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Case Report

# Cerebellar infarction risk in a mild COVID-19 case ☆

*Antigona Kabashi, MD, PhD[a], Henri Fero, MD[b], Artiona Gjini, MD[b], Diana Dhimitri, MD[b], Mimoza Spahiu, MD[b], Fjolla Hyseni, MD, PhD[c,*], Edlira Harizi, MD[d], Kledisa Shemsi, MD[e], Valon Vokshi, MD[f], Arlind Decka, MD[g], Asm al Amin, MD[h], Ahm Ataullah, MD[i], Livia Capi, MD[j], Sepideh Jahanian, MD[k], Fareeha Nasir, MD[l], Ivan N. Ayala, MD[m], Juna Musa, MD, MS[k], Jeton Shatri, MD, PhD[n], Kreshnike Dedushi, MD, PhD[o], Florim Leniqi, MD[p], Guri Hyseni, MD[q]*

[a] *Department of Radiology, AAB College, Clinical of Radiology, University Clinical Center of Kosovo, Prishtina, Kosovo*
[b] *GP German Hospital International, Tirana, Albania*
[c] *Department of Pediatrics, NYU Langone Health, 403 East 34 street, New York, NY, USA*
[d] *Neurology Department, Regional Hospital Durres, Tirana, Albania*
[e] *General Practitioner Doctor, Tirana, Albania*
[f] *Department of Anesthesiology and Reanimation, University Clinical Center of Kosovo, New York, USA*
[g] *Department of General Surgery, Westchester Medical Center, Valhalla, NY, USA*
[h] *Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh*
[i] *Sher-E-Bangla, Medical College Hospital, Barisal, Bangladesh*
[j] *University of Medicine of Tirana, Tirana, Albania*
[k] *Department of General Surgery, Mayo Clinic, Rochester, MN, USA*
[l] *Harlem Hospital Center, New York, USA*
[m] *KER Unit, Mayo Clinic, Rochester, MN, USA*
[n] *Clinical of Radiology, University Clinical Center of Kosovo. Faculty of Medicine, Department of Anatomy, University of Prishtina, Prishtina, Kosovo*
[o] *Clinical of Radiology, Faculty of Medicine University of Prishtina, University Clinical Center of Kosovo, Prishtina, Kosovo*
[p] *Faculty of Medicine, University of Gjakova, Gjakova, Kosovo*
[q] *Department of Pediatric Surgery, University Clinical Center of Kosovo, Prishtina, Kosovo*

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ABSTRACT

Thrombotic events in SARS-COV-2 disease patients are frequent, especially in patients with comorbidities such as heart failure, hypertension, cancer, diabetes mellitus, kidney failure, vascular disease, and other pulmonary illnesses. In severe cases, in particular those of hospitalized patients with other comorbidities, the development of thrombotic events in spite of anticoagulation therapy has been observed.

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* Corresponding author.
E-mail address: fjolla.hyseni@gmail.com (F. Hyseni).

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The main thrombotic events are pulmonary thromboembolism, cerebral ischemic stroke, and peripheral artery thrombosis. Despite the severity of SARS-COV-2 disease, some patients with the aforementioned comorbidities develop thrombotic events regardless of the severity of their SARS-COV-2 infection. In this setting, the cerebellum makes no exception as an uncommon, but still possible target for thrombotic events.



## Background

Cerebellar infarcts are relatively uncommon and represent ≈2% of all ischemic strokes [1]. Some of the potential mechanisms for cerebellar ischemic stroke such as cardiovascular disease, coagulation disorders, and hypercoagulability states are amongst the most common causes of cerebellar infarction.

Presenting neurologic deficits are primarily determined by the physiologic function of involved vascular territories. From caudal to rostral, obstruction of the posterior inferior cerebellar artery (PICA, also the most frequent location for a cerebellar infarct) leads to a headache and less commonly vomiting, vertigo, horizontal ipsilateral nystagmus, and truncal ataxia. Anterior inferior cerebellar artery (AICA) territory infarction more often leads to dysmetria, Horner's syndrome, unilateral hearing loss, and ipsilateral facial paralysis or anesthesia with contralateral hemi-body sensory loss of pain and temperature. Finally, obstruction of the superior cerebellar artery (SCA, located most rostral) tends to produce ataxia, dysarthria, and nystagmus with less vertigo, headache, and vomiting. However, presentation of hemorrhagic infarctions can often be atypical or symptoms overlap [2].

