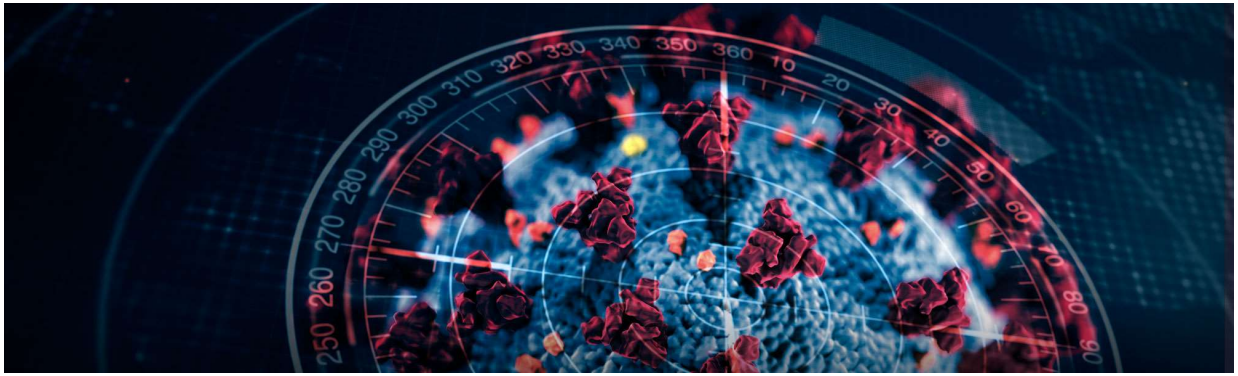
Neurologic Complications of COVID-19

Brain Summit 2020 Virtual Symposium

October 31st, 2020



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Disclosures

- I have no financial disclosures.
- I have served as an unpaid site Co-investigator for COVID-19 clinical trials sponsored by the NIH, Gilead, Regeneron, and Genentech.
- I will be discussing off-label investigational treatments for COVID-19.

Overview: COVID-19 Neurologic Disease

- Epidemiology
- Pathogenesis
- Clinical Spectrum of Neurologic Disease
- Treatment Considerations
- Impact on Non-COVID Neurology

Caveats About Available Data

- Mostly from small sample size, retrospective case series from chart reviews or registries
- Biases due to reporting and capture, lack of systematic and prospective data
- Limited mainly to hospitalized patients
- Most large cohorts lack in-person neuro consultation or advanced diagnostics
- Data starting to emerge on long-term symptoms and sequelae

Early Wuhan Experience

- Retrospective data from 3 Wuhan hosp. Jan-Feb 2020
- 214 hospitalized pts.
  - Mean age 52.7, 41% male
  - Severe disease (41%)
  - **36.4% had neuro sxs overall**
- Pts with severe disease were older, more comorbid dz and more likely to show neurologic manifestations (CVA, altered consciousness or muscle injury)
- **Limitations**: Sample size, chart review likely missed milder sxs

Table 1. Clinical Characteristics of Patients With COVID-19

Characteristic	No. (%) Total (N = 214)	Severe (n = 88)	Nonsevere (n = 126)	*P* value[a]
Age, mean (SD), y	52.7 (15.5)	58.2 (15.0)	48.9 (14.7)	
Age, y				
<50	90 (42.1)	24 (27.3)	66 (52.4)	<.001
≥50	124 (57.9)	64 (72.7)	60 (47.6)	
Sex				
Female	127 (59.3)	44 (50.0)	83 (65.9)	.02
Male	87 (40.7)	44 (50.0)	43 (34.1)	
Comorbidities				
Any	83 (38.8)	42 (47.7)	41 (32.5)	.03
Hypertension	51 (23.8)	32 (36.4)	19 (15.1)	<.001
Diabetes	30 (14.0)	15 (17.0)	15 (11.9)	.29
Cardiac or cerebrovascular disease	15 (7.0)	7 (8.0)	8 (6.3)	.65
Malignancy	13 (6.1)	5 (5.7)	8 (6.3)	.84
Chronic kidney disease	6 (2.8)	2 (2.3)	4 (3.2)	.69
Nervous system symptoms				
Any	78 (36.4)	40 (45.5)	38 (30.2)	.02
CNS	53 (24.8)	27 (30.7)	26 (20.6)	.09
Dizziness	36 (16.8)	17 (19.3)	19 (15.1)	.42
Headache	28 (13.1)	15 (17.0)	13 (10.3)	.15
Impaired consciousness	16 (7.5)	13 (14.8)	3 (2.4)	<.001
Acute cerebrovascular disease	6 (2.8)	5 (5.7)	1 (0.8)	.03
Ataxia	1 (0.5)	1 (1.1)	0	NA
Seizure	1 (0.5)	1 (1.1)	0	NA
PNS	19 (8.9)	7 (8.0)	12 (9.5)	.69
Impairment				
Taste	12 (5.6)	3 (3.4)	9 (7.1)	.24
Smell	11 (5.1)	3 (3.4)	8 (6.3)	.34
Vision	3 (1.4)	2 (2.3)	1 (0.8)	.37
Nerve pain	5 (2.3)	4 (4.5)	1 (0.8)	.07
Skeletal muscle injury	23 (10.7)	17 (19.3)	6 (4.8)	<.001

Mao L, et al. JAMA Neurol. 2020; 77(6):683-690.

