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Case Report

A case report of cerebral venous infarction due to venous sinus thrombosis as complication in a Covid-19 patient ☆,☆☆

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ABSTRACT

Coronavirus disease of 2019 (COVID-19) is a pandemic disease caused by a new corona virus known as SARS-CoV-2 (severe acute respiratory syndrome-coronavirus-2) and well known to increase the risk of developing venous thromboembolism; thus, patients with COVID-19 may present with neurological disorders. Venous thromboembolism is an important cause of morbidity and mortality in patients with COVID-19. We report a case of 35 years old woman who presented neurological disorders due to venous infarction and venous sinus thrombosis as complication in COVID-19. Clinicians and medical staff should be aware of neurologic symptoms and neurological deficit in background of COVID-19, which might even be the first presentation of this infection, thus prompting increased attention to disease presentation and early treatment with anticoagulation should be initiated in these settings. *

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Introduction

On 31 December 2019, the WHO China Country Office was informed a number of pneumonia of unknown etiology detected in Wuhan City, China, later called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. The neurological community were alerted to the high prevalence of anosmia and dysgeusia in early reports [2]. Severe neurological and neuropsychiatric presentations associated with COVID-19 have become increasingly apparent, including a patient with encephalitis in China in whom SARS-CoV-2 was identified in cerebrospinal fluid (CSF) [3]. COVID-19 infection was first recorded in Afghanistan in Herat province, in west region, neighboring to Iran [6]. As of April 22 2021, the Afghanistan Ministry of Public Health documented 58733 COVID-19 positive cases in the country [7].

Initially the mortality of this infection are related to severe acute respiratory distress syndrome, but later several publications showed that this infection caused an inflammatory response with severe systemic complications [8]. Venous thromboembolism has been shown to be an important cause of morbidity and mortality in patients with COVID-19, both in the general inpatient and in the intensive care unit (ICU) setting, and even in patients receiving therapeutic anticoagulation [9].

Case report

We report a case of cerebral venous infarction due to venous sinus thrombosis as complication in 35 years old female Covid-19 positive patient with no previous medical illness was admitted with shortness of breath, fever, dry cough and headache in Herat Regional Hospital, Herat, Afghanistan. She had no close contact with a COVID-19 patient. The patient had no history of drug and alcohol abuse and was a non-smoker.

Her vital signs including temperature of 39°C, a pulse rate of 105 beats per minute, a blood pressure of 120/80 mm Hg, a respiratory rate of 30 breaths per minute, and Oxygen saturation (SpO2) of 85 % while breathing ambient air and there was decreased air entry on the lung bases without any history of cardiovascular disease or respiratory disease. On physical abdomen examination her abdomen was soft with a palpable distended urinary bladder.

Non-contrast CT thorax recommended and *6 hours after admission* her CT scan done. Axial non-contrast CT thorax showed bilateral multilobar peripheral and subpleural consolidation indicative of COVID-19 [Fig. 1].

Also in this patient, A PCR nasal swab gave a positive result for the novel Corona virus (Covid-19).

Laboratory results showed high white blood cell count [14000/μL (reference range 4000-10,000/μL)]; hemoglobin [12.7 g/dL (reference range 12-16 g/dL)]; inflammatory marker C-reactive protein was elevated [73 mg/L) (reference range 0.0-8.0 mg/L)]; platelet [268,000/μL (reference range 150,000-350,000/μL)]; blood urea nitrogen [11 mg/dL (reference range 8-20 mg/dL); creatinine (serum) [0.9 mg/dL (reference range 0.7-1.3 mg/dL)]; sodium [140 mEq/L(reference range 136-145 meq/L)]; potassium [3.9 mEq/L (reference range 3.5-5.0 meq/L)] calcium [7.7 mg/dL (reference range 9-10.5 mg/dL)]; alkaline phosphatase was elevated [167 U/L (reference range 36-92 units/L)]; aspartate aminotransferase was elevated [53 U/L (reference rage 0-35 units/L)]; alanine aminotransferase [76U/L (reference rage 0-35 units/L)]; and negative troponin. [10]

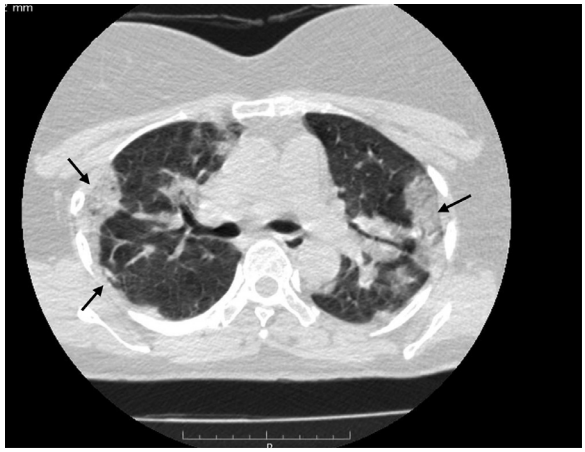

Fig. 1 – Chest CT image (lung window and axial view): *6 hours after admission*, showing bilateral areas of consolidation (arrows) in a peripheral distribution.

