Myoclonus in Patients With Coronavirus Disease 2019: A Multicenter Case Series

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Objectives: To describe the risk factors for and outcomes after myoclonus in a cohort of patients with coronavirus disease 2019.

Design: Multicenter case series.

Setting: Three tertiary care hospitals in Massachusetts, Georgia, and Virginia.

Patients: Eight patients with clinical myoclonus in the setting of coronavirus disease 2019.

Interventions & Measurements and Main Results: Outcomes in patients with myoclonus were variable, with one patient who died during the study period and five who were successfully extubated cognitively intact and without focal neurologic deficits. In five cases, the myoclonus completely resolved within 2 days of onset, while in three cases, it persisted for 10 days or longer. Seven patients experienced significant metabolic derangements, hypoxemia, or exposure to sedating medications that may have contributed to the development of myoclonus. One patient presented with encephalopathy and developed prolonged myoclonus in the absence of clear systemic provoking factors.

Conclusions: Our findings suggest that myoclonus may be observed in severe acute respiratory syndrome coronavirus 2 infected patients, even in the absence of hypoxia. This association warrants further evaluation in larger cohorts to determine whether the presence of myoclonus may aid in the assessment of disease severity, neurologic involvement, or prognostication. (*Crit Care Med* 2020; XX:00–00)

Key Words: coronavirus disease 2019; myoclonus; neurocritical care; neuroinfectious diseases

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a novel coronavirus that has caused a global pandemic of coronavirus disease 2019 (COVID-19) (1). SARS-CoV-2 has been identified in the cerebrospinal fluid (CSF) of patients with clinical meningoencephalitis in rare cases (2–6), and both SARS-CoV-2 and related coronaviruses have been isolated in neural tissue, including the brainstem, cerebrum, and thalamus (7–9).

Myoclonus is an involuntary movement common among critically ill patients. It is characterized by sudden, brief, and sometimes repetitive muscle contractions variably involving the face, extremities, and trunk and can be seen in association with specific neurologic disorders or can result secondarily from a host of toxic or metabolic triggers, including both mild and severe hypoxic brain injury and sedating medications (10). The significance of myoclonus in neuroprognostication is highly variable and depends on the underlying etiology and both clinical and neurophysiologic features (11).

Myoclonus has been observed in patients infected with other neuroinvasive RNA viruses (12). However, myoclonus has only rarely been reported in patients with COVID-19 (13). Here, we describe eight cases of COVID-19 complicated by myoclonus.

MATERIALS AND METHODS

This study was either approved or exempted by the institutional review boards of Boston Medical Center, Emory University Hospital, and Inova Fairfax Medical Campus.

RESULTS

Patient Characteristics

All patients were male, ranging in age from 28 to 73 (**Table 1**). Common comorbidities included diabetes mellitus in five of eight patients, hypertension (five patients), obesity (two patients), and chronic kidney disease (two patients). Obstructive sleep apnea was present in one patient.

TABLE 1. Characteristics and Outcomes of Patients With Myoclonus and Coronavirus Disease 2019