## Case presentation

Here, we present the case of a 58-year-old male patient presenting to our hospital with ataxia, vomiting, photophobia, headache, vertigo, and dysarthria. BP was 150/80 mmHg. Patient history: The patient refers that 3 weeks ago he had a fever for 3 days and consecutive myalgia and arthralgia. The patient was later diagnosed with SARS-COV-2 by a random serologic test. $O_2$ saturation was 97%-98% upon admission.

Risk factors include HTA, DMT2, alcohol abuse, sedentary lifestyle, dyslipidemia, and coronary artery disease.

Immediate non-contrasting head CT scan was performed upon admission. After patient rehabilitation at the Neurology department, further evaluation was made. Brain MRI was performed as shown in Fig. 3.

Cardiac ultrasound was performed, and it resulted in a normal ejection fraction of over 55% with diastolic dysfunction. Left atrium was dilated with 20 $cm^2$. Septal hypertrophy was found. PsAP was 25 mmHg.

## Discussion

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is related to arterial and venous thrombotic complications. According to previous studies and review articles, the rate of ischemic stroke development in COVID-19 is 5%, and it mostly develops within the first 10 days following the diagnosis of the disease [3]. Clinical manifestations of COVID-19 can be dominated by complications caused by systemic inflammation and a hypercoagulable state. The hypercoagulable state in patients with COVID-19 is associated with thromboembolic incidents including ischemic strokes. The incidence of cerebrovascular events seems to be higher than expected and their consequences are certainly very serious [4].

The main vascular complications in COVID-19 have been associated with elevated levels of fibrin/fibrinogen degradation products, D dimers, and inflammatory markers [5,6]. Preliminary data indicate increased blood viscosity in severe COVID-19 as a potential contributor to endothelial injury [7]. Thrombocytopenia has been noted in certain patients with COVID-19, with lower platelet counts associated with higher mortality [8]. It may result from increased "platelet consumption" as a consequence of extensive thrombosis in severe cases of COVID-19 [9]. Mostly, it is concomitant with multiple factors, such as prior medical conditions (thrombophilia, inflammatory bowel disease), transient situations (pregnancy, dehydration, infection), particular medications (oral contraceptives, substance abuse), and unpredictable events (head trauma), with which thrombus formation becomes more intensified.

SARS-CoV-2 is hypothesized to utilize the angiotensin-converting enzyme 2 receptor (which is expressed on glial cells and neurons) to permeate CNS cells, causing inflammation and damage to surrounding structures [10]. Dysregulation of angiotensin signaling induced by interaction of SARS-CoV-2 with ACE2, its cellular receptor, can initiate an increase in cell injury and/or death and inflammation in a range of cell types, providing a mechanistic model for COVID-19 pathobiology [11–15]. We identify mechanisms by which the dysregulation of angiotensin signaling in COVID-19 can promote thrombosis and enhance tissue injury by positive feedback mechanisms between inflammation and thrombosis [13]. Cerebral venous thromboembolism, with or without associated intracerebral hemorrhage, is much less frequent [16]. The underlying mechanism for the development of hypercoagulability and subsequent stroke in patients with COVID-19 is believed to be similar to the pathophysiology of more common systemic co-

