

# Stroke Revealing the Presence of SARS-CoV-2: Three Case Reports and a Review of the Literature

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## Abstract

**Introduction:** The 2019 Corona (Covid-19) disease is considered a potential extrinsic factor in the genesis or aggravation of a stroke. Patients with severe Covid-19 are more likely to develop stroke. **Comment:** We report three cases of stroke including one intracerebral haematoma and two cases of cerebral ischaemia revealing Covid-19. A history of hypertension was found in two of our patients, no notion of contagion was reported. Neurological signs were preceded by at least three days of infectious syndrome and respiratory distress syndrome. These were consistent and associated. Our patients all tested positive for Covid-19. They all had a positive CRP. Lung lesions of the alveolar, interstitial and alveolo-interstitial type were found on lung imaging. All our patients were treated with oxygen therapy, paracetamol, chloroquine and azythromicyne. In addition, acetylsalicylic acid for ischaemic strokes and nicardipine for haemorrhagic stroke. We have recorded two death and one recovery, the youngest (52 years old) with no cardiovascular risk factors. Neurological signs and symptoms can be attributed to neuroinvasion of the virus, and cerebral hypoxia. The diagnosis of Covid-19 is not a contraindication to endovascular treatment for a stroke. Stroke increase the death rate during Covid-19. **Conclusion:** Stroke is less frequent during Covid-19. Neurological signs are non-specific and combine respiratory and infectious signs. The treatment is multidisciplinary and has a variable prognosis.

## Keywords

Covid-19, Complication, Stroke, Guinea

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## 1. Introduction

Coronavirus disease 2019 (COVID-19) is considered a potential extrinsic factor in the onset or worsening of stroke (Siniscalchi & Gallelli, 2020).

In China, Li et al. (2020) report a 5% incidence of ischaemic strokes in patients hospitalised for COVID-19 in Wuhan.

In New York, over a two-week period, five patients who tested positive for COVID-19 were diagnosed with ischaemic stroke (Oxley et al., 2020).

Retrograde neuronal spread and the haematogenous route are the main mechanisms of central nervous system involvement (Siniscalchi & Gallelli, 2020).

This disease, whose symptoms are dominated by severe acute respiratory syndrome, is responsible for various neurological manifestations (Al Saiegh et al., 2020; Mao et al., 2020).

Severe infection or high levels of pro-inflammatory biomarkers indicate a significantly increased risk of ischaemic stroke, particularly in older people (Consoli et al., 2015; Siniscalchi et al., 2016).

The presence of coronavirus in the central nervous system has been confirmed in cerebrospinal fluid and in brain tissue during autopsies (Lau et al., 2004).

However, the optimal management of ischaemic strokes associated with COVID-19 remains unclear (Beyrouti et al., 2020).

## 2. Case Presentation

### First case:

A 72-year-old female patient with no known cardiovascular history was admitted with fever, dyspnoea, speech impairment and right-sided hemiplegia, which had developed over the previous nine days. The interview revealed no history of COVID-19 infection or recent travel abroad. The patient was drowsy, with well-coloured skin and mucous membranes and a WHO performance index of 3. Her vital signs on admission were as follows: BP = 130/80 mmHg; HR = 120 bpm; RR = 28 bpm; temperature = 39.8°C; Spo2 = 89%. Auscultation and percussion of the lungs revealed crackles and dullness in both lung fields, respectively. Neurological examination revealed a pyramidal syndrome of the right side of the body and motor aphasia with a NIHSS of 16. The electrocardiogram showed a regular sinus rhythm with no intracardiac conduction disturbance or abnormalities in the P, QRS and T waves. A chest X-ray (Figure 1) and brain scan (Figure 2) were performed, with the following conclusions: bilateral alveolar-interstitial pneumonia and total left sylvian infarction. Blood tests revealed a non-specific inflammatory syndrome with CRP = 15 mg/l and LDL cholesterol = 1.59 g/l. She received treatment consisting of oxygen therapy (5 l/min), amoxicillin combined with clavulanic acid (1200 mg/8 hours); paracetamol (500 mg/6 hours); Lovenox (4000 IU/24 hours) and acetylsalicylic acid (160 mg/24 hours). The epidemiological context prompted a PCR test for COVID-19 using a nasal swab from the patient, which proved positive. The tuberculin skin test was negative and D-dimer was not

measured. The patient's condition deteriorated and she died of respiratory distress one day later.



