STROKE IN THE TIME OF COVID-19
SPECIAL CONSIDERATIONS

For technical issues, please contact Felecia.Bryan@heart.org

The American Heart Association/American Stroke Association thanks Novartis for its support of the Quality Improvement Platform.

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HOST: DR MICHAEL FRANKEL

Since 1992, Dr. Michael Frankel has served as Chief of Neurology at Grady Memorial Hospital. He became the Director of the Marcus Stroke and Neuroscience Center at Grady when it opened in 2010. He is also a Professor of Neurology and Division Director of Vascular Neurology at Emory University School of Medicine.

Photo: The Wall Street Journal

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Disclosures (Frankel)

- Grant support
  - Industry
    - Nico Corporation: (ENRICH clinical trial)
  - NIH/CDC

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COVID-19 Data
5/7/2020

- Global Cases: 3,775,667
- Global Deaths: 264,406
- US Cases: 1,228,609
- US Deaths: 73,431

https://coronavirus.jhu.edu/map.html
Heart Attacks and Strokes Don’t Stop During Pandemics.
Call 911 right away if you have symptoms. Even while fighting the coronavirus, emergency systems stand ready to help.

heart.org
https://www.ahajournals.org.proxy.library.emory.edu/doi/pdf/10.1161/STROKEAHA.120.030023
CORRESPONDENCE

COVID-19 CASES

To rapidly communicate information on the global clinical effort against Covid-19, the Journal has initiated a series of case reports that offer important teaching points or novel findings. The case reports should be viewed as observations rather than as recommendations for evaluation or treatment. In the interest of timeliness, these reports are evaluated by in-house editors, with peer review reserved for key points as needed.

Large-Vessel Stroke as a Presenting Feature of Covid-19 in the Young

Thomas J. Oxley, M.D.
J. Mocco, M.D.
Shahram Majidi, M.D.
Christopher P. Kellner, M.D.
Hazem Shoirah, M.D.
I. Paul Singh, M.D.
Reade A. De Leacy, M.D.
Tomoyoshi Shigematsu, M.D.
Travis R. Ladner, M.D.
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Maryna Skliut, M.D.
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Presentation
Large Vessel Stroke as a Presenting Feature of COVID-19 in the Young

Stanley Tuhrim, MD
Dr. Tuhrim is the Director of the Division of Vascular Neurology and Vice-Chair for Clinical Affairs in the Department of Neurology. He is a Professor of Neurology and Geriatrics and Palliative Medicine in the Icahn School of Medicine at Mount Sinai.

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A Guidance Statement From the Society of Vascular and Interventional Neurology

Thanh N. Nguyen, MD, FRCPc; Mohamad Abdalkader, MD; Tudor G. Jovin, MD; Raul G. Nogueira, MD; Ashutosh P. Jadhav, MD; Diogo C. Haussen, MD; Ameer E. Hassan, DO; Roberta Novakovic, MD; Sunil A. Sheth, MD; Santiago Ortega-Gutierrez, MD, MSc; Peter D. Panagos, MD; Steve M. Cordina, MD; Italo Linfante, MD; Ossama Yassin Mansour, MD, PhD; Amer M. Malik, MD, MBA; Sandra Narayanan, MD; Hesham E. Masoud, MD; Sherry Hsiang-Yi Chou, MD; Rakesh Khatri, MD; Vallabh Janardhan, MD; Dileep R. Yavagal, MD; Osama O. Zaidat, MD; David M. Greer, MD; David S. Liebeskind, MD
Presentation

Neuroendovascular Considerations in Stroke Care During the COVID-19 Pandemic

Dr. Nogueira completed his training at the Massachusetts General Hospital (MGH)/Harvard Medical School (HMS). He is a Professor of Neurology at the Emory University School of Medicine and has been the Director of the Neuroendovascular Service at the Marcus Stroke & Neuroscience Center/Grady Memorial Hospital since its inception ten years ago.
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Questions & Answers at the end of all the presentations
Use the chat function to submit questions

- Large Vessel Stroke as a Presenting Feature of COVID-19 in the Young
- Neuroendovascular Considerations in Stroke Care During the COVID-19 Pandemic
- Venous Thromboembolism Considerations in Stroke Care During the COVID-19 Pandemic

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Stroke in the Time of Covid-19:
Large Vessel Stroke as a Presenting Feature of COVID-19 in the Young

Stanley Tuhrim, M.D.

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Daily Counts
This chart shows the number of positive cases by diagnosis date, hospitalizations by admission date and deaths by date of death from COVID-19 on a daily basis since March 3.
Hover over bars to see exact values.

Rates by Borough
This chart shows the number of positive cases per 100,000 people in each borough. It indicates the spread of COVID-19 relative to each borough’s population.

<table>
<thead>
<tr>
<th>Borough</th>
<th>Rate per 100,000 people</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Bronx</td>
<td>2,513</td>
<td>36,969</td>
</tr>
<tr>
<td>Staten Island</td>
<td>2,339</td>
<td>11,752</td>
</tr>
<tr>
<td>Queens</td>
<td>2,011</td>
<td>50,304</td>
</tr>
<tr>
<td>Brooklyn</td>
<td>1,585</td>
<td>42,996</td>
</tr>
<tr>
<td>Manhattan</td>
<td>1,069</td>
<td>20,121</td>
</tr>
<tr>
<td>Citywide</td>
<td></td>
<td>162,212</td>
</tr>
</tbody>
</table>
Total COVID-19 and PUI Hospitalized Cases by Day

- **COVID-19 Positive**
- **PUI**
2019 Mount Sinai Health System Stroke Mechanisms

- Large Artery Atherosclerotic Disease: 14%
- Cardioembolism: 21%
- Small Vessel Disease: 22%
- Stroke of Other Determined Source: 6%
- Cryptogenic: 37%

March 21-April 14 2020 Mount Sinai Hospital Stroke Mechanisms

- Large Artery Atherosclerotic Disease: 4%
- Cardioembolism: 26%
- Small Vessel Disease: 9%
- Stroke of Other Determined Source: 13%
- Cryptogenic: 48%
Mechanism of Large Vessel Occlusion Covid Cohort (March 21-April 14)

- Large Artery Atherosclerotic Disease: 57.1%
- Cardioembolism: 14.3%
- Small Vessel Disease: 14.3%
- Stroke of Other Determined Source: 0.0%
- Cryptogenic: 0.0%
### Mount Sinai Health System Endovascular Case Volumes

**March 21-April 30**

- **Q1 2020**: 49 cases (30 cases) + 9 cases
- **Q4 2019**: 48 cases
- **Q4 2019**: 59 cases (38 cases) + 38 cases
- **Q3 2019**: 52 cases
- **Q2 2019**: 51 cases
- **Q1 2019**: 53 cases

**Number of cases**

- Ranges from 48 to 59 cases
Clinical Characteristics of Five Young Patients Presenting with Large-Vessel Stroke.*

### Table 1. Clinical Characteristics of Five Young Patients Presenting with Large-Vessel Stroke.*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age—y</td>
<td>33</td>
<td>37</td>
<td>38</td>
<td>44</td>
<td>40</td>
</tr>
<tr>
<td>Sex</td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Male</td>
<td>Male</td>
</tr>
<tr>
<td>Medical history and risk factors for stroke</td>
<td>None</td>
<td>None</td>
<td>Hypertension, hyperlipidemia</td>
<td>Undiagnosed diabetes</td>
<td>Mild stroke, diabetes</td>
</tr>
<tr>
<td>Marfan’s syndrome</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>NIHSS score</td>
<td>10</td>
<td>15</td>
<td>12</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>At 24 hr</td>
<td>20</td>
<td>22</td>
<td>18</td>
<td>19</td>
<td>15</td>
</tr>
<tr>
<td>At last follow-up</td>
<td>22 (in-hospital)</td>
<td>25 (in-hospital)</td>
<td>25 (in-hospital)</td>
<td>20 (in-hospital)</td>
<td>17 (in-hospital)</td>
</tr>
<tr>
<td>Outcome status</td>
<td>Discharged home</td>
<td>Discharged to rehabilitation facility</td>
<td>Intensive care unite</td>
<td>Stroke unit</td>
<td>Discharged to rehabilitation facility</td>
</tr>
<tr>
<td>Time to presentation</td>
<td>24 h</td>
<td>16 h</td>
<td>16 h</td>
<td>16 h</td>
<td>16 h</td>
</tr>
<tr>
<td>Signs and symptoms of stroke</td>
<td>Headache, diplopia, dysphasia, dysarthria, dysphagia, dysphonia, sensory deficits</td>
<td>Headache, diplopia, dysphasia, dysphagia, dysphonia, sensory deficits</td>
<td>Headache, diplopia, dysphasia, dysphagia, dysphonia, sensory deficits</td>
<td>Headache, diplopia, dysphasia, dysphagia, dysphonia, sensory deficits</td>
<td>Headache, diplopia, dysphasia, dysphagia, dysphonia, sensory deficits</td>
</tr>
<tr>
<td>Vascular territory</td>
<td>Right internal carotid artery</td>
<td>Left middle cerebral artery</td>
<td>Left middle cerebral artery</td>
<td>Right middle cerebral artery</td>
<td>Right middle cerebral artery</td>
</tr>
<tr>
<td>Imaging for diagnosis</td>
<td>CT, CTA, MRI</td>
<td>CT, CTA, MRI</td>
<td>CT, CTA, MRI</td>
<td>CT, CTA, MRI</td>
<td>CT, CTA, MRI</td>
</tr>
<tr>
<td>Treatment for stroke</td>
<td>Intravenous (3 mg bolus)</td>
<td>Intravenous (3 mg bolus)</td>
<td>Intravenous (3 mg bolus)</td>
<td>Intravenous (3 mg bolus)</td>
<td>Intravenous (3 mg bolus)</td>
</tr>
<tr>
<td>COVID-19 symptoms</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>White cell count—per µL</td>
<td>7900</td>
<td>7900</td>
<td>7900</td>
<td>7900</td>
<td>7900</td>
</tr>
<tr>
<td>Platelet count—per µL</td>
<td>427,000</td>
<td>299,000</td>
<td>351,000</td>
<td>332,000</td>
<td>321,000</td>
</tr>
<tr>
<td>Prothrombin time—sec</td>
<td>13.3</td>
<td>12.4</td>
<td>12.4</td>
<td>12.8</td>
<td>13.3</td>
</tr>
<tr>
<td>Activated partial thromboplastin time—sec</td>
<td>35.0</td>
<td>35.7</td>
<td>35.7</td>
<td>36.4</td>
<td>37.6</td>
</tr>
<tr>
<td>Fibrinogen—mg/dL</td>
<td>501</td>
<td>570</td>
<td>790</td>
<td>440</td>
<td>531</td>
</tr>
<tr>
<td>D-dimer—µg/mL</td>
<td>400</td>
<td>52</td>
<td>2250</td>
<td>13,800</td>
<td>1700</td>
</tr>
<tr>
<td>Ferritin—µg/mL</td>
<td>180</td>
<td>190</td>
<td>190</td>
<td>190</td>
<td>190</td>
</tr>
</tbody>
</table>

* Reference ranges are as follows: platelet count, 150,000 to 450,000 per cubic millimeter; prothrombin time, 12.5 to 14.9 seconds; activated partial thromboplastin time, 25.5 to 34.9 seconds; fibrinogen, 175 to 410 mg per deciliter; and ferritin, 30 to 450 mg per milliliter. CT denotes computed tomography; CTA, CT angiography; CTP, CT perfusion; MRI, magnetic resonance imaging; NA, not applicable; PCR, polymerase chain reaction; and TIA, transitory ischemic attack.

