Pulmonary complications in neurosurgical patients

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ABSTRACT

Pulmonary complications are a major cause of morbidity and mortality in neurosurgical patients. The common pulmonary complications in neurosurgical patients include pneumonia, postoperative atelectasis, respiratory failure, pulmonary embolism, and neurogenic pulmonary edema. Postoperative lung expansion strategies have been shown to be useful in prevention of the postoperative complications in surgical patients. Low tidal volume ventilation should be used in patients who develop acute respiratory distress syndrome. An antibiotic use policy should be put in practice depending on the local patterns of antimicrobial resistance in the hospital. Thromboprophylactic strategies should be used in nonambulatory patients. Meticulous attention should be paid to infection control with a special emphasis on hand-washing practices. Prevention and timely management of these complications can help to decrease the morbidity and mortality associated with pulmonary complications.

Key words: Neurosurgery, post-operative, pulmonary complications

INTRODUCTION

Pulmonary complications, especially postoperative pulmonary complications, are an important cause of morbidity and mortality in neurosurgical patients. Postoperative mechanical ventilation is one of the important risk factors for the development of these complications. Also, craniotomy and intracranial pressure monitoring have been reported as important risk factors for development of pulmonary complications.[1,2] The various pulmonary complications that occur in the neurosurgical patients are vastly similar to those which occur in surgical patients; however a few of these occur predominantly in the neurosurgical patient (like neurogenic pulmonary edema (NPE)).[3] The commonest postoperative complications that have been reported in surgical patients as whole (well studied in thoraco-abdominal surgeries) include postoperative atelectasis, pneumonia, respiratory failure, and exacerbation of an underlying chronic lung disease.[4] A number of important physiological changes occur in the postoperative period following neurosurgery, which contribute to the development of pulmonary complications.[2] The consequent result to the development of these complications is an increased duration of hospital and intensive care unit (ICU) stay which further translates into a vicious circle in which the occurrence of various postoperative complications (like hospital-acquired pneumonia (HAP)) is further exacerbated. In fact, development of postoperative complications may also be associated with a poor neurological outcome in many situations.[1] Therefore, prevention, timely recognition, and appropriate management of these assume great importance for the neurosurgeon.

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INCIDENCE

Pulmonary complications especially with regard to neurosurgical patients have not been an extensively researched area. Only a few authors have focused on this aspect of management of neurosurgical patients. There is good evidence which supports neurosurgery as a risk factor for the development of postoperative pulmonary complications. In an American College of Physicians' (ACP) systematic review of identification of preoperative risk factors for pulmonary complications in noncardiothoracic surgery, neurosurgery was identified as a strong risk factor for the development of postoperative pulmonary complications (pooled estimated odds ratio 2.53 (1.84–3.47)). However, the specific surgery characteristics contributing to the development of these complications were not identified.[4,5]

Sogame *et al*. in a prospective study described the

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incidence of postoperative pulmonary complications and mortality in patients undergoing elective craniotomy and evaluated the association between preoperative and postoperative variables and postoperative pulmonary complications.[1] Over a period of two years, 236 patients were followed up. Postoperative pulmonary complications occurred in 58 patients (24.6%), and 23 patients (10%) died. On multivariate analyses, predictors for occurrence of postoperative pulmonary complications included the following: Infratentorial surgery ($P<0.0001$), mechanical ventilation ≥48 hours ($P<0.0001$), time spent in the ICU >3 days ($P<0.0001$), decrease in level of consciousness ($P<0.002$), duration of surgery ≥300 minutes ($P<0.01$), and previous chronic lung disease ($P<0.04$). The preoperative predictors for development of postoperative pulmonary complications were duration of surgery >300 min and previous chronic lung disease and patients with both these risk factors had a 60% probability of a postoperative pulmonary complication. The probability was 16.7% in patients without these risk factors. The most frequent postoperative pulmonary complication was purulent tracheobronchitis, followed by pneumonia, bronchospasm, and atelectasis.

The factors that contribute to the pathogenesis of pulmonary complications in neurosurgical patients are summarized in Table 1.

COMMON POSTOPERATIVE PULMONARY COMPLICATIONS

Pneumonia (HAP/health-care-associated pneumonia (HCAP)/ventilator-associated pneumonia (VAP))

Pneumonia is the most serious complication that occurs in hospitalized patients (surgical and nonsurgical). HAP is the second most common nosocomial infection that is associated with high morbidity and mortality, increased duration of hospital stay, and increased cost of treatment.[6] Endotracheal intubation is an extremely important contributing factor to the development of pneumonia in hospitalized patients, and in patients who are managed with noninvasive ventilation (NIV), nosocomial pneumonia occurs less frequently.[7] However, the routine use of NIV in neurosurgical patients is contraindicated in most cases. The definitions of HAP, VAP, and HCAP are summarized in Table 2.

