Lung maladies following aneurysmal subarachnoid haemorrhage

Gentle S. Shrestha

Abstract

Pulmonary complications are common in patients with aneurysmal subarachnoid haemorrhage. It is associated with adverse outcome in the form of higher incidence of vasospasm, poor neurological outcome, longer length of stay and higher mortality. These patients are at high risk for ventilator associated pneumonia. Preventive measures, early detection and timely appropriate treatment is important. Acute respiratory distress is common in patients with subarachnoid haemorrhage. Measures to minimize the second hit to the vulnerable lungs is crucial. Low tidal volume ventilation, application of PEEP and prone ventilation should be used with special emphasis to avoid hypercarbia and increase in intracranial pressure. Activation of sympathetic nervous system, release of catecholamine and damage to pulmonary endothelium can lead to neurogenic pulmonary edema. Treatment of underlying cause and supportive care is helpful. These patients are at high risk of deep vein thrombosis and pulmonary embolism. Measures to prevent deep vein thrombosis and administration of pharmacological prophylaxis when safe, needs to be considered.

Key words: Acute respiratory distress syndrome, aneurysmal subarachnoid haemorrhage, lung maladies, neurogenic pulmonary edema, pneumonia, pulmonary embolism

INTRODUCTION

In patients with aneurysmal subarachnoid haemorrhage (SAH), secondary complications are the important cause of morbidity and mortality.[1] Pulmonary complications are the important causes of non-neurological medical complications in patients with aneurysmal SAH.[2,3] Oxygenation disturbances occur in 43%–92% of patients with SAH.[4] In patients with brain injury and respiratory failure, mechanical ventilation is associated with longer Intensive Care Unit (ICU) and hospital stay, poor neurological outcomes, higher incidence of symptomatic vasospasm and increased mortality.[5,6] The lung maladies in patients with aneurysmal SAH occurs in the form of ventilator-associated pneumonia (VAP), acute respiratory distress syndrome (ARDS) and neurogenic pulmonary oedema (NPE).[7]

PNEUMONIA

Patients with neurological disorders are at increased risk for developing aspiration pneumonia and VAP in part due to decreased level of consciousness and aspiration of gastric contents.[8] VAP is the most common nosocomial infection in patients with SAH and is associated with longer duration of mechanical ventilation and ICU stay. It is an independent predictor of mortality and severe disability.[9,10] Methicillin-susceptible *Staphylococcus aureus* is the most common pathogen. Initiation of early enteral feeding, oral care and

Department of Anaesthesiology, Institute of Medicine, Tribhuvan University Teaching Hospital, Kathmandu, Nepal

Address for correspondence:
Dr. Gentle S. Shrestha, Department of Anaesthesiology, Institute of Medicine, Tribhuvan University Teaching Hospital, Maharajgunj, Kathmandu, Nepal.
E-mail: gentlesunder@hotmail.com

implementation of VAP prevention bundle can be beneficial to decrease the incidence of VAP. Unless contraindicated, daily interruption of sedation to assess readiness for extubation, maintaining endotracheal tube cuff pressure, hand hygiene, elevation of head-end of the bed, oral hygiene with chlorhexidine and stress-ulcer prophylaxis can together help decrease the incidence of pneumonia. The appropriate and timely institution of antibiotics and control of fever is necessary for a better outcome.[11-13]

ACUTE RESPIRATORY DISTRESS SYNDROME

ARDS is common in patients with injured brain. Patients with SAH represents 20–38% of patients with ARDS among brain-injured patients.[5,14] In brain-injured patients, use of high tidal volume ventilation in the days preceding the development of ARDS was found to be an important risk factor for the development of ARDS.[15] The high tidal volume may function as a second hit on the lung, which is already rendered vulnerable by the systemically released inflammatory mediators from the injured brain [Figure 1].[7] Furthermore, the lung injury may render the brain more sensitive to acute injuries.[7] Lung protective ventilation strategies using low tidal volume (6 ml/kg predicted body weight) limiting the plateau pressure to <30 cm H_2O, and adequate positive end-expiratory pressure (PEEP) has been used to prevent or limit ventilation-induced lung injury.[16] The consequent permissive hypercapnia can be deleterious in patients with raised intracranial pressure (ICP). Smaller studies have shown lung protective ventilation to be safe in patients with SAH.[17,18]

Application of PEEP in brain-injured patients can have one of the three responses – either the ICP will increase or decrease or there will be no change. If the applied PEEP results in alveolar recruitment with a consequent reduction in $PaCO_2$, the ICP will decrease. If PEEP induces sole hyperinflation without improvement on ventilation, the ICP may increase. If the applied PEEP

induces alveolar recruitment without a change in $PaCO_2$, the ICP may remain unchanged.[19] The effect of PEEP on cerebral haemodynamics would also depend on the elastance of the lungs. The effect is minimal in patients with low respiratory system compliance, like in patients with ARDS where PEEP is most indicated.[20] It is important to avoid alveolar overdistension and to maintain stable haemodynamics. There are emerging data about the safety of application of PEEP in patients with brain injury.[21] Furthermore, raising the head-end of the bed by 30° in patients with SAH, who are euvolemic, helps ameliorate the adverse effects of PEEP on ICP and cerebral perfusion pressure (CPP).[22] However, lung recruitment manoeuvre in patients with cerebral injury has been shown to adversely affect the cerebral haemodynamics and metabolism with only a marginal improvement in oxygenation. It should be applied with caution.[23] Larger well-defined studies are need to be performed to evaluate the safety and feasibility of lung protective ventilation strategies in these vulnerable patients and how the management needs to be individualised, weighing the risk and benefit in individual patients.