* The patients were screened for smoking, hypertension, hyperlipidemia, diabetes, aortic fibrosis, congenital heart failure, illicit drug use, and neck trauma.

* Scores on the National Institutes of Health Stroke Scale (NIHSS) range from 0 to 42, with higher numbers indicating more severe stroke.

Hypothesized Mechanisms of LVO in COVID-19

- Coagulopathy now well-recognized in COVID-19 disease state
- Extent seems to correlate with severity of respiratory disease
- Virally mediated disruption of endothelium (endotheliitis) leading to thrombus formation may also play a role in LVO
- SARS-CoV-2 virus may infect host cells via ACE2 receptors expressed in multiple organs and on vascular endothelial cells
- Viral particles and accumulations of inflammatory cells have been identified in multiple organs
Endothelial cell infection and endotheliitis in COVID-19

Cardiovascular complications are rapidly emerging as a key threat in coronavirus disease 2019 (COVID-19) in addition to respiratory disease. The mechanisms underlying the disproportionate effect of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection on patients with cardiovascular comorbidities, however, remain incompletely understood. 5

SARS-CoV-2 infects the host using the angiotensin converting enzyme 2 (ACE2) receptor, which is expressed in several organs, including the lung, heart, kidney, and intestine. ACE2 receptors are also expressed by endothelial cells.6 Whether vascular derangements in COVID-19 are due to endothelial cell involvement by the virus is currently

Figure: Pathology of endothelial cell dysfunction in COVID-19

Mount Sinai COVID-19 Anticoagulation Algorithm

Admitted patients with moderate or severe COVID-19

High Risk#

↑ \(O_2\) requirement
↑ D-dimers
↑ creatinine
↑ CRP

No

Apixaban 2.5-5mg PO BID^ or Enoxaparin SC 40mg QD

Yes

Heparin drip per PE protocol (goal PTT 70 - 110) or Enoxaparin SC 1mg/kg BID. Consider tPA protocol.

Admitted to an ICU?

No

Yes

CrCl >50

No

On RRT†

Yes

Apixaban 5mg PO BID† or Adjusted Dose Enoxaparin*

Yes

Obtain at baseline and daily:
- CBC, PT/PTT, D-dimer

Hold anticoagulation if:
- Platelet count <50,000; INR>1.5
- Evidence of current or recent bleeding

If patients take AC at home:
- May switch to therapeutic enoxaparin or heparin (as per algorithm) for the duration of hospitalization, unless contraindicated

Rivaroxaban may be used in place of Apixaban at any indication

Discharged COVID-19 patient on therapeutic anticoagulation while hospitalized

Consider Prophylactic AC for 2 weeks post discharge (Apixaban 5mg PO BID for 2 wks)

**Inclusion:** All admitted patients with moderate or severe COVID-19
**Exclusion:** High risk of bleeding as judged by treating physician

#High Risk: No precise metrics exist. Consider exam (eg \(O_2\) sat<90%, RR >24), ↑\(O_2\) requirement (eg, ≥4L NC), labs (eg, ↑d-dimers, C-reactive protein)

^Efficacy and dose not established; prophylactic or treatment doses acceptable

†RRT – Renal Replacement Therapy
‡ If ≥80 years of age or weight ≤60 kg, reduce apixaban to 2.5 mg BID
* If CrCl <30: enoxaparin 0.5mg/kg BID with anti-Xa level after 3rd dose
Mount Sinai COVID-19 Anticoagulation Algorithm

Definition of high risk for progression to ICU
- There is insufficient evidence to precisely define “high-risk” or provide specific cut-off values for individual factors
- Clinicians should consider a combination of exam findings (e.g., labored breathing, RR >24, decreased O₂ sat<90%), increased O₂ requirement (e.g., ≥4L NC), and lab biomarkers (e.g., elevated CRP, elevated creatinine, rising d-dimer >1.0).

Rationale for early anticoagulation
- Pathophysiology of COVID-19 associated respiratory disease is consistent with pulmonary vascular thromboemboli with increased dead space ventilation
- Autopsy studies have demonstrated venous thromboembolism in deceased coronavirus patients¹
- Early anticoagulation is necessary to prevent propagation of microthrombi at disease presentation
- Anticoagulation may be associated with decreased mortality²

Rationale for choice of anticoagulant
- Heparins bind tightly to COVID-19 spike proteins³,⁴
- Heparins also downregulate IL-6 and directly dampen immune activation⁵
- DOACs do not appear to have these anti-inflammatory properties
- Rivaroxaban can be used in place of Apixaban in this algorithm

References

Version 1.1 (April 9, 2020)
Association of Treatment Dose Anticoagulation with In-Hospital Survival Among Hospitalized Patients with COVID-19

Association of Treatment Dose Anticoagulation with In-Hospital Survival Among Hospitalized Patients with COVID-19

- Between March 14 and April 11, 2020, 2773 patients with laboratory-confirmed COVID-19 were admitted to the MSHS
- 786 received systemic anticoagulation during their hospital course
- Median time from admission to anticoagulation was 2 days
- Serious bleeding events occurred in 3% of AC and 1.9% non-AC but 1/3 of AC group’s bleeds occurred before anticoagulation
- AC group was more likely to require mech ventilation (30% vs 8%)
- In-hospital mortality for the 2 groups was similar, 22.5% in AC, 22.8% non-AC, but median survival was increased 21 vs 14 days
- Multivariate proportional hazard model longer duration of AC was associated with a reduced risk of mortality (HR .86/day, p<.001)

Large Vessel Stroke in the COVID ERA

Summary

• Stroke involving LVO seems to occur in patients with mild COVID-19 symptoms
• Stroke mechanism is not yet known but may be independent of hypercoagulable markers, or relate to a combination of a hypercoagulable state and direct endothelial damage (endotheliitis)
• Anticoagulation of hospitalized patients may improve outcome
• Other interventions, including those targeting the endothelium (ACEI?, statins?), may prove helpful
Neuroendovascular Considerations In Stroke Care During the COVID-19 Pandemic

Raul G Nogueira, MD

Professor in Neurology, Neurosurgery, and Radiology
Emory University

Director, Neuroendovascular Service
Grady Memorial Hospital
Atlanta, GA

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Disclosures:

Stryker’s Neurovascular Division
- DAWN Trial PI (unpaid)
- Trevo Registry Steering Committee
- Trevo-2 Trial PI
- Consultant

Medtronic
- SWIFT & SWIFT-PRIME Trial Steering Committee (unpaid)
- STAR Trial Core Lab

Penumbra (unpaid)
- 3-D Separator Trial Executive Committee

Neuravi /Cerenovus
- ARISE-II Trial Steering Committee (unpaid)
- ENDOLOW Trial and EXCELLENT Registry PI

Phenox, Anaconda, Corindus Robotics, Biogen, Genentech, Prolong Pharmaceuticals

Allm Inc - JOIN (unpaid)
- Free Consultant and Beta-Site
- FAST-ED App (Freeware)
- RESILIENT Trial Collaboration

IschemaView
- CRISP, SWIFT-PRIME, & DAWN Trials (unpaid)
- Speaker (paid)
- RESILIENT Trial Collaboration

Brainomix (unpaid)
- Research Software Usage
- RESILIENT Trial Collaboration

Viz-AI (Stock Options)
- Physician Advisory Board

Raul Nogueira, MD

Emory University
Before We Start....
INFODEMIC: A Strong Ally to COVID-19

Opinions are not FACTS!
INFODEMIC: A Strong Ally to COVID-19

Levels of evidence for COVID-19 data

- Case Control Studies
  - Cohort Studies
    - Meta Analyses
      - Randomized Trials
        - YouTube COVID-19 Conspiracies
          - Instagram Influencers
            - Family Group on WhatsApp

Quality of Information

OUR COLLECTIVE RESPONSIBILITY

The Pandemic Infodemic...
Thus far in the Covid-19 pandemic, we’ve observed that therapeutic management has often been initiated and altered on the basis of individual case reports and physician opinion, rather than of randomized trials. In these uncertain times, physicians fall prey to cognitive error and unconsciously rely on limited experiences, whether their own or others’, instead of scientific inquiry. We believe that physicians should be acting in concert with clinical equipoise. We should be skeptical of any purported therapeutic strategy until enough statistical evidence is gathered that would convince any “open-minded clinician informed of the results” that one treatment is superior to another.
COVID-19: Neurological Manifestations and Stroke
INVESTIGATIONS

Research shows COVID-19 is causing strokes in patients, including a Texas correctional officer

COVID-19 may increase blood cloting and blockage of brain blood vessels

by Henry Killworth, University College London

CT brain images highlighting acute ischaemic stroke. Credit: Jacob F...

Clinical observations of COVID-19 patients, who went on to have a stroke, suggest coronavirus may cause clots within blood vessels (arteries) in the brain, finds a team of neurologists from UCL and UCLH (the National Hospital for Neurology and Neurosurgery), London.

NJ cardiologist suffers stroke likely due to COVID-19 while recovering

Published 3 hours ago | Good Day | FOX 29 Philadelphia

NJ cardiologist suffers stroke likely due to COVID-19
Dr. Troy Barbee discusses his experience with recovering from COVID-19.

MEDPAGE TODAY

U.K. Report Backs Stroke as COVID-19 Complication
— More questions raised about anticoagulation timing, dose

by Jody Hyslop, Senior Staff Writer, MedPage Today May 4, 2020

Acute ischemic strokes were seen in six British COVID-19 patients, including two who had breakthrough strokes despite therapeutic anticoagulation, researchers reported.

Infectious Disease x COVID-19

Earning What You're Worth?

MSCF Desk

HealthcaresetPosition

MedPage Today

ACM Virtual Career Fair 2020
May 19, 2020 | 11:00am - 1:00pm

ACC Career Center


**Encephalitis in COVID 19**
## Guillain–Barré Syndrome Associated with SARS-CoV-2

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Onset of Neurologic Syndrome</th>
<th>Neurologic Signs and Symptoms</th>
<th>CSF Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7 Days after fever, cough, and ageusia</td>
<td>Flaccid areflexic tetraplegia evolving to facial weakness, upper-limb paresthesia (36 hr), and respiratory failure (day 6)</td>
<td>Day 2 (first lumbar puncture): normal protein level; no cells; negative PCR assay for SARS-CoV-2 Day 10 (second lumbar puncture): protein level, 101 mg/dl; white-cell count, 4 per mm³; negative PCR assay for SARS-CoV-2</td>
</tr>
<tr>
<td>2</td>
<td>10 Days after fever and pharyngitis</td>
<td>Facial diplegia and generalized areflexia evolving to lower-limb paresthesia with ataxia (day 2)</td>
<td>Day 3: protein level, 123 mg/dl; no cells; negative PCR assay for SARS-CoV-2</td>
</tr>
<tr>
<td>3</td>
<td>10 Days after fever and cough</td>
<td>Flaccid areflexia and facial weakness evolving to areflexia (day 2) and respiratory failure (day 5)</td>
<td>Day 3: protein level, 193 mg/dl; no cells; negative PCR assay for SARS-CoV-2</td>
</tr>
<tr>
<td>4</td>
<td>5 Days after cough and hyposmia</td>
<td>Flaccid areflexic tetraparesis and ataxia (day 4)</td>
<td>Day 5: normal protein level; no cells; negative PCR assay for SARS-CoV-2</td>
</tr>
<tr>
<td>5</td>
<td>7 Days after cough, ageusia, and anosmia</td>
<td>Facial weakness, flaccid areflexic paraplegia (days 2–3), and respiratory failure (day 4)</td>
<td>Day 3: protein level, 40 mg/dl; white-cell count, 3 per mm³; CSF/serum albumin ratio, 1.24; negative PCR assay for SARS-CoV-2</td>
</tr>
</tbody>
</table>

**Table 1. Characteristics of Five Patients with Guillain–Barré Syndrome after the Onset of Covid-19.†**

- **Antiangioidase Antibodies:**
  - Head: normal
  - Spine: enhancement of caudal nerve roots

- **MRI Results:**
  - Head: enhancement of facial nerve bilaterally
  - Spine: normal

- **Treatment and Outcomes at Week 4:**
  - Received 2 cycles of IVIG; had poor outcomes, including persistence of severe upper-limb weakness, dysphagia, and lower-limb paraplegia
  - Received IVIG; had improvement, including increase in ataxia and mild decrease in facial weakness
  - Received 2 cycles of IVIG; had poor outcomes, including ICU admission owing to neuromuscular respiratory failure and flaccid tetraplegia
  - Received IVIG; had mild improvement but unable to stand 1 mo after onset
  - Received IVIG and plasma exchange; had bacterial pneumonia during IVIG treatment, which delayed plasma exchange

---

†Covid-19 denotes coronavirus disease 2019, CSF cerebrospinal fluid, ICU intensive care unit, IVIG intravenous immune globulin, MRI magnetic resonance imaging, PCR polymerase chain reaction, and SARS-CoV-2 severe acute respiratory syndrome coronavirus 2.

