported improvement. In a study involving 26 patients, two had iso-electric EEG suggesting brain death and three of the five patients who had periodic discharges on EEG died a few days later. [34] In quantitative EEG of patients with COVID-19, greater delta-theta band spectral power, higher

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**EEG Abnormalities**

| EEG finding                              | Total | Frontal | Proportion of frontal vs. total EEG abnormalities |
|------------------------------------------|-------|---------|-------------------------------------------------|
| Focal slowing                            | 105   | 52      | 49.5%                                           |
| Lateralized/ multifocal Periodic discharges | 24    | 10      | 41.7%                                           |
| Lateralized/ multifocal Rhythmic discharges | 16    | 6       | 37.5%                                           |
| Focal/ multifocal Epileptiform discharges | 48    | 14      | 29.2%                                           |
| Seizures                                 | 12    | 2       | 16.7%                                           |
| Status epilepticus                       | 22    | 10      | 45.5%                                           |

**Fig. 3.** Frontal EEG findings compared to all brain regions.

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temporal variance, diversity in frequency band and spatial extent at baseline were associated with a better outcome [14].

Previous coronavirus epidemics are also known to cause neurological changes including seizures. The earliest available report of neurological complication of SARS–COV infection was a patient with respiratory failure and seizures, although EEG was not performed. [40] Not many reports of EEG findings are available - a normal EEG in a patient with generalized tonic clinic seizures due to SARS-CoV-1 virus [41] and diffuse slowing in a patient with MERS [42] have been reported. EEG reports are considerably more in COVID-19 compared to these previous coronavirus infections, maybe due to the larger extent of the current pandemic, and more frequent use of EEG including continuous EEG monitoring currently than in the past. [8,41,42]

This study has the shortcomings of other systematic reviews including lack of access to original data such as EEG waveforms. We understand that many normal EEGs will not be reported, and EEGs were possibly performed disproportionately on patients with neurological symptoms. Many patients with suspicion of clinical seizure-like events were placed on anti-seizure medications prior to EEG, which may have reduced the likelihood of detecting epileptiform changes. The diversity of findings reported could partly reflect different terminologies and reading styles used world-wide- for example generalized periodic discharges may be worded as “generalized discharges” or as “periodic discharges” in different papers. The term “triphasic waves” seemed to be used by authors, especially outside the US where the ACNS terminology may not be as widely adopted. However, we used the term “generalized periodic discharges with triphasic morphology” to be consistent with the currently accepted terminology. Even though background abnormalities like slow posterior rhythm, discontinuous background and decreased reactivity were detailed in some studies, several authors did not list them separately.

Several studies discussed labs and MRIs but failed to specify the
imaging results of the patients who had EEG. Only some studies specifically reported on EEGs without sedation. [43] While we attempted to quantify specific abnormalities such as frontal changes, many reports including large studies [4] did not have this level of detail, which could have led to under-estimating their relevance. It is difficult to find the total number of patients with EEG abnormalities because several studies do not specifically state the number of subjects with overlap of the reported abnormalities. Despite these limitations, several authors commented that greater use of EEG would help characterize more patients with COVID-19 related encephalopathy. [2,38,44] We would like to add that MedRxiv, BioRxiv and Research Square publish preprints references are preliminary reports of work that have not been peer reviewed. However, they are still important sources of information that speeds up dissemination of scientific information, which is especially important in a rapidly evolving situation like COVID-19.

EEG remains a crucial tool in the management of patients with neurological manifestations of COVID-19 especially with encephalopathy, seizures, and status epilepticus. Abnormalities when present include slowing, periodic discharges, epileptiform discharges, seizures and status epilepticus, indicating the presence of a localized dysfunction, nonspecific encephalopathy and cortical irritability in this condition. The extent of EEG abnormalities correlates with the diagnosis of COVID-19, duration of monitoring, pre-existing neurological conditions like epilepsy and severity of the disease. EEG abnormalities affecting the frontal lobe seems to be common in COVID-19 encephalopathy and has been proposed as a potential biomarker if recorded consistently. We encourage clinical alertness to identify such findings. It has been speculated that frontal EEG findings result from direct brain involvement in COVID-19, while more diffuse changes may result from either systemic involvement or more diffuse viral infection of the brain.

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Declaration of Competing Interest

The authors report no declaration of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.seizure.2020.10.014.

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