CONFERENCE PROCEEDING

Brain injury and the lung

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Neurogenic pulmonary oedema (NPE) is a clinical syndrome characterised by the acute onset of pulmonary oedema following a significant central nervous system (CNS) insult. While the exact locations and circuits involved in the CNS have not been clearly identified, this uncommon but potentially life-threatening condition may occur in the setting of acute, severe brain injuries including traumatic brain injury, intra-cerebral haemorrhage (ICH) as well as in seizures. The sudden rise in intracranial pressure (ICP) such as in subarachnoid haemorrhage (SAH) or ICH, hypothalamic involvement, rapidly occurring sympathetic surge and increased systemic vascular resistance have all been implicated in pathophysiology.[1-3]

Harvey Williams Cushing was the first to describe the connection between CNS injury and haemodynamic dysfunction.[4] The true incidence of NPE is unknown and most often underreported. The incidence of NPE in brain injury varied from 2% to 43%[5,6] in SAH to 20% in traumatic brain injury.[7]

The pathophysiology of NPE has been subject to debate with severity and acuity of the precipitating CNS event as the lead theory. Conditions that are associated with an abrupt and extreme elevation in ICP appear to be at greatest risk of being associated with NPE.[8] The abrupt increase is believed to give rise to an intense activation of the sympathetic nervous system and release of catecholamines. Although the exact source of sympathetic outflow has not been identified, certain centres have been implicated. These include the hypothalamus and the medulla.

Although ICP elevation plays an important role in NPE, it is unclear what occurs at the level of pulmonary endothelium. Several theories have been proposed: (1) Neuro-cardiac; (2) neuro-haemodynamic; (3) blast theory and (4) pulmonary venule adrenergic hypersensitivity. The details will be discussed during the presentation.

Two distinct clinical forms of NPE have been described. An early form of NPE with symptoms developing within minutes and a delayed form with symptoms developing within 12–24 h after CNS insult.

Because alternative conditions are common, NPE is a diagnosis by exclusion and requires documentation of non-cardiogenic pulmonary oedema.

The management of NPE to date has focused on treating the underlying neurologic conditions to decrease the ICP and autonomic surge.

During the presentation, we will discuss some diagnostic criteria and potential therapeutic options.

Management of NPE remains challenging. Understanding the pathophysiology of the disease and treating the cause aggressively will be key in management.

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