CASE REPORT

Non-Aneurysmal Subarachnoid Hemorrhage in COVID-19: A Case Report and Review of Literature

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Abstract

Background: Hemorrhagic cerebrovascular events, either due to aneurysmal rupture or spontaneous subarachnoid hemorrhage (SAH), are not rare in COVID-19. Several mechanisms such as coagulopathy, cytokine storm, viral endotheliopathy, hypertension, and immune modulation might play a role in the pathogenesis of SAH in COVID-19. This study aimed to report the first case of spontaneous non-aneurysmal SAH associated with SARS-CoV-2 from India. We briefly discussed the possible pathogenetic mechanisms underlying this process and succinctly reviewed the relevant literature.

Case report: We herein report a case of a non-comorbid young woman infected with SARS-CoV-2 presenting with thunderclap headache and eventually non-aneurysmal SAH, who recovered with conservative management.

Conclusion: Headache, although a very common clinical feature of COVID-19 itself, must be investigated in detail to identify alternate causes that may be life-threatening. This case also incites further enquiry into the possible pathogenic mechanisms of neurovascular complications in COVID-19.

Keywords: Neuro-COVID; subarachnoid hemorrhage; COVID-19; endotheliitis; coagulopathy.

1. Introduction

Owing to the abundance of medical literature, "Neuro-COVID" has now been accepted as a separate entity.[1] Both the central nervous system (CNS) and peripheral nervous system bear the impact of "Neuro-COVID".[1–7] Amongst the spectrum of involvement of CNS, hemorrhagic cerebrovascular events are not rare.[1,4] About 4.4% of COVID-19 patients suffer from intracranial hemorrhage, typically in the older age group, and in patients with comorbidities and on anticoagulation therapy.[8] Among these cases, almost 80% are caused by ischemic stroke.[9] Moreover, vascular events such as cerebral venous sinus thrombosis and reversal cerebral vasoconstriction syndrome have also been described. The thrombosis may be a consequence of cytokine storm or endothelial damage, leading to a state of hypercoagulability. [1, 5, 8]

Aneurysmal rupture-related or spontaneous subarachnoid hemorrhage (SAH) has been reported in association with severe acute respiratory syndrome coronavirus (SARS-CoV-2) infection.[10] Nevertheless, even in cases of non-aneurysmal SAH, whether an association with SARS-CoV-2 infection is causal or coincidental, remains elusive.[10,11] Several mechanisms, e.g., hypercytokinemia-related damage to vessel barrier, virus-mediated endotheliopathy, unpredictable hypertension , coagulopathy, and immune thrombocytopenia, among others, have been postulated to explain the pathogenesis of SAH in COVID-19.[12,13]

We herein report the first case of spontaneous non-aneurysmal SAH associated with SARS-CoV-2 from India who recovered with conservative management. Besides the addition of a new case to the tally of the "COVID-19 associated SAH" databox, we will also briefly discuss the possible pathogenetic mechanisms that might be underlying this process, and will succinctly review the relevant literature.

2. Case Report

A 35-year-old female without comorbidities presented with sudden onset thunderclap headache for the past 18 hours. She was a medical technologist by profession and she was not on anticoagulants. Before the onset of this severe headache, she had a mild fever for the last five days, which was associated with a sore throat, malaise, body ache, distressingly decreased smell, and taste sensations for which she was taking paracetamol on a required basis. On visiting emergency, her oropharyngeal and nasopharyngeal swab was tested for SARS-CoV-2 by real-time reverse transcriptase-polymerase chain reaction (RT-PCR), which came out to be positive.

She had a high-grade fever (39.8°C), tachycardia and was in extreme distress from the headache. Brief neurological examination revealed a Glasgow coma scale score of 12/15 and nuchal rigidity without Kernig's and Brudzinski's signs. Neuro-ophthalmological examination revealed minimal bilateral lateral rectus restriction and grade-1 papilloedema. No other focal neurological deficits were found.

