

Post-Covid Alpha Coma Pattern in A Huntington's Disease Patient: A Case Report and Electroencephalogram (Eeg) Literature Review

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Abstract:

Neurological complications of COVID-19 include encephalopathy and, less commonly, seizures. Electroencephalogram (EEG) findings are heterogeneous, with the alpha coma pattern rarely reported and typically observed during active infection. We describe a 49-year-old man with Huntington's Disease who developed an alpha coma EEG pattern two weeks after recovering from COVID-19-related encephalopathy. Initial EEGs showed diffuse slowing, while a later EEG revealed bifrontal alpha activity with partial reactivity. Brain MRI demonstrated cortical-subcortical hypoperfusion without contrast enhancement, and cerebrospinal fluid and immunological tests were unremarkable. The patient gradually improved, and the alpha pattern resolved. A literature review of 13 studies reporting EEG findings in COVID-19 patients revealed that generalized slowing is the most common abnormality, with alpha coma rarely observed and only during active infection. Our case uniquely documents the alpha coma pattern post-recovery, suggesting that chronic hypoxic injury may underlie this phenomenon. These findings highlight the potential for prolonged neurological sequelae after COVID-19 resolution.

Key words: alpha coma; covid-19; electroencephalography (eeg); huntington's disease; post-hypoxic encephalopathy

Introduction

The Coronavirus Disease 2019 (COVID-19) pandemic has been associated with a wide range of neurologic manifestations, attributed to: (i) indirect cytokine-mediated responses, (ii) direct central nervous system (CNS) invasion, and (iii) neural injury via angiotensin-converting enzyme 2 (ACE2) receptors [1]. Neurological symptoms have been reported in up to 36.4% of hospitalized COVID-19 patients, with encephalopathy occurring in approximately 31.8% and new-onset seizures in about 0.8% of cases [2].

Electroencephalogram (EEG) findings in COVID-19-associated encephalopathy are diverse, most commonly showing diffuse slowing or frontal intermittent rhythmic delta activity (FIRDA) [3]. The alpha coma pattern is exceedingly rare, and all documented cases have occurred during the acute phase of infection [4,5]. To our knowledge, this is the

first report of an alpha coma EEG pattern emerging after clinical recovery from COVID-19, in a patient with underlying Huntington's Disease (HD). We also provide a focused literature review of EEG findings in COVID-19 patients, with the aim of exploring whether post-infectious EEG abnormalities reflect direct viral neurotropism or indirect systemic mechanisms such as hypoxia or inflammation.

Methods

We conducted a structured literature review using the Medline (PubMed) database to identify studies published between January 2020 and December 2022. The search strategy included the following keywords and Boolean operators: ("COVID-19" OR "SARS-CoV-2") AND ("EEG" OR "electroencephalography" OR "encephalopathy" OR

“seizures”). Only English-language articles involving human subjects were included.

Studies were eligible if they reported EEG findings in patients with confirmed COVID-19 infection. We focused specifically on articles describing encephalopathy, altered mental status, seizures, or distinct EEG patterns (e.g., diffuse slowing, FIRDA, or alpha coma). Case reports, case series, and observational studies were included; editorials, reviews, and experimental studies were excluded.

Titles and abstracts were screened for relevance, followed by full-text review of selected articles. A total of 14 articles met the inclusion criteria and were analyzed qualitatively. Data extracted included patient demographics, clinical features, EEG findings, and timing of EEG in relation to COVID-19 onset.

