

# Covid-19 Concomitant with Acute Massive Cerebral Infarction: A Case Report and Review of Management

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## Abstract

Coronavirus mainly attacks lung cell of human body through the ACE2 receptor, leading to a series of pathophysiological abnormality such as ARDs and hypoxemia. In addition, it has been reported that Coronavirus might also lead to cardio cerebral vascular diseases since many heart related symptoms might appear in the coronavirus patients, which suggests that coronavirus might lead to damage on other tissues besides lung. According to the epidemiological survey, 40% of COVID-19 patients have been found to have cerebrovascular and chronic diseases. There are several cases of COVID-19 patients being found with acute cerebrovascular accident. Similarly, in 2003 when the SARS was pandemic, Peking University journal published and reported a SARS patient with acute cerebral infarction. A COVID-19 patient with massive cerebral infarction was treated in February 7, 2020. The diagnosis and treatment are reported as follows.

**Keywords:** Coronavirus pneumonia; Acute massive cerebral infarction; Risk factor; Treatment

## CASE REPORT

An elder man of 55 years was admitted to this hospital on Feb. 8, 2020 and he was found with expectoration for 14 days. During this time, he was not found with any other obvious inducement such as expectoration and cough. Following that, he appeared the symptom of aggravation in 4 days and left limb weakness for 2 days. His cough did not have obvious circadian and his sputum has white foam. He was not given with diagnosis and treatment. As the cough aggravated for consecutive four days, and chest tightness, asthma and headache symptoms occurred [1-3]. At this time, he still could tolerate the pain. By Feb. 6, he felt numbness in his left upper limb, and he started to be treated. Since the morning of February, the patient's left lower limb was disable and he was admitted to the Neurology department in our hospital, undergoing CT examination. The examination result indicated that he had multiple lacunar cerebral infarction, and he also had pneumonia, suggesting that he had high likelihood of viral pneumonia. The patient was quickly transferred to the Fever Clinic department of the hospital and was performed with throat swab test [4]. In addition, he was treated with oral levamlodipine, clopidogrel, aspirin and butylphthalide. His coronavirus nucleic test showed that

he had coronavirus. When he was under the observation of the hospital, his situation became worse [5-7]. Symptoms such as vomiting, nausea and severe headache appeared. He was given with CT examination, and this result showed that he had multiple and severe lacunar infarction and brain degeneration. He was isolated as a coronavirus patient and he was sent to the isolated ward.

The patient was a driver and he was healthy, and he took regular health examination and did not show any problems in his body. He was scored at 14 points when he was collected in the NIHSS. His body temperature was 36-degree, P 69 times/min, R 19 times/min, BP 179/114mmHg SPO2 94% (without oxygen). In addition, he had a clear mind, stable breathing. He responded well when he was given with some test questions. During percussion, he did not show abnormal unclear problems. His heart-beat rate was 69 times/min. His muscle strength of his right limb was graded to be 5, and left side limb was graded to be 1 [8].

After he was admitted to the hospital, his auxiliary examination showed the coagulation function was prothrombin 12.4 second, fibrinogen was 3.82g/L, activated partial thromboplastin time was 32.3

second, international standard ratio was 0.94, D-dimer quantitative was 1.33 ug/ml, fibrin degradation product rate 3.97ug/ml. His blood routine examination shows that his white blood cell was  $3.83 \times 10^9/L$ , neutrophil  $2.40 \times 10^9/L$ , lymphocyte number  $1.16 \times 10^9/L$ , neutrophil 62.90%. His biochemical parameter was: potassium 3.01 mmol/l, sodium 138 mmol/L, chlorine 99 mmol/L, calcium 2.28 mmol/l, urea 2.60 mmol/l, creatinine 62.7 umol/l, aspartate aminotransferase 40u/L, albumin 48.8 g/l, total bilirubin 35.8 umol/l, lactate dehydrogenase 363u/L, glucose 10.25 mmol/l, carbon dioxide 28.0 mmol/l, uric acid The results were as follows: 195.9 umol/l, troponin < 0.012 ng/ml, procalcitonin 0.020 ng/ml, high sensitivity CRP 17.13

mg/l, saa203.1 mg/l,  $\beta$  2-macroglobulin 1.61mg/l, phospholipase 2374u/L [9].