She had mild respiratory distress (respiratory rate: 20/min), occasional dry cough, normal oxygen saturation in room air (SpO_2 96%), blood pressure and blood glucose. Neither she nor any of her family members reported any neurological illness. She was married, had a child of six years, and had no high-risk behaviours, strenuous exercise, recent trauma or drug therapy.

Complete blood cell count revealed neutrophilic leucocytosis, neutrophil to lymphocyte ratio of 3:1, raised erythrocyte sedimentation rate (78 mm/hour), C-reactive protein (56 mg/L), lactate dehydrogenase (560 U/L), ferritin (600 ng/mL) and high D-dimer (866 ng/mL) levels. Blood glucose, thyroid and renal function tests were normal. Liver function tests revealed mild elevation of transaminases (SGPT 80U/L, SGOT 60U/L). High-resolution computed tomography (CT) of the thorax revealed a severity score of 10/25. Abdominal ultrasound examination ruled out polycystic kidney disease; electrocardiogram and echocardiography were otherwise normal.

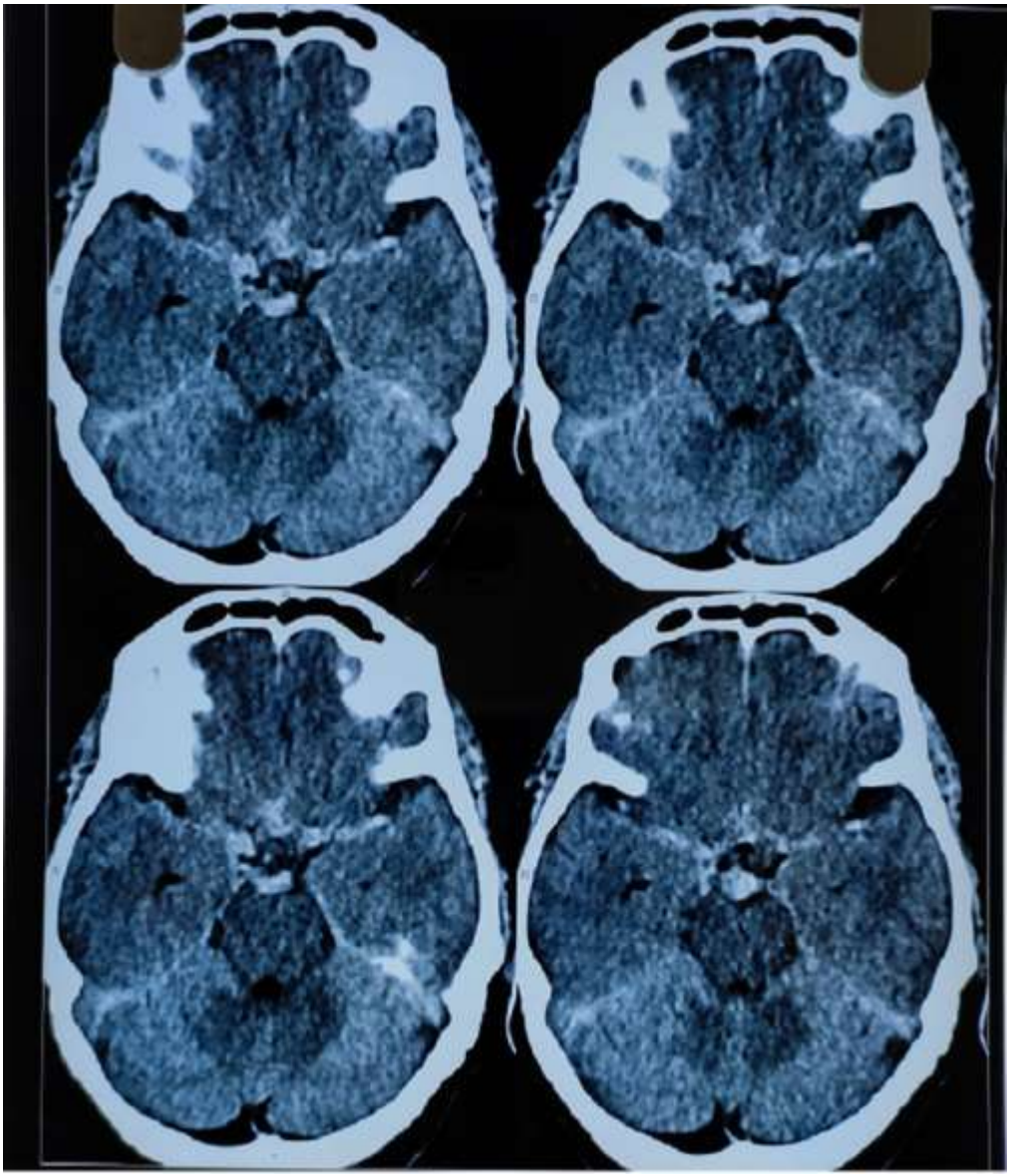

Figure 1: Non-contrast CT scan of the brain revealing hyperdense areas (blood density) involving basal cisterns and both sylvian fissures, suggestive of subarachnoid hemorrhage.

A non-contrast CT scan of the brain showed subarachnoid hemorrhage involving basal cisterns, interhemispheric fissure (feathery outlines) and both sylvian fissures. Due to infrastructural shortcomings, digital subtraction angiography (DSA) could not be performed within 48 hours, but CT-angiography of cerebral vessels could not demonstrate any evidence of cerebral aneurysms or arteriovenous malformations. Later, after discharge from the hospital (COVID-19 RT-PCR negative status), DSA was performed, which also failed to demonstrate any vascular pathology (Figure 2).

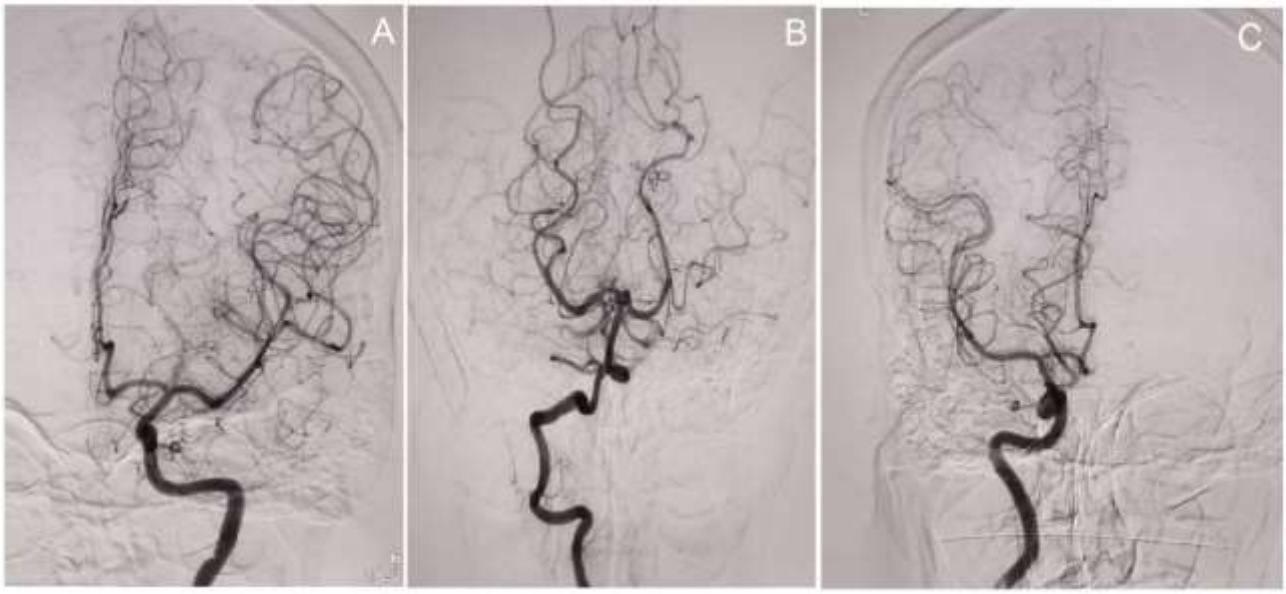

Figure 2: Digital subtraction angiography showing no abnormality.