According to her laboratory data and imaging findings, she received inpatient medical and supportive treatment including supplemental oxygen for COVID-19 and her status improved, but soon after worsening dyspnea, palpitation, lethargy, confusion, blurring of vision, and generalized weakness and initially the cranial nerves examination was normal and her pupils were reactive to light and accommodation, subsequently complained of severe headache, unconsciousness and disoriented. Her serum glucose level at the time of unconsciousness was normal (110 mg/dL).

Subsequently after *3 days of admission*, CT examination of the brain was done and demonstrated hyperdensity within distal superior sagittal and right transvers sinus suggestive of venous sinus thrombosis, remainder of non-contrast imaging shows no significant brain abnormalities especially no mass lesion, infarct, hemorrhage, extra-axial collection or ventricular changes and left transverse sinus spared [Fig. 2].

Patient was admitted to the intensive care unit (ICU) and received heparin-based prophylaxis started as first-line treatment for dural venous thrombosis.

Over the next few days, the patient's mental status and consciousness progressively deteriorated, leading to a Glasgow Coma Scale (GCS) score of seven. Her neurological status exacerbated despite the improvement of oxygen level, clinical status, and radiological findings of lung involvement. She developed arrhythmia, confusion and new-onset hemiplegia.

Subsequently *7 days after admission* a non-contrast CT and CTA (Angiogram)/CTV (Venogram) scan showed multiple hyperdensity blurring the grey-white matter differentiation in the right frontal and both parietal lobes suggestive of venous sinus infarction hemorrhages [Fig. 3] on top of right transverse and superior sagittal sinuses thrombosis [Fig. 4] evidenced by showing filling defects within on CTA/CTV [Fig. 5].

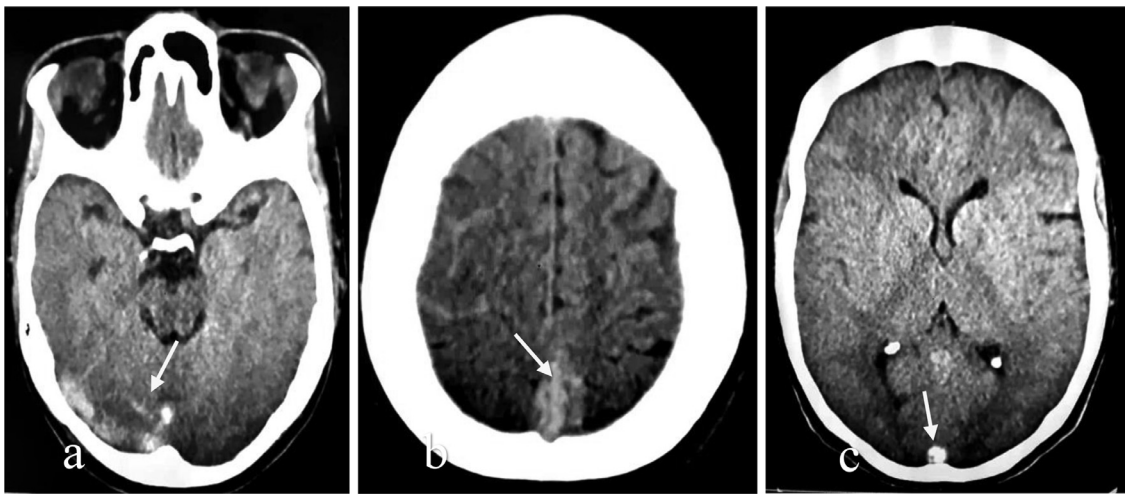

Fig. 2 – Non-enhanced CT brain images (axial view): *Three days after admission* showing areas of abnormal hyperattenuation in right transverse sinus (A), superior sagittal sinus (B, C) consistent with cerebral venous thrombosis (arrows), and no significant brain abnormalities especially no mass lesion, infarct, hemorrhage, extra-axial collection or ventricular changes and left transverse sinus spared.