The CT examination result showed: 1. He had multiple lacunar cerebral infarction; 2. He suffered cerebral degenerative changes; 3. He had high possibility of viral pneumonia considering the pneumonia (Figure 1). His ECG was normal, and he was performed with coronavirus nucleic acid test by the mean on throat swab in Yaohai District Center for Disease Control on Feb. 7, 2020. The CT examination showed: 1. He had massive cerebral infarction in right frontotemporal occipital lobe. 2. He had multiple lacunar infarction; 3. He had brain degeneration (Figure 2); Considering his thrombolysis without external intervention, The diagnosis and

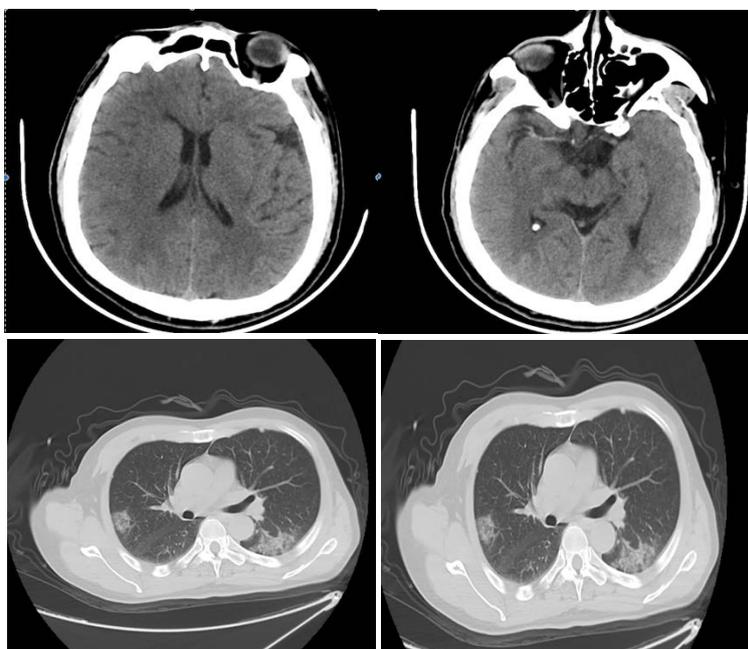


Figure 1: The head and lung CT examination of Feb. 7, 2020

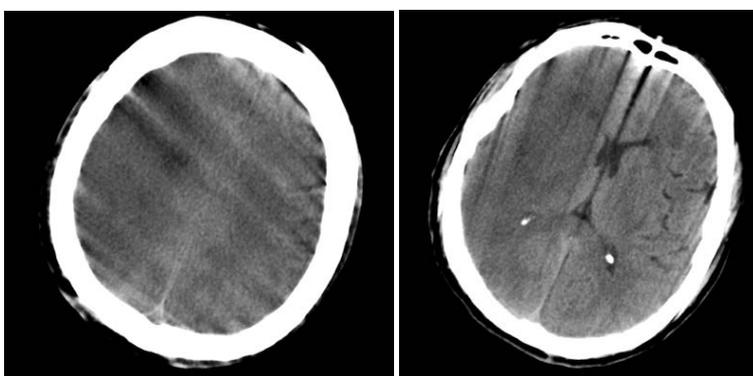


Figure 2: The head CT examination of Feb. 8, 2020

treatment of experts from neurology department was that he was treated with oxygen therapy, dehydration, mild hypothermia treatment, intracranial pressure, platelet aggregation, improving cerebral circulation, blood pressure control, cerebral circulation and lipid regulation. Towards his viral infection and lopinavir, he was treated with Lianhua Qingwei orally together with acid suppression, stomach protection, tonifying and phlegm resolving. On Feb.8. 2020, his parameter was: temperature 36.3-degree, P 78 times/min, R 19 times/min, bp 165/82 mmhg and SpO2:97%(3L/min). The patient still had headache, expectoration, cough and little amount of sputum. His consciousness was clear, and he did not show convulsion. In addition, his lung sign was the same as before. On Feb. 9. 2020, his consciousness became worse and he was in the state of somnolent. His GCS score was down to 7 points. His coronavirus pneumonia was a circle shape with a diameter of 3mm. He was given with dehydration treatment and it was enhanced subsequently. The consciousness of the patient decreased significantly further at that time. The patient was in a coma and his GCS score was down to 6 point. According to Anhui Provincial Health commission, he was transferred to a designated hospital for furthermore treatment. He had cerebral hernia and central respiratory was failed for him. Even though he was given with endotracheal intubation for mechanical ventilation, he died of central respiratory and circulatory failure.

## DISCUSSION

To summarize the patient and his family information, he is a middle age adult and he keeps a good shape, also he has healthy body and does not drink alcohol. Despite that he used to drink alcohol while he was not addicted, and he got rid of alcohol for more than 10 ears. According to his examination report, he was healthy and free from high blood pressure, obesity and heart disease. He did not have poor healthy evidence and he was free from OSAS disease. Meanwhile he did not intake medicine for long term maintenance. The only bad habit for him was the long hour and static working. He was working as a driver for over 5 years and he was sitting on the seat for more than 9 hours every day. The examination of @@ could not reflect whether he had the problem of the venous thrombosis of lower extremity. This should be supported by the examination of Doppler ultrasound of lower extremity.