Results and Case Description

Case Presentation

We describe the case of a 49-year-old man with a 15-year history of Huntington's Disease (HD), manifesting mood depression, cognitive

impairment, and gait instability, treated with haloperidol 1 mg/day. In November 2020, he presented with his first generalized tonic-clonic seizure. At admission, he tested positive for SARS-CoV-2. Chest CT revealed typical COVID-related ground-glass opacities. Brain CT showed HD-related subcortical atrophy, and EEG demonstrated generalized delta slowing consistent with cortico-subcortical dysfunction. Levetiracetam 500 mg BID was initiated, [6] but the patient's condition deteriorated rapidly, requiring high-concentration oxygen via Venturi mask. Delirium developed and was managed with IM aripiprazole and IV diazepam, replacing haloperidol. After one month, despite COVID-19 recovery (confirmed by swab negativity), he remained in a comatose state (GCS 8: O3, V1, M4), prompting transfer to our neurology unit. Follow-up CT revealed new cortical and subcortical hypoperfusion. EEG again showed frontal-predominant delta slowing with overlying 14–16 Hz monomorphic activity (likely related to sedation). Despite partial sedation withdrawal, coma persisted. The patient then developed a bacterial superinfection. Two weeks post-negative swab, a second EEG revealed a bifrontal alpha coma pattern (10–11 Hz, low-voltage, transient), with limited reactivity to eye opening (Figure 1a-b).

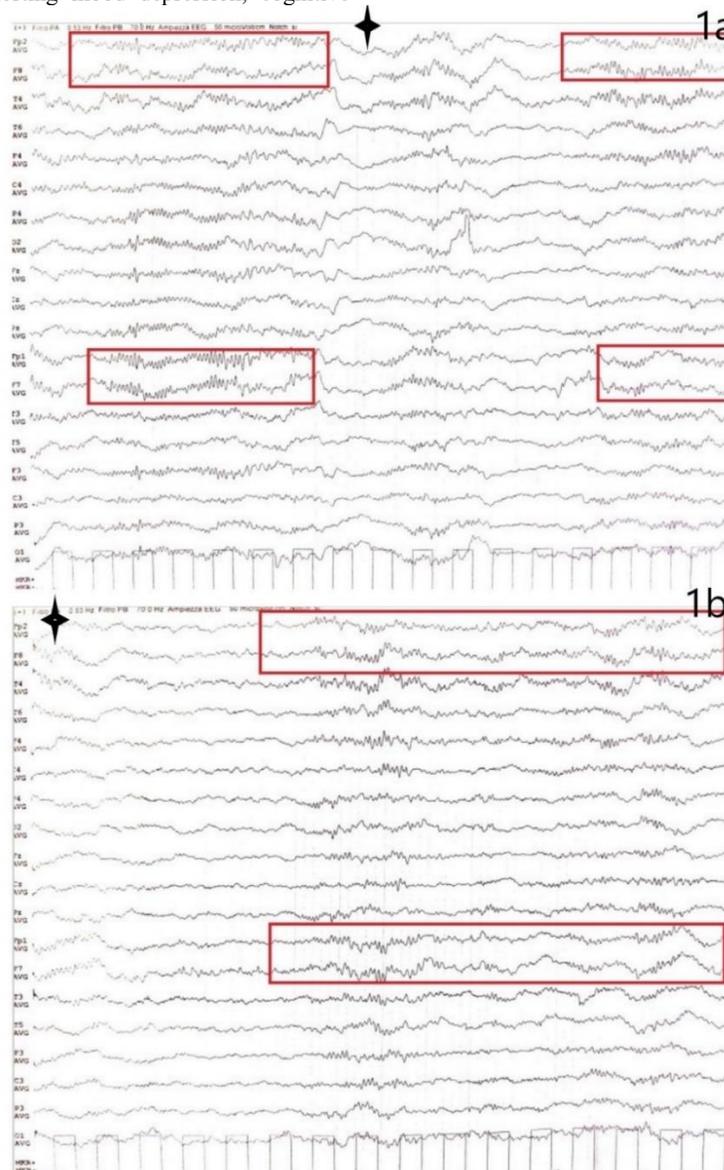


Figure 1 A-B: EEGs show low-voltage, frontally predominant alpha activity at 10–11 Hz, without an anterior–posterior gradient. Red rectangles and black arrows highlight bifrontal alpha bursts. Black stars mark eye opening, where the alpha rhythm transiently disappears, indicating partial reactivity consistent with post-hypoxic alpha coma.

