Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Letter to the editor

Unusual case of acute cerebral infarction due to large proximal ICA floating thrombus in the setting of severe COVID-19 infection

A 63-year-old right-handed man without vascular risk factors was referred for sudden left-sided hemiparesis.

A week ago, he was diagnosed with COVID-19 on the basis of moderate fever, cough and a positive reverse-transcriptase polymerase chain reaction test (RT-PCR) for SARS-CoV-2.

The patient was admitted with left-sided hemiplegia, dysarthria, homonymous lateral hemianopia and hemineglect. The initial National Institutes of Health Stroke Scale (NIHSS) score was 15.

General examination revealed a body temperature of 38,5 °C and an elevated respiratory rate of 24 breaths per minute. His peripheral oxygen saturation was 78% on room air and 92% on oxygen therapy (3L/min) via nasal cannula.

Brain Computed Tomography (CT) and CT angiography (CTA) performed in emergency showed subtle cortical right insular hypoattenuation (ASPECT score 8), a large intraluminal floating thrombus (ILT) appended to the proximal right internal carotid artery (ICA) wall (Fig. 1, A) and a distal occlusion of the right middle cerebral artery (MCA) non accessible to a mechanical thrombectomy. The patient was non-atherosclerotic and no plaque was noted in close contact to the floating thrombus. CT perfusion disclosed a large area of hypoperfusion throughout the right MCA territory consistent with penumbra.

He received intravenous thrombolysis (IVT) with recombinant tissue plasminogen activator (rt-PA) 140 min after symptoms onset.

24 hours after admission, the patient rapidly developed acute respiratory distress syndrome associated with a worsening of his neurological deficit.

Control brain imaging was compatible with an extensive MCA territory infarct (Fig. 1, B). We noted the persistence of the large ILT adherent to the right proximal ICA wall as well as a novel nonocclusive ILTwithin the cavernous segment of the right ICA (Fig. 1, C).

Chest CT showed diffuse ground glass opacities involving both lungs compatible with a severe COVID-19 infection. No pulmonary embolism was found (Fig. 1, D).

Intravenous unfractionated heparinotherapy was contraindicated because of the recent extensive brain infarction and because the patient benefited from IVT within 24 hours.

The etiological assessment including cardiac monitoring, tranthoracic echocardiography, and a complete immunologic workup (including the search of antiphospholipid antibodies) was unremarkable. Testing for the JAK2-V617F mutation was also negative.

The diagnosis of right MCA infarct due to large ILT of ICA in the course of severe COVID-19 infection was made.

Unfortunately, respiratory failure and sepsis lead to the patient's death.

ILT of the cervical arteries is an uncommon finding among patients with ischemic stroke. ILT can occur in many situations such as in the setting of an arterial injury (on ulcerated plaques, in cases of arterial dissections or external compression), from an embolic source or in association with an hereditary or non hereditary coagulopathy. However, in some cases the cause remains undetermined (up to 16% in some series). Atherosclerotic disease represents the most common cause of ILT (up to 82% in elderly patients) and mostly occurs in patients with high-grade arterial atheromatous stenosis and with conventional risk factors. Spontaneous or post-traumatic artery dissections affect particularly younger patients. The remaining patients with no identifiable cause should be investigated for underlying hypercoagulable state (such as JAK2 mutation, lupus anticoagulant) but also for malignancies, infections or vasculitis depending on the context. Cardioembolism is not a common etiology of ILT and often coexist with other potential causes [1,2].

In a large COVID-19 cohort, neurologic symptoms (such as cerebrovascular events, impaired consciousness, and muscle injury) were seen in 36% of patients particularly in those with severe infection [3].

While not fully understood, immune response to COVID-19 infection could lead to systemic inflammation, coagulopathy and local endotheliitis [2].

ILT occurring in the setting of COVID-19 infections are usually appended to an atheromatous plaque possibly because thromboinflammation related to COVID-19 may