These definitions are important because these help in identification of factors that are likely to be associated with the presence of multidrug-resistant (MDR) organisms as a cause of pneumonia and therefore are critically important in deciding the choice of antibiotic therapy and subsequent management. The major difference is that early HAP/VAP is likely to be caused by antibiotic sensitive organisms and associated with a better prognosis. On the other hand, late HAP/VAP is likely caused by antibiotic-resistant (MDR) organisms and associated with higher morbidity and mortality.[7] The common causative organisms in early-onset HAP/VAP include methicillin-sensitive *Staphylococcus aureus*, *Haemophilus influenzae*, *Streptococcus pneumoniae*, Enterobacteriaceae, and anaerobes. In late-onset HAP/VAP, the usual causative pathogens are virulent, multiantibiotic-resistant microorganisms like *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Klebsiella pneumoniae*, *methicillin-resistant S. aureus*, and vancomycin-resistant enterococcus. This is the reason as to why the late-onset infections are usually associated with a high morbidity and mortality. Whenever a patient with pneumonia is being managed, it is extremely important to be aware of the associated risk factors that increase the likelihood of an MDR pathogen being the underlying etiology. Head trauma is a risk factor for

Table 1: Factors contributing to the pathogenesis of pulmonary complications in neurosurgical patients

Respiratory depression—central (due to traumatic brain injury, central nervous system disease, sedative use) or peripheral (muscle disease or neuromuscular blockade)
Spinal cord injury—injury to the spinal cord at high cervical levels can lead to impairment of diaphragmatic function, decreased ability to cough, and decreased ability to handle secretions. Injury at thoracic cord levels can lead to weakness of thoracoabdominal muscles leading to paradoxical respiration, decreased cough strength, and reduction in functional residual capacity
Hyperventilation in patients with raised intracranial pressure—hyperventilation leads to hypocapnia and respiratory alkalosis. This has been found to be associated with increase in the intrapulmonary shunt and shift of the oxyhemoglobin dissociation curve to the left. This can lead to worsening of hypoxemia
Neurogenic pulmonary edema
Impaired consciousness, abnormal swallowing mechanism, delayed gastric emptying, depressed gag reflexes, and decreased gastrointestinal motility can predispose to aspiration
Nasotracheal or orotracheal intubation—predisposes patients to bacterial colonization and nosocomial pneumonia
Associated traumatic airway injuries—in patients who have sustained neurotrauma, there can be associated significant airway injuries like tracheobronchial disruptions, traumatic tracheoesophageal fistula,or diaphragmatic rupture
Associated upper airway injuries—traumatized oropharyngeal tissue, tongue edema, dental trauma, oral secretions, bronchospasm, and oral bleeding can predispose to upper airway obstruction leading to impairment of ventilation
Prolonged immobility can predispose to deep venous thrombosis and pulmonary embolism
Associated malnutrition can lead to neuromuscular weakness and predisposition to infections

S. aureus infection.[7,8] The risk factors for MDR pathogens have been summarized in Table 3.

The bacteriological profile and resistance patterns vary depending on study population, place of study, and geographical boundaries and also over time in the same hospital settings. Therefore, it is critically important to remember that each treating unit be aware of their local hospital epidemiology. In many centers, early HAP/VAP has been reported to be caused by MDR organisms. In the Indian scenario, Gram-negative organisms are the most common cause of HAP/VAP.[9]

DIAGNOSTIC STRATEGY (CLINICAL/ MICROBIOLOGICAL)

The purpose of the diagnostic strategy is to establish whether the patient has pneumonia or not and if yes, then what is the causative organism. The diagnosis of pneumonia should be considered when there is appearance of new or progression of existing radiological infiltrates on the chest radiograph along with new-onset fever, purulent secretions, leukocytosis, and decline in oxygenation status. In patients with acute respiratory distress syndrome (ARDS), the diagnosis of pneumonia should be considered with any one of the above parameters. Another important consideration is in patients who have unexplained hemodynamic instability or worsening hypoxemia during mechanical ventilation.[7,10]

