RESEARCH ARTICLE

ACUTE STROKE IN YOUNG PATIENT: ALSO THINK ABOUT CORONAVIRUS!
(ABOUT A CASE AND A REVIEW OF THE LITERATURE)

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Abstract

Certainly, the respiratory clinical symptomatology remains the most classic, however, covid 19 is recently considered to be a vasculitis affecting several organs, and which is the origin of other extra-pulmonary manifestations, in particular thromboembolic disease (both venous and arterial) which has increasingly become a dramatic complication to be feared during this pandemic, due to its negative impact on the prognosis of affected patients, increasing the need for intensive care and the death rate. The mechanism is multifactorial, associating endothelial damage, an abnormality of hemostasis, and immunothrombosis[1]. We report the case of a 39-year-old female patient who had acute ischemic stroke (AIS) by embolization of a thrombus located in the left ventricle, whose his covid 19 disease was subsequently diagnosed, and whose evolution was later marked by clinical worsening, so the patient was transferred to intensive care. The interest of this case report is to show the need to acquire the reflex to think about a covid 19 disease in case of AIS (especially for the young) even in the absence of other obvious respiratory signs, as well as to shed light on the mechanism and the particularity of diagnostic and therapeutic management of this kind of complications.
On clinical examination, we note a Glasgow coma score (GCS) at 15, with left hemiplegia, preserved sensitivity, and normal olfactory faculty.

Brain CT revealed a right fronto-parieto-temporal AIS. The electrocardiogram shows a regular sinus rhythm, incomplete right bundle branch block, and negative T waves in the apicalateral.

A transthoracic echocardiogram (TTE) was performed showing normal sized heart chambers with a normal ejection fraction, and the presence of a floating pedicle thrombus whose implantation base appears to be at the level of the lateral wall of the left ventricle measuring 24x9mm.

Subsequently anosmia and respiratory distress was installed, so a PCR of SARS-COV-2 was done, coming back positive, and a thoracic CT objectifying an aspect in favor of a Covid 19 disease, corads 5.

The patient was put on conventional treatment for Covid 19 disease, with curative HBPM-based anticoagulation and simple anti-platelet aggregation.

The evolution was marked by neurological alteration (GCS a 10), requiring transfer to intensive care, intubated for respiratory and neurological criteria.

**Discussion:**

Despite the respiratory tropism covid 19 disease, the rate of neurovascular involvement, and thromboembolic events remains non-negligible.

AIS during COVID 19 can possibly be due to two phenomena: a direct neurotropism of SARS-CoV-2 or a prothrombotic hyperinflammatory state [3] and therefore an embolic origin, as is most likely the case. case of our patient.

Numerous studies have made it possible to advance the understanding of the mechanisms, prevention and treatment of thromboembolic events (TEEs) associated with COVID-19, ultimately concluding that there are three main mechanisms:

Firstly, a disease of the endothelium [4] which constitutes an interface between the blood and the tissues and whose role is to maintain vascular homeostasis through its anti-aggregating and anticoagulant properties. After SARS-CoV-2 enters the airways, damage to alveolar epithelial cells and pulmonary endothelial cells will occur and cause a local inflammatory storm spreading to endothelial cells and causing generalized endothelitis.

Second, and like any viral infection, sepsis secondary to COVID-19 results in the release of pro-inflammatory cytokines, thus causing a cytokine storm responsible for an imbalance between procoagulant and fibrinolytic factors leading to the formation of thromboses. Hypoxia is also involved by its effect stimulating the synthesis of procoagulant factors which can go in severe forms up to disseminated intravascular coagulation [5].

Finally, an immunothrombosis hypothesis [6] is suggested in the mechanism of thromboembolic events during covid 19, and which is of particular interest to platelets. These normally play the role of a key mediator of the relationship between inflammation and coagulation. During covid 19 infection, SARS Cov2 causes platelet hyperreactivity thus increasing thrombotic phenomena.

Since covid 19 is an inflammatory disease affecting the arteries and veins, the accumulation of inflammatory cells can be observed in the endothelium of different organs [7] including the kidney (renal tubular necrosis with severe renal failure), the heart (with risk of embolization in the coronary circulation, and systemic can thus affect several organs as is the case of our patient), the small intestine causing a mesenteric ischemia, the lungs, and the lower limbs.

Indeed, in an article in the journal Intensive Care Medicine, a study was conducted in China [8] reporting a high risk of life-threatening thromboembolic complications in Covid-19 patients with respiratory distress syndrome. acute. These thromboembolic complications set in despite the initiation of preventive or therapeutic anticoagulant treatment.
Of the 150 patients included in this study, 64 presented with such complications, mainly pulmonary embolism in 25 patients (more than 16% of cases). Half of the cases were diagnosed with pulmonary embolism 5 days after admission to intensive care. Mesenteric ischemia was suspected in 5 patients (ie 3% of cases). Other patients developed a stroke [9].