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8
Age, yr, sex	47, male	28, male	73, male	64, male	66, male	72, male	62, male	71, male
Method of testing for COVID-19	Nasopharyngeal swab	Nasopharyngeal swab negative × 2, nasopharyngeal aspirate positive	Nasopharyngeal swab	Nasopharyngeal swab	Nasopharyngeal swab	Nasopharyngeal swab	Nasopharyngeal swab negative × 2, bronchoalveolar aspirate positive	Nasopharyngeal swab
Duration of symptoms prior to presentation (d)	4	3	9	9	2	7	7	7
Presenting symptoms	Headache, odynophagia, dry cough, tactile fevers	Fevers, dyspnea, odynophagia, chest pain, nausea, vomiting, and abdominal pain	Cough, generalized weakness	Cough, dyspnea, fever	Cough, dyspnea, fever	Nausea, vomiting, productive cough	Cough, dyspnea, fevers	Confusion, gait disturbance
Type of myoclonus	Generalized, stimulus-induced	Generalized, stimulus-induced	Torso and upper extremities, stimulus-induced	Upper extremities, stimulus-induced	Upper extremities and face, spontaneous	Generalized, multifocal, stimulus-induced	Generalized, stimulus-induced	Generalized, action-induced; lingual
Duration of myoclonus	3–5 min, lasted 2 d	Minutes, lasted 1 d	Seconds to minutes, lasted 2 d	Seconds to minutes, lasted 1 d	Minutes, lasted 2 d	Seconds to minutes, persistent for > 2 wk	Minutes, lasting > 10 d	14 d
Timing of myoclonus	HD 4, 1 d post-intubation	HD 5, 1 hr post-intubation	HD 1, immediately post-intubation	HD 2, 1 d post-intubation	HD 10, immediately post-extubation	HD 9, immediately post-extubation	HD 5, 2 d post-intubation	HD 0
Resolved?	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes
Medications resolving myoclonus	Ketamine, dexmedetomidine	Lorazepam, midazolam, dexmedetomidine	Levetiracetam	Dexmedetomidine	Dexmedetomidine	Valproic acid, levetiracetam, lorazepam, dexmedetomidine	Valproic acid, primidone, clonazepam, lorazepam	Levetiracetam, valproic acid
Provoking medications	Propofol, hydromorphone	Propofol, hydromorphone	Propofol	Fentanyl	Fentanyl	Fentanyl	Propofol	None
Brain imaging	Head CT, no acute findings	Head CT, no acute findings	Head CT, no acute findings	MRI brain with equivocal right temporal T2 hyperintensity	Head CT, no acute findings	Head CT, no acute findings	No imaging	MRI brain with diffuse pachymeningeal enhancement
Cerebrospinal fluid findings	Not available	Not available	Cell count 1 cell/μL Glucose 120 mg/dL Protein 83 mg/dL	Cell count 2 cells/μL Glucose 111 mg/dL Protein 37 mg/dL	Not available	Not available	Not available	Cell count 1 cell/μL Glucose 54 mg/dL Protein 44 mg/dL
Electroencephalogram data	Not available	Not available	Not available	Not available	Not available	Bifrontal sharp waves, resolved after intubation, although to represent muscle artifact	Moderate generalized dysfunction, occasional low-amplitude sharp transients, myogenic activity	Mild diffuse background slowing

(*Continued*)

TABLE 1. (*Continued*). Characteristics and Outcomes of Patients With Myoclonus and Coronavirus Disease 2019

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8
Glucose range (RR 70–100 mg/dL)	131–316	59–218	90–416	112–142	186–216	184–286	97–290	54–97
Sodium range (RR 135–145 mmol/L)	130–138	134–138	131–147	140–145	149–151	145–154	136–160	143–145
Magnesium range (RR 1.6–2.6 mg/dL)	1.6–2.8	2.1–2.2	2.3	2.4–2.8	2.4–2.6	1.9–2.0	1.9–2.7	1.8–2.0
Calcium range (RR 8–10.5 mg/dL)	7.1–10.2	7.5–7.8	8.4–10.8	7.4–7.8	8.8–9	7.8–8.5	7.7–9.1	8.4–9.6
Bicarbonate range (RR 19–28 mmol/L)	17–27	21–26	24–28	28–31	25–26	22–25	15–26	22–27
Maximum blood urea nitrogen (RR 7–25 mg/dL)	41	12	44	39	170	48	76	19
Maximum ammonia (RR 10–80 µg/dL)	Not available	Not available	40	Not available	Not available	25	Not available	Not available
Administered dialysis?	Yes	No	No	Yes	Yes	No	No	No
Thyroid-stimulating hormone (RR 0.45–4.5 mU/L)	Not available	Not available	Not available	2.09	Not available	0.97	Not available	0.12
Hypoxia? (oxygen saturation nadir)	Yes, 86%	Yes, 86% (transiently 30% during intubation)	Yes, 80%	No	No	No	Yes, 72%	No
Maximum temperature, °F	104.4	102.9	102.4	100.9	101.3	99.3 (minimum 92.5)	103.2	99.0
Maximum D-dimer (RR < 243 ng/mL)	1,201	1,132	10,779	19,957	> 60,000	3,580	2.47	390
Maximum C-reactive protein (RR 0–5 ng/mL)	530	311.2	307.9	439.6	392.4	333	30.3	14.9
Medical comorbidities	Diabetes mellitus, obstructive sleep apnea, morbid obesity, hypertension	Diabetes mellitus, hyperlipidemia	Diabetes mellitus, hypertension, stage 3 chronic kidney disease	Hypertension	Diabetes mellitus, hypertension, obesity	Diabetes mellitus	Hypertension	Hypertension, stage II chronic kidney disease
Treatments administered for COVID-19	Azithromycin, hydroxychloroquine, anakinra	Azithromycin, hydroxychloroquine, anakinra	Azithromycin, hydroxychloroquine	Hydroxychloroquine	Azithromycin	Hydroxychloroquine	Azithromycin, hydroxychloroquine	None

