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Severe Covid 19 Pneumonia and Neurological Disorders in A Field Hospital: Benefit of Bedside Ultrasound

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ABSTRACT

Introduction: The Covid 19 is multisystem disease, with a cerebro-vascular manifestation including, ischemic, embolic and hemorragique strocke.

Case Report: Patient is 80 years old, with a history of chronic smoking that has been stopped for 30 years and ischemic heart disease. Presenting for a fortnight a cough with fever and myalgia and dyspnea. A thoracic tomodensitometry had objectified an aspect of Covid 19 pneumonia with more than 75% of the pulmonary parenchyma, and a PCR SARS-COV 2 performed on a positive nasopharyngeal sample. He admitted to intensive care unit because he had a respiratory distress with high oxygen flow. The evolution during the first 24 hours of hospitalisation was one of respiratory aggravation, followed on the second day by the installation of a predominantly brachial left hemiparesis, without pupillary anomaly. A cerebral ultrasound scan revealed a hyperchoic image in the cerebral parenchyma suggesting intracerebral hemorrhage, without any image of midline deviation, with a normal Doppler study. The patient had worsened respiratory distress requiring mechanical ventilation with protective ventilation by trying to keep a PaCO2 close to normal and stopping anticoagulation for one week. The result was unfavourable with death on the ninth day of admission.

Keywords: Severe pneumonia; Neurological disorder; Ultrasound; Cerebral hematoma

INTRODUCTION

The Covid 19 pandemic, due to the new SARS-COV 2 virus[1], initially described as a respiratory disease, quickly developed its multisystem profile. Among the organs affected is cerebrovascular damage with a high morbidity and mortality, mostly ischaemic but also hemorrhagic.

Through this observation, we describe the diagnosis interest of cerebral ultrasound in hypoxemic patient with severe Covid 19 pneumonia.

CASE REPORTS

Patient is 80 years old, with a history of chronic smoking that has been stopped for 30 years and ischemic heart disease under anti-platelet therapy with acetyl salicylic acid, presenting for a fortnight a cough with fever and myalgia, without digestive disorders, evolving towards the installation then the aggravation of a dyspnea motivating the admission to the emergency room, where a thoracic tomodensitometry had objectified an aspect of Covid 19 pneumonia with more than 75% of the pulmonary parenchyma made of diffused frosted glass, with a PCR SARS-COV 2 performed on a positive nasopharyngeal sample. The patient was then transferred to the intensive care unit at the companion hospital after a 24-hour stay in the emergency department. On admission, the physical examination found a confused patient with a Glascow score of 13/15, no focal deficit with a normal pupillary examination, 80% pulsed oxygen saturation in ambient air reduced to 96% under 15 litres per minute of oxygen via a tank mask, polypnoea at 35 cycles per minute, blood pressure at 114/75 mmHg and heart rate at 76 beats per minute at a steady rate, capillary blood glucose was correct and the patient was apyretic. The biological assessment found a biological inflammatory syndrome with CRP at 207 mg/l, ferritinemia at 1020 ng/ml, LDH at 1050 IU/l, elevated troponin Ic at 1200 ng/l, white blood cell count at 6000/mm^3, lymphopenia at 690/mm^3 and neutrophils at 4520/mm^3, renal, hepatic and electrolyte balance was normal.

The treatment was high flow nasal oxygen therapy, with 80% FIO_2 and a flow rate of 60 L/min, allowing saturation at 95%, with third generation cephalosporin-based antibiotic therapy, corticotherapy and low molecular weight heparin thromboprophylaxis, in addition to the usual treatment. The evolution during the first 24 hours of hospitalisation was one of respiratory aggravation, followed on the second day by the installation of a predominantly brachial left hemiparesis, without pupillary anomaly. A cerebral ultrasound scan revealed a hyperchoic image in the cerebral parenchyma (Figures 1,2) suggesting intracerebral hemorrhage, without any image of midline deviation, with a normal Doppler study with systolic velocities at 65 and 70 cm/s left and right respectively, and diastolic velocities at 30 and 40 cm/s in the same order. The patient had worsened respiratory distress requiring mechanical ventilation with protective ventilation by trying to keep a PaCO2 close to normal and stopping anticoagulation for one week. The result was unfavourable with death on the ninth day of admission.