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Spanish Experience: ALBACOVID Registry

- Retrospective data from 2 Spanish hosp. Mar 2020
- 841 hospitalized pts.
  - Mean age 66.4, 56% male
  - Comorbid dz common
  - Severe disease (39%)
  - **57.4% had neuro sxs overall**
- Non-specific sxs and anosmia/ dysgeusia seen early in less severe dz
- Altered consciousness (19.6%) seen in older pts, more severe dz
- Neuro complications cause of death in 4.1% of all deceased
- **Limitations**: Retrospective, did not account for other causes of sxs, 1/3 on full dose anti-coagulation

Table 3 Neurologic manifestations of coronavirus disease 2019

	Total, n = 841	Nonsevere, n = 512 (60.9%)	Severe, n = 329 (39.1%)	OR	95% CI	*p* Value
Any	483 (57.4)	270 (52.7)	213 (64.7)	1.65	1.2–2.2	0.001
Nonspecific symptoms						
Myalgias	145 (17.2)	101 (19.7)	44 (13.4)	0.63	0.4–0.9	0.02
Headache	119 (14.1)	81 (15.8)	38 (11.6)	0.70	0.5–1.1	0.08
Dizziness	51 (6.1)	34 (6.6)	17 (5.2)	0.77	0.4–1.4	0.38
Syncope	5 (0.6)	5 (1)	0	NA		0.07
Symptoms related to cranial nerves						
Anosmia	41 (4.9)	32 (6.3)	9 (2.7)	0.42	0.2–0.9	0.02
Dysgeusia	52 (6.2)	39 (7.6)	13 (4)	0.49	0.3–0.9	0.04
Disorders of consciousness						
Any	165 (19.6)	37 (7.2)	128 (38.9)	8.18	5.5–12.2	<0.001
Depressed level of consciousness						
Total	117 (13.9)	21 (4.1)	96 (29.1)	9.63	5.9–15.8	<0.001
Somnolence	73 (62.4)	17 (81)	56 (58)	NA		0.15
Stupor	34 (29.1)	3 (14.3)	31 (32.3)			
Coma	10 (8.5)	1 (4.8)	9 (9.4)			
Bradypsychia, disorientation	85 (10.1)	17 (3.3)	68 (20.7)	7.59	4.4–13.2	<0.001
Acute confusional syndrome	69 (8.2)	20 (3.9)	49 (14.9)	4.31	2.5–7.4	<0.001
Peripheral nervous system manifestations						
Dysautonomia	21 (2.5)	15 (2.9)	6 (1.8)	0.61	0.2–1.6	0.31
AIDP	1	1	0	NA		NA
Muscle damage						
HyperCKemia	73 (9.2)	28 (5.9)	45 (14.2)	2.64	1.6–4.3	<0.001
Rhabdomyolysis	9 (1.1)	2 (0.4)	7 (2.2)	5.34	1.1–25.9	0.02
Myopathy	26 (3.1)	4 (0.8)	22 (6.7)	9.13	3.1–26.7	<0.001
Cerebrovascular manifestations						
Ischemic stroke	11 (1.3)	7 (1.4)	4 (1.2)	0.88	0.3–3.1	0.85
Intracranial hemorrhage	3 (0.4)	0	3 (0.9)	NA		0.03

Romero-Sanchez RM, et al. Neurology 2020; 95:e1060-70.

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US Experience: Northwestern Cohort

- Retrospective data from 10 Chicago hosp. Mar-Apr 2020
- 509 hospitalized pts.
  - Mean age 58.5, 55% male
  - Severe disease (on MV; 26.3%)
  - **Neuro sxs: 63% at admit, 82% overall**
- Myalgias, HA, encephalopathy, dizziness, dysgeusia and anosmia most common (accounted for 91% of neuro sxs overall)
- Encephalopathy independently associated with 30d mortality (**OR 2.92**)
- **Limitations**: Retrospective, less than 10% had in-person neuro consultation or advanced dx

Table 2. Timing of neurologic manifestations by Covid-19 severity.

	At time of Covid-19 symptom onset				Any time during Covid-19			
	Overall	Non-severe Covid-19 disease	Severe Covid-19 disease	*P*	Overall	Non-severe Covid-19 disease	Severe Covid-19 disease	*P*
N	509	375	134		509	375	134	
Any neurologic manifestations *n*(%)	215 (42.2)	160 (42.7)	55 (41.0)	0.822	419 (82.3)	295 (78.7)	124 (92.5)	0.001
Number of neurologic manifestations *n*(%)				0.595				<0.001
0	294 (57.8)	215 (57.3)	79 (59.0)		77 (15.1)	70 (18.7)	7 (5.2)	
1	145 (28.5)	106 (28.3)	39 (29.1)		146 (28.7)	116 (30.9)	30 (22.4)	
2	53 (10.4)	39 (10.4)	14 (10.4)		133 (26.1)	93 (24.8)	40 (29.9)	
3	13 (2.6)	12 (3.2)	1 (0.7)		101 (19.8)	71 (18.9)	30 (22.4)	
4 or more	4 (0.8)	3 (0.8)	1 (0.7)		52 (10.2)	25 (6.7)	27 (20.1)	
Myalgias *n* (%)	134 (26.4)	99 (26.6)	35 (26.1)	1	228 (44.8)	172 (45.9)	56 (41.8)	0.476
Headache *n* (%)	84 (16.5)	64 (17.1)	20 (14.9)	0.662	192 (37.7)	149 (39.7)	43 (32.1)	0.143
Encephalopathy *n* (%)	9 (1.8)	8 (2.1)	1 (0.7)	0.507	162 (31.8)	49 (13.1)	113 (84.3)	<0.001
Dizziness/vertigo *n* (%)	26 (5.1)	24 (6.4)	2 (1.5)	0.047	151 (29.7)	111 (29.6)	40 (29.9)	1
Dysgeusia *n* (%)	24 (4.7)	17 (4.5)	7 (5.2)	0.931	81 (15.9)	64 (17.1)	17 (12.7)	0.293
Anosmia *n* (%)	18 (3.5)	14 (3.7)	4 (3.0)	0.897	58 (11.4)	47 (12.5)	11 (8.2)	0.233
Syncope *n* (%)	6 (1.2)	4 (1.1)	2 (1.5)	1	22 (4.3)	15 (4.0)	7 (5.2)	0.726
Rhabdomyolysis *n* (%)	2 (0.4)	1 (0.3)	1 (0.7)	1	18 (3.5)	1 (0.3)	17 (12.7)	<0.001
Orthostatic hypotension *n* (%)	—	—	—	—	16 (3.1)	10 (2.7)	6 (4.5)	0.458
Ischemic stroke *n* (%)	—	—	—	—	7 (1.4)	2 (0.5)	5 (3.7)	0.022
Movement disorder *n* (%)	1 (0.2)	1 (0.3)	0 (0.0)	1	4 (0.8)	2 (0.5)	2 (1.5)	0.610
Seizure *n* (%)	2 (0.4)	2 (0.5)	0 (0.0)	0.966	4 (0.8)	4 (1.1)	0 (0.0)	0.528
Focal motor deficits *n* (%)	—	—	—	—	3 (0.6)	2 (0.5)	1 (0.7)	1
Ataxia *n* (%)	—	—	—	—	2 (0.4)	1 (0.3)	1 (0.7)	1
Polyneuropathy *n* (%)	—	—	—	—	2 (0.4)	0 (0.0)	2 (1.5)	0.117
Encephalitis *n* (%)	—	—	—	—	1 (0.2)	1 (0.3)	0 (0.0)	1
Focal sensory deficits *n* (%)	1 (0.2)	0 (0.0)	1 (0.7)	0.591	1 (0.2)	0 (0.0)	1 (0.7)	0.591
Hemorrhagic stroke *n* (%)	—	—	—	—	1 (0.2)	0 (0.0)	1 (0.7)	0.591
Polyradiculitis *n* (%)	1 (0.2)	0 (0.0)	1 (0.7)	0.591	1 (0.2)	0 (0.0)	1 (0.7)	0.591