She was kept under close monitoring of vital parameters and was put on maintenance intravenous fluids, intravenous acetaminophen for pain relief, nimodipine, remdesivir, methylprednisolone, and other supportive therapies. Her vital parameters, and serum electrolytes were being monitored closely to detect any early complications related to either SAH or COVID-19 itself. She was discharged home after three weeks, with good functional status (modified Rankin Scale score = 1).

3. Discussion

In spite of a significant decrease in mortality over the last two decades, SAH has remained a highly morbid neurological emergency and result in high "productive-years" loss and societal cost.[14] While the majority (85%) of the cases of non-traumatic SAH result from ruptured cerebral aneurysms, in about 10% of cases, no bleeding source can be identified. Arterial hypertension, smoking, excessive alcohol consumption, and recreational drug abuse are the most commonly associated modifiable risk factors for non-traumatic SAH.[14] Our patient did not have any single modifiable or non-modifiable risk factor except sex.

Table 1: Clinical characteristics of subarachnoid hemorrhage cases in COVID-19.

Age/Sex	Presentation	Brain imaging/Angiography	Treatment	Complications and outcome
50/man[11]	Weakness, dry cough and impaired consciousness	SAH in bilateral parasagittal areas originating from Willis polygon	Not reported	COVID-19 associated pneumonia; death
60/man[25]	Chest pain, headache and confusion	SAH with intraventricular hemorrhage	Blood pressure control / calcium channel blocker	Death
Two cases:[26] (1) 74/man; (2) 53/man	COVID-19 and type 1 respiratory failure	Case 1: multifocal small volume convexity SAH; normal magnetic resonance angiography Case 2: multifocal curvilinear foci of sulcal high density consistent with SAH	Supportive therapy	Ongoing rehabilitation
Case series (five cases – 3 men and 2 women)[27]	Neurologic symptoms (one), respiratory symptoms (three) and both in one	Case 1: subcortical hemorrhage and perimesencephalic cistern SAH Case 2: left fronto-parietal SAH Case 3: bilateral fronto-temporo-parietal SAH Case 4: diffuse SAH Case 5: left fronto-parietal SAH and multiple small embolic strokes Angiography unremarkable in three cases; right ICA occlusion and left ICA stenosis in one case; not reported in one case	Anticoagulants	Death (five cases)
9/man[28]	Cardiopulmonary arrest, low Glasgow coma scale, respiratory symptoms, abdominal pain, headache and fatigue	Hyperdensity in basal cisterns, and in interhemispheric and bilateral Sylvian fissures	Intravenous dopamine, fresh frozen plasma, meropenem, vancomycin, azithromycin, oseltamivir, levofloxacin, lopinavir/ritonavir, and hydroxychloroquine	Non-oliguric renal failure, brain edema; death
Case series (four cases – one man and three women)[10]	Fever, cough, anosmia, headache, dyspnea and myalgia	1) Fisher grade IV SAH and incipient hydrocephalus; DSA: ruptured saccular aneurysm in posterior communicating segment of right ICA 2) Fisher III SAH; DSA: vasospasms without aneurysms nor arteriovenous malformations Case 3: Fisher IV SAH; DSA: not reported Case 4: Fisher III SAH with acute interhemispheric acute subdural hemorrhage; normal DSA	Ceftriaxone, vancomycin, meropenem, azithromycin, oseltamivir, nimodipine, anti-vasospasm therapy and external ventricular drain	Rehabilitation (two), recovery (one) and death (one)
56/man[20]	Dyspnea and hypoxic respiratory failure	Small diffuse bilateral fronto-parietal and intraventricular hemorrhage	Unfractionated heparin and extracorporeal membrane oxygenation	Pulmonary embolism; discharged with stable hemodynamic and neurological status
66/woman[30]	Post-cardiac arrest with return of spontaneous circulation. One-week history of dry cough, shortness of breath and general malaise	Extensive SAH extending into the suprasellar cistern, Sylvian and interhemispheric fissures, effacement of fourth ventricle, and diffuse cerebral edema	Vancomycin, cefepime, mannitol, hypertonic saline, azithromycin and hydroxychloroquine	Death