This research has found some potential coronavirus infection factors leading to the increase of brain injury. Firstly, coronavirus patients might not have apparent

and significant symptoms in term of their early clinic symptoms, while their disease severity will quickly aggravate, and patients will display the chest tightness, fever and white lung symptoms in a short time. At present, the mechanism of coronavirus cells is linked to their activation through the angiotensin-converting enzyme. The excessive activation of lung cells leads to the inflammation. The feedback look of inflammation cells will lead to the cell damage while since ACE2 are highly expressed in the blood of human body, it is highly possible that the inflammatory induces the damage of blood vessel. Secondly, anxiety and stress level of patients are also influential and risk factors for cerebrovascular disease. Especially since the outbreak of COVID-19, people feel stress and anxious, which leads to many unhealthy psychology and physical reaction. The catecholamine release in human body result in the microcirculation, vasospasm and disorder. In worse case, death might also occur. Thirdly, coronavirus mainly attacks lung cells and the symptoms of elder and weak patients are obvious. The hypoxemia is also a significant phenomenon (SPO2 is less than 93 when the oxygen inhalation is lower than 300). Hypoxia leads to the low level of energy required for the metabolism, and this causes oxygen free radicals and acidosis, eventually it will destroy the cell membrane's phospholipid layer. Thirdly,

According to the 1291 patients' data collected by Beijing 2003 clinic cases, academician Hu Shengshou and his team found that the proportion of SARS patients with cardiovascular and cerebrovascular disease accounted for 17%. The newest update from 99 hospitalized case of COVID-19 published in Lancet Journal shows that 40% patients had chronic diseases including cerebrovascular and cardiovascular disease. Especially elder patients have found significant large presence of chronic diseases. Their immunity is undermined and impacted by COVID-19. Even though COVID-19 mainly attacks the tissue and cells with ACE2 while the current epidemiological data shows the relationship between COVID-19 and cardiovascular disease. At this point, further study and experiment are needed.

In summary, when patients are affected with COVID-19, their body cells will be destroyed directly, which will subsequently result in hypoxemia, inflammatory storms and causes cerebrovascular damage or vasospasm. For the suspicious cases especially those patients who are under vascular risk, if they have appeared the symptoms of frequent cough, headache, hypoxia discomfort or adverse emotion state. They should be given with frequent monitor and evaluation, which can provide precautious measures for the subsequent treatment.

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