†On CSF analysis, all the patients had a normal glucose level and IgG index and a polyclonal pattern on electrophoresis. The normal range for the protein level is 15 to 45 mg per deciliter.

†An enzyme-linked immunosorbent assay was used to test for antibodies to GM1, GQ1b, and GD1b.
Patients **severe infection** (n=88/214; 41.1%) (as per the international guidelines for community-acquired pneumonia were older and more often had other underlying disorders but had **less typical symptoms such as fever** (40 [45.5%] vs 92 [73%], P<0.001) and **dry cough** (30 [34.1%] vs 77 [61.1%], P<0.001).

**Neurological symptoms** were significantly **more common in severe cases** as compared with non-severe cases (40 [45.5%] vs. 38 [30.2%], P<0.05).

**Neurological symptoms** included **stroke** in **5.7%** (n=5; 4 ischemic; 1 hemorrhagic), impaired consciousness in **14.8%** and muscle injury in **19.3%** of the 88 patients with **severe disease**.

Patients with CNS symptoms had lower lymphocyte and platelet counts and higher blood urea nitrogen levels.

There is a neuroinvasive potential of SARS-CoV2. Notably, infection of SARS-CoV has been reported in the brains from both patients and experimental animals, where the brainstem was heavily infected ([J Med Virol.](https://doi.org/10.1002/jmv.25728) 2020 Feb 27. doi: 10.1002/jmv.25728. [Epub ahead of print])
Clinical neurological findings in COVID-19

• Chinese autopsy reports of endovasculitis, endothelial damage, cerebral edema, hyperemia, and neurodegeneration in SARS-CoV-2

• Other neurological complications of critical illness, such as critical illness myopathy, increased thrombotic risk, and cerebral hypoperfusion, may result from the extended intensive care stays.
### Table 1. Clinical Characteristics of Five Young Patients Presenting with Large-Vessel Stroke.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age — yr</td>
<td>33</td>
<td>34</td>
<td>39</td>
<td>44</td>
<td>49</td>
</tr>
<tr>
<td>Sex</td>
<td>Female</td>
<td>Male</td>
<td>Male</td>
<td>Male</td>
<td>Male</td>
</tr>
<tr>
<td>Medical history and risk factors for stroke?</td>
<td>None</td>
<td>None</td>
<td>Hyperlipidemia, hypertension</td>
<td>Undiagnosed diabetes</td>
<td>Mild stroke, diabetes</td>
</tr>
<tr>
<td>Medications</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>None</td>
<td>Aspirin (81 mg), atorvastatin (80 mg)</td>
</tr>
<tr>
<td>NIHSS score</td>
<td>19</td>
<td>13</td>
<td>16</td>
<td>23</td>
<td>13</td>
</tr>
<tr>
<td>On admission</td>
<td>13</td>
<td>13</td>
<td>16</td>
<td>23</td>
<td>13</td>
</tr>
<tr>
<td>At 24 hr</td>
<td>17</td>
<td>11</td>
<td>16</td>
<td>23</td>
<td>11</td>
</tr>
<tr>
<td>At last follow-up</td>
<td>13 (on day 14)</td>
<td>5 (on day 15)</td>
<td>NA; intubated and sedated, with multigorgan failure</td>
<td>19 (on day 12)</td>
<td>7 (on day 4)</td>
</tr>
<tr>
<td>Outcome status</td>
<td>Discharged to rehabilitation facility</td>
<td>Discharged home</td>
<td>Intensive care unit</td>
<td>Stroke unit</td>
<td>Discharged to rehabilitation facility</td>
</tr>
<tr>
<td>Time to presentation — hr</td>
<td>28</td>
<td>16</td>
<td>8</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Signs and symptoms of stroke</td>
<td>Hemiplegia on left side, facial droop, gaze preference, homonymous hemianopia, dysarthria, sensory deficit</td>
<td>Reduced level of consciousness—dysphasia, hemiplegia on right side, dysarthria, sensory deficit</td>
<td>Reduced level of consciousness, gaze preference to the right, left homonymous hemianopia, hemiplegia on left side, stances</td>
<td>Reduced level of consciousness, global dysphasia, hemiplegia on right side, gaze preference</td>
<td>Reduced level of consciousness—hemiplegia on left side, dysarthria, facial weakness</td>
</tr>
<tr>
<td>Vascular territory</td>
<td>Right middle cerebral artery</td>
<td>Left middle cerebral artery</td>
<td>Right posterior cerebral artery</td>
<td>Left middle cerebral artery</td>
<td>Right middle cerebral artery</td>
</tr>
<tr>
<td>Imaging for diagnosis</td>
<td>CT, CTA, CTP, MRI</td>
<td>CT, CTA, MRI</td>
<td>CT, CTA, CTP, MRI</td>
<td>CT, CTA, MRI</td>
<td>CT, CTA, CTP</td>
</tr>
<tr>
<td>Treatment for stroke</td>
<td>Aipasibin (5 mg twice daily)</td>
<td>Clot retrieval, aipasibin (5 mg twice daily)</td>
<td>Clot retrieval, aspirin (81 mg twice daily)</td>
<td>Intravenous t-PA, clot retrieval, hemicraniectomy, aspirin (81 mg daily)</td>
<td>Clot retrieval, stent, aspirin (325 mg daily), glyco- gen (75 mg daily)</td>
</tr>
<tr>
<td>Covid-19 symptoms</td>
<td>Cough, headache, chills</td>
<td>No symptoms; recently exposed to family member with PCR-positive Covid-19</td>
<td>None</td>
<td>None</td>
<td>Lethargy</td>
</tr>
<tr>
<td>White-cell count — per mm³</td>
<td>7800</td>
<td>9900</td>
<td>5500</td>
<td>9000</td>
<td>4900</td>
</tr>
<tr>
<td>Platelet count — per mm³</td>
<td>427,000</td>
<td>299,000</td>
<td>135,000</td>
<td>372,000</td>
<td>255,000</td>
</tr>
<tr>
<td>Prothrombin time — sec</td>
<td>13.3</td>
<td>13.4</td>
<td>14.4</td>
<td>12.8</td>
<td>15.2</td>
</tr>
<tr>
<td>Activated partial thromboplastin time — sec</td>
<td>25.0</td>
<td>42.7</td>
<td>27.7</td>
<td>26.9</td>
<td>37.0</td>
</tr>
<tr>
<td>Fibroinogen — mg/dl</td>
<td>501</td>
<td>370</td>
<td>739</td>
<td>443</td>
<td>531</td>
</tr>
<tr>
<td>D-dimer — mg/ml</td>
<td>460</td>
<td>52</td>
<td>2230</td>
<td>13,800</td>
<td>1750</td>
</tr>
<tr>
<td>Ferritin — ng/ml</td>
<td>7</td>
<td>136</td>
<td>1564</td>
<td>987</td>
<td>596</td>
</tr>
</tbody>
</table>

*Reference ranges are as follows: platelet count, 150,000 to 450,000 per cubic millimeter; prothrombin time, 12.3 to 14.9 seconds; activated partial thromboplastin time, 25.4 to 34.9 seconds; fibrinogen, 175 to 450 mg per deciliter; D-dimer, 0 to 500 mg per milliliter; and ferritin, 30 to 400 mg per milliliter. CT denotes computed tomography; CTA CT angiography; CTP CT perfusion; MRI magnetic resonance imaging. NA not applicable, PCR polymerase chain reaction, t-PA tissue plasminogen activator.

The patients were screened for smoking, hypertension, hyperlipidemia, diabetes, atrial fibrillation, congestive heart failure, illicit drug use, and neck trauma.

Scores on the National Institutes of Health Stroke Scale (NIHSS) range from 0 to 42, with higher numbers indicating more severe stroke.

### COVID-19 CASES

To rapidly communicate information on the global clinical effort against Covid-19, the Journal has initiated a series of case reports that offer important teaching points or novel findings. The case reports should be viewed as observations rather than as recommendations for evaluation or treatment. In the interest of timelines, these reports are evaluated by in-house editors, with peer review reserved for key points as needed.

Large-Vessel Stroke as a Presenting Feature of Covid-19 in the Young
## Stroke in COVID-19