Liotta EM, et al. Annals Clin Transl Neuro 2020; doi: 10.1002/acn3.51210

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Overview: COVID-19 Neurologic Disease

- Epidemiology
- **Pathogenesis**
- Clinical Spectrum of Neurologic Disease
- Treatment Considerations
- Impact on Non-COVID Neurology

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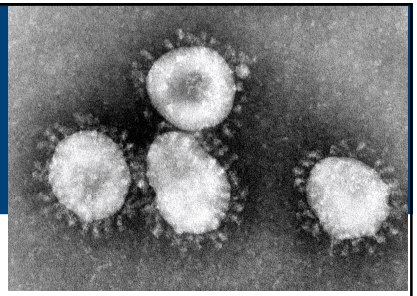
Prior Coronaviruses



- Large, enveloped positive strand RNA viruses, so named because of their “crown” viral spikes
- 4 Genera: alpha, beta, gamma, delta
- Four endemic strains (229E, NL63, OC43, HKU1) account for about 30% of adult uncomplicated URIs
- 2 highly pathogenic emerging beta-CoVs over past 20 yrs
  - SARS-CoV (Severe acute respiratory syndrome-CoV): 2002
  - MERS-CoV (Middle East respiratory syndrome-CoV): 2012
- 3 HCoV shown to infect neurons: **229E, 0C43, SARS-CoV**
- Reports of serious CNS and PNS neurologic sequelae from SARS and MERS (0.1-0.4% of cases) but pandemic scale much smaller

Ellul MA, et al. Lancet Neurol 2020; 19: 767-83.

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Potential COVID-19 Neuropathogenic Mechanisms

- **Direct Viral Neuroinvasion (not proven yet)**
  - Transsynaptic retrograde transfer into CNS
  - Entry via olfactory nerve
  - Infection of vascular endothelium
  - Leukocyte migration across BBB
- Systemic inflammation, coagulopathy and immune dysregulation
- Sequelae of organ failure and critical illness

Zubair AS, et al. JAMA Neurol. 2020; 77(8):1018-27.
Iadecola C, et al. Cell 2020; 183(1): 16-27.

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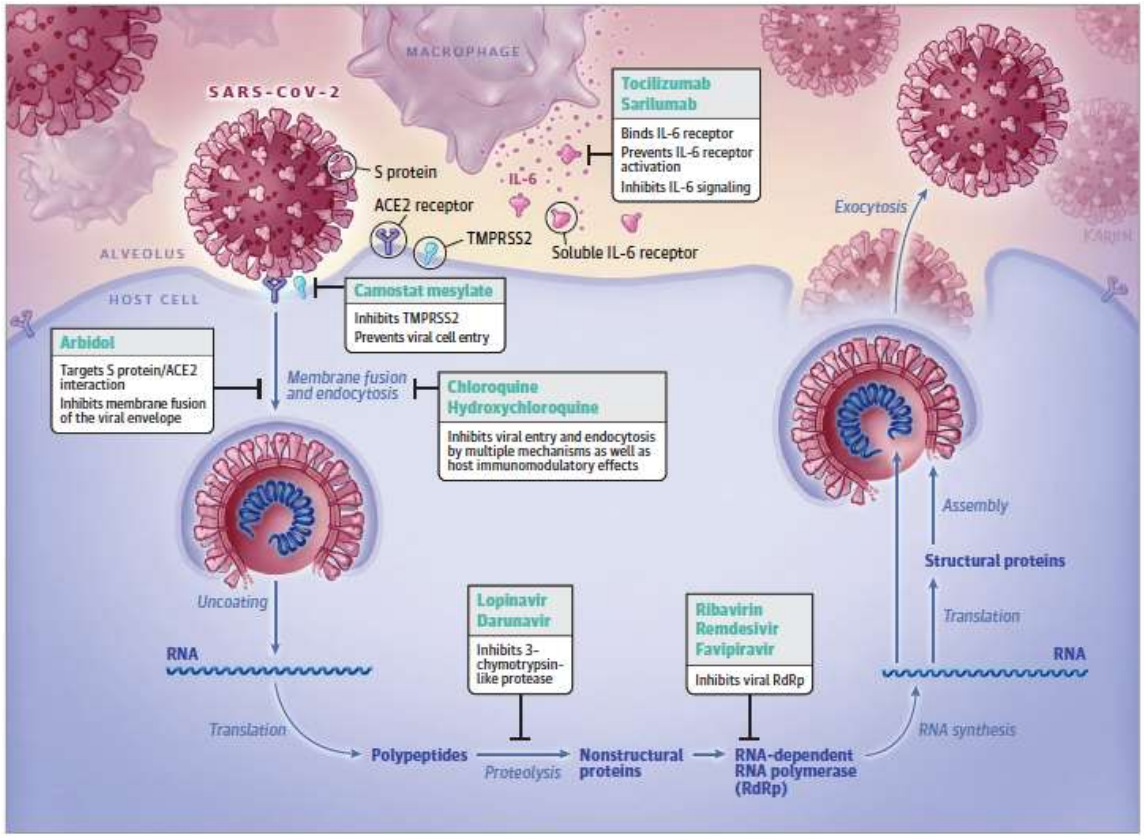
SARS-CoV-2 Life Cycle: ACE-2 Receptor



