finished products from China such as nebulizers, high-filtration masks, and thermometers. Due to the shortage of products, goods are either unavailable in drug stores or are sold at high prices, beyond the budget of low- and middle-income households. Similarly, hand sanitizer is scarce in drug stores. Even though the government is educating people to use masks and hand sanitizer, the shortage of materials is a concern that must be dealt with swiftly.

The Indian government has implemented a strict and timely quarantine policy for returning workers, either in a hospital or at home. Violators are prosecuted by law, and adhering to strict discipline has become a crucial mandate. Furthermore, spraying alcohol on roads, vehicles, public trains, and personnel to disinfectant people has no value. Vast quantities of alcohol spray are detrimental to human health.[4] Health education must provide advice based on scientific evidence. The spread of unscientific information (eg, drinking cow urine to counter the coronavirus) must be totally stopped. Close monitoring to facilitate a better understanding of the epidemiology and transmission pattern of the SARS-CoV-2 virus across all states is vital. The government needs to consider the effectiveness of public health policies in terms of their social implications in practice.[5]

Both central and state governments across India have taken several scientific control measures to weed out the spread of the SARS-CoV-2 virus. The prime minster also initiated a disaster fund for the South Asian Association for Regional Cooperation (SAARC)

block nations to assist neighboring nations. Within India, several states have allocated a special fund to deal with the pandemic. On March 25, 2020, a total lockdown of all states across the nation was undertaken for 21 days to control the community spread of the virus. The World Health Organization has praised India's response. India should continue the massive efforts against the SARS-CoV-2 virus along with the already successful "Clean India" campaign promoted by the prime minister because adopting better hygiene may contribute to minimizing the spread of this dangerous pandemic.

References

1. Wang C, Horby PW, Hayden FG, Gao GF. A novel coronavirus outbreak of global health concern. *Lancet* 2020;395:470–473.
2. Advisory: travel and VISA restrictions related to COVID-19. India Bureau of Immigration website. https://boi.gov.in/content/advisory-travel-and-visa-restrictions-related-covid-19-0. Published 2020. Accessed April 17, 2020.
3. Chatterjee P. Indian pharma threatened by COVID-19 shutdowns in China. *Lancet* 2020;395:675.
4. Coronavirus disease (COVID-19) technical guidance: infection prevention and control. World Health Organization website. https://www.who.int/emergencies/diseases/novelcoronavirus-2019/technical-guidance/infection-prevention-and-control. Published 2020. Accessed April 17, 2020.
5. Heymann DL, Shindo N. WHO Scientific and Technical Advisory Group for Infectious Hazards. COVID-19: what is next for public health? *Lancet* 2020;395:542–545.

Could COVID-19 represent a negative prognostic factor in patients with stroke?

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To the Editor—Coronavirus infectious disease 2019 (COVID-19) is a highly contagious disease that has become a worldwide pandemic. Coronaviruses (CoVs), positive-stranded RNA viruses, are known to cause respiratory or intestinal infections in humans and animals.[1] Coronaviruses are known to affect the cardiovascular system.[2]

The SARS-CoV-2 virus uses the enzyme 2 receptor (ACE2) to gain entry into cells,[3] and these receptors have been revealed in the neuronal and glial cells of the human brain. Thus, they may be a potential target of SARS-CoV-2, which might explain the death of olfactory cells in patients with COVID-19.[1] CoVs can enter the central nervous system through 2 distinct pathways: retrograde neuronal diffusion or hematogenous diffusion. The spread of SARS-CoV-2 through the cribriform plaque of the ethmoid bone during an initial or subsequent infection phase can lead to brain involvement. In the systemic circulation, the presence of ACE2

receptors on both capillary and neuronal endothelial cells could be responsible for the subsequent spread and damage to the cerebral nervous system without substantial inflammation. The presence of CoVs in the cerebral nervous system has been confirmed in the cerebrospinal fluid and brain tissues of patients during autopsies.[4,5]

Several symptoms indicative of CNS involvement are present in approximately one-third of COVID-19 patients: dizziness, headache, impaired consciousness, ataxia, epilepsy, and acute cerebrovascular disease.[1] Changes in the coagulation system (ie, D-dimer and platelet abnormalities)[2,6] and in inflammatory biomarkers (eg, interleukin-6, C-reactive protein, and ferritin)[7] have been reported in COVID-19 patients. In patients with stroke, the presence of COVID-19 could be a potential extrinsic factor in the genesis or worsening of stroke. Infection or high levels of proinflammatory biomarkers indicate significantly increased risk of ischemic stroke, especially in the elderly.[8-10] The onset or worsening of a stroke in these patients could be caused either by direct damage of the CoVs on the nervous system and/or by an activation of the

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mechanisms of COVID-19 inflammation induced as well coagulation disorders. As the disease spreads and new evidence emerges, we need to identify the existence of additional pathophysiological mechanisms of stroke in COVID-19 patients. We should establish a prospective registry of these patients to better identify the factors most responsible for a possible greater onset or worse prognosis of stroke in these patients and to identify and/or predict a better or lesser response of these patients to thrombolytic treatments.