(*Continued*)

TABLE 1. (*Continued*). Characteristics and Outcomes of Patients With Myoclonus and Coronavirus Disease 2019

Variables	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8
Outcome at the time of manuscript submission	Deceased	Discharged home; cognitively intact at discharge without further myoclonus	Discharged home; cognitively intact at discharge without further myoclonus	Extubated; alert and cognitively intact without further myoclonus	Extubated; alert and cognitively intact without further myoclonus	Reintubated 2 d after extubation, remains intubated at the time of manuscript submission; encephalopathic during sedation vacations with continued, improved myoclonus	Discharged home on valproic acid, clonazepam, and primidone with continued, improved myoclonus; cognitively intact	Discharged to rehabilitation; improved cognition at discharge without further myoclonus

COVID-19 = coronavirus disease 2019, HD = hospital day, RR = reference range.

Ranges for glucose, sodium, magnesium, calcium, bicarbonate, blood urea nitrogen, ammonia, and temperature are recorded during the 24 hr before the onset of myoclonus. Hypoxia and oxygen saturation nadir are recorded for the 72 hr prior to the onset of myoclonus. Thyroid-stimulating hormone level and maximum D-dimer and C-reactive protein levels are recorded for the hospitalization as a whole.

Features of COVID-19

Presenting symptoms of COVID-19 included headache, odynophagia, fever, dyspnea, chest pain, nausea, vomiting, abdominal pain, and both dry and productive cough. One patient did not have respiratory or gastrointestinal symptoms but developed confusion and gait disturbance prior to presentation (case 8). In all cases, COVID-19 was identified based on characteristic clinical features and positive polymerase chain reaction testing for SARS-CoV-2 from nasopharyngeal or respiratory specimens. In two cases (numbers 2 and 7), two successive nasopharyngeal swab samples tested negative for SARS-CoV-2, and the diagnosis was made only with a third sample (nasopharyngeal aspirate in case 2 and bronchoalveolar lavage in case 7). Patients presented with between 2 and 9 days of respiratory and systemic symptoms. In six cases, patients had an elevated temperature during the 24 hours prior to the onset of myoclonus (100.9–104.4°F). In all cases, patients had elevated C-reactive protein (14.9–530 mg/L) and D-dimer (2.47 to > 60,000 ng/mL) levels during their hospitalization. One patient also had significant spontaneous hypothermia during the 24 hours prior to myoclonus onset (92.5°F). Seven patients had respiratory failure requiring intubation during their hospitalization, while one patient was not critically ill and did not require intensive care. Treatments administered for COVID-19 included hydroxychloroquine (six patients), azithromycin (five patients), and anakinra (two patients).