**Figure 1.** Alveolar-interstitial pneumonia.



**Figure 2.** Total left sylvian infarct (opacification of the glassy type) and cardiomegaly.

### Second case:

This also concerns a 62-year-old housewife who has been known to have hypertension for three years and has been receiving irregular treatment. She was admitted for a dry cough, dyspnoea and weakness in the left side of her body, which had been developing for three days. On admission, she presented with respiratory distress (RR = 28 cycles/min; Spo<sub>2</sub> = 88%; HR = 124 beats/min), metabolic syndrome (BP = 160/90 mmHg; blood glucose = 1.54 g/l; waist circumference = 115 mm) and right hemiplegia with an NIHSS = 10. A chest X-ray revealed bilateral opacities predominantly in the basal regions (**Figure 3**), and a brain CT scan (**Figure 4**) revealed capsular ischaemia. The electrocardiogram showed left ventricular hypertrophy, the cardiac Doppler ultrasound concluded hypertrophic cardiomyopathy with an ejection fraction of 62%, and the supra-aortic trunk ultrasound showed intima-media thickening of the common carotid arteries.

Blood tests showed CRP = (+) 40, HDL cholesterol = 0.48 g/l, LDL cholesterol = 1.68 g/l, and triglycerides = 1.28 g/l.



**Figure 3.** Bilateral opacity with a basilar predominance.



**Figure 4.** Right capsular infarct.

The COVID-19 test was positive.

The patient was transferred to the intensive care unit at the Donka Epidemiological Treatment Centre, where she died.

**Third case:**

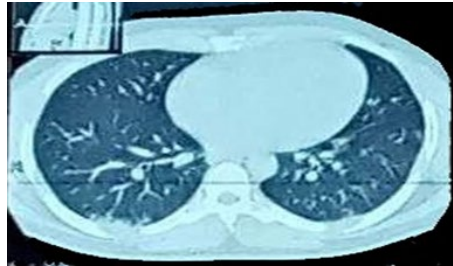
This 52-year-old accountant, who had been suffering from high blood pressure for one year and was taking 10 mg of perindopril, woke up with hemiplegia after four days of fever, headaches, chest pain and dyspnoea. His vital signs on admission were as follows: BP = 180/100 mmHg; HR = 110 beats/minute; RR = 32 cycles/minute; Spo2 = 94%; temperature = 39.6°C.

The electrocardiogram was normal. A chest CT scan (**Figure 5**) revealed ground-glass opacities suggestive of bilateral interstitial pneumonia, and a brain CT scan (**Figure 6**) revealed capsulolenticular hyperdensity suggestive of a haemorrhagic stroke. His oropharyngeal swab tested positive for COVID-19. CRP = (+) 53 and HDL cholesterol level = 0.41 g/l. The patient received oxygen therapy at 5 l/min, injectable nicardipine, paracetamol, chloroquine and azithromycin. In addition to the improvement in the above-mentioned signs during hospitalisation, the COVID-19 control test performed 7 days after admission was negative.

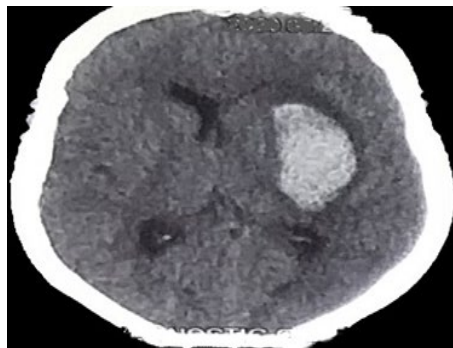
### 3. Discussion

On 12 April 2020, Guinea had 2213 cases of COVID-19. Many patients presented

with symptoms suggestive of brain damage; however, to date, no cases of focal neurological deficit suggestive of stroke have been observed in these patients. Although COVID-19 mainly affects the lungs, it can also cause multisystem involvement involving the central nervous system (Huang et al., 2020; Xu et al., 2020).



**Figure 5.** Bilateral interstitial pneumonia.



**Figure 6.** Hyperdense capsulo-lenticular lesion.

Patients with severe forms of the disease are more likely to develop neurological manifestations than those with mild or moderate forms (Mao et al., 2020).

Although the youngest patient reported was 33 years old, middle-aged and elderly people are the most represented in the literature (Oxley et al., 2020; National Health Commission of the People's Republic of China, 2020).