### Table 1. Baseline characteristics of COVID-19 patients with new onset of CVD during infection

<table>
<thead>
<tr>
<th>Type of CVD</th>
<th>Subtype of AIS</th>
<th>Age, y</th>
<th>Sex</th>
<th>Smoking History</th>
<th>Drinking History</th>
<th>Blood pressure (mm Hg)</th>
<th>Fasting Blood-glucose (mmol/L)</th>
<th>Type of COVID-19 Patients (Severe/Non-Severe)</th>
<th>Onset Time of SAR-CoV-2 Infection</th>
<th>Onset Time of CVD</th>
<th>Treatment of CVD</th>
<th>Outcome Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIS</td>
<td>Small vessel</td>
<td>70</td>
<td>M</td>
<td>No</td>
<td>No</td>
<td>110/70</td>
<td>5.44</td>
<td>Severe</td>
<td>01/26/20</td>
<td>02/23/20</td>
<td>Antiplatelet</td>
<td>Survival</td>
</tr>
<tr>
<td>AIS</td>
<td>Large vessel stenosis</td>
<td>75</td>
<td>F</td>
<td>No</td>
<td>No</td>
<td>110/67</td>
<td>6.03</td>
<td>Severe</td>
<td>01/24/20</td>
<td>02/15/20</td>
<td>Antiplatelet</td>
<td>Survival</td>
</tr>
<tr>
<td>AIS</td>
<td>Cardiac embolic</td>
<td>89</td>
<td>F</td>
<td>No</td>
<td>No</td>
<td>97/64</td>
<td>6.77</td>
<td>Non-severe</td>
<td>02/19/20</td>
<td>02/19/20</td>
<td>Anticoagulant</td>
<td>Survival</td>
</tr>
<tr>
<td>AIS</td>
<td>Large vessel stenosis</td>
<td>91</td>
<td>F</td>
<td>No</td>
<td>No</td>
<td>192/97</td>
<td>6.7</td>
<td>Severe</td>
<td>02/01/20</td>
<td>02/10/20</td>
<td>Anticoagulant</td>
<td>Survival</td>
</tr>
<tr>
<td>AIS</td>
<td>Large vessel stenosis</td>
<td>72</td>
<td>F</td>
<td>No</td>
<td>No</td>
<td>155/89</td>
<td>7.93</td>
<td>Severe</td>
<td>02/01/20</td>
<td>02/12/20</td>
<td>Anticoagulant</td>
<td>Survival</td>
</tr>
<tr>
<td>AIS</td>
<td>Cardiac embolic</td>
<td>71</td>
<td>M</td>
<td>Yes</td>
<td>No</td>
<td>142/67</td>
<td>16.25</td>
<td>Severe</td>
<td>01/31/20</td>
<td>02/07/20</td>
<td>Anticoagulant</td>
<td>Death</td>
</tr>
<tr>
<td>AIS</td>
<td>Cardiac embolic</td>
<td>86</td>
<td>M</td>
<td>Yes</td>
<td>No</td>
<td>110/72</td>
<td>13.81</td>
<td>Severe</td>
<td>01/24/20</td>
<td>02/11/20</td>
<td>Antiplatelet</td>
<td>Death</td>
</tr>
<tr>
<td>AIS</td>
<td>Large vessel stenosis</td>
<td>82</td>
<td>F</td>
<td>Yes</td>
<td>No</td>
<td>140/83</td>
<td>24.2</td>
<td>Severe</td>
<td>02/02/20</td>
<td>02/16/20</td>
<td>Antiplatelet</td>
<td>Death</td>
</tr>
<tr>
<td>AIS</td>
<td>Small vessel</td>
<td>78</td>
<td>M</td>
<td>Yes</td>
<td>No</td>
<td>156/82</td>
<td>11.0</td>
<td>Severe</td>
<td>01/17/20</td>
<td>01/17/20</td>
<td>Antiplatelet</td>
<td>Death</td>
</tr>
<tr>
<td>AIS</td>
<td>Large vessel stenosis</td>
<td>57</td>
<td>M</td>
<td>No</td>
<td>No</td>
<td>127/83</td>
<td>13.24</td>
<td>Non-severe</td>
<td>02/06/20</td>
<td>02/07/20</td>
<td>Anticoagulant</td>
<td>Survival</td>
</tr>
<tr>
<td>AIS</td>
<td>Small vessel</td>
<td>66</td>
<td>F</td>
<td>No</td>
<td>Yes</td>
<td>98/62</td>
<td>8.67</td>
<td>Non-severe</td>
<td>02/11/20</td>
<td>02/17/20</td>
<td>Anticoagulant</td>
<td>Survival</td>
</tr>
<tr>
<td>CVD</td>
<td>Small vessel</td>
<td>32</td>
<td>M</td>
<td>Yes</td>
<td>Yes</td>
<td>130/80</td>
<td>8.23</td>
<td>Severe</td>
<td>02/09/20</td>
<td>02/23/20</td>
<td>Anticoagulant</td>
<td>Survival</td>
</tr>
<tr>
<td>CVD</td>
<td>CH</td>
<td>62</td>
<td>M</td>
<td>Yes</td>
<td>No</td>
<td>150/80</td>
<td>5.81</td>
<td>Severe</td>
<td>01/23/20</td>
<td>02/01/20</td>
<td>Death</td>
<td></td>
</tr>
</tbody>
</table>

- The patients of COVID-19 were confirmed by SARS-CoV-2 RT-PCR positive in throat swab and viral-like pneumonia in chest CT.

Abbreviations: COVID-19, Coronavirus disease 2019; CVD, Cerebrovascular disease; AIS, Acute ischemia stroke; CH, Cerebral hemorrhage; CVST, Cerebral Venous Sinus Thrombosis; F, Female; M, Male

Six consecutive AIS pts b/w 04/6-16/20 at the Queen Square, London, UK COVID-19 confirmed by RT-PCR; Age 53-85 years; 5/6 males

Time COVID Sx to Stroke Sx: 8-24 days but preceded by 2 days in 1 case

All six pts had LVO with D-dimer levels ≥1000 (range, 1,080->80,000)

All six pts had elevated ferritin and LDH levels

Lupus anticoagulant positive in 5/6 pts but Anticardiolipin and Anti-β2-glycoprotein-1 negative in 5/6

Three pts had multi-territory infarcts

Two had concurrent venous thrombosis

In two ischemic strokes occurred despite therapeutic anticoagulation

Our data cannot confirm a causal relationship between SARS-CoV-2 and ischaemic stroke, since competing vascular risk factors and mechanisms were present in most patients; four of six had hypertension, and two had AF.
Thrombosis Research 191 (2020) 9 – 14

Venous and arterial thromboembolic complications in COVID-19 patients admitted to an academic hospital in Milan, Italy

Corrado Lodigiani\textsuperscript{a,h}, Giacomo Iapichino\textsuperscript{c}, Luca Carengo\textsuperscript{c}, Maurizio Cecconi\textsuperscript{b,c}, Paola Ferrazzi\textsuperscript{a}, Tim Sebastian\textsuperscript{c,d}, Nils Kucher\textsuperscript{d}, Jan-Dirk Stuhti, Clara Sacco\textsuperscript{a}, Bertuzzi Alexia\textsuperscript{f}, Maria Teresa Sandri\textsuperscript{g}, Stefano Barco\textsuperscript{d,h}, on behalf of the Humanitas COVID-19 Task Force

388 patients (median age 66 years, 68% men, 16% ICU) consecutive symptomatic pts with laboratory-proven COVID-19 admitted to a university hospital in Milan, Italy

Table 3
Venous and arterial thromboembolic events in hospitalized COVID-19 patients.

<table>
<thead>
<tr>
<th>Thromboembolic events</th>
<th>Intensive care unit</th>
<th>General ward</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>% of closed cases</td>
<td>% of imaging tests performed*</td>
</tr>
<tr>
<td>At least one thromboembolic event</td>
<td>8</td>
<td>16.7% (95%CI 8.7–29.6%)</td>
<td>–</td>
</tr>
<tr>
<td>VTE</td>
<td>4</td>
<td>8.3%</td>
<td>22%</td>
</tr>
<tr>
<td>PE (± DVT)</td>
<td>2</td>
<td>4.2%</td>
<td>25%</td>
</tr>
<tr>
<td>Isolated pDVT</td>
<td>1</td>
<td>2.1%</td>
<td>7%</td>
</tr>
<tr>
<td>Isolated dDVT</td>
<td>0</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Catheter-related DVT</td>
<td>1</td>
<td>2.1%</td>
<td>50%</td>
</tr>
<tr>
<td>Ischemic stroke</td>
<td>3</td>
<td>6.3%</td>
<td>–</td>
</tr>
<tr>
<td>ACS/MI</td>
<td>1</td>
<td>2.1%</td>
<td>–</td>
</tr>
</tbody>
</table>

ACS, acute coronary syndrome; DVT, deep vein thrombosis; MI, myocardial infarction; pDVT, proximal deep vein thrombosis; dDVT, distal DVT; PE, pulmonary embolism; VTE, venous thromboembolism.

Overall: 9/362 (2.5%)
ICU: 3/48 (6.3%)
Incidence of thrombotic complications in critically ill ICU patients with COVID-19

F.A. Klok\textsuperscript{a,*}, M.J.H.A. Kruip\textsuperscript{b}, N.J.M. van der Meer\textsuperscript{c}, M.S. Arbous\textsuperscript{d}, D.A.M.P.J. Gomers\textsuperscript{e}, K.M. Kant\textsuperscript{f}, F.H.J. Kaptein\textsuperscript{a}, J. van Paassen\textsuperscript{d}, M.A.M. Stals\textsuperscript{a}, M.V. Huisman\textsuperscript{a,1}, H. Endeman\textsuperscript{e,1}

184 consecutive ICU patients (mean age 64 years, 76% men, median duration 7 days [IQR 1-13]) with laboratory-proven COVID-19 pneumonia in 3 Dutch Hospitals.

<table>
<thead>
<tr>
<th>Type of event</th>
<th>Number of cases</th>
<th>Relevant details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary embolism</td>
<td>25</td>
<td>– 18 cases with at least PE in segmental arteries, 7 cases PE limited to subsegmental arteries</td>
</tr>
<tr>
<td>Other venous thromboembolic events</td>
<td>3</td>
<td>– 1 proximal deep-vein thrombosis of the leg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>– 2 catheter related upper extremity thrombosis</td>
</tr>
<tr>
<td>Arterial thrombotic events</td>
<td>3</td>
<td>– All ischemic strokes</td>
</tr>
</tbody>
</table>

Note: acute pulmonary embolism was diagnosed with CT-pulmonary angiography, deep vein thrombosis/upper extremity vein thrombosis was diagnosed with ultrasonography, strokes were diagnosed with CT.

**ICU: 3/184 (1.6%)**
31 y/o man flu-like sx: later SAH from a ruptured PICA aneurysm

62 y/o woman no COVID sx: AIS due to LMCA occlusion s/p MT with massive hemorrhagic conversion requiring a decompressive hemicraniectomy 10 days later

Both patients’ CSF repeatedly negative on real-time PCR analysis despite concurrent neurological disease

? Underlying inflammatory and hypercoagulable state may incite cerebrovascular disease without disruption of the blood–brain barrier
COVID-19: Stroke as a Disease Modifier
Cerebrovascular disease is associated with an increased disease severity in patients with Coronavirus Disease 2019 (COVID-19): A pooled analysis of published literature

Gaurav Aggarwal, Giuseppe Lippi and Brandon Michael Henry

<table>
<thead>
<tr>
<th>Study</th>
<th>Sample size</th>
<th>Outcomes</th>
<th>Severe patients/ craveators</th>
<th>Non-severe patients/ craveators</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gent et al.1</td>
<td>1099</td>
<td>Admission to ICU, PM, Death</td>
<td>173 (15.7%) 52 (49-65) 42%</td>
<td>4 (2.3%) 90 (89.3%) 45 (34-57) 42%</td>
</tr>
<tr>
<td>Qin et al.2</td>
<td>452</td>
<td>Respiratory distress/ insufficiency</td>
<td>206 (43.5%) 61 (51-49) 45.8%</td>
<td>8 (2.3%) 166 (36.7%) 53 (41.25-62) 51.8%</td>
</tr>
<tr>
<td>Ruan et al.3</td>
<td>150</td>
<td>Death</td>
<td>68 (45.3%) 65 (15-81) 28%</td>
<td>7 (17%) 81 (54.6%) 50 (46-81) 33%</td>
</tr>
<tr>
<td>Wang et al.4</td>
<td>138</td>
<td>Clinical variables, PM, death</td>
<td>36 (26.1%) 66 (57-70) 39%</td>
<td>6 ( 4.7%) 107 (79.9%) 51 (37-62) 48%</td>
</tr>
<tr>
<td>Yang et al.5</td>
<td>52</td>
<td>Death</td>
<td>32 (61.5%) 64 (11-2) 34%</td>
<td>7 (22%) 20 (39.3%) 51 (12-9) 30%</td>
</tr>
<tr>
<td>Zhang et al.6</td>
<td>140</td>
<td>Respiratory distress/ insufficiency</td>
<td>58 (41.4%) 64 (25-87) 43%</td>
<td>2 (2.3%) 81 (58.6%) 55 (26-78) 54%</td>
</tr>
</tbody>
</table>

*Age data presented as median (Q3-Q1) or mean (SD); PM: mechanical ventilation; ICU: intensive care unit; NR: not reported.