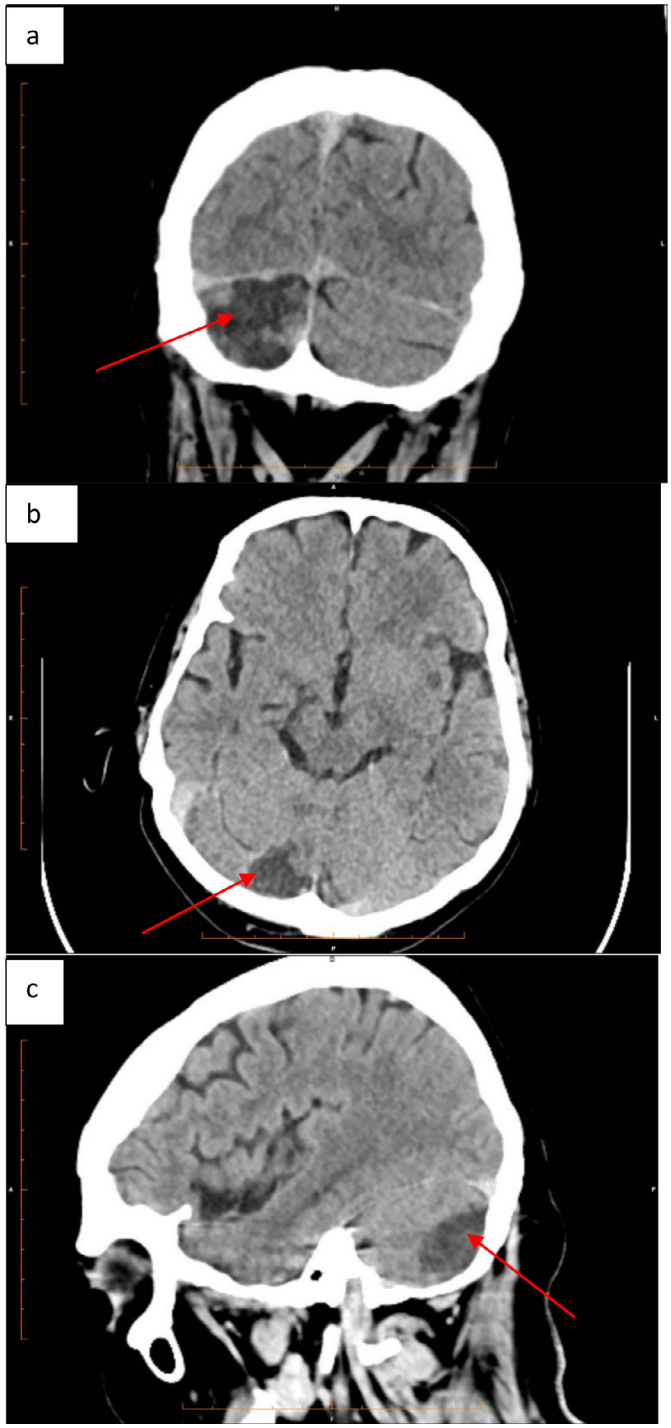


**Fig. 1 – Head CT—Non-contrast right cerebellar hypodensity, suggesting an ischemic lesion, in transverse, sagittal and coronal view (A-C red arrows).**

agulopathies, such as disseminated intravascular coagulation or thrombotic microangiopathy [17].

In addition to these mechanisms, the risk for thrombus formation is exacerbated by atherosclerosis, hypertension, DMT2, and alcohol abuse.

A recent study identified that patients with CNS symptoms had lower lymphocyte levels, platelet counts, and higher blood urea nitrogen levels compared with those without CNS symptoms [18]. Laboratory monitoring for coagulopathy such as D-dimer, fibrinogen, platelet count, PT as well as inflammatory parameters like IL-6, C-reactive protein, ferritin, and procalcitonin can aid in satisfying patients at high risk for thromboembolic events [5]. Intravenous thrombolytic therapy should be considered as with all other stroke patients. Anecdotal data do not suggest any serious safety concerns for intravenous tissue plasminogen activator use, however tPA has not been specifically studied in the setting of COVID-19 [19,20].