Brain MRI confirmed chronic cortico-subcortical hypoperfusion, with pre-existing caudate atrophy but no contrast enhancement. We considered two potential mechanisms: a post-infectious autoimmune encephalitis or prolonged hypoxic injury. CSF analysis (normal proteins, glucose, cell count, negative antibodies, and PCR panel) did not support autoimmune etiology. Nevertheless, empiric treatment with IV immunoglobulins and broad-spectrum antibiotics was initiated; levetiracetam was increased to 750 mg BID. MRI findings were more suggestive of chronic hypoxia than inflammation. Absence of contrast enhancement and presence of new hypoperfusion changes further supported this. Over time, the patient's clinical condition improved. EEG showed disappearance of the alpha coma pattern, replaced by reactive theta and delta activity. Subsequent EEGs revealed reappearance of sleep patterns including spindles and K-complexes. He was discharged one month later with improved consciousness (GCS 12: O4, V2, M6). At present, he has a Unified Huntington Disease Rating Scale score of 56, with moderate dementia,

mood instability, and the inability to walk unaided. Levetiracetam is ongoing with reported benefit.

Literature Review Results

We identified 13 relevant studies, the majority of which described EEGs performed during active infection. Only one study reported EEG follow-up after infection. [4] In most cases, EEG was requested to assess for encephalopathy. Generalized slowing was the most common finding, followed by focal slowing, triphasic waves, burst suppression, and epileptiform discharges. [5] However, no consistent EEG pattern emerged as predictive of outcome. New-onset seizures were uncommon and mainly generalized; focal seizures were rare and typically linked to underlying cerebrovascular lesions. EEG improvement after infection was generally reported, but post-infection patterns remain understudied. (Table 1) [9-20].

Author/year	Patients	Timeline	Findings
Saez-Landete/2022[4]	11	15 acute; 14 follow-ups	Acute: 6.6% symmetric posterior alpha, no reactivity Follow-up: 42.8% symmetric posterior alpha; 35% no reactivity
Pellinen/2020[9]	111	During infection	61% encephalopathy 9% seizure exacerbation 21% paroxysmal activity (unclear cause) 57% generalized slowing, 30% epileptiform
Hwang/2022[10]	192	During infection	88.5% generalized slowing 15.6% focal slowing 3.6% burst suppression 3.1% attenuation 3.1% normal EEG
Gogia/2021[11]	19	During infection	Survivors (13): 36% generalized slowing; 16% normal EEG Non-survivors (6): 66% generalized slowing; 17% seizures
Corazza/2020[12]	28	During infection	35.7% generalized slowing 10.7% slow posterior activity 3.6% burst suppression 7.1% triphasic waves 25% diffuse attenuation.
Galanopoulou/2020[13]	22	During infection	40.9% epileptiform discharges 88.9% frontal sharp waves 63.6% seizure-like events
Besnard/2020[14]	42	During infection	28.6% normal EEG 21.4% slight slowing rhythm 19% unspecific anomalies 9.5% focal/diffuse epileptic pattern 21.4% encephalopathic patten
Pastor/2020[15]	20	During infection	Posterior dominant rhythm in theta band Sharp waves in 4/20 patients.
Tantillo/2022[16]	93	During infection	97% generalized slowing 31% generalized attenuation 17% generalized periodic discharges 15% generalized sharp waves 43% epileptiform abnormalities
Canham/2020[17]	10	During infection	Generalized slowing consistent with encephalopathy Asymmetry and triphasic waves associated with cerebrovascular changes
Chen/2020[18]	5	During infection	Diffuse slowing and generalized rhythmic delta activity 2/5 epileptiform discharges reaching 2–3 Hz
Niguet/2021[19]	33	During infection	85% slow activity 42% unreactive activity 21% low-voltage activity 61% rhythmic or periodic delta waves
Petrescu/2020[20]	36	During infection	42.5% EEG alterations 32.5% generalized multifocal or rhythmic periodic discharges

Koutroumanidis/2020 ³	13	Non-recovery after sedation suspension	5 cases of alpha coma pattern
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Table 1: Review of literature

Discussion

This case is the first to document a post-infectious alpha coma EEG pattern emerging after clinical and virological recovery from COVID-19 in a patient with underlying Huntington's Disease (HD). While previous studies have reported alpha coma patterns predominantly during the acute phase of infection, often implicating viral neurotropism or immune-mediated encephalopathy as key mechanisms, [3] our findings challenge this framework by revealing a delayed manifestation likely driven by secondary hypoxic injury.