Figure 1. Results of meta-analysis showing association of cerebrovascular disease with severity of disease and mortality in patients with COVID-1.

Conclusion: There is a ~2.5-fold increase in odds of severe COVID-19 illness with a history of cerebrovascular disease.
A More Definite Problem: The COVID-19 Collateral Damage on the Non-COVID-19 Stroke Care...
COVID-19 in Stroke Care: USA
Artificial intelligence recruited to find clues about COVID-19

by Gopal Ratnam
Methodology

Data downloaded from Viz Analytics™, a dynamic, live clinical intelligence software package, were analyzed from suspected stroke patients from 38 US hospitals. Number of patients, scans, age, and LVO alerts were analyzed over a period of 25 weeks, between November 4, 2019 - April 26, 2020. CT Perfusion core was measured by rCBF < 30%.

The weekly mean number of CTAs, LVOs and core volume, and median age, were plotted versus time, relative to the incidence of COVID cases. The collection period was divided into pre and post Covid period, defined as before March 1st 2020, and after March 1st, 2020.

P values were calculated to compare means of pre and post Covid period, with significance demonstrated at <0.05.
COVID-19 in Stroke Care: USA

**Summary Statistics**

**Study period:** November 4th, 2019 - April 26, 2020

Total Number of Patients: 75,079  
Total number CTAs in study: 11,801  
Total Number of CTPs in study: 3,616  
Total Number and % of LVOs: 1,594, 13%

Median Age for the Total Population: 63 +/- 18  
Median Age for the LVO Population: 67 +/- 16  
Average Core Volume for the Total Population: 9 +/- 28  
Average Core for the LVO Population: 22 +/- 40
National CTA v LVO

Nationally: Significant Drop in CTAs. LVO volume steady

Mean CTA/wk: 532.6
35% Reduction
P=0.0003

Mean CTA/wk: 410

Mean CTA/wk: 69.8
P=0.069

Mean CTA/wk: 59.1

# CTAs performed  Number of LVOs  # Covid deaths
National Median Age of LVO

Trend in Median Age

Median Age LVO: 69.8 yrs

Median Age LVO: 66.5 yrs

P=0.0137
National Av Core Volume

Mean Core Volume: 20.9 cc

Mean Core Volume: 27.6 cc

P=0.107
COVID-19 in Stroke Care: Italy
Compared with the same period in 2019, we have observed a half of minor strokes, TIAs, and transfers from spokes, along with longer onset-to-door and door-to-treatment times for major strokes.

- Intravenous thrombolysis: decreased by 26%
- Bridging therapy (combined IVT and MT): decreased by 30%
- Primary MT: increased by 41% - “most of these patients had very serious strokes arriving late, sometimes too late”
Piacenza province (about 280,000 inhabitants) - one of the epicenters of the Italian epidemic, listing 2,276 cases at the time of writing.

Over the past 5 years (2015–2019): annual average of 612 new cases of ischemic stroke, with a monthly average of 51 cases, 21% LVOS.

Between February 21, 2020 (first SARS-CoV-2 patient recorded in Italy – in Codogno, a nearby city), and March 25, 2020, there were only 6 admissions from the Casualty Department for ischemic stroke (2 transient ischemic attacks, 1 cardioembolic LVO, and 3 lacunar stroke).
COVID-19 in Stroke Care: Barcelona

Courtesy of Marc Ribo, MD
Comparación de la letalidad del Covid-19 en las diferentes regiones más afectadas

El número de fallecidos por Covid-19 desde el primer día en que se registraron 10 muertes

Lombardia  Catalunya  Madrid  Hubei  Nueva York

El salto final de la curva de Hubei se debe a un cambio en el recuento de fallecidos en China

Fuente: John Hopkins, Ministerio de Sanidad, Dipartimento della Protezione Civile

LA VANGUARDIA
Evolució de pacients COVID-19

Evolució diària de casos COVID-19 a l'Hospital Universitari Vall Hebron. Talls de 0 a 24h

Data |
--- |
24/03 |
25/03 |
26/03 |
27/03 |
28/03 |
29/03 |
30/03 |
01/04 |
02/04 |
03/04 |
04/04 |
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06/04 |
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18/04 |
19/04 |
20/04 |
21/04 |

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<th>Ingressats a crítics</th>
<th>Ingressos nous</th>
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<th>Derivacions altres centres URG</th>
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<td>317</td>
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<td>13/04</td>
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<td>277</td>
<td>260</td>
<td>247</td>
<td>252</td>
</tr>
<tr>
<td>14/04</td>
<td>294</td>
<td>277</td>
<td>260</td>
<td>247</td>
<td>252</td>
<td>237</td>
</tr>
</tbody>
</table>

En cas que un pacient, en un mateix dia, hagi sigut traslladat entre diferents UTs (Planta / UClús) pot comptabilitzar-se per duplicat. Talls de dia de 0 a 24h
Patients admitted for a suspected stroke in 2020: 147 (44%)
Vs 2019: 185 (56%)
NIHSS on admission

Lines show Median
Arrival to the Hospital

Own means (p=0.025)
Stroke type

%LVO


p=0.08


%TIA


p=0.04
Los infartados están llegando tarde por miedo a ir al hospital

Cardiólogos de diez centros catalanes piden a sus enfermos que no esperen.

“Aquí gente está pasando sus infartos en casa por miedo al coronavirus”, alerta Antoni Bayés, responsable de ambulancias de Emergencias Mediques en el Hospital Vall d’Hebron.

Natàlia Pérez de la Ossa: "Gran part de la población amb síntomas lleves de malalties que no són el coronavirus s'està quedant a casa. Això ens preocupa"
LOWER NUMBER OF ADMISSIONS
BUT
SIMILAR NUMBER OF REPERFUSION TREATMENTS
ASPECTS on admission

Bar chart showing the percentage of ASPECTS<6 for COVID-19 patients compared to non-COVID patients. The chart indicates a significant increase in the percentage of ASPECTS<6 for COVID-19 patients in 2020 compared to 2019.
EVT: LTSW TO GROIN

**Left Diagram**
- **Y-axis**: LTSW + GROIN
- **X-axis**: Cohort
- **Years**: 2019, 2020
- **Data Points**: 250, 350

**Right Diagram**
- **Y-axis**: LTSW + GROIN
- **X-axis**: Cohort
- **Years**: 2019, 2020
- **Data Points**: 250, 350, 370

Bars show Median.
24 NIHSS

\[ p = 0.05 \]
A binary logistic regression analysis adjusted by age and NIHSS score showed 2 independent predictors of early mortality:

Patients in 2020-C (OR 3.1 CI 1.1-8.8 p=0.03)

SARS-CoV-2 infection (OR 7.1 CI 1.8-29, p<0.01)
First COVID+ EVT: March 26

March 26 – April 9:
16 EVT (8 COVID+ 50%)
   (5 died first days due to respiratory failure)
SICH: 0%

7 IV tPA Tx in COVID+: 0% SICH
The COVID-19 Collateral Damage to Stroke Care:

• The pandemic is a major healthcare disaster with significant impact on stroke care (and likely other cardiovascular emergencies)

• High complexity:
  • home confinement and the fear of hospital consultation
  • Impact of Pre- and Intra-Hospital Work-Flow
  • Potential greater severity of stroke in COVID+ patients

We Must Also Focus on the COVID-19 Impact on Non-COVID Diseases...
3. Inform the emergency medical system and the public that these centers will be protected and will remain fully operational even during crises.
4. Improve education of health professionals and the public, especially those who are at high risk of stroke, to recognize stroke and call emergency medical services immediately to be taken to one of the designated stroke centers so as to avoid significant delay in transferring patient from one hospital to the other.
Procedural Aspects: How Much PPE?
215 pregnant women on delivery unit

Four women (1.9%) had fever or other symptoms of Covid-19 on admission, and all 4 women tested positive for SARS-CoV-2.

Nasopharyngeal swabs were obtained from 210 women with no Covid-19 symptoms, 29 (13.7%) were positive for SARS-CoV-2. One (3.4%) positive patient developed COVID-19 fever.

88% of SARS-CoV-2 positive patients on admission had no symptoms at presentation!
89 SNF Residents

64% (n=57, age, 78.6±9.5y) tested positive for SARS-CoV-2 either during the point-prevalence surveys, clinical evaluation, or postmortem examination

76 residents participated in the first point-prevalence survey:
- 35% (17): typical symptoms
- 8% (4): only atypical symptoms
- 56% (27): no new symptoms
  - 89% (24) new symptoms within 7 (median, 4 [IQR, 3-5]) days

56% of SARS-CoV-2 positive patients on first survey had no symptoms at that time!
In conclusion, we have estimated that viral shedding of patients with laboratory-confirmed COVID-19 peaked on or before symptom onset, and a substantial proportion of transmission probably occurred before first symptoms in the index case.
Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV-2)

Ruiyun Li1*, Sen Pei2*,†, Bin Chen3*, Yimeng Song4, Tao Zhang5, Wan Yang6, Jeffrey Shaman2†

Estimation of the prevalence and contagiousness of undocumented novel coronavirus [severe acute respiratory syndrome–coronavirus 2 (SARS-CoV-2)] infections is critical for understanding the overall prevalence and pandemic potential of this disease. Here, we use observations of reported infection within China, in conjunction with mobility data, a networked dynamic metapopulation model, and Bayesian inference, to infer critical epidemiological characteristics associated with SARS-CoV-2, including the fraction of undocumented infections and their contagiousness. We estimate that 86% of all infections were undocumented [95% credible interval (CI): 82–90%] before the 23 January 2020 travel restrictions. The transmission rate of undocumented infections per person was 55% the transmission rate of documented infections (95% CI: 46–62%), yet, because of their greater numbers, undocumented infections were the source of 79% of the documented cases. These findings explain the rapid geographic spread of SARS-CoV-2 and indicate that containment of this virus will be particularly challenging.

Wölfel R¹, Corman VM², Guggemos W³, Seilmaier M³, Zange S¹, Müller MA², Niemeyer D², Jones TC²,⁴, Vollmar P¹, Rothe C⁵, Hoelscher M⁵, Bleicker T², Brünink S², Schneider J², Ehmann R¹, Zwirglmaier K¹, Drosten C⁶, Wendtner C⁷.