Table 2: Definitions of HAP, VAP, and HCAP

HAP	Pneumonia that occurs 48 h or more after admission, which was not incubating at the time of admission
HCAP	Pneumonia developing in any patient who was hospitalized in an acute care hospital for 2 or more days within 90 days of the infection, received recent intravenous antibiotic therapy, chemotherapy, or wound care within the past 30 days of the current infection or attended a hemodialysis clinic
VAP	Pneumonia that arises more than 48–72 h after endotracheal intubation
	Early VAP/HAP is defined as pneumonia occurring within first 4 days of hospitalization
	Late VAP/HAP is defined as pneumonia occurring after 5 days of hospitalization

HAP – Hospital-acquired pneumonia; HCAP – Health-care-associated pneumonia; VAP – Ventilator-associated pneumonia; Reference: American Thoracic Society Documents. Am J Respir Crit Care Med 2005. 171; 388–416

Table 3: Risk factors for infection with a MDR organism in HAP, HCAP, and VAP

Antimicrobial therapy in preceding 90 days
Current hospitalization of 5 days or more
High frequency of antibiotic resistance in the community or in the specific hospital unit
Presence of risk factors for HCAP Hospitalization for 2 d or more in the preceding 90 days Residence in a nursing home or extended care facility Home infusion therapy (including antibiotics) Chronic dialysis within 30 days Home wound care Family member with MDR pathogen
Immunosuppressive disease and/or therapy

HAP – Hospital-acquired pneumonia; HCAP – Health-care-associated pneumonia; VAP – Ventilator-associated pneumonia; Reference: American Thoracic Society Documents. Am J Respir Crit Care Med 2005;171:388-416

There are two approaches to the diagnosis of HAP/VAP. These include

1. Clinical strategy
2. Bacteriologic strategy

Clinical strategy

The clinical strategy is based on a combination of clinical suspicion of pneumonia (the presence of a new or progressive radiographic infiltrate plus at least two of following: Fever greater than 38°C, leucocytosis or leucopenia, and purulent secretions) and a semiquantitative evidence of infection based on Gram staining and culture of tracheal aspirates. The antibiotics are selected based on the risk factors for specific pathogens and knowledge of local patterns of antibiotic resistance and organism prevalence. The main advantage of this strategy is that it requires no specialized microbiological methods for diagnosis and all patients suspected of having pneumonia are treated promptly. However, this strategy does have few limitations which include over usage of antibiotics, treatment of noninfectious conditions like pneumonia and failure of early recognition of extra pulmonary infections.[7,11]

Bacteriologic strategy

The bacteriologic strategy relies on use of quantitative cultures of lower respiratory secretions [endotracheal aspirates, bronchoalveolar lavage (BAL) or protected specimen brush specimens collected with or without a bronchoscope] to define both the presence of pneumonia and establish the etiologic pathogen.[7] Microbiological growth above a threshold concentration is required for the diagnosis and growth below the threshold is assumed to be due to colonization or sample contamination. The advantages of this approach include targeted antibiotic therapy, separation of colonizers from infecting pathogens, and avoidance of over-usage of antibiotics. The limitations of this strategy include likelihood of false-negative cultures especially when antibiotics have been administered in preceding 72 h, absence of a "gold standard" with which diagnostic results can be compared, and the overall general consensus by most investigators that clinically unstable patients with signs of infection be administered broad-spectrum antibiotics regardless of the bacteriologic results. Quantitative cultures can be performed on endotracheal aspirates or samples collected

either bronchoscopically or nonbronchoscopically, and each technique has its own diagnostic threshold and methodological limitations. The choice of method depends on local expertise, experience, availability, and cost.

Clinical Pulmonary Infection Score

Clinical pulmonary infection score (CPIS) is a tool that was described to objectify the diagnosis of patients with suspected VAP. CPIS combines clinical, radiographic, physiological (PaO_2/FiO_2), and microbiologic data into a single numerical result. CPIS >6 has demonstrated a good correlation with the presence of pneumonia as defined by quantitative cultures of bronchoscopic and nonbronchoscopic BAL specimens.[12] The score can be calculated as described in Table 4. A modified CPIS was described by Singh *et al.* which did not rely on culture data to guide clinical management.[13]

In the Indian setting where resource limitation can be a constraint, a tracheal aspirate Gram stain along with CPIS can be a useful strategy for diagnosis of VAP which can help in guiding initial empirical therapy. A negative tracheal aspirate for Gram stain should point toward a nonpulmonary cause of fever and in patients with CPIS >6 HAP/VAP should be suspected. In patients with a modified CPIS ≤6 but a strong suspicion of VAP/HAP, reevaluation should be done after 48–72 h. Patients in whom initial modified CPIS is ≤6 on day 1, a repeat score of ≤6 after bacteriology results makes diagnosis of HAP/VAP less likely. It is also important to remember that therapy should not be delayed in clinically unstable patients for the purpose of performing diagnostic sampling and a combined clinical and bacteriological strategy yields better results in diagnosing HAP/VAP.