Sanders JM, et al. JAMA 2020; 323 (18):1824-36.

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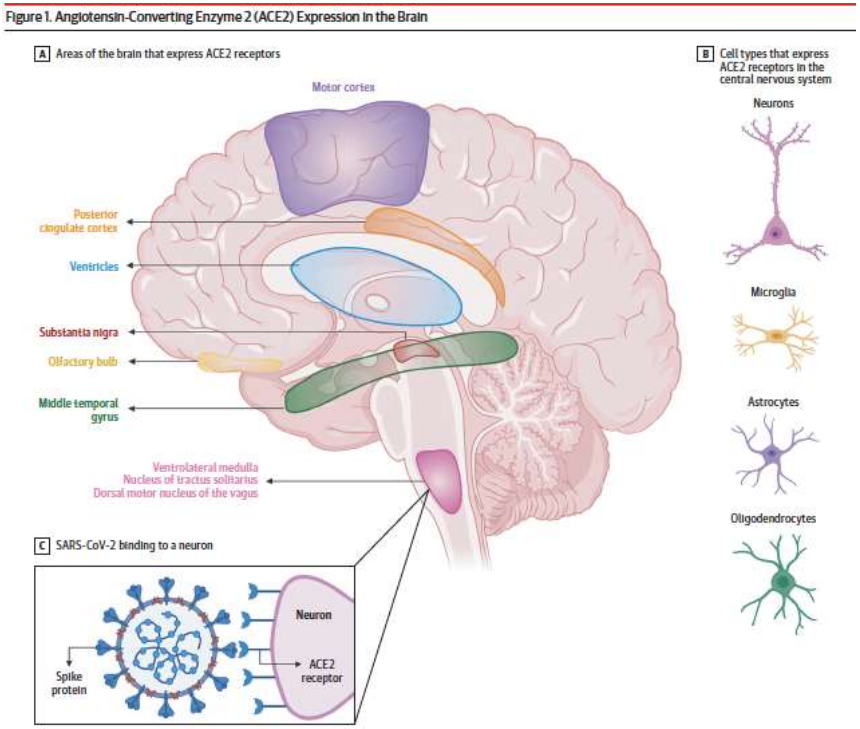
ACE-2 Receptor Expression in CNS

- *In vitro* and transgenic mouse studies show ACE-2 dependent neuronal infection and death
- Inconsistent autopsy and CSF or tissue studies to confirm ACE2 expression or viral invasion
- Virus may use non-canonical receptors basigin or neuropilin-1



Zubair AS, et al. JAMA Neurol. 2020; 77(8):1018-27.
Iadecola C, et al. Cell 2020; 183(1): 16-27.

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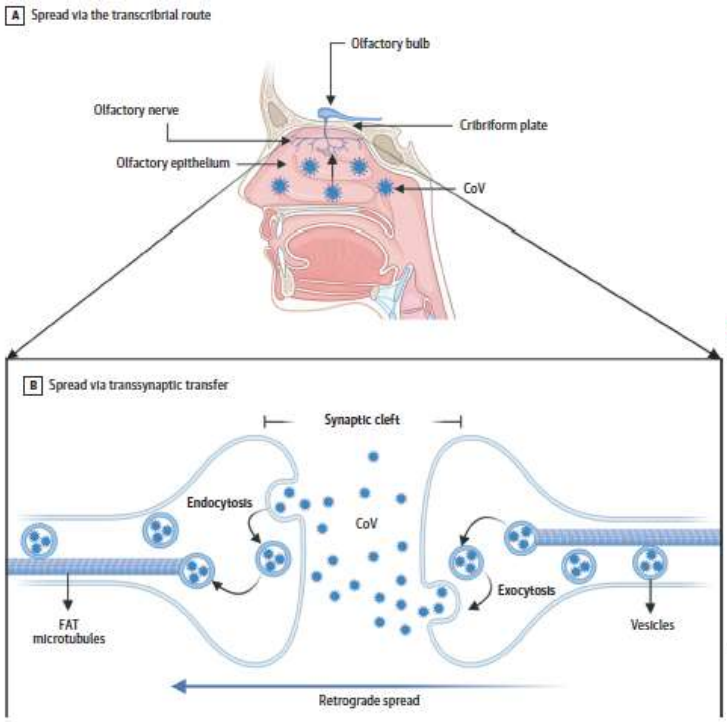
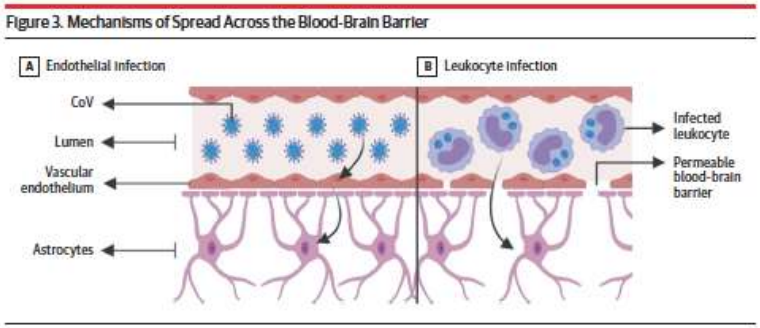
Potential CNS Entry Mechanisms



- Multiple potential mechanisms of CNS entry but **none definitively proven yet**



Zubair AS, et al. JAMA Neurol. 2020; 77(8):1018-27.
Jadecola C, et al. Cell 2020; 183(1): 16-27.