+ Author information

Abstract
Coronavirus disease 2019 (COVID-19) is an acute infection of the respiratory tract that emerged in late 2019¹,². Initial outbreaks in China involved 13.8% of cases with severe courses, and 6.1% of cases with critical courses³. This severe presentation may result from the virus using a virus receptor that is expressed predominantly in the lung²,⁴; the same receptor tropism is thought to have determined the pathogenicity-but also aided in the control-of severe acute respiratory syndrome (SARS) in 2003⁵. However, there are reports of cases of COVID-19 in which the patient shows mild upper respiratory tract symptoms, which suggests the potential for pre- or oligosymptomatic transmission⁶-⁸. There is an urgent need for information on virus replication, immunity and infectivity in specific sites of the body. Here we report a detailed virological analysis of nine cases of COVID-19 that provides proof of active virus replication in tissues of the upper respiratory tract. Pharyngeal virus shedding was very high during the first week of symptoms, with a peak at 7.11 × 10⁸ RNA copies per throat swab on day 4. Infectious virus was readily isolated from samples derived from the throat or lung, but not from stool samples-in spite of high concentrations of virus RNA. Blood and urine samples never yielded virus. Active replication in the throat was confirmed by the presence of viral replicative RNA intermediates in the throat samples. We consistently detected sequence-distinct virus populations in throat and lung samples from one patient, proving independent replication. The shedding of viral RNA from sputum outlasted the end of symptoms. Seroconversion occurred after 7 days in 50% of patients (and by day 14 in all patients), but was not followed by a rapid decline in viral load. COVID-19 can present as a mild illness of the upper respiratory tract. The confirmation of active virus replication in the upper respiratory tract has implications for the containment of COVID-19.
Infection Among Hospital Staff in Barcelona

<table>
<thead>
<tr>
<th>Role</th>
<th>Total Staff</th>
<th># COVID+</th>
<th>% COVID+</th>
</tr>
</thead>
<tbody>
<tr>
<td>MD</td>
<td>1348</td>
<td>71</td>
<td>5.3%</td>
</tr>
<tr>
<td>Nurse</td>
<td>1745</td>
<td>261</td>
<td>14.9%</td>
</tr>
<tr>
<td>Nurse Aid</td>
<td>1042</td>
<td>143</td>
<td>13.7%</td>
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<tr>
<td>Transport</td>
<td>417</td>
<td>34</td>
<td>8.2%</td>
</tr>
<tr>
<td>Residents</td>
<td>579</td>
<td>30</td>
<td>5.2%</td>
</tr>
<tr>
<td>Technician</td>
<td>649</td>
<td>33</td>
<td>5.1%</td>
</tr>
<tr>
<td>Administration</td>
<td>622</td>
<td>30</td>
<td>4.8%</td>
</tr>
<tr>
<td>Others</td>
<td>119</td>
<td>17</td>
<td>14.2%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>6731</strong></td>
<td><strong>619</strong></td>
<td><strong>9.2%</strong></td>
</tr>
</tbody>
</table>
Procedural Aspects: How Much PPE?

- History is limited in the setting of Acute Stroke
- High Rates of Asymptomatic and Atypical Patients (≈ 55-90%)
- Higher viral shedding in the upper (vs. lower) respiratory tract vs. SARS and Influenza
- Early and Pre-Symptomatic Spread (≈45-55%)

Do Not Trust COVID-19 Screening to Make Intra-Procedural Decisions Regarding PPE!
We are the Gladiators in this Battle – A Gladiator would never go to a fight without a strong shield....
Mechanical thrombectomy in the era of the COVID-19 pandemic.

Emergency preparedness for neuroscience teams.

A guidance statement from the Society of Vascular & Interventional Neurology

Cover Title: Mechanical thrombectomy in the era of the COVID-19

Thanh N. Nguyen, MD FRCPc,1 Mohamad Abdalkader, MD,2 Tudor G. Jovin, MD,3 Raul G. Nogueira, MD,4 Ashutosh P. Jadhav, MD,5 Diogo C. Haussen, MD,4 Ameer E. Hassan, DO,6 Roberta Novakovic, MD,7 Sunil A. Sheth, MD,8 Santiago Ortega-Gutierrez, MD,9 MSc, Peter D. Panagos, MD,10 Steve M. Cordina, MD,11 Italo Linfante, MD,12 Ossama Yassin Mansour, MD PhD,13 Amer M. Malik, MD, MBA14 Sandra Narayanan, MD,5 Hesham E. Masoud, MD,15 Sherry Hsiang-Yi Chou,5,16 MD, Rakesh Khatri, MD,17 Vallabh Janardhan, MD,18 Dileep R. Yavagal, MD,14 Osama O. Zaidat, MD,19 David M. Greer, MD,1 David S. Liebeskind, MD.20
Every acute stroke patient (direct presenting to ED or in transfer) should be triaged for symptoms and signs of COVID-19, including potential contact.

If there is positive screening for COVID-19, this patient should wear a surgical mask and immediately be placed in a negative pressure room in the ED.

If unknown assume it is a possible case!

Downsize stroke team and minimize exposure

PPE including full sleeved gown, surgical mask, eye protection (face shield or equivalent) and gloves.
Pre-hospital and Emergency Department care of acute LVO

• Personal protection should be escalated to N95 mask, hair cap, double gloves, shoe covers in the setting of contact with confirmed COVID-19 patient and/or aerosolizing events such as coughing, sneezing, nebulizer treatment, suctioning, nasogastric tube placement, bag mask ventilation, CPR, and intubation.

• A dedicated CT scan room for COVID-19 patients should be established if multiple CT rooms are available.

• As it would minimize exposure to emergency department and CT suite personnel a “direct to the angiography suite” approach should be considered for stable transferred patients with stroke symptoms onset within 24 hours, particularly if the time from the outside hospital imaging to arrival is less than two hours and CT ASPECTS is $\geq$ 7.
Airway Management for MT

• The anesthesiologist should be alerted early of a COVID-19 or suspect patient.
• Local policies for intubation and general anesthesia versus conscious sedation differ at different centers – not the best time for changes!
• If appropriate, consider conscious sedation as first-line:
  • to protect anesthesiologists from exposure
  • to protect our patients from unnecessary intubation
  • conserving mechanical ventilator and ICU resources.
• Converting a patient from conscious sedation to general anesthesia in the middle of the procedure in the angiography suite should be avoided due to high risk of aerosolization in a positive pressure room.
• **Converting** a patient from conscious sedation to general anesthesia in the middle of the procedure in the angiography suite **should be avoided** due to high risk of aerosolization in a positive pressure room.

• Early and controlled intubation is preferred if:
  • Acute respiratory distress/ hypoxemia/ high oxygen requirement
  • Unable to protect their airway/ low GCS (<9)
  • Agitation, uncooperative
  • Active vomiting
  • Active cough

• **These are not isolated reasons to intubate on our opinion:**
  • Dominant cerebral hemisphere occlusions
  • Aphasic patients
  • High NIHSS (>15)
General Endotracheal Anesthesia

• Only essential personnel
• Avoid high flow pre-oxygenation
• Rapid sequence induction using video-laryngoscopy (most experienced person available to intubate)
• Vasopressors immediately available.
• Maintain SBP >140mmHg, SpO₂ >94%, normocarbia
• HEPA (High Efficiency Particulate Air) filter on ETT and CO₂ sampling line
• Avoid circuit disconnections
• Extubate preferably in a negative pressure location avoiding coughing
General Endotracheal Anesthesia

- An “aerosol box” can be used as a cover as an additional measure of PPE protection during intubation.
Monitored Anesthesia Care

- Patient should wear surgical mask
- Avoid high flow nasal cannula oxygen
- Careful titration of sedation to avoid oro- or nasopharyngeal airway insertion or chin lift/jaw thrust.
- Consider use of expiratory viral filter on oxygen masks.
SNACC Consensus Statement:

Anesthetic Management of Endovascular Treatment of Acute Ischemic Stroke During COVID-19 Pandemic: Consensus Statement from Society for Neuroscience in Anesthesiology & Critical Care (SNACC)

Endorsed by Society for Vascular & Interventional Neurology (SVIN), Society for NeuroInterventional Surgery (SNIS), Neurocritical Care Society (NCS), and European Society of Minimally Invasive Neurological Therapy (ESMINIT) and American Association of Neurological Surgeons (AANS) and Congress of Neurological Surgeons (CNS) Cerebrovascular Section

Deepak Sharma, MD, DM1 Mads Rasmussen, MD, PhD2 Ruquan Han, MD, PhD3 Matthew Whalin, MD, PhD4 Melinda Davis, BMed, FANZCAe Andrew Kofke, MD MBA FCCM FNCSf Lakshmkumar Venkatraghvan MDg Radoslav Raychev MD, FAHA8 Justin F. Fraser, MD, FAANS, FAHA9

Society of NeuroInterventional Surgery recommendations for the care of emergent neurointerventional patients in the setting of covid-19

Justin F Fraser •,1 Adam S Arthur •,2,3 Michael Chen,4 Michael Levitt,5 J Mocco,6 Felipe C Albuquerque,7 Sameer A Ansari,8 Guilherme Dabus,9 Mahesh V Jayaraman,10 William J Mack,11 James Milburn,12 Maxim Mokin •,13 Sandra Narayanan,14 Ajit S Puri,15 Adnan H Siddiqui •,16,17 Jenny P Tsai18 Richard P Kluczni9

• Authors have reported to us, and we have determined, that they have no relationships relevant to the content of this document to disclose.
UNDOCUMENTED COVID STATUS

Screening for fever and respiratory symptoms should be part of the screening of all potential neurointerventional patients. Intubation of these patients prior to transportation to the angiography suite should be considered, especially in patients with risk factors for intra-procedural intubation as noted above. Given that thrombectomy is such a time-sensitive procedure, that family members are often not available to provide a complete medical history, and that a neurologically impaired patient may not be able to answer screening questions, it is recommended that patients of unknown COVID status be treated as high risk for COVID-positive (see above), provided institutional resources are available.

DOCUMENTED COVID-POSITIVE STATUS

Patients with COVID-positive documentation (or those presumed positive; see below) should be treated with maximum safety precautions. Intubation, extubation, suction, and active CPR may result in aerosolization of respiratory secretions, increasing the risk of exposure to personnel. Intubated patients pose less of a transmission risk to neurointerventional staff given that their ventilation is managed through a closed circuit. Nonetheless, disruption of the circuit (such as for a cuff leak, suctioning, endotracheal tube manipulation) can release additional aerosolized secretions. Therefore, we recommend standard institutional protocols with a low threshold for intubation of stroke thrombectomy COVID-19-positive patients prior to transport to the angiography suite, ideally in a negative pressure environment. For instance, patients with dominant hemisphere occlusions, very high National Institutes of Health Stroke Scale score or a low Glasgow Coma Scale score, or posterior circulation occlusions (as well as any patient with significant symptomatic respiratory difficulty) should be considered for prophylactic intubation as
The threshold for tracheal intubation will need to be altered by the situation presented and is likely to be impacted by availability of equipment and personnel. In general, the threshold for the use of GA for EVT may be reduced during COVID-19 pandemic. If the anesthesiologist has any concerns for possible urgent conversion from MAC to GA during EVT, it is advisable to start with GA. However, not all patients undergoing EVT need to be intubated solely for the purpose of reducing the risk to healthcare personnel. In fact, intubation may increase the risk of aerosolization and hence, the exposure.

My Suggestions:
Should Ischemic Stroke Patients with Aphasia or High National Institutes of Health Stroke Scale Score Undergo Preprocedural Intubation and Endovascular Treatment?

Ameer E. Hassan, DO,*† Malik M. Adil, MD,* Haralabos Zacharatos, DO,* Basit Rahim, MD,* Saqib A. Chaudhry, MD,* Wondwossen G. Tekle, MD,† and Adnan I. Qureshi, MD*

Abstract

BACKGROUND: Presence of aphasia or severe neurologic deficits is considered an indication for preprocedural intubation (PPI) for endovascular treatment (ET) in acute ischemic stroke patients. We determined the feasibility, technical success rates, and outcomes of ET without PPI in 2 groups of patients: those with aphasia and those with an admission NIHSS score of 20 or more.

METHODS: The rates of intraprocedural intubation (IPI), good functional outcome at discharge (modified Rankin Scale score of 0-2), mortality, and intracerebral hemorrhage (ICH) were compared between those who did or did not undergo PPI in the above-mentioned patient groups.

