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Case Report

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Hypoxic convulsions induced by adult respiratory distress syndrome in Covid-19 patient

Abstract

Covid 19 is one of the major infections that primarily attacks the respiratory tract. The most common symptoms at onset of COVID-19 illness are temperature, cough, dyspnea, hemoptysis and diarrhea. In severe cases, patients may develop severe pneumonia - often billateral, acute respiratory distress syndrome (ARDS) and multi-organ failure including perimyocarditis. Hypoxic convulsions is a rare finding, however it is associated with very dangerous disease and poor prognosis. This current report presents severe Covid-19 patient with ARDS and severe hypoxia accompanied with hypoxic seizures after exclusion of meningoencephalitis and other possible etiologies for epileptic attack.

Keywords: covid19, hypoxia, convulsions, ARDS, epilepsy

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Introduction

Prevalence of epilepsy in the world at 2019 was 70 million patients,[1] however there are few reports about Covid-19 induced hypoxia and seizures without encephalitis.[1-6] Epileptic attack due to hypoxia in adults is an uncommon condition. Seizures due to hypoxia, known as hypoxic convulsions, develop due to hypoxic-ischemic encephalopathy (HIE).[1-2,7-8] HIE is caused by a deficiency of appropriate oxygen supply to the cerebrum.[1,5-9] The condition is well known during birth or shortly after the delivery in newborns and children. Seizures are the result of severe electrical activity in the brain.[1,3,8] Manifestations of hypoxic convulsions are similar to those of idiopathic epilepsy, however epileptic seizures due to acute hypoxia – hypoxemia are also often accompanied by severe pathological changes such as neuronal ischemic necrosis and developing of partial cortical softening regions due to impaired blood brain barrier permeability.[2,3,7-12] It leads to higher neuronal excitability and lower the epileptic seizure threshold.[9-12]

Similar condition is seen in chronic hypobaric hypoxia in high-altitude regions where low oxygen tension leads to various neurological diseases such as epilepsy.[6]

During hypoxia the cerebral blood circulation decreases due to elevation of hematocrit and blood viscosity. Covid-19 infection leads to changes in autonomic nerve activity, and vasoconstriction induced by interleukins and cytokines released by vessels endothelium.[4] Hypoxia stimulates excitation of microglia by inducing oxidative stress and neuroinflammation.[13-14]

Glial tissue includes astrocytes, microglia and oligodendrocytes, microglia cells which function as CNS phagocytic cells.[13-14]

Abnormal activation and proliferation of astrocytes caused by chronic hypoxia can increase neuronal electrical excitability at the level of neurotransmitters such as GABA by reducing it secretion and enhance seizure susceptibility. Hypoxia causes impairment of the blood–brain barrier by increasing its permeability by neuro-inflammation and oxidative stress.[1,2 8-14]

Chronic hypoxia damages the physiological function of the blood–brain barrier by increasing its permeability and triggers series of neurotoxic reactions through the above-mentioned side effects of neuro-inflammation and oxidative stress.[4-8,10-12]

Case presentation

A 72 year old male was admitted to the ICU ward due to shortness of breath, productive cough and desaturation (89% room air) that started three days prior to admission. The patient was diagnosed as suffering from severe Covid 19 infection. Prior medical history did not contain any chronic illnesses. Physical examination findings included, dyspnea, tachypnea, tachycardia, fever, peripheral cyanosis. Auscultation revealed rales and crackles in both lungs. Chest X-ray findings included patchy opacifications in both lungs.

Blood test revealed: hemoglobin-13g/dl, WBC-3400 per microliter, 18% lymphocytes (lymphopenia). CRP was 14mg/dl (normal values 0-0.5mg/dl), urea 55mg/dl, creatinine 1.3mg/dl, other tests were unremarkable.