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- Epidemiology
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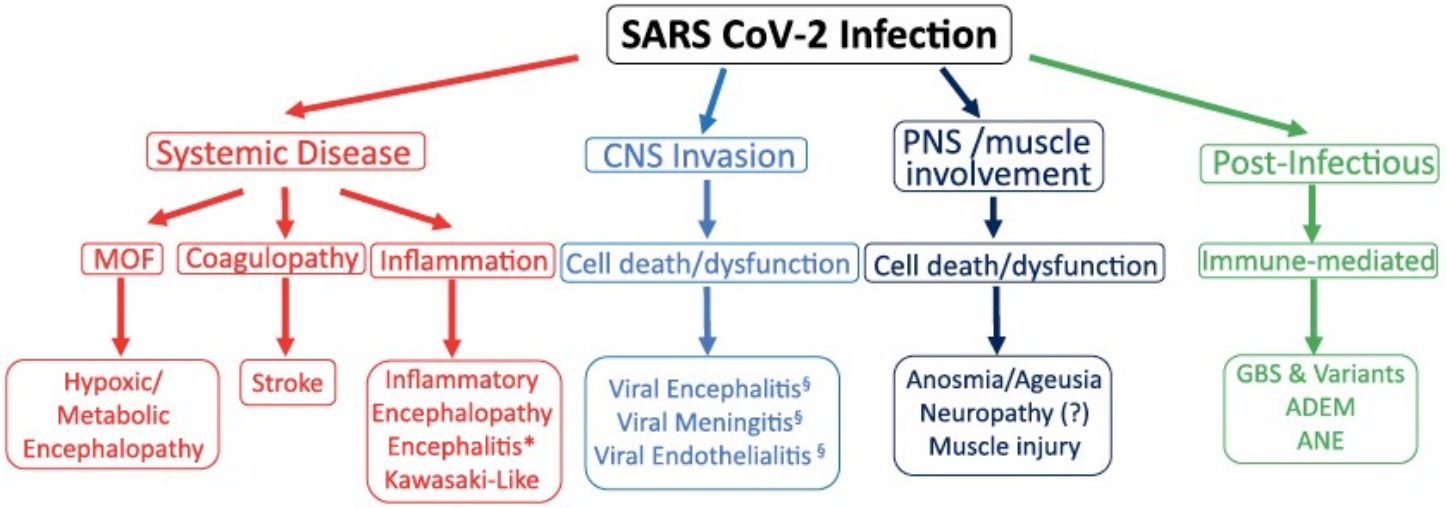
Clinical Spectrum of Neurologic Disease

- Most reported neurologic symptoms have been non-specific such as myalgias, headache, dizziness, and disorders of taste and smell



*Evidence of CNS inflammation without direct neuroinvasion
§ Direct evidence of neuroinvasion (positive PCR in CSF or positive biopsy)

Koralnik IJ, et al. Ann Neurol. 2020; 88:1-11.

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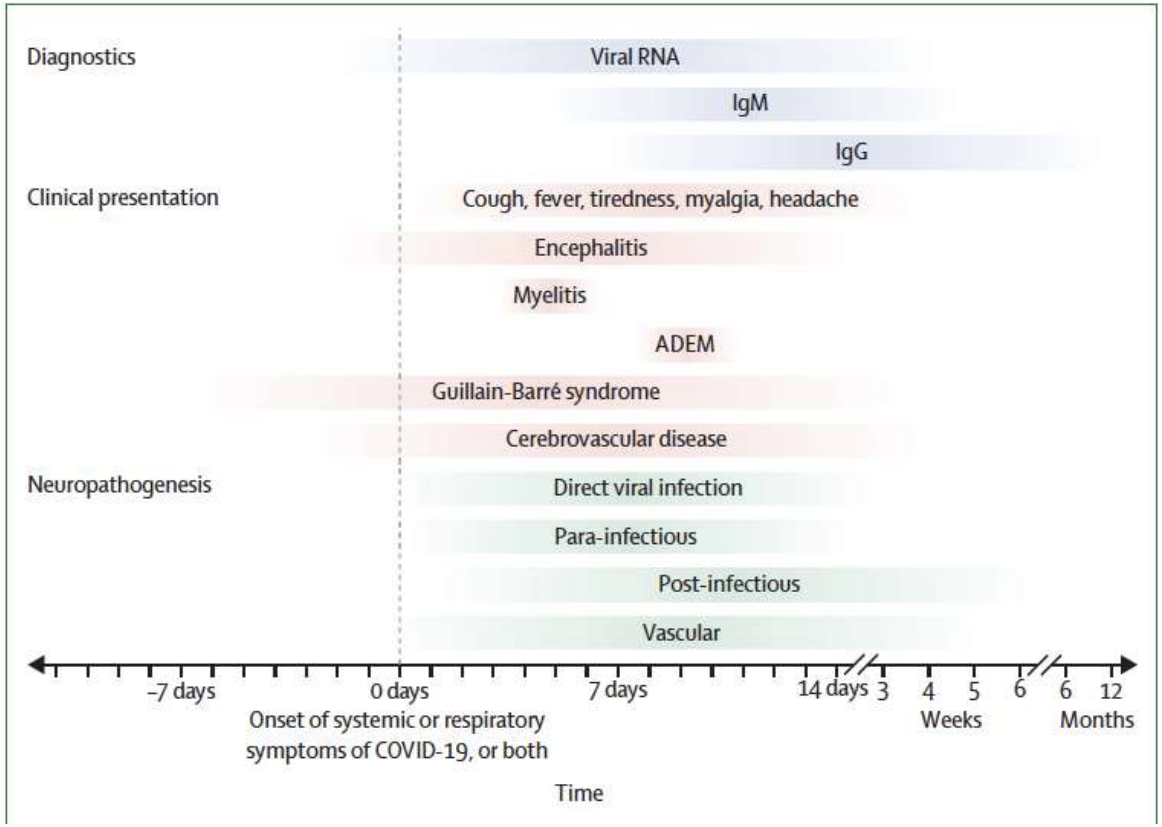
Time Course of Clinical Disease