RESULTS: A total of 60 (50%) of 120 patients with aphasia underwent ET without PPI; 6 of 60 patients required IPI. The odds of any ICH (odds ratio [OR] 6.3) and in-hospital mortality (OR 9.3) were significantly higher in those undergoing PPI. In the second analysis, 36 (39%) of 93 patients with an NIHSS score of 20 or more underwent ET without PPI; 6 of 57 patients required IPI. The risk of any ICH (OR 7.6) and in-hospital mortality (OR 5.0) was higher among patients who underwent PPI. The rates of good outcome at discharge were significantly lower among patients with aphasia (OR .1, 95% confidence interval [CI] .04-.2) or those with an NIHSS score of 20 or more (OR .07, 95% CI .005-.9) with PPI compared with those without PPI.

CONCLUSIONS: Despite the risk of IPI, patients with aphasia or an admission NIHSS score of 20 or more who underwent ET with PPI had lower rates of good outcomes and higher rates of ICH and death.

Aphasia (n=60): 10% (n=6) Conversions to GA
NIHSS ≥20 (n=36): ? Conversions Rate
Good outcomes at discharge lower with GA (OR .07, P=0.04)
Good outcome at discharge only 1 of 6 patients who converted
Agitated Confusional States in Patients With Right Hemisphere Infarctions

JAMES W. SCHMIDLEY, M.D., AND ROBERT O. MESSING, M.D.

SUMMARY Patients with infarctions in the territory of the right middle cerebral artery (RMCA) sometimes present with an agitated confusional state. We reviewed clinical data on 46 patients with RMCA infarcts and compared neurologic findings in patients with and without agitated confusion. Neither of the two patients presenting with agitated confusion showed obvious localizing neurologic signs; subtle motor, visual field and sensory deficits referable to the infarcted regions were present, but difficult to elicit because of the mental state. In contrast, all but one of the patients without agitated confusion had prominent motor and sensory signs. Infarction of the RMCA territory may cause agitated confusion in patients without prominent localizing signs; the initial neurologic findings may suggest a metabolic encephalopathy. However, the possibility of a cerebrovascular cause should not be dismissed in confused and agitated patients who have no definite lateralizing signs.

PATIENTS WITH INFARCTIONS in the territory of the right middle cerebral artery (RMCA) may present with an agitated confusional state and a paucity of lateralized deficits.1 We have encountered two such patients in three years. A detailed description of this syndrome has been published only once,1 and we were unable to find any information concerning the frequency of this presentation among patients with RMCA infarctions. We therefore undertook this study to ascertain how common this presentation was, and to determine whether there were any clinical features distinguishing those patients presenting with agitated confusion from other patients with RMCA territory infarctions.

Methods

We reviewed the records of patients with RMCA strokes who were seen by the Neurology Services of San Francisco General and University of California, Moffitt Hospitals, between July 1, 1979 and June 30, 1982. Patients with coma, metabolic derangement, sepsis, preexisting dementia, or other conditions capable of causing an abnormal mental state were excluded from consideration, as were those with frank intracerebral hemorrhage on computed tomographic (CT) scan, and those whose CT scan and examination indicated lacunar infarction.2 During the period of the study, a CT scan was a standard part of the investigation of all patients with unexplained confusion. Forty-six patients fulfilled these criteria. Orientation and level of consciousness were recorded in all cases. An agitated confusional state was defined by the presence of disorientation, distractibility, agitation, impaired cognition and perceptual errors (illusions, delusions or hallucinations). CT scans confirmed the presence of cerebral cortical infarction in 35 patients. Of the remaining 11 patients, four had only deep cerebral infarctions on CT scan and seven showed no lesion on CT scan (in six of these seven, CT scans were done within 24 hours of onset of neurologic symptoms). All 11, however, had sensory deficits suggesting parietal cortical infarction, such as agraphesthesia, astereognosis, extinction to double simultaneous stimulation, impaired sensory localization, unilateral neglect, and anosognosia. They were therefore included in the study. Two patients with infarctions in the right internal carotid artery (RICA) distribution were grouped with RMCA stroke patients.

Results

The two patients who presented with an agitated confusional state are described briefly. Both were seen

\[\text{Stroke Oct 15, No 5, 1984}\]

\[\text{From the Department of Neurology, School of Medicine, University of California, San Francisco, California 94143}\]

\[\text{Address correspondence to Dr. James Schmidley, Department of Neurology, School of Medicine, University of California, San Francisco, California 94143}\]

\[\text{Received November 21, 1983; accepted February 22, 1984.}\]
Grady Memorial Hospital  2010 to 2/18/2020

ALL patients: 2127
  • MAC: 1651
  • GA: 476
  • Converted: 26 (1.22%)

Anterior circulation: 1849
  • MAC: 1499
  • GA: 350
  • Converted: 22

Posterior circulation 278
  • MAC: 152
  • GA: 126
  • Converted: 4

Anterior Circulation stroke
  • Left sided lesion: 1003
  • MAC: 788
  • GA: 215
  • Converted: 10

All NIHSS > 6 and anterior circulation : 1728
  • MAC 1393
  • GA: 335
  • Converted: 19 (1.36%)

Left side lesion and NIHSS > 6: 935
  • MAC: 734
  • GA: 201
  • Converted: 7 (0.95%)

Anterior circulation with NIHSS > 15: 1058
  • MAC: 801
  • GA: 257
  • Converted: 10 (1.24%)

Left sided with NIHSS > 15: 669
  • MAC: 498
  • GA: 171
  • Converted: 4 (0.80%)
<table>
<thead>
<tr>
<th>Predictor</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>p</th>
<th>AUC</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.99</td>
<td>0.97 – 1.02</td>
<td>0.56</td>
<td>0.46</td>
<td>0.34 – 0.57</td>
</tr>
<tr>
<td>BMI (continuous)</td>
<td>0.99</td>
<td>0.94 – 1.06</td>
<td>0.93</td>
<td>0.48</td>
<td>0.35 – 0.61</td>
</tr>
<tr>
<td>BMI (&gt;30)</td>
<td>1.10</td>
<td>0.46 – 2.64</td>
<td>0.84</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>NIHSS (continuous)</td>
<td>0.96</td>
<td>0.90 – 1.02</td>
<td>0.18</td>
<td>0.42</td>
<td>0.29 – 0.55</td>
</tr>
<tr>
<td>NIHSS (&gt;15)</td>
<td>0.62</td>
<td>0.28 – 1.36</td>
<td>0.23</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Echo EF</td>
<td>1.004</td>
<td>0.98 – 1.03</td>
<td>0.77</td>
<td>0.53</td>
<td>0.42 – 0.64</td>
</tr>
<tr>
<td>Location</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right vs Left</td>
<td>1.48</td>
<td>0.62 – 3.53</td>
<td>0.38</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Basilar vs Left</td>
<td>2.73</td>
<td>0.90 – 8.27</td>
<td>0.08</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
The summary is that only 19 of 1,393 patients (1.36%) with anterior circulation strokes and NIHSS $\geq 6$ were converted from MAC to GA.

Neither left-sided occlusion or NIHSS $>15$ seem to be reasonable predictors for conversion.

In fact, not even their combination seems to mean much.
Anesthesia Management for MT:

• Patient-Centric: Workflow, Times, Drop in Blood Pressure

• Consider that intubation will often be expected to be done at the a negative pressure room in the ED which will increase the risks to the ED personnel and overload them even more.... Similarly, for extubation will expose ICU personnel and take ICU resources.

Do you best to avoid conversions but stick with what you know best...
Angiography Room Post Treatment Care
Reviewing Our Objectives

• COVID-19: Neurological Manifestations and Stroke
  • WHO Concept of Infodemic's
  • Data on Neurological Diseases and Stroke
  • Potential Mechanisms for Stroke
    • Neurotropism
    • Clotting Disorder

• A More Definite Problem: The COVID-19 Collateral Damage on the Non-COVID-19 Stroke Care

• Rationale for Standard Protection
  • Asymptomatic Patients
  • Pre-symptomatic Spread

• Anesthesia: A Change is just Different not Necessary Better...
  • Understanding the Pre-COVID data: Multicentric Non-Anesthesia Trials vs Monocentric Anesthesia Trials – Stick with what you do well...
  • ? Predictors of MAC to GA Conversion
  • Best practices

• Post-treatment Care – Now :)
When neurointerventionists take a selfie...
When neurointerventionists take a selfie...

Thank you
COVID-19 Associated Coagulation Disorders

K Kathleen W. Chester, PharmD, BCCCP, BCGP
Olivia J. Morgan, PharmD, BCCCP, BCGP
Marcus Stroke & Neuroscience Center
Grady Health System

The opinions expressed during this webinar are those of the speaker and do not necessarily reflect the opinions, recommendations or guidance of American Heart Association. For more information about AHA, visit heart.org.
Disclosure Statement

The following individuals have nothing to disclose concerning possible financial or personal relationships with commercial entities (or their competitors) that may be referenced in this presentation:

– Katleen W. Chester, PharmD, BCCCP, BCGP
– Olivia J. Morgan, PharmD, BCCCP, BCGP

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COVID-19 Associated Coagulopathy (CAC) Presentation Outline

- Epidemiology and pathogenesis
- Clinical/Laboratory presentation
- Prophylaxis
- Treatment

CACD = COVID-19 Associated Coagulation Disorder
Earliest Report of Coagulopathy


Elevations in D-Dimer and reductions in platelet count in majority with severe infection

Severity of COVID-19 Infection

- Asymptomatic infections
- Mild infections
- Moderate
- Severe
- Death
Severe COVID-19 and VTE

**China**
N = 81

- 20 (25 %) DVT only
- No baseline chemoprophylaxis
- PE incidence unreported
- Routine VTE screening

**Netherlands**
N = 184

- 50 (27 %) VTE
- Prophylactic doses lower than standard in some patients
- Evaluated on clinical suspicion
- PE (n= 25, 13.5 %)

**France**
N = 26

- 18 (69 %) VTE
- Admission prophylaxis or anticoagulation
- Routine VTE screening
- PE (n= 6, 23 %)

Cui et al. 2020. doi: 10.1111/JTH.14830
Pathogenesis

Viral entry, replication, and ACE2 down-regulation

SARS-CoV-2 spike protein binding to ACE2

Local or systemic infection or sepsis

Angiotensin-(1–9) → Angiotensin I → Angiotensin II

ACE inhibitors

ACE

ARBs

Angiotensin II type 1 receptor

Acute lung injury
Adverse myocardial remodeling
Vasoconstriction
Vascular permeability

Physiol Rev 98: 505–553, 2018
Mechanisms of Thrombosis

Hypercoagulable

Immobility
Hypoxia
Inflammatory cytokines
Activation of coagulation
Angiotensin II
Upregulation of plasminogen activator inhibitor (PAI-1)
Release of tissue factor
Autoimmune mechanisms

Endothelial injury/dysfunction
Stasis
### Laboratory Findings

<table>
<thead>
<tr>
<th>Abnormalities</th>
<th>Disease Severity</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Lymphopenia</td>
<td>- D-Dimer*</td>
<td>- D-Dimer*</td>
</tr>
<tr>
<td>- ↑ CRP</td>
<td>- Thrombocytopenia</td>
<td>- Thrombocytopenia</td>
</tr>
<tr>
<td>- ↑ Ferritin</td>
<td>- PT</td>
<td>- PT</td>
</tr>
<tr>
<td>- ↑ FDP</td>
<td>- IL-6</td>
<td></td>
</tr>
<tr>
<td>- ↑ aPTT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Fibrinogen</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Thrombocytopenia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ↑ D-dimer</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ↑ PT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ↑ IL-6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

D- dimer most strongly and consistently associated with disease severity and mortality.