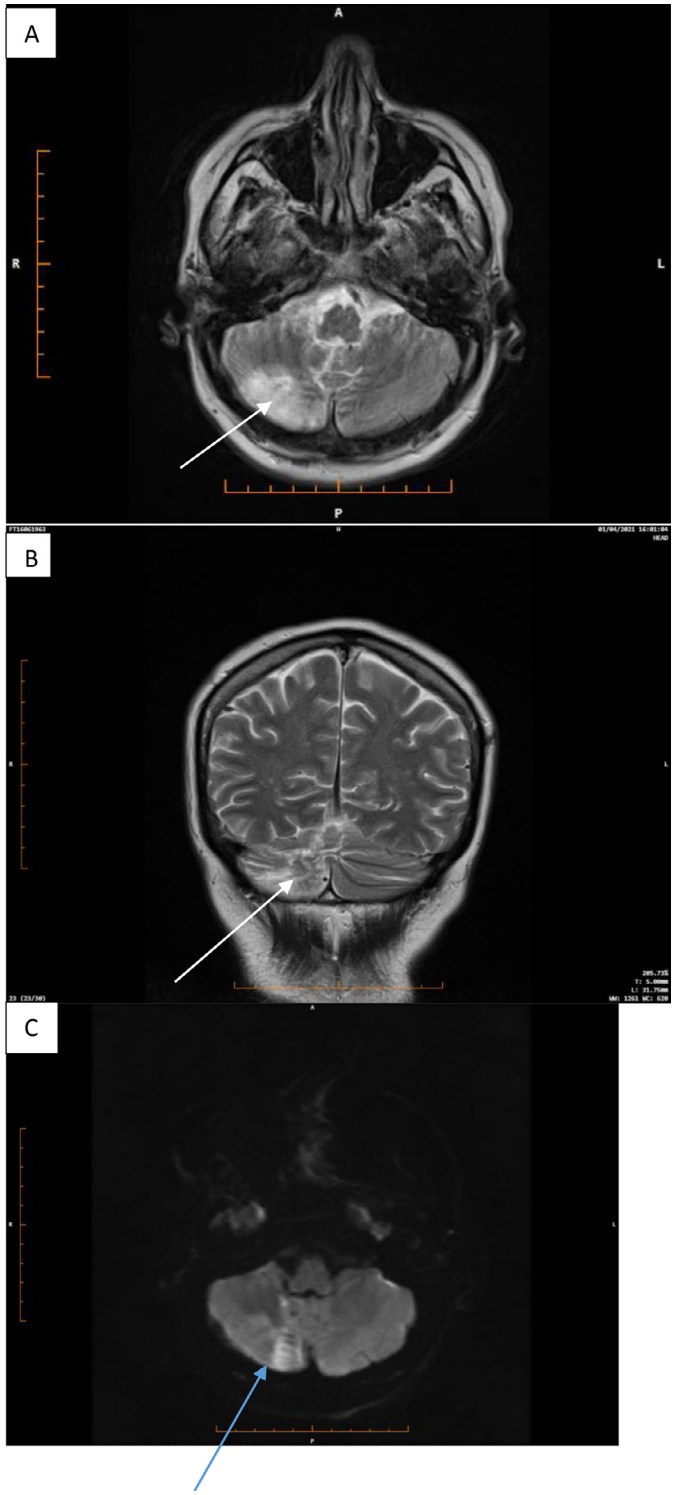


**Fig. 2 – Head MRI—T2, and TIRM multiplanes. Hyper intense right cerebellar lesion on axial and coronal T2 (A and B, white arrow) DWI (C, blue arrow) with restricted diffusion and low ADC values (D, white arrow) suggestive of an acute infarction, in the vascular territory of the right medial branch of PICA. TOF (E, yellow).**