Prone ventilation has been a standard of care for patients with severe ARDS. The beneficial effects are in the form of improved oxygenation and reduction in mortality when applied for the duration of >12 h/day.[24] In patients with SAH, the beneficial of prone ventilation on the improvement of cerebral oxygenation by improving arterial oxygenation seems to overweigh the adverse consequence of increase in ICP and decrease in CPP. Careful monitoring of ICP in the patients during prone ventilation can be prudent.[25]

NEUROGENIC PULMONARY OEDEMA

NPE presents as an acute onset of pulmonary oedema following a significant central nervous system (CNS) insult. Patients usually present with bilateral infiltrates, PaO_2/FiO_2 ratio of <200, no evidence of left atrial hypertension, presence of severe CNS injury enough to cause significant increase in ICP and the absence of other common causes of ARDS (sepsis, aspiration, massive blood transfusion, etc.).[26] The incidence of NPE ranges from 23% to 71% of the hospitalised patients with SAH. Risk factors for developing NPE following SAH are higher clinical and radiographic grade at presentation, increasing age, increasing time to securing of aneurysm and vertebral origin of aneurysm.[27,28] These patients have a higher mortality rate approaching 10%.[29]

Sudden, rapid and significant elevation of ICP, followed by intense activation of the sympathetic nervous system with the release of catecholamine, has been considered as a key pathophysiological mechanism for NPE.[26] However, the real pathophysiological events
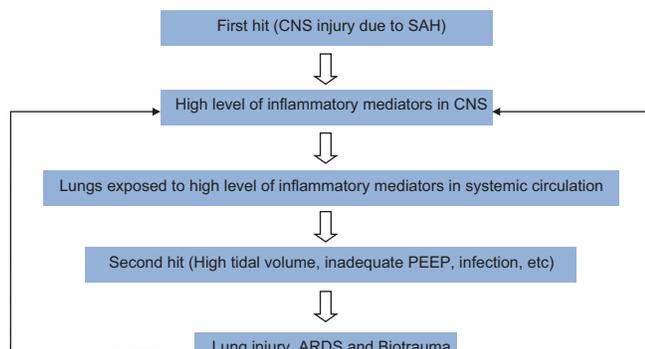
Figure 1: Double-hit model in the patient with brain injury

First hit (CNS injury due to SAH)

High level of inflammatory mediators in CNS

Lungs exposed to high level of inflammatory mediators in systemic circulation

Second hit (High tidal volume, inadequate PEEP, infection, etc)

Lung injury, ARDS and Biotrauma

at the level of pulmonary vasculature remain elusive. Several clinicopathological explanations have been proposed.

Neurocardiac neurogenic pulmonary oedema

The massive sympathetic discharge following SAH can induce direct myocyte injury causing hypokinesia, diminished lusitropy and diastolic dysfunction, with subsequent cardiogenic pulmonary oedema.[30]

Neurohaemodynamic neurogenic pulmonary oedema

The CNS insult is followed by an abrupt increase in systemic resistance, followed by translocation of fluid from high resistance systemic circulation to low resistance pulmonary circulation and subsequent pulmonary oedema.[31]

Blast theory

During the catecholamine surge, in addition to the increase in hydrostatic pressure, acute rise in pulmonary capillary pressure leads to structural damage of pulmonary endothelium, which leads to vascular leakage and persistent protein-rich pulmonary oedema.[32]

Pulmonary venule adrenergic hypersensitivity

The exaggerated sympathetic discharge directly affects the integrity of pulmonary endothelium integrity, and the oedema develops regardless of the changes in systemic and pulmonary circulation. The presence of α- and β-adrenoceptors in pulmonary vascular bed supports the theory.[26]

The symptoms often resolve spontaneously over 24–48 h but may persist in patients with ongoing brain injury and raised ICP. Treatment of NPE is mainly focused on treating the underlying neurological condition to limit the sympathetic discharge responsible for lung injury.[26] Various measures for reduction of raised ICP have shown to be beneficial to improve oxygenation.[29] Few case reports have shown the effectiveness of α-blocking agents for rapid improvement of patients with NPE.[33]

PULMONARY EMBOLISM

Patients with SAH are at higher risk for deep vein thrombosis (DVT) and pulmonary embolism. The incidence of DVT varies from 1.5% to 18%. Poor-grade patients are at highest risk.[34,35] The measures to prevent DVT like the use of sequential compression devices should be used routinely in all the patients as soon as they are admitted to hospital.[36] Venous thromboembolism prophylaxis should be considered with unfractionated heparin at least 24 h after an aneurysm has been secured by coiling or surgical clipping.[37]

CONCLUSION

Pulmonary complications in patients with aneurysmal SAH are the major cause of morbidity and mortality. With the improving critical care management of SAH, the clinicians should consider early detection and appropriate management of pulmonary complications to improve patient outcome.

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Conflicts of interest
There are no conflicts of interest.

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