Ellul MA, et al. Lancet Neurol 2020; 19:767-83.

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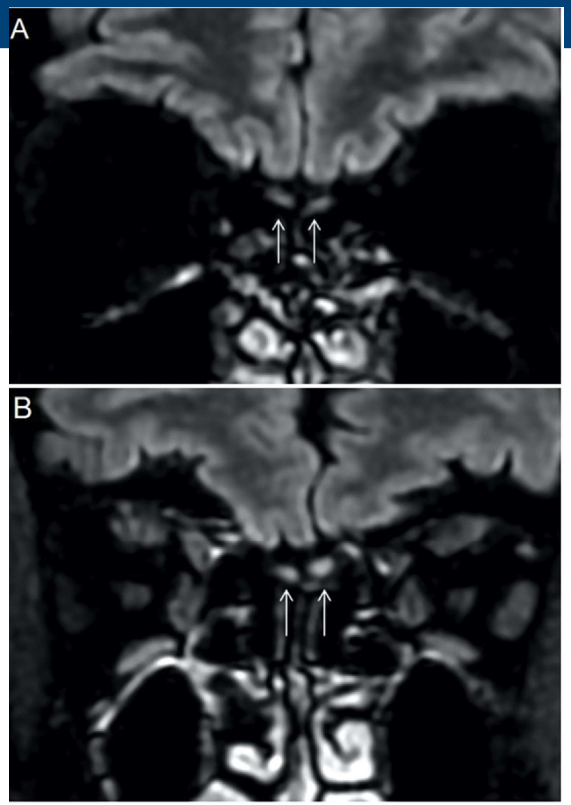
Disorders of Taste and Smell

- Anosmia and dysgeusia common sxs of COVID-19, may be initial sx in ≅ 10% of cases
- Wide range of incidence: 5% to > 80%, higher in Europe than in Asia
- European multicenter study of 417 mild-moderate COVID-19 found that 86% and 88% of pts have olfactory and gustatory dysfunction, 12% as first symptom[1]
- 80% of those without nasal obstruction or rhinorrhea were anosmic
- 44% recovery rate of olfaction at 8 days



MRI hyperintensity of olfactory bulb in anosmia

1. Lechien JR, et al. Eur Arch Otorhinolaryngol. 2020 Aug; 277 (8): 2251-61.
2. Chetrit A, et al. J Infection 2020 Jul 30; S0163-4453 (20) 30509-0.

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Encephalopathy/Altered Consciousness

- Most common serious CNS manifestation; incidence up to 20-30% in hospitalized pts, > 50% of pts with ARDS
- Multifactorial: Toxic-metabolic, hypoxia, sepsis, organ failure, drugs, etc.
- Risk factors similar to those for ICU delirium
- Imaging typical of post-hypoxic leukoencephalopathy from ARDS of any etiology
- Autopsy series of 18 pts some acute hypoxic/ischemic changes and chronic neuropathology
- Smaller subset may have CNS inflammation on CSF but negative viral PCR; ? Role for steroids

Solomon IH, et al. N Engl J Med 2020; 383:989.

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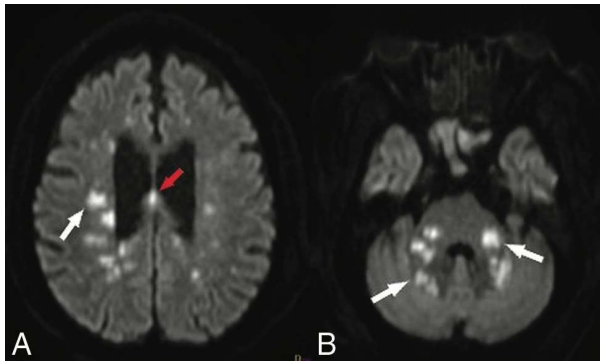
Cerebrovascular Disease

- Incidence in observational studies: Ischemic CVA 0.5-3% in hospitalized pts, up to 5% in ICU, ICH 0.2-0.9%
- Timing: Generally 1-3 weeks after COVID-19 sxs onset
- Multifactorial: Traditional CVA risk factors, infxn/ inflammation, hypercoaguability, cardioembolic, etc.
- One cohort study found aOR 7.6 for CVA in COVID compared to influenza (1.6% vs. 0.2%)
- More rare CVA subset may be in younger adults without risk factors or possibly due to vasculitis or endothelialitis



DWI Deep White Matter Ischemic Lesions

- Management should follow std. care for non-COVID stroke though various preventive AC strategies under study

Merkler AE, et al. JAMA Neurol 2020; doi:10.1001/jamaneurol.2020.2730.
Hanafi R, et al. Amer J of Neuroradiol 2020; doi:10.3174/ajnr.A6651.