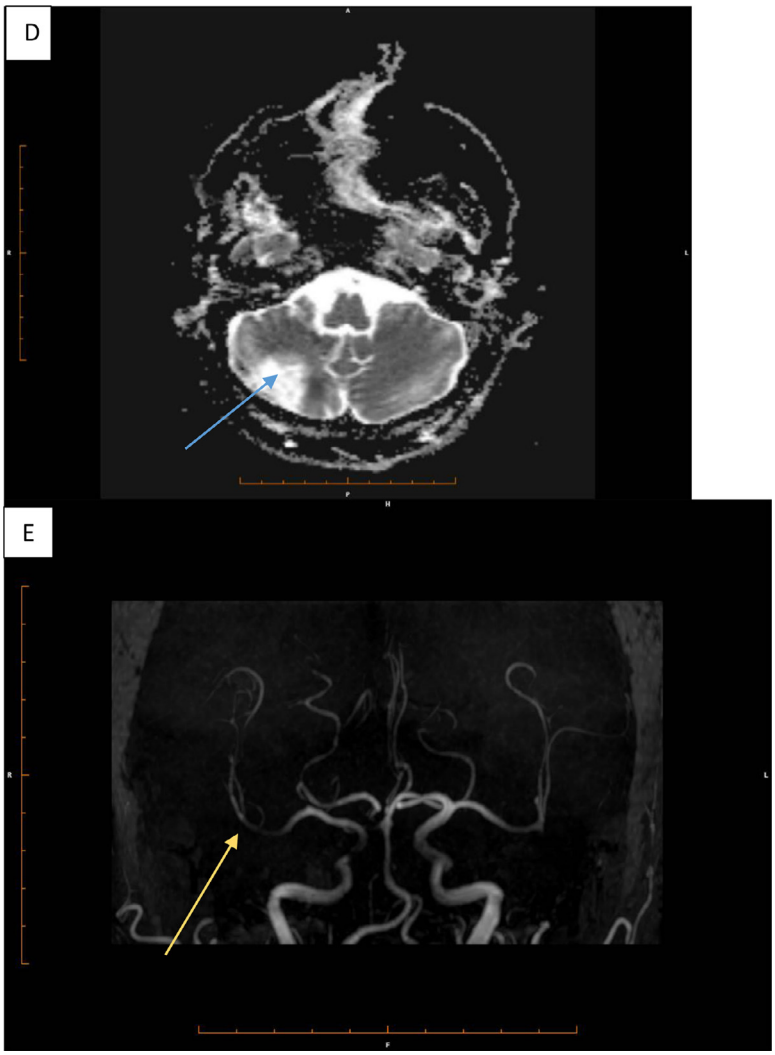


**Fig. 2 – Continued**

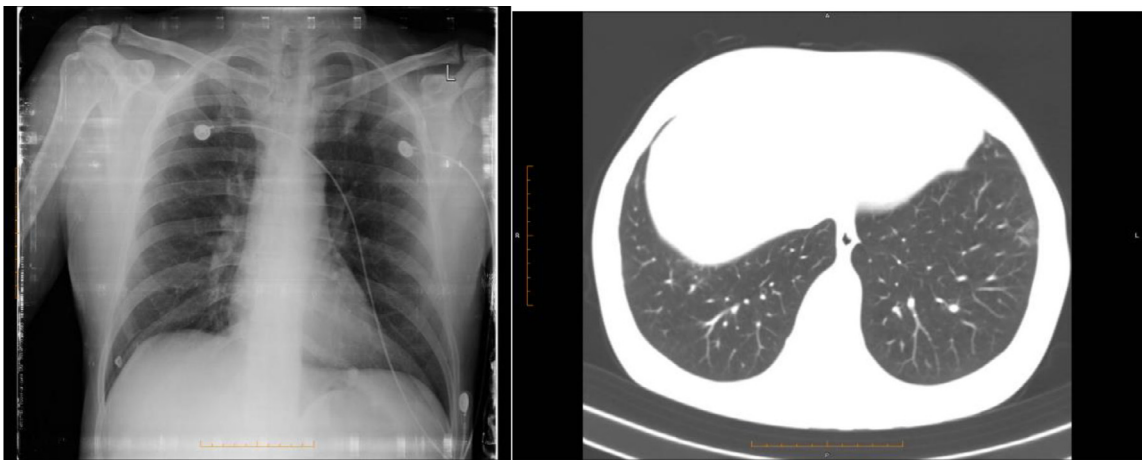

**Fig. 3 – Chest X-ray shows no signs of pulmonary involvement. Chest CT, 2 weeks after COVID infection, shows mild ground-glass opacities with peripheral distribution (yellow arrow), CTSS 2\25 points.**

## Conclusions

Despite the severity of the SARS-COV-2 pneumonia, thrombotic events are likely to happen in case of comorbidities. According to this case, we can hypothesize that even in light cases of SARS-COV-2 pneumonia with no evidence of pulmonary involvement, patients with chronic disease have a higher risk of developing thrombotic events and the cerebellum may be one of the targeted organs.

## Patient consent

We obtained written, informed consent for publication from the patient.

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