Myoclonus Risk Factors and Onset

In four of eight cases, the onset of myoclonus was within 24 hours of intubation, while in two cases, it occurred immediately post-extubation, and in one case, it occurred 2 days after intubation. In four cases, significant hypoxia was documented during the 72 hours before the onset of myoclonus (oxygen saturation range 30–86% by pulse oximeter). Metabolic derangements were noted during the 24 hours before the onset of myoclonus in all cases, including hypo- and hyperglycemia (glucose range 54–416 mg/dL), hypo- and hypernatremia (sodium range 130–160 mmol/L), and uremia (blood urea nitrogen range 12–170 mg/dL). Three of eight cases required renal replacement therapy for renal failure. In seven cases, neuroactive medications with the potential to provoke myoclonus were used for sedation, including propofol, hydromorphone, and fentanyl.

Myoclonus Semiology and Duration

In seven cases, myoclonus was stimulus- or action-induced, with just one case of spontaneous myoclonus. In five cases, myoclonus was generalized (Supplemental Video 1, Supplemental Digital Content 1, http://links.lww.com/CCM/F731; and Supplemental Video 2, Supplemental Digital Content 2, http://links.lww.com/CCM/F732 [legend, Supplemental Digital Content 3, http://links.lww.com/CCM/F733]), while in one case, it was limited to the upper extremities and in the remaining two, the torso or face were also involved. The duration of myoclonus ranged from 1 to 14 days. Two patients (cases 6 and 7) continued to have myoclonus at the time of manuscript submission, including one patient who was discharged home.

Ancillary Neurologic Testing

Seven of eight patients had brain imaging, including five patients with CT and two with MRI (cases 4 and 8). No acute findings were seen on any of the head CTs, which were all done without contrast. The MRI in case 4 showed a temporal T2 hyperintensity of unclear significance without associated enhancement with gadolinium administration. The MRI in case 8 showed diffuse pachymeningeal enhancement. Twenty-four hour electroencephalography was obtained in three cases during myoclonus (cases 6, 7, and 8) and revealed significant myogenic activity and generalized cerebral dysfunction in all cases. CSF studies were obtained in three cases (3, 4, and 8) and revealed normal nucleated cell counts in all cases, with mildly elevated protein of 83 in case 3. SARS-CoV-2 testing was not available from CSF.

Myoclonus Treatments and Outcomes

Medications used with improvement of myoclonus included valproic acid, primidone, clonazepam, lorazepam,

levetiracetam, dexmedetomidine, midazolam, and ketamine. Outcomes were variable, with one patient who was deceased at the time of manuscript submission, one who remained intubated, three who were discharged home cognitively intact, one who was discharged to rehabilitation with cognitive improvement, and two who were extubated, with improved mental status and without focal neurologic deficits. In five cases, the myoclonus completely resolved within 2 days of onset, while in three cases (cases 6, 7, and 8), it persisted for 10 days or longer. The presence of hypoxia did not clearly correlate with either the duration of myoclonus or the functional outcome: one patient who did not experience hypoxia remains intubated with continued myoclonus, while three patients who did experience hypoxia were discharged home cognitively intact. One patient continued to have myoclonus at discharge, although it was significantly improved on valproic acid, clonazepam, and primidone. A second patient who remains intubated continues to have myoclonus during sedation breaks.

DISCUSSION

Initial reports suggest that 5% of patients with COVID-19 will become critically ill, requiring intensive care (14). Limited literature suggests that critically ill COVID-19 patients are more likely to have neurologic complications, including cerebrovascular events, impaired consciousness, and skeletal muscle injury (15, 16). It is unclear whether these complications represent true CNS invasion by the virus or whether they result more generally from critical illness. More recent reports have described encephalitis or meningoencephalitis in patients with COVID-19 with or without respiratory symptoms (2–6). In these cases, SARS-CoV-2 has been identified in the CSF. Notably, early studies have found that the rates of both mortality and critical illness are higher in male patients with COVID-19 than in their female counterparts, a finding reflected in the demographics of the all-male cohort described here (17).

