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Cerebral Stroke with Sepsis during COVID-19 Infection

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Abstract

Aim: This case report includes the stroke with sepsis during the COVID-19 infection in a male patient of 70 years age.

Introduction: This is a patient hospitalized for COVID-19 interstitial pneumonia. After a few days of therapy with oasegeno, antivirals and hydroxychloroquine, the patient was discharged. He returned to the emergency department for confusion, fever and dysarthria. The TAC finding of ischemic and hemorrhagic areas has placed us in great difficulty regarding therapeutic treatment.

Conclusion: Although neurological symptoms are not frequent in coronavirus epidemics, the high number of patients with SARS-CoV-2 infection may explain the presence of the virus in the central nervous system and increase the probability of early or delayed onset neurological symptoms. Follow-up of patients with the SARS-CoV-2 epidemic should include careful assessment of the central nervous system.

Keywords: Central nervous system; Coronavirus; Neurological symptoms; SARS-CoV-2; COVID-19; Stroke; Interstitial pneumonia; Sepsi

Introduction

One of the emerging characteristics of the COVID-19 pandemic is coagulopathy, defined as "sepsis-induced coagulopathy" (SIC), with high D-dimer and fibrinogen values [1,2].

SIC is a precursor state of DIC, related to a systemic inflammatory response induced by infection with endothelial dysfunction and microthrombosis with organ failure, usually without bleeding [3].

Stroke is emerging as a complication of the COVID-19 pandemic; in fact neurological symptoms have been described in many cases: anosmia, hypogeusia and convulsions, up to stroke [2]. We report a case of stroke with cerebral septic emboli during COVID-19 infection.

Clinical Case Presentation

A male patient of 70 years old was admitted in the Emergency Room for dyspnoea and fever lasting for 7 days on 09/03/2020. He underwent naso-pharyngeal swab for COVID-19, which resulted positive, blood tests showed: leukocytes 10.560, platelets 358.000, CRP of 86.1, blood gas analysis with pO_2: 46.2, oxygen saturation 96%. Chest x-rays showed diffuse opacities of the interstitium with bilateral diffusion. He was hospitalized and treated with oxygen therapy at 12 litres per minute at a FiO_2 of 60%, therapy with hydroxychloroquine 200 mg × 2/day and lopinavir/ritonavir 200/50 cp × 2/day for 7 days. He was discharged on 3/19/2020 having normal general conditions. After 3 days the patient was again brought to the emergency room because he presented with elevated fever and confusional state, aphasia and dysarthria. The head CT-scan showed hemorrhagic material in the right frontal lobe and occipital lobes bilaterally,with extensive ischemic areas in the occipital area. The Chest-X-ray was unchanged compared to the previous hospitalization. Blood chemistry tests showed: white blood cells 16,780, platelets 19,000, D-dimer above 20, LDH 380 U/L (min value 248), procalcitonin 3.07 (n.v. 0-0.51 ng/ml), PCR 285.2 (n.v. 0-5), fibrinogen 269 mg/dl (n.v. up to 200), troponin 655.3ng/L (n.v. 0-34.2). The patient was intubated and admitted to the Intensive Care Unit where he underwent brain MRI with multiple that showed supratentorial and subtentorial findings of multiple alterations due to hemosiderinic deposition from the hemorrhagic component, at the frontal-right and left frontal-parietal-radiated crown, occipital temporum bilaterally at the level of the head of the caudate and cerebellar nucleus bilaterally. After injection of contrast medium, the findings are compatible with septic embolization of ischemic lesions. The patient's clinical condition worsened until death.

Discussion

The world is currently focusing on the COVID-19 pandemic, with most admissions and complications related to lung dysfunction. This case is a rare presentation of the COVID-19 infection which exhibits a larger inflammatory process involving the respiratory systems, but which progresses to cause neurological dysfunction. Infection-induced endothelial cell dysfunction causes excessive thrombin production with the stopping of fibrinolysis and increased clotting capacity [3,4]. Furthermore, hypoxia found in severe forms of COVID-19

cases can stimulate thrombosis not only by increasing the blood viscosity but also the Transcription Factor levels [5]. Excessive suppression of fibrinolysis caused by overproduction of Plasminogen Activator Inhibitor (PAI) with potential prothrombotic effects is a hallmark of DIC associated with sepsis [4-6] with rapid progression to multi-organ failure and death. Many studies show that a serious COVID-19 infection is associated with increased coagulopathy related to the severity of the disease. The marked increase in D-dimer, as in our case and the reduction of platelets are consistent with the progressive activation of coagulation [7,8]. The X-ray picture in our clinical case has highlighted the presence of septic emboli, raising the doubt that it was septicemia with cerebral localization of coronavirus.

Hung et al. They reported a case of coronavirus-associated encephalitis (SARS-CoV) severe acute respiratory syndrome; CSF cell count, protein and glucose were normal, but SARS-CoV-2 RNA was detected in CSF [9]. Another clinical case from China described acute myelitis as a complication of COVID-19 which had flaccid weakness of the lower limbs and persistent fever; no lumbar puncture was performed but the authors believed that the likely cause of myelitis was a post-viral inflammatory process [10]. As illustrated by this unusual case of septic cerebral embolism, with evidence of a wider multisystem inflammatory response and the development of neurological symptoms over time, it is important to be vigilant for late non-respiratory manifestations of COVID-19 infections. Furthermore, the difficulty of the case is linked to the simultaneous presence of ischemic areas and hemorrhagic areas in various areas of the brain, placing strong doubts on the possible therapeutic choice.

Conclusion

DIC is a complication having high mortality of septic patients, characterized by the systemic activation of coagulation. The DIC associated with sepsis is secondary to the suppression of endothelial dysfunction-induced fibrinolysis which can rapidly continue towards multi-organ failure and death. In addition, many studies have confirmed that cerebrovascular disease is associated with an increase in disease severity in positive COVID-19 patients. However, many other studies are needed to understand this relationship with COVID-19 disease.

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