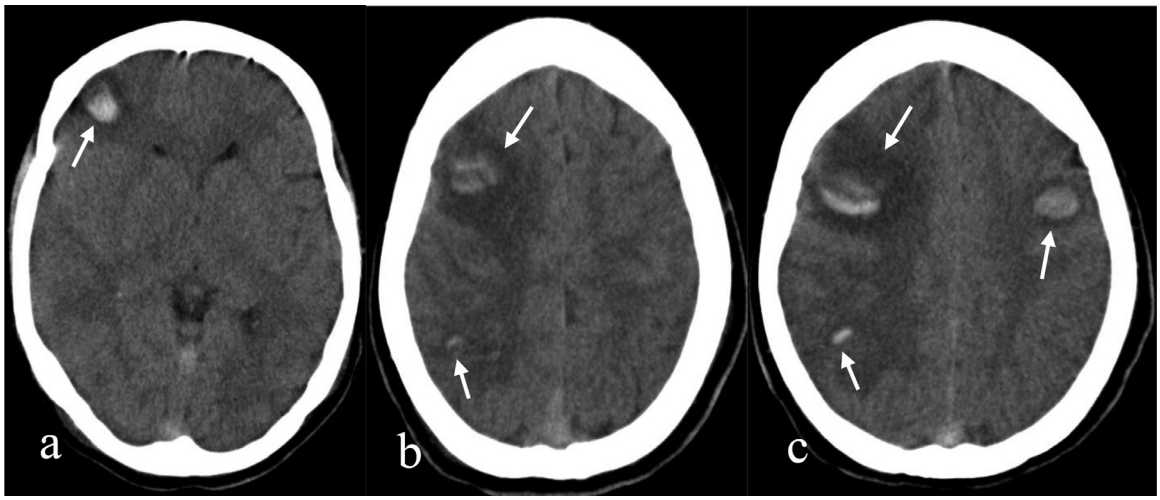

Fig. 3 – Non-enhanced CT brain images (axial view): *Seven days after admission* showing multiple areas of hypodensity blurring the gray-white matter differentiation in right frontal lobe (A) and both parietal regions (B, C) and contain parenchymal hemorrhages (white arrows).

Discussion

Covid-19, the disease caused by SARS-CoV-2, was recognized as a substantial global health emergency and SARS-CoV-2 was declared a pandemic on March 11, 2020 [1] and Covid-19 infection causing multiple organ injuries includes respiratory, gastrointestinal and renal system. However, nervous system involvement after infection with Covid-19 has been rare [11,12]. And a patient with acute necrotising encephalopathy in Japan [4] and cases of cerebrovascular disease [2,5]. There are different mechanisms for the etiology of neurologic injuries in the cases who suffer from viral infections such as COVID-19, including direct neuronal damage, extreme immune response through cytokine storm, unintended host immune response and the effects of systemic illness [11]. There are several studies suggested significantly increased risk of thromboembolism and venous and arterial thromboses in patients with COVID-19 despite administration of standard prophylaxis, especially in those patients requiring intensive care unit support [13-15].

In our case presented here, the occurrence of severe headache, unconsciousness and disoriented with chest imaging indicative of COVID-19 pneumonia suggests likely the central nervous system involvement by COVID-19. COVID-19 is usually diagnosed with the results of a real-time reverse transcription–polymerase chain reaction (RT-PCR) test of a nasopharyngeal specimen obtained with a swab. In the begin-

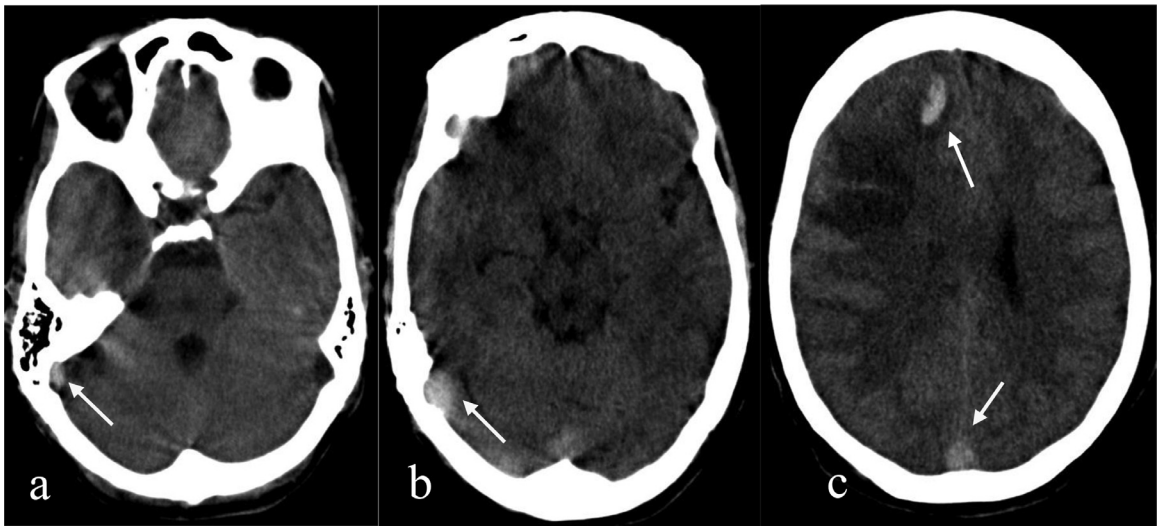

Fig. 4 – Non-enhanced CT brain images (axial view): *Seven days after admission* showing right transverse sinus and superior sagittal sinus hyperdensity due to venous sinus thrombosis (A,B,C yellow arrows) associated with right frontal lobe hemorrhage (c white arrow).(Color version of figure is available online)