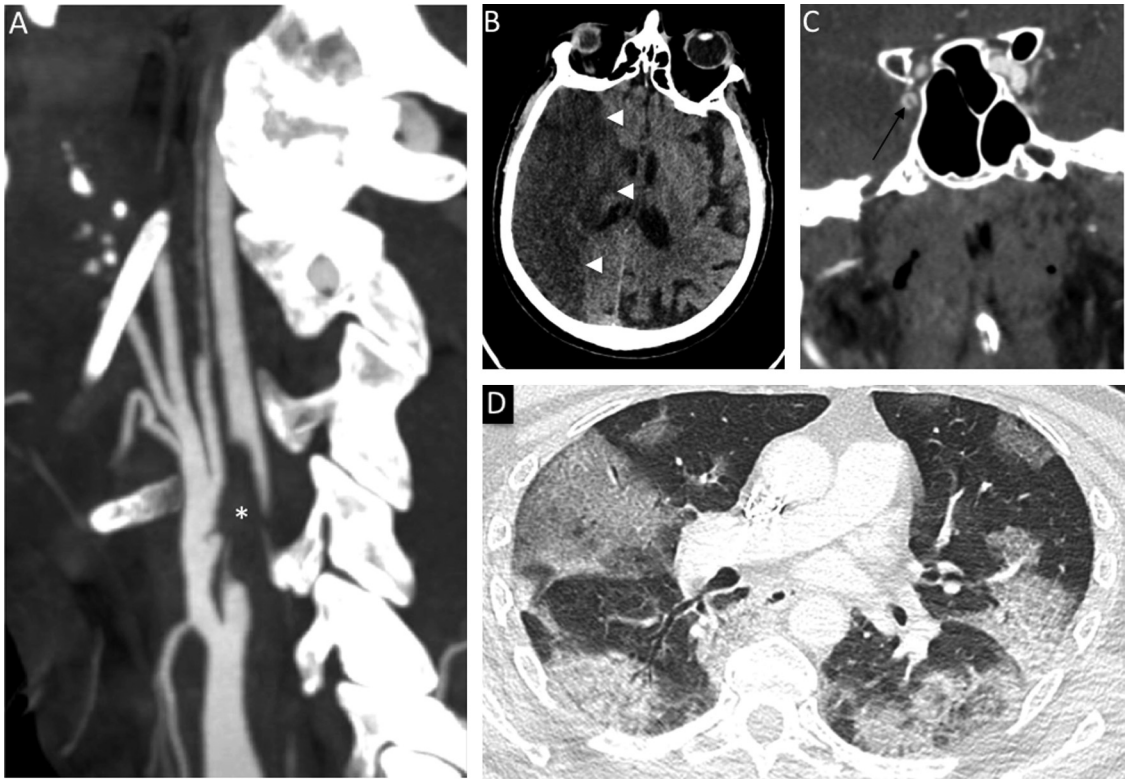

Fig. 1 – CTA showed a huge, nonocclusive ILT adherent to the right proximal ICA wall. Note the absence of atherosclerotic plaque adjacent to the thrombus (A, asterisk). CT scan performed at 24 hours disclosed a clear hypodensity within the right hemisphere consistent extensive right MCA territory infarct (B, arrowhead) and a novel nonocclusive ILT within the cavernous segment of the right ICA (C, arrow). Chest CT demonstrated peripheral ground-glass pulmonary findings consistent with severe COVID-19 infection (D).

preferentially affect areas of atheromatous disease [4,5]. ICA, aortic arch or common carotid artery can be involved [4,5].

To our knowledge ILT affecting intracranial portion of the ICA in the setting of COVID-infection has not yet been reported.

Our case highlights that ILT can also involve nonatherosclerotic arteries in the course of COVID-19 infection, presumably in relation with heightened thrombotic proclivity in the infectious context.

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Dr Benjamin Hebant, MD: study concept and design.

Dr Patrick Ahtoy, MD, Dr Bertrand Bourre, MD, Dr David Maltête, PhD, Dr David Wallon, PhD: critical revision of the manuscript for important intellectual content.

Disclosure of interest

Dr Benjamin Hebant, Dr Patrick Ahtoy, Dr Bertrand Bourre, Dr David Maltête, Dr David Wallon declare that they have no competing interest.

Ethical approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants included in the study.

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Gait ataxia due to chronic cerebrospinal fluid overshunting: A case report

1. Case Report

A 63-year-old man with a ventriculoperitoneal shunt implanted five years ago for post-trauma hydrocephalus presented with gait impairment and upper extremity dysesthesia which had been worsening for five months. On neurological examination there was a stomping gait, a positive Romberg sign, and decreased vibratory sensation (2/8 at the great toe). There was no motor deficit but symmetric hyperreflexia of the upper and lower limbs was noted. The Babinski signs were negative. Postural headache was absent and a lumbar puncture recorded low cerebrospinal fluid (CSF) pressure.

Brain and cervical spine magnetic resonance imaging revealed characteristic features of intracranial hypovolemia including diffuse enhancing, thickened meninges, and sinus venous enlargement (Fig. 1). Massively dilated epidural veins and giant subarachnoid cysts caused severe canal stenosis with, predominantly, compression of the posterior column. Vascular imaging of the cervical spine showed engorgement of the upper cervical epidural venous system (Fig. 2).

On the basis of these imaging and clinical data, the patient was taken to the operating room, the indwelling medium pressure valve was replaced with an adjustable valve (Sophysa Polaris®, France) with a functional range from 30 to 200 mm of water. The valve was empirically set to open at a pressure of 110 mm to 170 mm of water to raise the intracranial pressure but allow for adjustment if the patient did not clinically improve. Post-operatively, the patient's neurological status improved, and he no longer required an assistive device to ambulate. Six months later, the patient showed minimal symptoms but he refused magnetic resonance follow-up.

2. Discussion

Our case is a good example of the Monro-Kellie doctrine, by which the volume of CSF decreases with the intact volume of the skull and brain leading to an increase in blood volume by increasing flow blood to the cerebral sinuses and meningeal thickening [1]. Increased blood flow and dilatation of the spinal cord epidural plexus is explained by the presence of anastomoses between the venous structures of the suboccipital and upper cervical regions: the anterior condylar vein and the vertebral venous plexuses; thus the Monro-Kellie doctrine also seems to be applicable to the intraspinal compartment [2].

Hypotension intracranial is a clinico-radiological syndrome, in which the most common symptoms are orthostatic headache, diplopia, neck pain, nausea, and vomiting [3]. The phenomenon has been related to overshunting CSF, spinal surgery, craniotomy, lumbar puncture, and spontaneous leaks of CSF [4]. Myelopathy is rare in patients with intracranial