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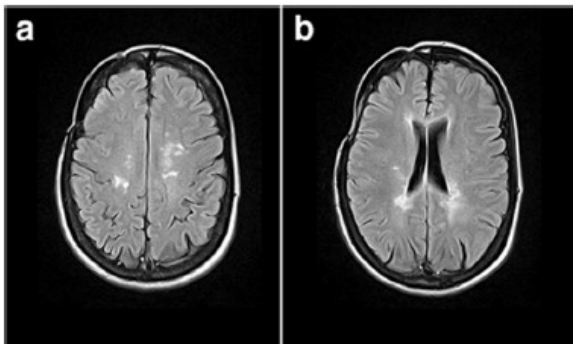
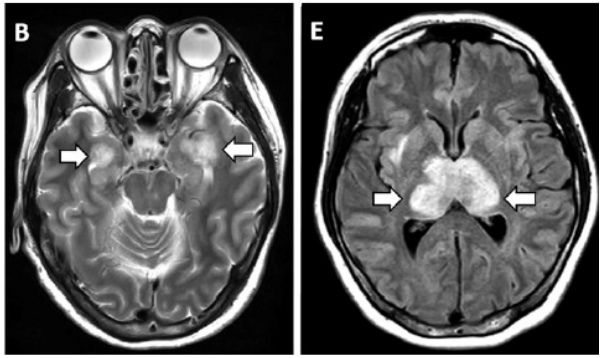
Autoimmune/Post-Infectious Sequelae

- **Guillain-Barre Syndrome** (GBS) and variants[1]
  - AIDP, AMAN, Miller Fisher syndrome all described with COVID-19
  - ? Worse clinical outcomes associated with COVID-19, not as common as with Zika
- **Acute Disseminated Encephalomyelitis** (ADEM)[2]
  - Cases with brain or spinal involvement, increasing numbers with hemorrhagic lesions
- **Acute Necrotizing Encephalopathy** (ANE)[3]
  - Case report of this rare complication, usually seen with influenza or other viruses
  - Characteristic symmetric, bilateral hemorrhagic lesions in thalami



T2 FLAIR of ADEM Case



T2 FLAIR Images of ANE Case

1. Zubair AS, et al. JAMA Neurol. 2020; 77(8):1018-27.; 2. Zanin L, et al. Acta Neurochir. 2020; 162:1491-94.; 3. Poyiadji N ,et al. Radiology 2020; 296:E119-120.

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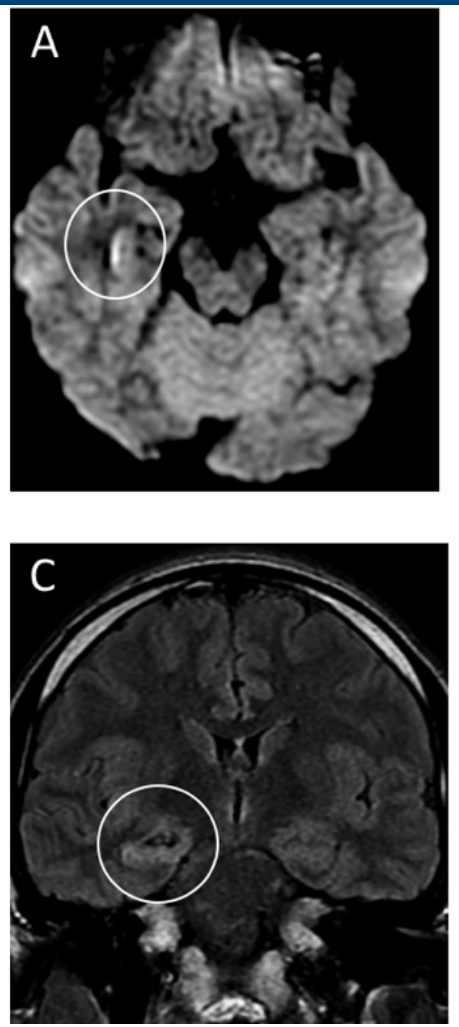
Viral Encephalitis

- Rare reports of suspected viral encephalitis with inflammation and detection of SARS-CoV-2 PCR in CSF

- First case of viral encephalitis reported in 24 yo Japanese male
  - 9 days of HA, fever, sore throat followed by seizures, meningismus and AMS
  - CSF: elevated OP, 12 WBCs (monocyte predominance), **+ SARS-CoV-2 PCR in CSF but negative NP swab**
  - DWI hyperintensity R lateral ventricle; FLAIR hyperintensity in R mesial temporal lobe and hippocampus
  - Outcome uncertain at time of publication



Moriguchi et al. Int J Infect Dis 2020; 94: 55-58.

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Summary of COVID-19 Associated Neurologic Clinical Presentations

TABLE. Neurologic Conditions Associated with SARS-CoV-2 Infection

Disease entity	Presentation	Supportive Neurodiagnostic testing	Pathogenesis
Encephalopathy	Altered mental status	MRI: non-specific EEG: abnormal (slow) CSF: nl cells and Pro CSF SARS-CoV-2 RT-PCR: NEG	Multiple organ failure Hypoxemia Systemic Inflammation Endothelialitis
Encephalitis	Altered mental status and CNS dysfunction	MRI: non-specific (? WM changes) EEG: abnormal (slow, +focal) CSF: pleocytosis & elev. Pro CSF SARS-CoV-2 RT-PCR: NEG	CNS inflammation
Viral encephalitis	Altered mental status and CNS dysfunction	MRI: new abnormality EEG: abnormal (slow, ±focal) CSF: Pleocytosis and elev. Pro CSF SARS-CoV-2 RT-PCR: POS Brain Tissue: POS (Ag or RNA)	Brain parenchymal neuro-invasion
Viral meningitis	Headache, nuchal rigidity	MRI: meningeal enhancement, CSF: pleocytosis & elev. Pro CSF SARS-CoV-2 RT PCR: POS	Subarachnoid invasion
Stroke	Focal motor or sensory deficit	MRI: ischemia or bleed, abnormal coagulation factors, increased inflammatory markers	Coagulopathy
Anosmia/ageusia	Olfactory or taste dysfunction	Abnormal smell/taste tests	? Peripheral vs central neuro-invasion
ADEM	Headache, acute neurologic symptoms	MRI: hyperintense FLAIR lesions with variable enhancement	Postinfectious
Guillain-Barre syndrome	Flaccid muscle weakness	CSF: increased protein, nl WBC CSF SARS-CoV-2 RT-PCR: NEG EMG/NCS: abnormal	Postinfectious
Muscle injury	Myalgia	CK elevated	Myopathy or myositis?