Case series (four cases – two men and two women)[21]	Loss of consciousness, fever and dyspnea	Case 1: diffuse spontaneous SAH and intraventricular hemorrhage (Fisher IV). DSA: anterior communicating artery aneurysm Case 2: massive spontaneous SAH (Fisher V) Case 3: not reported Case 4: Fisher IV SAH; DSA: no aneurysm	Not reported	Death (three cases)
41/woman[17]	Respiratory failure secondary to interstitial pneumonia	Edema involving posterior body and splenium of corpus callosum and minimal parietal convexity SAH	Remdesivir	Oedema, acidosis and shock; death
75/woman[18]	Acute hypoxia	Large left temporal intraparenchymal hemorrhage, SAH and left acute/subacute subdural hemorrhage	Remdesivir, enoxaparin, vancomycin, ceftriaxone and milodrine	Death
68/woman[19]	Sudden-onset headache, fever and vomiting	SAH (Fisher II) from right posterior communicating aneurysm	Nimodipine, hydration, low-molecular-weight heparin and steroids	Recovery
64/man[29]	Sudden onset right-sided weakness, numbness and dysarthria	Acute infarct of left thalamus and left temporo-occipital lobe and a small SAH on left parietal sulcus	Aspirin	Recovery
21/woman[16]	Fever, headache and altered sensorium for five days	Subarachnoid bleed with diffuse edema	Hydroxychloroquine, oseltamivir, mannitol, hydrocortisone, vancomycin, and ceftriaxone	Death
54/woman[22]	Body ache, fever, and hypoxia.	Diffuse SAH with intraventricular extension and sulcal effacement	Aspirin, heparin, sarilumab, epoprostenol and desmopressin	Cerebral circulatory arrest; death
64/man[31]	Not reported	Multiple foci of supratentorial intraparenchymal hemorrhage in both hemispheres, SAH and subcortical microbleeds	Low-molecular-weight heparin	Recovery
Case series (three cases – one man and two women)[23]	Acute hypoxic respiratory failure	Case 1: acute SAH and intraparenchymal hemorrhage within posterior fossa Case 2: brain anoxia with scattered SAH, subdural hematoma and tonsillar herniation Case 3: large intraparenchymal hemorrhage and cerebral edema, with transtentorial herniation	Apixaban, unfractionated heparin and enoxaparin	Death
55/woman[24]	Acute respiratory distress. Sudden loss of consciousness with mydriasis and coma on day 10	Acute ischemia with wide hemorrhagic infarction in the absence of vascular malformation, and SAH on cerebellar tentorial margin	Tocilizumab and low-molecular-weight heparin	Death

Abbreviations: DSA=Digital subtraction angiography; ICA=Internal carotid artery; SAH=subarachnoid hemorrhage.

Albeit extremely rare, viral encephalitis can present with SAH.[15] Several reports of SAH in COVID-19 patients are present in the literature (summarized in Table 1).[10,11,16–31] SARS-CoV-2 may gain access to CNS via two principal routes, namely the hematogenous and the retrograde neuronal (axonal) dissemination. In the hematogenous entry, SARS-CoV-2 may enter the CNS through the blood-brain barrier (BBB) by any of the three mechanisms- (1) by direct entry into CNS via infection of the endothelial cells, (2) via angiotensin-converting enzyme-2 (ACE-2) receptors expressed on the endothelium, or (3) via migration by infecting the monocytes and macrophages.[1,2,4,10] In the neuronal entry, SARS-CoV-2 enters the CNS via the olfactory neurons through the nose.[1-4,10]