The timing of the alpha coma pattern—appearing two weeks after negative SARS-CoV-2 testing—strongly suggests that ongoing viral invasion or acute neuroinflammation are unlikely causes. Notably, prior reports have associated alpha coma with bilateral MRI abnormalities involving the basal ganglia and brainstem, areas integral to the reticular activating system, which is critical for maintaining consciousness. [3] In contrast, our patient's neuroimaging demonstrated diffuse cortico-subcortical hypoperfusion without basal ganglia inflammation or contrast enhancement, consistent with chronic hypoxic injury rather than immune-mediated damage. The absence of inflammatory markers in the CSF further supports this interpretation.

Alpha coma, characterized by unreactive, frontally predominant alpha-frequency rhythms (8–13 Hz), has historically been linked to poor prognosis, particularly following cardiac arrest. [7–8] However, emerging evidence indicates that alpha coma after respiratory hypoxia or prolonged hypoxemia can have a more favorable prognosis, especially if some EEG reactivity is preserved. Our patient's EEG evolution—transitioning from an alpha coma pattern to the re-emergence of normal sleep architecture elements such as spindles and K-complexes—correlates with his gradual clinical improvement and partial recovery of consciousness. This EEG trajectory aligns with the notion that post-hypoxic alpha coma may be reversible under certain conditions.

Critically, this case underscores the complexity of interpreting EEG patterns in COVID-19 survivors, particularly those with pre-existing neurodegenerative disorders like HD, who may be more vulnerable to hypoxic insults. It highlights the importance of differentiating between active infectious encephalopathy and secondary injury mechanisms when assessing late neurological deterioration. Such differentiation has direct clinical implications for management strategies, including the decision to pursue immunotherapy versus supportive care targeting hypoxic brain injury.

While we cannot categorically exclude a subclinical immune-mediated process, the convergence of clinical timeline, neuroimaging, and CSF findings collectively favors chronic hypoxia as the primary driver. This case thus broadens the spectrum of COVID-19-related neurological sequelae, revealing that alpha coma patterns can arise as delayed manifestations rather than exclusively during acute infection.

Future studies should prioritize longitudinal EEG monitoring and multimodal neuroimaging in post-COVID-19 patients to better characterize the timing, mechanisms, and prognostic significance of delayed encephalopathic patterns. Additionally, investigation into the

interplay between neurodegenerative diseases and COVID-19-related hypoxic injury is warranted to optimize patient outcomes.

Conclusions

This case expands current understanding of COVID-19-related EEG abnormalities by documenting the emergence of an alpha coma pattern after infection resolution, diverging from prior reports that emphasize acute-phase autoimmune mechanisms. Our findings suggest that prolonged hypoxia during severe respiratory illness can induce delayed alpha coma EEG changes, especially in vulnerable patients with underlying neurodegenerative conditions. We advocate for continued EEG surveillance in post-COVID-19 patients with persistent altered consciousness, as early recognition of post-hypoxic complications may inform prognosis and guide tailored therapeutic interventions. Recognizing this delayed pattern is essential to avoid misdiagnosis and unnecessary immunomodulatory treatments, ultimately improving patient management in the evolving landscape of COVID-19 neurological care.

Ethic statement:

Ethical approval was not required for this case report, as determined by the Ethics Committee of the University of Naples Federico II. The report is based on a retrospective review of anonymized clinical data and did not involve any experimental intervention. Written informed consent for publication, including use of clinical information and video material, was obtained from the patient and his legal guardian (wife).

The authors confirm that they have read and adhered to the journal's guidelines on ethical publication and affirm that this work complies with those standards.

Disclosures:

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Conflicts of Interest

The authors declare no conflicts of interest relevant to this work.

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