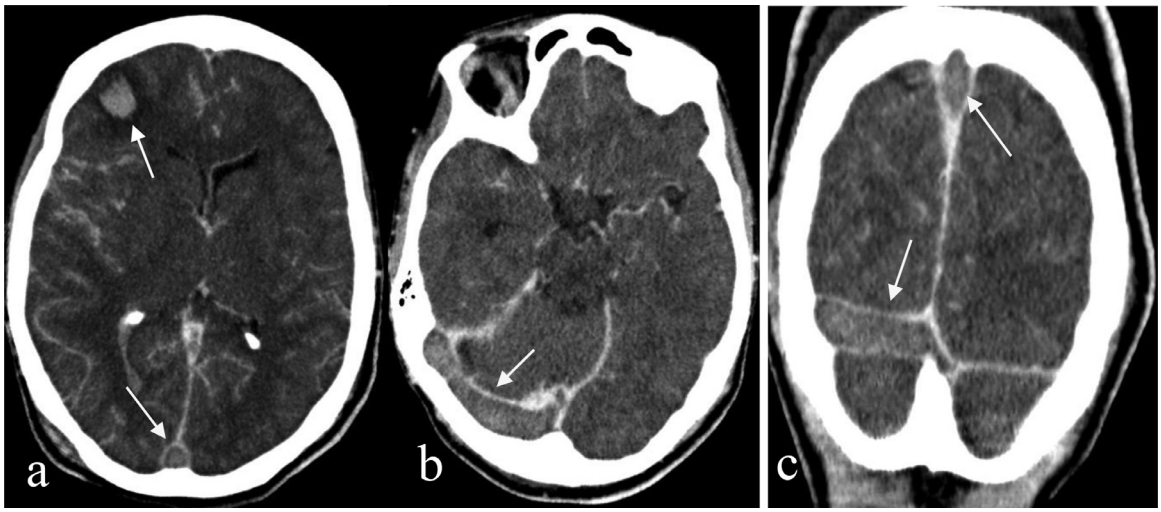

Fig. 5 – CT Venogram of brain images (axial A, B) and coronal (C) views): *Seven days after admission* showing filling defects in the superior sagittal sinus (A, C yellow arrows) and in the right transverse venous sinus thrombosis (B, C yellow arrows) associated with right frontal lobe venous infarct hemorrhage (A white arrow). (Color version of figure is available online)

ning of this crisis in Afghanistan in case of (RT-PCR) shortage in our region, radiology especially the chest CT scanner stands as a important role in the diagnosis and complications of COVID-19 infection. We aim to compare the CT results in diagnosis of complications of COVID-19 infection in a case of cerebral venous infarction due to venous sinus thrombosis. Chest CT scan done 6 hours after admission showing pattern of bilateral ground- glass opacity and bilateral areas of consolidation in a peripheral distribution [Fig. 1] was noticeable and the most cause of brain injury was a hypoxic state for days, although thromboembolic events could be also influential, and we cannot rule out this possibility. Subsequent non-contrast CT scan and CT venogram of brain after three and seven days showing venous sinus thrombosis and venous infarct hemorrhage [Figs. 3-5]. However, serious cases have been reported to experience of disorientation, loss of consciousness, stupor, coma, and paralysis (6). The most likely mechanism of focal neurologic deficits could be ischemic brain injury induced by the virus through triggering a cytokine storm [11].

The optimal choice of anticoagulant and duration of [16]. treatment for cerebral venous sinus thrombosis is not known [17]. We elected to use infused curative heparine for our patient and then shown a positive response to treatment with stable condition and we anticipate continuing anticoagulation for a minimum of 3 months [18], but will continue to review treatment.

This report highlights both the necessity to consider cerebral venous sinus thrombosis in patients presenting with

headache, unconsciousness, disorientation or focal neurological deficits and systemic anticoagulation (e.g. heparin and warfarin) is still the first-line treatment for dural venous thrombosis and anticoagulation is usually required even in the setting of venous hemorrhage [19,20].

In conclusion the reported case affirm this important point that we should be aware of neurologic symptoms such as symptoms of cerebrovascular accidents in the setting of COVID-19, which might even be the first presentations of this infection or more commonly along with respiratory symptoms and need increased attention to disease presentation and early treatment with anticoagulation should be initiated.

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