References

1. Jin H, Hong C, Chen S, Zhou Y, et al. Consensus for prevention and management of coronavirus disease 2019 (COVID-19) for neurologists. *Stroke Vasc Neurol* 2020 April 1 [Epub ahead of print]. doi: 10.1136/svn-2020-000382.
2. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential effects of coronaviruses on the cardiovascular system: a review. *JAMA Cardiol* 2020 Mar 27 [Epub ahead of print]. doi: 10.1001/jamacardio.2020.1286.
3. Baig AM, Khaleeq A, Ali U, Syeda H. Evidence of the COVID-19 virus targeting the CNS: tissue distribution, host-virus interaction, and proposed neurotropic mechanisms. *ACS Chem Neurosci* 2020;11:995–998.
4. Lau KK, Yu WC, Chu CM, et al. Possible central nervous system infection by SARS coronavirus. *Emerg Infect Dis* 2004;10:342–344.
5. Xu J, Zhong S, Liu J, et al. Detection of severe acute respiratory syndrome coronavirus in the brain: potential role of the chemokine Mig in pathogenesis. *Clin Infect Dis* 2005;41:1089–1096.
6. Tang N, Bai H, Chen X, Gong J, Li D, Sun Z. Anticoagulant treatment is associated with decreased mortality in severe coronavirus disease 2019 patients with coagulopathy. *J Thromb Haemost.* 2020 Mar 27 [Epub ahead of print]. doi: 10.1111/jth.14817.
7. Ruan Q, Yang K, Wang W, et al. Clinical predictors of mortality due to COVID-19 based on analysis of data o f 150 patients from Wuhan, China. *Intensive Care Med* 2020 Mar 3 [Epub ahead of print]. doi: 10.1007/s00134-020-05991-x.
8. Consoli D, Vidale S, Aguglia U, et al. Previous infection and the risk of ischaemic stroke in Italy: the IN2 study. *Eur J Neurol* 2015;22:514–519.
9. Siniscalchi A, Iannacchero R, Anticoli S, et al. Anti-inflammatory strategies in stroke: a potential therapeutic target. *Curr Vasc Pharmacol* 2016;14:98–105.
10. Siniscalchi A, Gallelli L, Malferrari G, et al. Cerebral stroke injury: the role of cytokines and brain inflammation. *J Basic Clin Physiol Pharmacol* 2014;25:131–137.

Early phases of COVID-19 management in a low-income country: Bangladesh

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To the Editor—The World Health Organization has emphasized the importance of diagnostic testing in tracking and managing COVID-19, and most high-income economies have adopted widespread population testing schemes. The United States now leads the way, with >370,000 tests performed as of March 26, 2020.[1] This level of testing starkly contrasts with low-income economies such as Bangladesh, where an almost contrarian strategy seems to have been adopted that is arguably masking the true national spread of the virus.

From the first reported case of COVID-19 in Bangladesh on March 8 until March 28, 1,068 samples were tested by the Institute of Epidemiology, Disease Control and Research (IEDCR) in Dhaka.[2] The IEDCR was the sole institute in Bangladesh with testing facilities for COVID-19 until March 26, when a second facility was given testing rights. Centralized testing in these underresourced public institutions has been unable to effectively respond to the wave of suspected COVID-19 patients.

Even at this initial stage with limited confirmed cases, busy telephone hotlines and lack of timely testing for symptomatic patients raised concerns regarding Bangladesh's preparedness. In addition, the Bangladesh government has not sought to proactively limit community transmission from primary cases thus far. With a population of 161 million and a total of 1,169 ICU beds,[3] this inadequate strategy could potentially devastate Bangladesh's health system with multiple outbreaks.

This risk is compounded by thousands of Bangladeshi workers returning from COVID-19–struck countries and poor adherence to self-quarantine recommendations due to limited education and monitoring mechanisms. This situation is particularly problematic for Bangladesh because a significant portion of returning workers (ie, significant sources of SARS-CoV-2) reside in rural areas outside Dhaka and thus carry the virus to some of the most vulnerable and ill-equipped communities. This situation was likely worsened by the government declaring a 10-day holiday without travel restrictions from March 26 to April 5, which encouraged millions of city workers to leave Dhaka and return to their rural communities.[4]