Ciu et al. 2020. doi: 10.1111/JTH.14830
## Laboratory Findings

<table>
<thead>
<tr>
<th></th>
<th>Non-survivor (n = 54)</th>
<th>Survivor (n = 137)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prothrombin Time (PT) &gt; 16s</td>
<td>7 (13%)</td>
<td>4/128 (3 %)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>D-Dimer &gt; 1000 ng/mL</td>
<td>44 (81 %)</td>
<td>28/118 (24 %)</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Non-survivor (n = 54)</th>
<th>Survivor (n = 137)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelets &lt; 100 x 10⁹/L</td>
<td>11 (20%)</td>
<td>2 (1 %)</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Platelets count, x 10⁹/L</td>
<td>165.5 (107 – 229)</td>
<td>220 (168 – 271)</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

Zhou F, et al. Lancet. 2020. DOI: [https://doi.org/10.1016/S0140-6736(20)30566-3](https://doi.org/10.1016/S0140-6736(20)30566-3)
Laboratory Findings
D-Dimer and Fibrinogen

# Comparison of CAC with SIC and DIC

<table>
<thead>
<tr>
<th>Item</th>
<th>Score</th>
<th>SIC Range</th>
<th>DIC Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count (-10⁹/L)</td>
<td>2</td>
<td>&lt; 100</td>
<td>&lt; 50</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>100 to &lt; 150</td>
<td>50 - 99</td>
</tr>
<tr>
<td>FDP / D-Dimer</td>
<td>3</td>
<td>-</td>
<td>Strong increase</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-</td>
<td>Moderate increase</td>
</tr>
<tr>
<td>PT Prolongation (INR for SIC)</td>
<td>2</td>
<td>&gt; 1.4</td>
<td>≥ 6 s</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>&gt; 1.2 to 1.4</td>
<td>3 to 6 s</td>
</tr>
<tr>
<td>Fibrinogen (g/mL)</td>
<td>1</td>
<td>-</td>
<td>&lt; 100</td>
</tr>
<tr>
<td>SOFA score</td>
<td>2</td>
<td>≥ 2</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Total score for DIC or SIC</td>
<td>≥ 4</td>
<td>≥ 5</td>
<td></td>
</tr>
</tbody>
</table>

CAC = COVID associated coagulopathy  
FDP = fibrin degradation products  
DIC = disseminated intravascular coagulopathy  
SIC = sepsis induced coagulopathy

Comparison of CAC with SIC and DIC

<table>
<thead>
<tr>
<th>Item</th>
<th>Score</th>
<th>SIC Range</th>
<th>DIC Range</th>
<th>CAC Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Platelet count (-10^9/L)</td>
<td>2</td>
<td>&lt; 100</td>
<td>&lt; 50</td>
<td>100 - 200</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>100 to &lt; 150</td>
<td>50 - 99</td>
<td></td>
</tr>
<tr>
<td>FDP / D-Dimer</td>
<td>3</td>
<td>-</td>
<td>Strong increase</td>
<td>D-dimer elevated (&gt;2000-3000)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>-</td>
<td>Moderate increase</td>
<td></td>
</tr>
<tr>
<td>PT prolongation (INR for SIC)</td>
<td>2</td>
<td>&gt; 1.4</td>
<td>≥ 6 s</td>
<td>Prolonged 3 - 6 s days 10, 14</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>&gt; 1.2 to 1.4</td>
<td>3 to 6 s</td>
<td></td>
</tr>
<tr>
<td>Fibrinogen (g/mL)</td>
<td>1</td>
<td>-</td>
<td>&lt; 100</td>
<td>Normal-elevated: typically &gt; 100</td>
</tr>
<tr>
<td>SOFA score</td>
<td>2</td>
<td>≥ 2</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Total score for DIC or SIC</td>
<td></td>
<td>≥ 4</td>
<td>≥ 5</td>
<td></td>
</tr>
</tbody>
</table>

CAC may mimic DIC, but atypical of SIC

- Less prominent thrombocytopenia
- Less consumption of coagulation factors
- Normal or increased fibrinogen

CAC = COVID associated coagulopathy
FDP = fibrin degradation products
DIC = disseminated intravascular coagulopathy
SIC = sepsis induced coagulopathy

CACD Laboratory Monitoring

Non-critically ill patients

- Daily CBC
- D-dimer

Critically-ill patients

- Daily CBC
- DIC panel
- [Bi-weekly MOCHA panel and PAI-1]

Markers of inflammation

- Consider IL-6 if significant change in clinical condition

DIC panel: PT, aPTT, fibrinogen, d-dimer; PAI-1: Plasminogen activator inhibitor-1
MOCHA: Markers of Coagulation and Hemostasis Activation Panel (Fibrinogen Activity, Prothrombin Fragment 1+2, Thrombin/Antithrombin Complex)

Heparin and SARS-CoV 2

Heparin reduces proinflammatory proteins such as IL-6

Heparin or LMWH mostly in prophylactic doses

Management Strategies for CACD

- Quick identification of patients at increased risk for thromboembolism
- Prompt initiation of VTE prophylaxis and frequent laboratory monitoring
- Escalation of prophylactic intensity in patients with more severe coagulopathy
- Escalation of anticoagulation in patients with suspected VTE
CACD Patient Stratification

All patients should receive anticoagulation unless contraindicated

**Group A**
- Patients who do not have a clear indication for full dose anticoagulation
- D-Dimer < 6 times ULN (< 3,000 ng/mL)

**Group B**
- Patients at increased risk for VTE
- D-Dimer > 6 times ULN (> 3,000 ng/mL)

**Group C**
- High suspicion or confirmed VTE

---

Thachil J. J Thromb Haemost. 2020. GHS Hematology
Group A: Prophylactic Anticoagulation

LMWH should be considered as first line agents in the absence of contraindications:

- Active bleeding
- Platelet count < 25 x 10⁹/L
- Severe renal impairment
- Invasive procedures within 12 hours

Enoxaparin doses up to 0.5 mg/kg SQ day may be considered

- On average, patients will require enoxaparin doses between 30 – 50 mg SQ daily
- Patients who cannot receive LMWH may get UFH 5000 units SQ 8 - 12 hours or SCDs

Routine VTE prophylaxis guidance in stroke patients should be observed

LMWH: Low Molecular Weight Heparin, UFH: Unfractionated Heparin, SCD: Sequential Compression Device

Elevation in d-dimer levels is a common finding in patients with COVID-19, and may correlate with detection of VTE

- Does not currently warrant routine investigation for acute VTE in absence of clinical manifestations or other supporting information

Clinicians are considering intermediate-dose anticoagulation in patients with d-dimer > 6 times ULN (> 3,000 ng/L)

Enoxaparin 1 mg/kg/day SQ to target Anti-Xa levels 0.3 - 0.5

- Anti-Xa levels should be checked 4 hrs after 3rd dose; recheck after 3rd dose with every dose change, or with significant change in clinical status
- Low intensity UFH infusions targeting similar anti-xa levels should be considered in patients unable to receive LMWH

GHS Hematology; Bikdeli B. J Am Coll Cardiol. 2020
Group C: Therapeutic Anticoagulation

Patients with confirmed or suspected VTE

Enoxaparin 1 mg/kg SQ Q12 hours should be considered first line to achieve Anti-Xa goal of 0.6 - 1

- High intensity UFH infusions targeting similar anti-xa levels should be considered in patients unable to receive LMWH

Heparin resistance has been reported due to reduced anti-thrombin levels and other procoagulant factors

- Failure to achieve goal Anti-Xa levels despite adequate doses (UFH > 35,000 units/ 24 hrs or LMWH > 300-500units/kg/ day) should prompt ordering an anti-thrombin level and hematology consult
- May warrant use of direct thrombin inhibitor (DTI) while inpatient and DOAC upon discharge

Patients with COVID-19 and an alternative indication for anticoagulation (atrial fibrillation, mechanical heart valve) should be converted to LMWH or UFH in the acute setting

LMWH: Low Molecular Weight Heparin, UFH: Unfractionated Heparin  
DOAC: Direct Oral Anticoagulant  
GHS Hematology; Bikdeli B. J Am Coll Cardiol. 2020
Transitioning Between Treatment Groups

**Group A**

- If daily d-dimer reports > 6 times ULN (> 3,000 ng/L)
- Trend levels and consider advancing to Group B

**Group B**

- If daily d-dimer reports < 6 times ULN (< 3,000 ng/L)
- Consider continuing Group B or deescalating to Group A if high bleeding risk
- Confirmed or clinical suspicion of VTE should advance patients to Group C

**Group C**

- Continue therapeutic anticoagulation

---

Bikdeli B. J Am Coll
COVID-19 Associated Coagulopathy (CAC)

LMWH prophylaxis may decrease thrombin generation

- Long acting antiplatelet therapies should be discontinued unless benefit outweighs risk

There is no evidence that correction of laboratory parameters with blood products will improve outcomes

In a patient with CAC who is actively bleeding:

- Transfuse platelets if the platelet count is less than $50 \times 10^9/L$
- Fresh frozen plasma if the INR is above 1.8
- Order fibrinogen concentrate or cryoprecipitate if the fibrinogen level is less than 1.5 g/L

The hemostatic effectiveness of tranexamic acid (TXA) is unknown in this setting and is not recommended
## Anticoagulant Dosing Considerations

### Obesity
- **Group A**
  - Enoxaparin 0.5 mg/kg SQ daily (Max: 80 mg SQ daily)
  - UFH SQ every 8 hours should be considered
- **Group B and C**
  - Consider enoxaparin 0.8 mg/kg/day to avoid supratherapeutic antithrombin III levels

### Pregnancy
- Concomitant hypercoagulable state
- Can contribute to elevated d-dimers
- Utilize clinical judgment when escalating anticoagulation
- LMWH is the drug of choice
- DOAC therapy has not been formally evaluated

### Heparin Induced Thrombocytopenia (HIT)
- Consider fondaparinux for prophylactic and full anticoagulation
- Requires adjustments for weight and renal impairment
- Alternatively, direct thrombin inhibitors can be considered
- Consider hematology consult

---

GHS Hematology; Bikdeli B. J Am Coll Cardiol. 2020
Tissue Plasminogen Activator (t-PA)

Proposed as a salvage treatment for COVID-19 patients with decompensating respiratory function when mechanical ventilation or extracorporeal membrane oxygenation (ECMO) is not available

Currently, there is limited clinical experience and no clinical trial data to promote routine use in COVID patients with acute respiratory distress syndrome (ARDS)
Discharge Considerations

Extended prophylaxis with LMWH or direct oral anticoagulants (DOACs) can reduce the risk of VTE

- Limited data supports use of anticoagulation at discharge in all patients admitted for COVID-19, but optimal duration is unknown

Patients with confirmed VTE should receive a minimum of 3 months of therapeutic anticoagulation

Decision to use thromboprophylaxis at discharge should consider the individual patient’s VTE risk factors

- Financial feasibility, compliance, laboratory monitoring, drug interactions, bleeding risk, immobility
- VKA or aspirin therapy can be considered in patients not appropriate for LMWH or DOAC therapy

LMWH: Low Molecular Weight Heparin, VTE: Venous Thromboembolism
VKA: Vitamin K Antagonist

GHS Hematology; Bikdeli B. J Am Coll Cardiol. 2020
With COVID-19, what we do today may be history tomorrow. We must continuously learn as we treat patients with this disease.
Select Resources

• American Heart Association/American Stroke Association
• International Society of Thrombosis and Hemostasis
• American Society of Hematology
• Journal of the American College of Cardiology
• National Blood Clot Alliance
• American Society of Health-System Pharmacists
QUESTIONS?

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Grady Health System
On behalf of the American Heart Association/American Stroke Association, thank you.

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