COMMENTARY

Brain-lung Interaction in Neurotrauma in COVID-19 Patients

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Abstract

The recently described coronavirus (SARS-CoV-2) has produced a series of pathological changes after infection of the human body. A significant percentage of infected critically ill patients with COVID-19 will require multiple intensive care strategies to give appropriate support to increase the possibility of favorable evolution. The new coronavirus could invade using the respiratory mucosa and to infect various cell types successively creating a severe inflammatory response. Patients with cerebral neurotrauma have elements associated with the primary and secondary lesions. Lung injury impact brain with hypoxia, hypercapnia, hypocapnia, mediators release, presence of neurotoxic factors, and endothelial activation. On the other hand, brain injury impacts lungs due to increase in intracranial pressure (ICP). There is development of neuroinflammatory phenomena, the activation of sympathetic nervous system, and the presence of intense dopaminergic activity through the hypothalamic-pituitary-adrenal axis. Studies have demonstrated injury at the ultrastructural level in type II pneumocytes after traumatic brain injury.

Keywords: Lung, Neurocritical care, Neurotrauma.

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The recently described coronavirus (SARS-CoV-2) has produced a series of pathological changes after infection of the human body.[1] A significant percentage of infected critically ill patients with COVID-19 will require multiple intensive care strategies to give appropriate support to increase the possibility of favorable evolution.[2] The new coronavirus could invade using the respiratory mucosa and infect various cell types successively creating a severe inflammatory response.[3,4]

Patients with cerebral neurotrauma present unique challenges for the intensivist. This presentation has elements associated with the primary and secondary lesions.[5] Lung injury impacts the brain with hypoxia, hypercapnia, hypocapnia, mediators release, presence of neurotoxic factors, and endothelial activation. On the contrary, brain injury impacts the lungs due to an increase in intracranial pressure. There is a development of neuroinflammatory phenomena, the release of catecholamines, and the presence of intense dopaminergic activation. Studies have demonstrated injury at the ultrastructural level in type II pneumocytes after traumatic brain injury.[6,7]

It is well known that brain injury produces the release of large amounts of catecholamines, which leads to increased capillary permeability due to sympathetic alteration.[8] Hemodynamic changes are also the result of the action of catecholamines, increasing the hydrostatic pressure, and damaging the blood-gas barrier. The release of inflammatory mediators arises in response to hemodynamic changes, causing endothelial injury and ischemia.[7,8] Covid ARDS requires prone ventilation due to profound hypoxemia. Intracranial hypertension is can happen with the positional change and change in the dynamics of venous return. Bedside placement of intracranial monitoring for TBI and prone position can help to decide when and how long to keep the position. Prevention of DVT in TBI patients with Covid is possible with chemoprophylaxis. It is tricky with active DVT and PE to initiate full dose anticoagulation with the risk of conversion of TBI to ICH. The evidence is robust of a high incidence of clotting in Covid patients. Most of the centers with TBI and active Covid will require full dose chemoprophylaxis and close neuro watch for ICH. A nonbolus approach with a goal of aPTT to 40 to 60 is one of the approaches suggested in the literature.

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From the experience of China and Italy, we know that between 30 and 80% of COVID-19 patients will require mechanical ventilation.[9,10] A recent meta-analysis showed that up to 15% of COVID-19 patients develop ARDS.[11] Respiratory failure is associated with increased mortality in patients with TBI.[12] Patients with COVID-19 present a greater complexity in the context of neurotrauma, the neuroinvasion of the coronavirus can become an aggravation of the lesions and originate from cerebral injury. This leads to further dysfunction in lung-brain interaction.

Recommended strategies to maintain brain-lung interaction within the controlled state in patients with a combined presentation of COVID-19 and traumatic brain injury would be: Maintain 30-degree head-up tilt with straight head position, reduce alveolar overdistension in inspiration, minimize instrumental dead space, optimizes respiratory rate to reduce intrinsic PEEP and overdistension, close respiratory monitoring for any early decline, neuromonitoring with minimum ICP probe. Extracorporeal membrane oxygenation (ECMO) support may also be started depending on available resources. The multiorgan clinical approach should be the strategy to approach a patient with respiratory failure and neurotrauma in the context of the new pandemic by COVID-19.[12]

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