Koralnik IJ, et al. Ann Neurol. 2020; 88:1-11.

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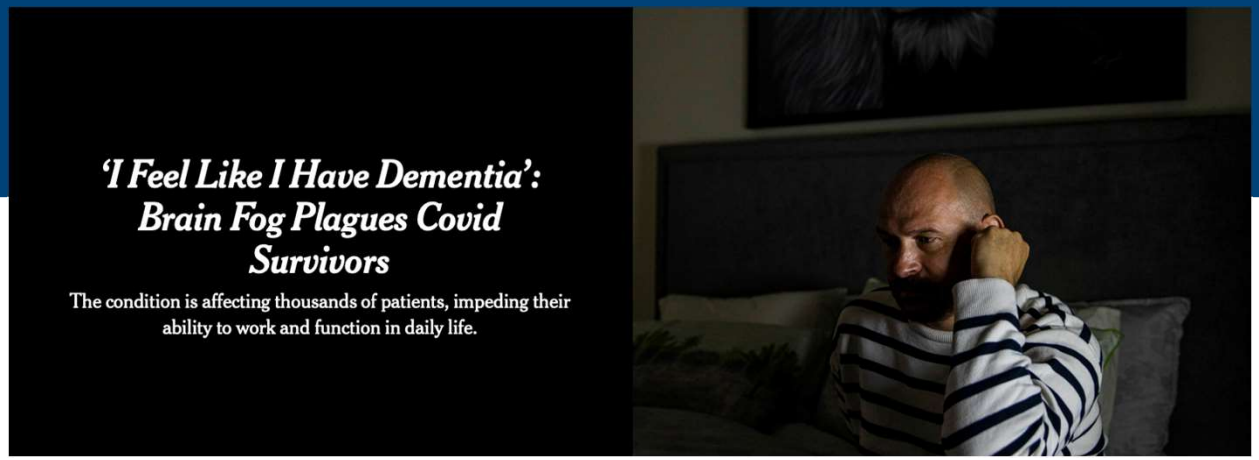

- Long-term sequelae of COVID-19 in so-called "long haulers"
- COVID-19 "brain fog": memory loss, confusion, dizziness, trouble focusing or grasping for words
- French study in 120 pts > 100 days after illness showed 34% with memory loss and 31% sleep disorders and 28% with concentration difficulties[1]
- Cause and management unclear although some suggest it is similar to PTSD following trauma or critical illness

NY Times 10/11/20 https://www.nytimes.com/2020/10/11/health/covid-survivors.html

1. Garrigues E, et al. J Infect 2020 Aug 25; doi:10.1016/j.jinf.2020.08.029.

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Overview: COVID-19 Neurologic Disease

- Epidemiology
- Pathogenesis
- Clinical Spectrum of Neurologic Disease
- **Treatment Considerations**
- Impact on Non-COVID Neurology

Treatment Considerations

- COVID treatments impact on CNS
  - Hydroxychloroquine: CNS side effects, can trigger myasthenic crisis or seizures; no efficacy proven for COVID-19
  - Tocilizumab: poor CNS penetration, cases of cerebral TMA
  - ICU care/mgmt: delirium, critical illness neuropathy/ myopathy all commonly seen
  - Vaccines: potential for rare CNS side effects such as transverse myelitis
- Management of Neurologic Conditions
  - No change in mgmt. of CVA, seizures, GBS or other conditions related to COVID-19, but **infection control and delivery of care more challenging**
  - Best Rx for coagulopathy in critically ill remains unclear

Zubair AS, et al. JAMA Neurol. 2020; 77(8):1018-27.

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COVID-19 Risk in Premorbid Neurologic Conditions

- Cerebrovascular disease and chronic neurologic diseases (e.g., dementia, ALS) possible risk factors for more severe COVID-19 per CDC[1]
- No strong evidence to suggest patients with neurologic disease on IS therapies at higher risk of infection, most guidance recommends continuation
- One case report of myasthenic crisis triggered by COVID-19[2]
- Older age, obesity, CV risk factors and more severe disability at baseline appear to be stronger risk factors for severe COVID-19[3]

1. https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/people-with-medical-conditions.html
2. Delly F, et al. J Neurol Sci 2020; 414:116888.
3. Louapre C, et al. JAMA Neurol 2020 Jun 26; 77(9):1-10.

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COVID Impact on Non-COVID Care

- Decreased or delays in Stroke care
  - 30-40% decrease in Stroke admissions during pandemic
  - Impact of lockdowns, overwhelmed health systems, patients avoiding or delaying care?
- Rise of Tele-medicine in Neuro (see prior talk)
- Widening disparities in care for underserved populations

Kansagra AP, et al. NEJM 2020; 383:400-401.
Josephson SA, et al. JAMA 2020; 324(12): 1139-40

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Conclusions

- Growing data on the incidence and spectrum of acute neurologic complications related to COVID-19, long-term data still needed
- Relative contributions of direct viral neuroinvasion, inflammation, vascular and autoimmune mechanisms unclear
- Most common neuro sxs are mild except encephalopathy in older pts or severe disease
- Chronic neurologic disease may be risk factor for more severe COVID-19 disease, but current guidance advise no change in baseline treatment except usual precautions
- COVID-19 has altered care delivery models and widened care disparities in Neurology as in all of medicine

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