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Spontaneous subdural hematoma in a young COVID-19-confirmed patient without comorbidities: A case report

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Abstract

The majority of acute subdural hematomas are due to trauma, and cases of spontaneous subdural hematoma are rare. This report aims to provide an overview of subdural hematoma associated with COVID-19 virus. We described a case of a 22-year-old female without comorbidities and confirmed COVID-19 with spontaneous subdural hematoma on non-contrast computed tomography scan. This was the first case encountered in our hospital. There is no published cases yet in the Philippines. Mechanisms linking cerebrovascular events to COVID-19 are hypothesized. First, it has been postulated that COVID virus is neurotropic toward angiotensinogen-converting enzyme-2 receptors and it can invade and directly damage cerebral vessels. Second, entry of the virus into the cells results in marked reduction in angiotensinogen-converting enzyme-2 levels which could contribute to the development of intracranial hemorrhage. Third, COVID-19 patients usually develop a systemic hyperinflammatory syndrome characterized by fulminant hypercytokinemia which may mediate vascular remodeling and predispose to intracranial hemorrhage. COVID infection should be considered as one of the differentials in patients presenting with neurological symptom. More research needs to be performed to understand the pathogenic mechanisms behind each of these disorders and better treat such patients with suitable drugs in a timely manner.

Keywords

Subdural hematoma, COVID infection, central nervous system

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Introduction

There are various pathological processes that could lead to acute spontaneous subdural hematoma like hypertension, vascular malformations, hematological malignancies, infection, hypervitaminosis, coagulopathy, cocaine abuse, and alcoholism.[1] Clinical symptoms of subdural hematoma include vomiting, headache, conscious disturbance, visual impairment, and brain herniation.[2] Spontaneous subdural hematoma has not been previously associated with any viral infection such as COVID. Although COVID-19 is a respiratory disease, several studies have reported neurological complications in COVID-19 patients. This report aims to discuss the possible pathogenesis and mechanisms of cerebral vascular sequelae such as spontaneous subdural hematomas, probably secondary to COVID-19.

Case report

In late March 2021, a 22-year-old female, Filipino, was brought to the emergency room due to headache followed by loss of consciousness. The patient was apparently well until 1 day prior to admission, when she allegedly complained of headache, uncharacterized. No associated trauma, no cough, no vomiting, nausea, fever, diarrhea, body malaise,or weakness of the extremities. Patient self-medicated with Paracetamol tablet. Then 1 h prior to consult, patient sent a text message to her brother asking for help due to her headache. However, her brother found her unconscious, lying on the bed, hence she was immediately brought to the emergency room.

Past medical history of the patient was unremarkable. No history of taking antiplatelet or anticoagulant medications, and no previous bleeding problems. No illicit drug use and no alcohol abuse.

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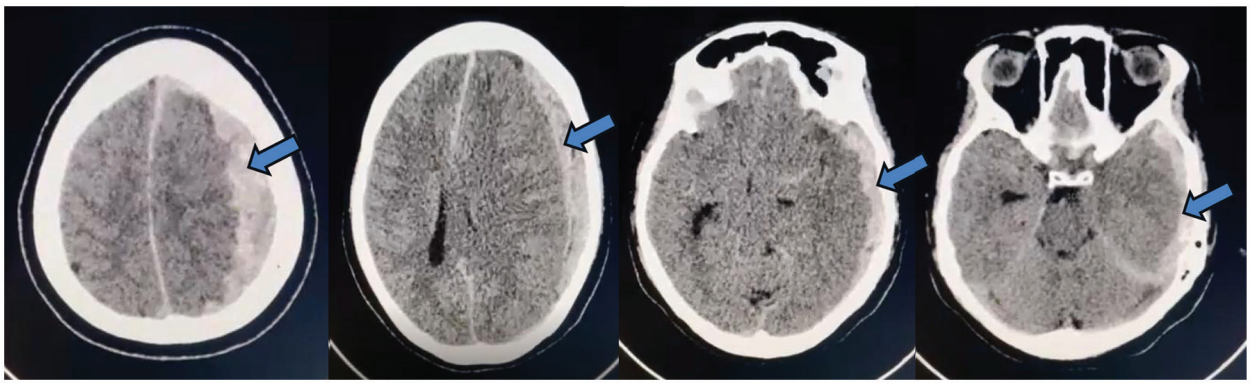

Figure 1. Cranial non-contrast computerized tomography scan of the patient on admission showing acute subdural hemorrhage on the left cerebral convexity (1.9 cm maximal thickness) with compression of the adjacent cortical sulci and left lateral ventricle (arrow). Rightward subfalcine herniation (1.2 cm) (http://patlog.qmmc.com/procedures.php).

Table 1. CBC, coagulation tests, and inflammatory markers of the patient done on admission.

Test	Result	Reference range
Prothrombin time	12.2 s	
International normalized ratio	0.93	
Partial thromboplastin time	27.10 s	
CBC	White blood cells 9.5×10^9/L	5–10
	Neutrophils 93	
	Lymphocytes 5 L	
	Hemoglobin 127 g/L	140–180
	Hematocrit 0.39 vol%	0.40–0.54
	Platelet 252×10^9/L	150–450
Lactate dehydrogenase	186.98 units/L	140–280
C-reactive protein	78.50 mg/L	<6
Ferritin	<6 mg/L	<6

CBC: complete blood count.