These patients often have other cerebrovascular risk factors; some, with no known history, develop their first acute ischaemic stroke (National Health Commission of the People's Republic of China, 2020).

The average time between COVID-19 diagnosis and stroke onset is estimated at 12 days (Li et al., 2020).

On the one hand, neurological signs and symptoms may be attributed to direct neuroinvasion by the virus; on the other hand, they may result from cerebral hypoxia secondary to pulmonary failure (Bersano & Pantoni, 2020). On the other hand, endothelial dysfunction is a major pathophysiological mechanism responsible for most of the clinical manifestations of COVID-19. Cardiovascular events occurring after COVID-19 infection appear to be linked to persistent immune dysregulation. This immune dysregulation can trigger activation of the coagulation pathway. The formation of significant microclots *in vivo*, both during the acute phase of COVID-19 and during long COVID-19, appears to be a relevant mecha-

nism responsible for the persistence of symptoms and cardiovascular events (Boccatonda et al., 2023).

Chest X-ray was the main method of pulmonary examination in our patients. It has a sensitivity of 69% for the diagnosis of COVID-19; characteristic lesions include consolidations (47%), ground-glass opacities (33%), with peripheral (41%), lower (50%) and bilateral (50%) distribution (Caruso et al., 2020).

However, chest CT scans performed on one of our patients proved to be significantly more effective, with a sensitivity of 88%, reaching 97% when combined with a positive CRP (Ai et al., 2020).

Increased systemic inflammation and coagulation abnormalities appear to play a key role in the onset of stroke during COVID-19 (Li et al., 2020).

Numerous studies conclude that respiratory infection is an independent factor in the onset of acute cerebrovascular disease; this association was observed in all of our patients (Elkind, 2007; Warren-Gash et al., 2018).

The most frequently reported central neurological manifestations during COVID-19 are headaches, drowsiness, epileptic seizures and motor deficits (Mao et al., 2020).

In our series of three stroke cases, two were cerebral ischaemias. Data from the literature also show a predominance of ischaemic stroke during COVID-19, probably related to the coagulopathy and vascular endothelial dysfunction associated with the infection (Zhou et al., 2020; Chen et al., 2020).

However, as in our series, cases of cerebral haemorrhage have also been reported, particularly in the Wuhan cohort (Mao et al., 2020).

Brain lesions may be multiple, although this was not observed in our patients. Recurrences may occur despite effective anticoagulation (Beyroufi et al., 2020).

In patients with acute ischaemic stroke with suspected or confirmed COVID-19, management is based on multidisciplinary collaboration between neurologists and infectious disease specialists. Endovascular treatment of stroke during COVID-19 is not contraindicated, although it has not been performed in our patients (Smith et al., 2020).

The combination of hydroxychloroquine and azithromycin was used as medical treatment in our patients, in accordance with the recommendations in force at the beginning of 2020 (Recovery Collaborative Group, 2021; Beigel et al., 2020). Although subsequent data did not confirm the efficacy of this therapeutic strategy, it was based on the initial protocols. A retrospective study subsequently questioned its efficacy, highlighting the need for further research to deepen our understanding (Mehra et al., 2020).

Clinical deterioration appears to be linked to hyperactivation of inflammatory mediators, leading to a potentially fatal inflammatory storm as the disease progresses (Jin et al., 2020). For cases 1 and 2, both of whom died, D-dimer testing could not be performed due to the unacceptability of the test and in order to minimise the risk of contamination. However, a D-dimer level above 1 µg/mL could help to identify patients at risk of poor prognosis at an early stage (Zhou et al., 2020).

SARS-CoV-2 infection manifests itself in varying degrees of severity, with an estimated mortality rate of between 2 and 4%, which may be higher in the event of a stroke (Wu et al., 2020).

These observations highlight the need for clinicians to remain highly vigilant regarding the risk of stroke in COVID-19 patients with acute neurological deficits.

Therefore, any acute neurological deficit in a COVID-19 patient should raise suspicion of a cerebrovascular event and warrant rapid assessment.

#### 4. Conclusion

Stroke is less common than respiratory symptoms, but it remains a serious complication. This does not imply any clinical specificity of stroke, but neurological signs are generally associated with an infectious syndrome and/or respiratory distress.

Management is multidisciplinary. The prognosis depends not only on the severity of the condition, but also on the timeliness and quality of care.

#### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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