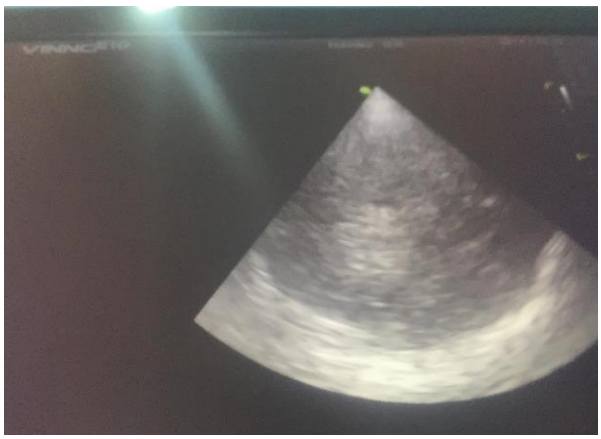

Figure 1: Hyperechoic images in cerebral parenchyma founded with cerebral ultrasound through temporal window

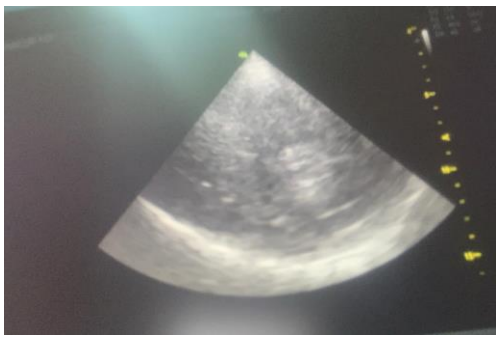

Figure 2: Hyperechoic images in cerebral parenchyma founded with cerebral ultrasound through temporal window

DISCUSSION

Several publications have enriched the literature on the subject of cerebrovascular impairment during Covid 19 disease, which shares several risk factors with stroke,[2] in particular arterial hypertension, diabetes,[3] advanced age[4] and a high level of D-dimer.[5] The incidence of intracerebral haemorrhages associated with SARS-COV 2 infection remains variable according to the publications, ranging from 0.5%,[6] 1.1%.[7]

The exact pathophysiology remains debated, but etiopathogenic hypotheses have been put forward including a direct role of SARS-COV 2 via the ACE 2 receptor present in the central nervous system, and on the vascular endothelium,[8] via abnormalities of the blood-brain barrier and cerebral self-regulation.[9] These abnormalities may be of indirect origin caused by the cytokine storm induced by the SARS-COV 2 infection.[10] Other mechanisms include dysregulation of systemic blood pressure by imbalance in the balance between angiotensin 2 and angiotensin 1,[11,12] as well as a broad spectrum of bleeding disorders, varying between hypocoagulability by thrombocytopenia, prolongation of the quinck time and hyperfibrinolysis,[13] as well as a high level of tissue plasminogen activator and thrombomodulin,[14] and hypercoagulability fomenting intracerebral haemorrhage.[15]

The diagnosis of an intracerebral haemorrhage is based on a non-injected CT image which is the standard gold. Now in our case, the realization of this imaging was delicate for two reasons, the first being the transport of a patient in respiratory distress that could require invasive ventilation, or while he was intubated and ventilated his transport by respecting the rules of a protective ventilation with a risk of derecruitment due to iterative disconnections and the non perfection of the transport ventilators. The second is the absence of scanner equipment in a companion hospital designed in the midst of a pandemic to manage the wave and the exhaustion of the existing health structures. Hence the use of cerebral ultrasonography at the patient's bedside which allows accurate descriptions of sonographic features of cerebral anatomy are available in the literature.[16] Some reports have shown hematomas, midline shifts, and ventricular enlargements can be detected using a low-frequency probe through the temporal bone of an intact skull.[17]

CONCLUSION

Cerebral ultrasound can be a diagnostic alternative to the patient's bedside, allowing both an anatomical diagnosis of the lesions and a functional diagnosis via the Doppler study of intracerebral hemodynamics.

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