Patient's vital signs: BP 110/70, CR 64, RR 19, T 36.6, and SpO_2 98% at room air, with capillary blood glucose of 146 mg/dL. Patient was drowsy, disoriented, and inconsistently answered questions and followed commands appropriately. Pupils were anisocoric, 2 mm on the right and 4 mm on the left, equally reactive to light. Primary gaze was at midline. Patient had brisk corneal reflex, no facial asymmetry, with intact gross hearing, and withdrew to pain on all extremities.

Immediate non-contrast computerized tomography scan (Figure 1) revealed an acute subdural hemorrhage, left cerebral convexity (1.9 cm maximal thickness), with compression of the adjacent cortical sulci and left lateral ventricle, with rightward subfalcine herniation (1.2 cm). Computerized tomography angiography revealed no aneurysm or arteriovenous malformation.

Ideally, patient should have been screened for coagulation factors deficiency, but such tests were not readily available. Baseline blood counts, coagulation tests, and inflammatory markers were within normal (Table 1).

The patient was referred to neurosurgery service, and a left frontotemporoparietal craniotomy with evacuation of subdural hematoma was done. Decompressive hemicraniectomy was not done because brain parenchyma was not edematous on inspection post-evacuation of hematoma. No identifiable source of bleeding and no noted cerebral vessel malformation. Medical decompression was started after surgery. Postoperatively, patient became fully awake, oriented, followed command, answered appropriately without motor or sensory deficit. Patient's reverse transcription polymerase chain reaction tested positive for COVID-19, but chest X-ray was unremarkable, and she had no respiratory symptoms. Hence, the patient completed 14 days of quarantine in the hospital and was discharged without neurologic deficits.

Discussion

Prior to COVID-19 pandemic, Awaji et al. cited that spontaneous subdural hematoma accounts to 0.7%–6.7% of all acute subdural hematoma with mortality rate as high as

37.2%.[3] At the start of the pandemic, COVID-19 was considered to be a respiratory disease and neurological involvement was considered to be rare. However, a variety of cerebrovascular disorders has been recently reported, probably due to endothelial dysfunction or hypercoagulability. These disorders include ischemic stroke, intracerebral hemorrhage, and cerebral venous sinus thrombosis.[4] According to a study in Wuhan, neurological complications are around 36.4%, whereas another study from Spain reported neurological symptoms in 57.4% of the patients.[5] The most common neurological manifestations include headache, lethargy, unstable gait, ataxia, and seizures, in addition to peripheral nervous system manifestations such as loss of taste and smell, vision impairment, and nerve pain. The most serious developing neurological diseases include polyneuritis, Guillain–Barré syndrome, meningitis, encephalitis, and encephalopathy, in addition to cerebral hemorrhage and infarction.[6] The exact incidence of intracranial hemorrhage among patients with COVID-19 is still unknown as cases are rare, unlike that of ischemic stroke which occurs about 2.3% in a study based in Wuhan and 0.9% according to a study in New York.[1]

While the precise mechanisms linking cerebrovascular events to COVID-19 remain uncertain, possible hypotheses have been put forward. First, it has been postulated that COVID-19 virus is neurotropic toward angiotensinogen-converting enzyme (ACE-2) receptors present in the central nervous system and can invade and directly damage cerebral vessels. Second, entry of the virus into the cells results in marked reduction in ACE-2 levels which could contribute to the development of intracranial hemorrhage. This is supported by preclinical studies in which an inverse relationship between ACE-2 levels and the occurrence of hypertension has been observed. Vicenzi et al. in their study of 40 COVID-19 patients demonstrated significant rise in the systemic blood pressure with the deterioration in the pulmonary function, even in patients without prior hypertension. Third, a subset of COVID-19 patients usually develops a systemic hyperinflammatory syndrome characterized by fulminant hypercytokinemia which may mediate vascular remodeling and predispose to intracranial hemorrhage.[7]

But how does the virus set off a cascade of events that cause so much destruction to the blood vessels? COVID-19 infection affects the vascular system and blood's coagulation properties, injuring vascular walls and causing blood clots to form in both large and microscopic blood vessels. It affects the microcirculation, causing endothelial cell swelling and damage (endothelitis), microscopic blood clots (microthrombosis), capillary congestion, and damage to pericytes that are integral to capillary integrity and barrier function, tissue repair (angiogenesis), and scar formation.[8]

Gogia et al. reported the first case of COVID-19-associated hyperacute subdural hematoma along with extensive intracerebral hemorrhage and subarachnoid hemorrhage in a 75-year-old patient. This individual was on double antiplatelet (aspirin and clopidogrel) treatment for coronary artery disease.[5] Our patient was neither on antiplatelet nor on anticoagulant.

In a report of two cases of COVID-19-associated spontaneous subacute subdural hematoma by Tabibkhooei et al., the index patients were both elderly with respiratory symptoms and decline in consciousness: a 69-year-old man with hypertension, diabetes mellitus, and ischemic heart disease and an 84-year-old woman with hypertension.[9] In contrast, our patient was young, without respiratory symptoms, and with unremarkable medical and personal history that would have predisposed her into hemorrhage.

Conclusion

Neurological manifestations of COVID-19 are increasingly recognized, and spontaneous subdural hematoma is a rare but a part of a wide spectrum of neurological conditions. Interestingly, many COVID-19 cases may present with pure neurological manifestations including stroke, Guillain–Barré syndrome, and meningoencephalitis. Although the current case definition of COVID-19 illness includes a primary respiratory involvement, many cases have been reported with neurological illness being the earliest manifestation of COVID-19 or with sole neurologic symptoms only. Thus, COVID-19 infection should be considered in the list of differentials in cases with new-onset unexplained neurologic disease.

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

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