The exact pathogenetic mechanisms underlying spontaneous SAH in COVID-19 are not yet well understood. However, evidence points towards an inflammatory and hypercoagulative state, facilitated by SARS-CoV-2 induced endothelitiis and microvascular dysfunction (Figure 3). Indeed, COVID-19 infection may cause injury to the vascular endothelium, making them vulnerable to rupture, especially at a pre-existing susceptible site (e.g. aneurysm).[12] The endotheliopathy may be attributed to the arterial wall damage inflicted by the adhesion of leukocytes to the endothelium and the creation of a microenvironment characterised by an excess production of anti-proteases and free radicals. The endothelial damage may induce a state of hypercoagulation by Tissue Factor-VIIa pathway (by release of Tissue Factor from activated macrophages) and by the Intrinsic Pathway (by release of collagen from damaged endothelium and Factor-VIII/vWF from Weibel-Pallade Bodies).[11] This will, in turn, lead to an aggravated activation of the fibrinolytic system, thus increasing the bleeding risk. Furthermore, many of the patients, perhaps already having neuroendothelial dysfunction, are treated with anticoagulant therapy, which, combined with the effects of pre-existing comorbidities such as obesity and arterial hypertension, may lead to hemorrhage.[32,33] Our patient had both hyperinflammation and endotheliopathy, which were suggested by the elevated erythrocyte sedimentation rate, LDH, C-reactive protein and ferritin levels, as well as neutrophilic leukocytosis (a neutrophil-to-leukocyte ratio of 3:1) and absence of any aneurysm in the DSA.