In addition, 28 of the 29 patients on dialysis for kidney failure developed a clot that blocked the hemodialysis circuit.

All the major series mention the occurrence of stroke [10], with a frequency ranging from 1.7% [11] to 6% [12].

The rate of AIS in CoVid-19 patients seems relatively low, but a higher proportion occurs in young people [13], as is the case in our patient, and are often more severe compared to strokes occurring in people not infected with SARS-CoV-2.

AIS can settle within a median of 10 days after the onset of the first symptoms of COVID 19 [11,14], as it can initiate the clinical symptomatology before the onset of respiratory signs in rare cases, such as our patient.

Clinically and biologically, it has been shown a peculiarity in AIS attributed to Covid 19 disease compared to other AIS. In fact, in a study conducted in New York in March 2020, for patients with CoVid-19 disease, 0.9% of patients presented with AIS, a concomitant comparison was made between those admitted for AIS and CoVid-19, and those admitted for AIS only concluded that patients infected with sars-CoV-2 were younger, such as our patient's case, and had more extensive AIS assessed by NIHSS score (19 vs 8), had a d-dimer level more increased (10,000 vs 525), and had a higher mortality rate (63.6% vs 9.3%) [14].

The diagnostic means in case of AIS with COVID 19 remain the same, in particular: Brain CT which should be performed urgently which has a sensitivity of 92% [15]. It can be coupled with the realization of a brain angio-CT which allows visualization of the arteries of the supraortic trunks with a venous phase should be carried out in the event of suspicion of Cerebral venous thrombosis (CVT)

Brain MRI, when it is possible in an emergency, it constitutes the reference examination. It allows to date the AIS with uncertain onset. It makes it possible to diagnose small-sized AIs, thus to identify old and recent cerebral infarctions (even from the first hour by highlighting the cytotoxic edema which develops in the ischemia focus).

Other work-ups in search of etiology are necessary, in particular an electrocardiogram, transthoracic echocardiography in search of an intracavitary thrombus such as the case of our patient, ultrasound of the supra-aortic trunks, inflammatory work-up, hemostasis workup specialized, d-dimer, fibrinogen,platelets, TP, TCA, antithrombin III, protein C, protein S.

For the management, in the AIS with a measurable neurological deficit, thrombolysis (within 3 hours with a maximum delay of 4.5 hours) must be considered, and the precise inclusion and exclusion criteria are stipulated in guidelines [16].

COVID 19 disease should not modify the inclusion and exclusion criteria for mechanical thrombectomy according to guidelines of the Neuro-Interventional Surgery Society [17].

Thrombectomy with a stent retriever is indicated if the following criteria are met: mean rankin score 0-1 before stroke; AIS treated with recombinant tissue plasmogen (rtPA) <4.5h due to internal carotid artery or middle cerebral artery occlusion (MCA, m1); age ≥18 years; national institutes of Health stroke scale (NIHSS) score ≥6 [18].

Concerning anticoagulation and antiplatelet aggregation in case of AIS during the COVID19 pandemic, experts recommend the prescription of anticoagulants in the acute phase for patients who have benefitted from thrombolysis or thrombectomy and in them infection with SARS-COV-2 was suspected or confirmed.

In the absence of thrombolysis or thrombectomy [19], in patients with suspected or confirmed infection a single or double antiplatelet aggregation should be initiated with curative dose anticoagulation if a cardio-embolic origin of
the stroke has been identified (as it is the case for our patient). The recommended doses for antiplatelet drugs and anticoagulants are the same regardless of COVID19 status.

A Chinese study published in the Journal of Thrombosis and Haemostasis [20], reports that abnormal results of biological markers of coagulation, in particular high levels of D-dimer and fibrin degradation products, are associated with a poor prognosis in Covid-19 patients with pneumonia with a thromboembolic event. It is also noted that the alertness disorder and the extent and topography of the AIS constitute considerable prognostic elements [21].

**Conclusion:**
Being an inflammatory viral disease, COVID 19 is accompanied by venous thromboembolic complications, but also arterial and microvascular complications which are becoming more and more considerable, putting the patient's vital prognosis at risk.

AIS constitutes 6%, hence the need for additional research to better elucidate the pathophysiological mechanisms as well as the diagnostic and therapeutic features of these patients in order to properly control their management, and therefore to minimize the transfer rate in intensive care, and mortality.

We aim through our case report to shed light on this neurovascular complication, which can inaugurate symptomatology and therefore mask its initial etiology being the COVID19 disease.

**Conflict Of Interest**
The authors declare no competing interest.

**Figure Legends:**
Figure 1: Thoracic CT showing an aspect in favor of covid 19 disease (25% of the lung parenchyma).
Figure 2: Cerebral CT showing a right fronto-temporo-parietal ischemic stroke.
Figure 3: Transthoracic echocardiography (Parasternal short-axis and apical 4 chamber view) showing an intracavitary thrombus (left ventricle)
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