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Morphological Changes in the Lungs in Brain Injuries

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Abstract: The brain and lungs strongly interact via complex pathways from the brain to the lungs but also from the lungs to the brain. The main pulmonary disorders that occur after brain injuries are neurogenic pulmonary edema, acute respiratory distress syndrome, and ventilator-associated pneumonia, and the principal brain disorders after lung injuries include brain hypoxia and intracranial hypertension. All of these conditions are key considerations for management therapies after traumatic brain injury and need exceptional case-by-case monitoring to avoid neurological or pulmonary complications.

Keywords: traumatic brain injuries, neurocritical care, neurogenic pulmonary edema, lung injury, ventilator-induced lung injury.

Introduction Traumatic brain injury (TBI) is one of the leading causes of death and disability worldwide, with enormous economic and social consequences [1]. TBI results from direct damage to the brain caused by an external, physical force, which can lead to a state of decreased consciousness, physical functioning, or deterioration of cognitive ability [2]. Many secondary lesions in patients with TBI occur in a medium-long term period, and the appearance of brain and pulmonary complications is highly prevalent. Direct brain injury and altered levels of consciousness decrease the protection of the airways and alter the natural defense barrier, which adds to reduced mobility and multiple pathophysiological deficits inherent to the injury [3] and triggers several lung injuries, like neurogenic pulmonary edema (NPE), acute respiratory distress syndrome (ARDS), and ventilation-associated pneumonia (VAP). In the same way, lung injuries can affect the brain due to alterations in pulmonary physiology having repercussions at the systemic level, leading to neurological disorders which can be triggered mainly by hypoxia and intracranial hypertension [3,4].

The brain and lungs share fundamental connections that are compromised in patients with TBI. This injury becomes a vicious cycle that worsens patient condition (Figure 1). Considering the increasing global incidence of TBI, deficient access to healthcare in many parts of the world, and inadequate methods of treatment [1], we present a narrative review of the available evidence on the mechanisms of brain-lung interaction in patients with trauma as well as new therapeutic implications and possible management strategies that could reduce brain and pulmonary complications and improve the prognosis of patients in order to provide a basis for future research.

HISTORY Extracranial complications frequently occur in patients with TBI; among them, pulmonary disorders are common and deteriorate patient condition, leading to serious neurological outcomes. Brown-Séquard [5] was the first to describe the interaction between TBI and the lungs in 1871 in his experimental traumatic injury to the pons of guinea pigs that resulted in pulmonary hemorrhage and edema. In 1969, Simmons et al. [6] evaluated an autopsy series of patients who died from TBI that reported the vast majority of those who died within few minutes

after trauma to have pulmonary edema, which allowed them to affirm that TBI is decisive in the development of pulmonary edema with consequent respiratory dysfunction. In 1976, Theodore and Robin introduced the "explosion theory" to explain NPE in patients with TBI [7] that declared an excess of catecholamines and consequent positive regulation of sympathetic signal transduction to be responsible for the increase in pulmonary venous pressure and transudative edema. Finally, although NPE was estimated to have an incidence of only 1% after cerebral trauma in 1997, a mortality of 60%–100% was demonstrated regardless of its etiology, which triggered the alarms to consider it a life-threatening condition after it was demonstrated to increase of intracranial pressure (ICP), where immediate therapeutic interventions are essential [8].

Likewise, in terms of frequent pulmonary complications, ARDS is also notable. Its concept has evolved from the old name idiopathic pulmonary anasarca, postulated by Laënnec in 1821, until its recent definition in Berlin [9]. In 1967, Ashbaugh et al. [10] presented the concept of ARDS as respiratory distress syndrome, which was improved in 1988 by Murray et al. [11] by including the description of a multi-section system. Later, the European-American Consensus Conference conceptualized the criteria for acute lung injury (ALI). However, it was decided not to use the ALI term and to stratify ARDS into three levels of severity in the more recent Berlin definition [9].

In 1997, Bratton and Davis [12] evaluated the incidence of ALI in comatose patients after isolated TBI and found that it was 20%, showing a three-fold greater probability of dying or surviving in a vegetative state. In a study published in 2003, it was reported that 31% of patients with severe TBI developed ALI, with a greater number of days in mechanical ventilation (MV), a worse neurological outcome, and mortality of 38% [13]. The presence of ALI or ARDS complicates the treatment of patients with TBI because hypoxia causes additional damage to the brain and because therapies used to protect the lungs and improve patient oxygenation can reduce cerebral blood flow and increase ICP [14]. In a 20-year retrospective cohort study of patients with TBI, an increase in the prevalence of ALI or ARDS was found, and a greater association with comorbidities, such as congestive heart failure, hypertension, chronic obstructive pulmonary disease, chronic kidney disease, and liver failure, as well as increased risk of sepsis, multi-organ dysfunction, and in-hospital mortality was found [15].

There is a high frequency of infection in patients with TBI due to a predisposition attributed to compromised host defenses after brain trauma and the need for MV in these patients [16]. In 1992, Piek et al. [17] evaluated the determinants of recovery after severe brain trauma and found that pneumonia was the second most frequent complication (40.6%) on the fifth to tenth days after the trauma; furthermore, they emphasized the fact that many of them were not preventable in critically ill patients and could only be managed through quick diagnosis, prompt etiology identification, and opportune treatment [17]. Later, pneumonia was found in 60% of 125 patients with closed head trauma. Of which, 47.8% corresponded to early pneumonia and were associated with lower scores on the Glasgow coma scale (GCS), longer intubation time, intensive care duration, and hospitalization [18].

VAP is among the most important subtypes of nosocomial infections, and the incidence of this type of pneumonia in patients with brain injury ranges from 28% to 40% [19]. In 1999, Ranieri et al. [20] demonstrated for the first time that MV induced a cytokine response in the lungs and plasma, which was associated with higher rates of multi-organ failure. However, they suggested that attenuation of this inflammatory response could be a useful strategy to minimize overstretching and cell recruitment in lungs, with possible improvements in clinical outcomes. In 2004, Bronchard et al. [21] published a prospective observational study of patients with TBI who required tracheal intubation for neurological reasons and ventilation for at least 2 days and reported a 41.3% incidence of early-onset pneumonia with low arterial oxygen pressure (PaO_2), decreased inspired fraction of oxygen (FiO_2), fever, hypotension, and intracranial hypertension.

They concluded that early-onset pneumonia led to secondary lesions and neurological deterioration.

Between the organs through different pathways. Nevertheless, almost always after a TBI, only the brain effect at the pulmonary level is considered, with very little consideration of the detrimental reverse consequences. Acute lung damage caused by ARDS or MV can worsen neurological functions in patients with TBI by humoral, neuronal, and cellular communications and by the interaction of numerous elements released locally or systemically in trauma response. Hence, therapeutic strategies should be able to minimize the impact of TBI in critical patients, both at the pulmonary and neurological levels.

MV can have a beneficial effect on oxygenation and cerebral perfusion in patients with TBI. However, the estimation of tidal volume, PEEP, and RMs is also necessary in addition to considering systemic and cerebral parameters, such as ICP, $PaCO_2$, PaO_2, and properties of the lungs and chest wall due to the close relationship that they have to the elevation of ICP. Finally, we conclude that the management of patients with TBI should be established on a case-by-case basis considering individual characteristics to avoid the appearance of neurological and pulmonary complications that influence prognosis or constitute long-term sequelae with greater morbidity and mortality, additional costs to health services, and the inevitable decrease in patient quality of life. Although new strategies targeting the inflammatory cascade are being investigated, experimental and clinical studies are needed to evaluate the brain and lung level effects