Myoclonus in critically ill patients can result from a range of etiologies, including medication toxicity, cerebral hypoxia, traumatic brain or spinal cord injury, metabolic derangements, and inflammatory and infectious encephalitides (10). The myoclonus observed in patients with COVID-19 may reflect multiple contributing factors. Myoclonus has been reported in the setting of other neuroinvasive RNA viruses (9, 10), and nervous system involvement by COVID-19 may contribute. In our series, metabolic derangements occurred in all cases, consistent with preliminary reports suggesting that hyponatremia and early renal failure may be common features of COVID-19 (18). Seven of the patients included in our series had some exposure to sedating medications with the potential to precipitate myoclonus. Finally, four of the patients in our series experienced periods of significant hypoxia, although the presence of hypoxia did not correlate with either functional outcome or duration of myoclonus. Notably, one patient who was not critically ill and did not require intubation developed prolonged myoclonus and encephalopathy in the setting of pachymeningeal enhancement on MRI.

Myoclonus occurs in 20% of cardiac arrest patients within 72 hours of cardiopulmonary resuscitation (19), but less is known about the frequency of myoclonus in patients who experience hypoxia without cardiac arrest. Preliminary reports suggest that paucisymptomatic hypoxia may be common in patients with COVID-19, even prior to hospital presentation (20). Patients with COVID-19 may therefore experience silent and prolonged hypoxia prior to admission that could contribute to the occurrence of myoclonus in this particular patient population.

Although ancillary neurologic testing, including neuroimaging, electroencephalography, and CSF studies, may be helpful in characterizing the etiologies of myoclonus, availability of these studies during a pandemic may be limited. In our series, CSF studies and electroencephalography were available for just three patients, while MRI brain was available for only two. Given limited resources, critical care providers must rely on clinical findings to determine whether to pursue available ancillary testing. In our experience, for patients with movements consistent with a diagnosis of myoclonus and without concern for seizure (suggested by a nonfocal neurologic examination and normal consciousness), electroencephalography may be safely deferred. Conversely, in situations where a patient has altered consciousness and movements that cannot be clinically distinguished between myoclonus and seizure, a trial of an anti-seizure medication may be considered, as this could potentially treat both conditions. For patients with altered consciousness and myoclonic movements who do not respond to anti-seizure medication trial, we strongly recommend pursing electroencephalography.

The significance of myoclonus in neuroprognostication for critically ill patients is highly variable, as is treatment responsiveness. Both are determined largely by etiology and neuroanatomic localization; cortical myoclonus caused by toxic or metabolic encephalopathy may be partially or completely reversible with medication cessation or correction of metabolic derangements, while myoclonus associated with cerebral infection or hypoxic injury is more often treatment refractory (10). Although myoclonic status epilepticus following hypoxic brain injury was previously thought to be associated with a high mortality rate, more recent studies suggest that a minority of patients with myoclonus after hypoxic brain injury have favorable outcomes, more often in those without epileptiform activity and with a preserved background rhythm (21), although a broad range of outcomes can also be seen depending on the electrographic pattern observed (22). A less common post-hypoxic action myoclonus, Lance-Adams syndrome, may occur after a cardiopulmonary arrest and is associated with a favorable neurologic prognosis (23).

In our cohort, outcomes after myoclonus were highly variable, with one deceased patient and five who were successfully extubated cognitively intact without focal neurologic deficits. In five cases, the myoclonus completely resolved

within 2 days of onset, while in three cases, it persisted for 10 days or longer.

In cases of COVID-19, myoclonus may be multifactorial and represent a combination of hypoxia, medication toxicity, and direct or para-infectious complications of the virus itself. Our findings suggest that myoclonus may be observed in SARS-CoV-2 infected patients regardless of respiratory failure. This association warrants further evaluation in larger cohort studies to determine whether the presence of myoclonus may aid in the assessment of disease severity, neurologic involvement, or prognostication.

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