Treatment with remdesivir and dexamethasone were started. Oxygen support was given as well and oxygen saturation level raised to 96%. Following 4 days presented improvement in the patients' state. Fever and cyanosis disappeared and respiratory rate dropped to 18 breathes per minute. Laboratory tests did not change significantly except which CRP which raised to 16mg/dl. One week following admission the patient developed severe respiratory distress and needed mechanical ventilation. Consecutive chest X-ray revealed opacifications and large regions with lung fibrosis. CT scan of the lungs revealed findings consistent with adult respiratory distress syndrome (ARDS). The patient continued treatment with remdesivir for 10 days. Dexamethasone was continued and the dose was elevated. Blood cultures were negative however because of possibility of superinfection treatment with ceftriaxone and azithromycin were added and on day12 after admission antibiotics were changed to piperacillin-tazobactam for 5 days and later to imipenem and amikacin.

The patient state was periodically improving and deteriorating. Several extubations were done following intubation several days later due to respiratory instability.

Oxygen saturation was 98% at the beginning of mechanical ventilation and it decreased during the continuous treatment to 86% on

100% percent of oxygen. Repeated chest CT showed severe bilateral pneumonia with aggravation of fibrosis (opacifications enlarged and pneumocysts appeared).

On day 20 of hospitalization and 11 day on mechanical ventilation the patient developed general convulsions whereas oxygen saturation was 87% both on pulse oximeter and in arterial blood gases, pH- 7.21, pO2-86 mmHg, pCo2-90 mmHg, bicarbonate - 28mmol/l. To exclude technical problems the apparatus (of mechanical ventilation) was changed 3 times and qualified technicians inspected the ventilation machine several times. Head CT didn't reveal any signs of brain infarct, edema or abscess. CSF from lumbar puncture – showed clear, colorless fluid, Pressure: 70 to 180 mm H_2O 3 leukocytes, glucose-70mg/dl, chloride-110, protein-15mg/dl, EEG showed generalized rhythmic delta slow waves without epileptic foci. Midazolam was the only neurotropic medicine given for effective ventilation, which was discontinued on 13 day after hospitalization. All antibiotics were discontinued – to exclude antibiotic neurotoxicity. Patients oxygen saturation variated from 86% to 93%. High doses of steroids and later tocilizumab were added. Convalescent Covid-19 plasma exchange was performed without any improvement. Seizures mostly appeared when the oxygen saturation was less than 89%. Between the epileptic attacks the patient was somnolent. Convulsions were treated by Leveteracitam, however it did not significantly changed the frequency of seizures. Anoxic Brain Damage was considered.

The patient's condition continued to deteriorate secondary to severe catabolic state. Bilateral pulmonary embolism were detected on day 22 after admission. Blood tests showed severe hypoalbuminemia of 1.4mg/dl (received albumin together with hyper-alimentation). 32 days following admission the patient deceased.

Comments

Multiple studies have shown relationship between chronic hypoxia and occurrence of epilepsy.[1,3,4,7]

The Covid19 virus interacts with alveolar epithelial cells and stimulates oxidative stress and severe inflammatory response (hyper-inflammation) with prominent elevation of tumor necrosis factor alpha, interleukins especially, IL-6, IL-12 and IL-15.[5]

The current report describes uncommon finding of Covid-19 infection hypoxic convulsion which developed due to severe bilateral pneumonia and progressive pulmonary fibrosis. Treatment with mechanical ventilation did not managed to elevate oxygen saturation above 86%. Other causes for seizures in Covid-19 as meningoencephalitis, vascular thrombosis and antibiotic neurotoxicity were excluded by normal CSF analysis and normal CT of brain.

High dose antiepileptic treatment did not halt the convulsions completely. The only possible treatment was lung transplantation, which was excluded due to his poor physical condition and low albumin levels.

During the viral phase in the first week of hospitalization the patient's condition was stable. The following 10 days were characterized by severe deterioration including ARDS development and the need for mechanical ventilation. Although given maximal oxygen support the patient's state continued to deteriorate including bilateral pneumonia and lung fibrosis which accompanied with permanent hypoxic encephalopathy and hypoxic convulsions.

Conclusion

Covid-19 is a common infection which can lead to ARDS. Hypoxic encephalopathy and convulsions are rare conditions which is important to recognize for early treatment.

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Conflicts of interest

None to declare.

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