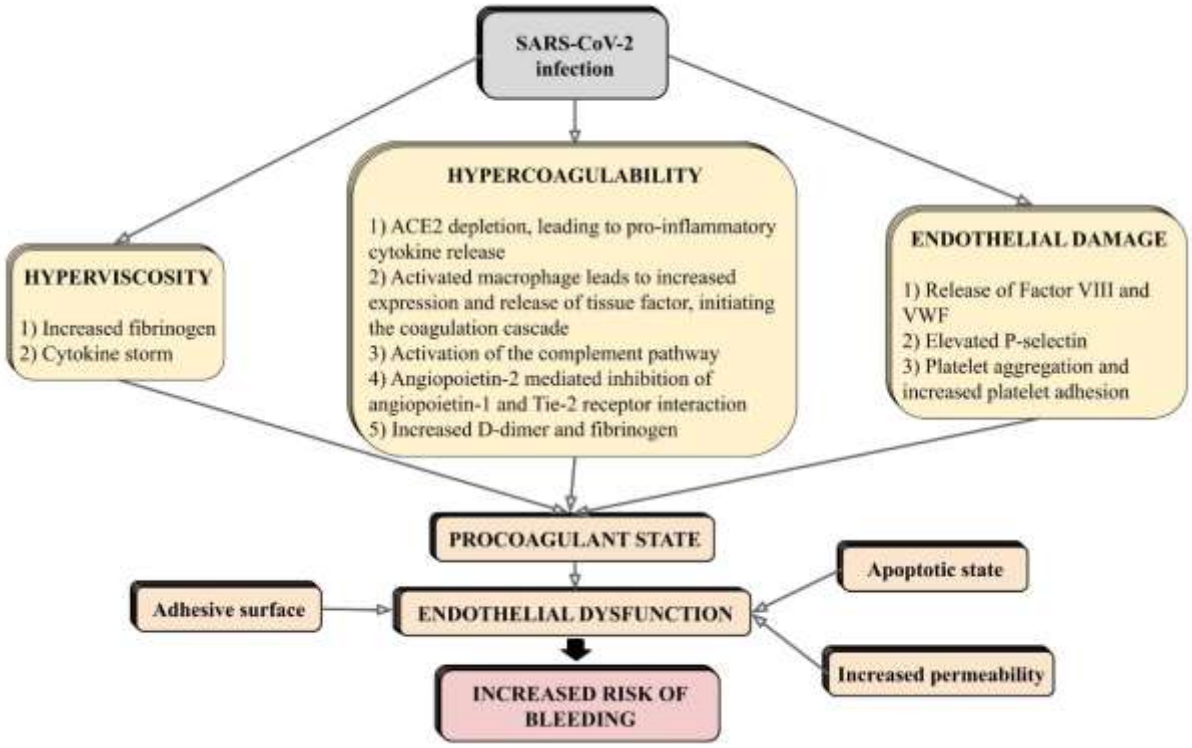

Figure 3: Potential mechanisms mediating increased risk of spontaneous subarachnoid hemorrhage in COVID-19. SARS-CoV-2 infection induces a prothrombotic state via hypercoagulability, hyperviscosity and endothelial damage. The underlying factors act in an interrelated manner to cause endothelial dysfunction. The increased P-selectin expression causes the endothelial surface to become adhesive. Diminished angiotensin-converting enzyme-2 expression causes angiotensin to increase, which in turn increases the permeability of the surface. The damaged endothelium itself releases angiopoietin-2 that antagonizes angiopoietin-1 by competitively binding to Tie-2, the latter of which takes part in anti-inflammatory and anti-apoptotic signalling pathways. These factors combined, increases the risk of spontaneous hemorrhage.

DSA remains the gold standard investigation for diagnosing vascular malformations resulting in SAH.[14] However, as previously mentioned, in about 10-15% of cases, DSA fails to delineate a definite culprit bleeder, especially in perimesencephalic SAH.[14] In our patient, both initial CT angiography and follow-up DSA were negative, similar to many previously reported cases.[10,11] Different factors may contribute to a 'negative DSA' which include blood in cisterns hiding the aneurysm, thrombus within the aneurysmal sac, proximal and distal vasospasm resulting in non-filling of an aneurysm, proximity of skull bone impairing the image, very small aneurysm, venous bleed, and a technically insufficient examination of the posterior circulation.[14] 'Negative DSA' in spontaneous SAH associated with COVID-19 further incites doubts as to whether the pathogenesis of SAH in these patients and the general population are similar or dissimilar.

Neurological manifestations are more common among severe COVID-19 patients than non-severe ones. Headache is a common symptom of SARS-CoV-2 infection with or even without (in most cases) true neuroinvasion.[1] In our case too, the patient presented with a headache. However, the features of the headache were highly

suggestive of an underlying serious neurological emergency. Similar thunderclap headache in a similar background of an admitted patient with SARS-CoV-2 infection can be expected in reversible cerebral vasoconstriction syndrome,[6] intracerebral hemorrhage with intraventricular extension,[32] cerebral venous sinus thrombosis,[5] lobar hemorrhage with stretching of meninges,[33] and rarely, pituitary apoplexy.[7]

The intentions of presenting this case are manifold. First, to the knowledge of the authors, this is the first case of spontaneous non-aneurysmal SAH associated with SARS-CoV-2 being reported from India. Secondly, this case incites further enquiry into possible pathogenetic mechanisms behind neurovascular manifestations of COVID-19. Thirdly, headache, although a very common clinical feature of COVID itself, must be investigated in detail to identify alternate causes that may be life threatening. However, more epidemiological and clinical studies are required to establish a correlation between SAH and COVID-19.

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Ethics: Informed consent was taken from the patient.

Authors contributions:
Conception and Organization of the project: RG, DR, AR, AM, SD, SKP, and JBL.
Execution: RG, DR, and AR.
Writing of the first draft: RG and AR.
Review and critique of the manuscript: DR, AM, SD, SKP and JBL.

Data availability: The data that support the findings of this study are available from the corresponding author, JB-L, upon reasonable request.

Conflict of interest/Disclosures:
Ritwik Ghosh has no potential conflicts to disclose.
Dipayan Roy has no potential conflicts to disclose.
Adrija Ray has no potential conflicts to disclose.
Amrita Mandal has no potential conflicts to disclose.
Shambaditya Das has no potential conflicts to disclose.
Shyamal Kanti Pal has no potential conflicts to disclose.
Julián Benito-León has no potential conflicts to disclose.

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