Table 4: Clinical pulmonary infection score

Temperature (°C)	Points
≥36.5 and ≤38.4	0
≥38.5 and ≤38.9	1
≥39 and ≤36.5	2
Blood leukocytes (/mm³)	
≥4,000 and ≤11,000	0
< 4,000 or >11,000	1
+ band forms ≥50%	Add 1 point
Tracheal secretions	
Absence of tracheal secretions	0
Presence of nonpurulent tracheal secretions	1
Presence of purulent tracheal secretions	2
Oxygenation: PaO_2/FIO_2	
>240 or ARDS	0
<240 and No ARDS	2
Pulmonary radiography	
No infiltrate	0
Diffuse (or patchy) infiltrate	1
Localized infiltrate	2
Progression of pulmonary infiltrate	
No radiographic progression	0
Radiographic progression (after CHF and ARDS excluded)	2
Culture of tracheal aspirate	
Pathogenic bacteria cultured in rare or light quantity or no growth	0
Pathogenic bacteria cultured in moderate or heavy quantity	1
Same pathogenic bacteria seen on Gram stain	Add 1 point

References: Pugin and colleagues. (12), Singh and colleagues. (13)

TREATMENT

Arterial blood gas and saturation monitoring should be done routinely. Blood cultures and samples of lower respiratory tract secretions should always be obtained from all patients with suspected HAP/VAP before initiating or changing antibiotics. Samples can include an endotracheal aspirate, BAL, or protected brush specimen. A diagnostic thoracocentesis should be performed in patients with pleural effusions to guide appropriate management. After a decision to initiate treatment of HAP/VAP has been taken, timely initiation of empiric antibiotic therapy which is appropriate, adequate, and optimal should be done. After the microbiological and sensitivity results are available, an attempt to de-escalate antibiotics should always be done. Short-course antimicrobial therapy should be preferred and prolonged continuation of antibiotics despite clinical resolution of pneumonia should be discouraged. Ideally, every ICU should have its own antibiotic policy to initiate empiric antibiotic therapy in suspected nosocomial infections. Any deviation from the policy should be based on strong evidence. Formulation of antibiotic policy should be based on culture and sensitivity reports obtained over the previous month or an active surveillance program wherein twice-weekly endotracheal aspirate sampling is performed for all mechanically ventilated ICU patients.

Neurogenic Pulmonary Edema

NPE is a syndrome characterized by the acute onset of pulmonary edema following significant central nervous system (CNS) injury. The diagnosis is made after the exclusion of other plausible causes. Characteristically, the onset occurs within minutes to hours after a neurologic insult and usually resolution occurs within 72 h. The typical presentation is acute development of dyspnoea, tachypnoea, and hypoxemia within minutes. Pink, frothy sputum is commonly seen and bilateral crackles are audible on auscultation. If untreated or inappropriately managed, the condition is associated with high mortality.[3] The most commonly associated CNS injury is subarachnoid hemorrhage accounting for more than two-thirds of reported cases. The incidence is 23% following subarachnoid hemorrhage, 20% following severe head injury, and about 33% among patients with status epilepticus. Rare causes

include multiple sclerosis, brain tumor, encephalitis, cervical spine injury, and ischemic stroke.[14]

The etiology is proposed to be related to a catecholamine surge associated with significant CNS injury that results in cardiopulmonary dysfunction, increased pulmonary hydrostatic pressure, and increased lung capillary permeability related to the inflammatory response. Two distinct clinical forms—cardiogenic and noncardiogenic—have been proposed, distinction between which may have a therapeutic implication. Diagnostic criteria for noncardiogenic subset of NPE have been proposed which include (1) bilateral infiltrates; (2) PaO_2/FiO_2 ratio <200; (3), no evidence of left atrial hypertension; (4) presence of CNS injury (severe enough to have caused significantly increased ICP); and (5) absence of other common causes of acute respiratory distress or ARDS. Measurement of serum catecholamines may be helpful in this subgroup and a trial of an α-adrenergic blocking agent, such as phentolamine, can be considered. Overall, treatment is usually supportive.[3]

Lung Atelectasis

A number of factors contribute to the development of lung atelectasis in neurosurgical patients. These include depression of the respiratory drive, impaired consciousness, cervical and thoracic spine injury, pain contributing to hypoventilation, bronchial obstruction from mucous plugs or aspiration of particulate matter, and compression of adjacent lung by traumatic pneumothorax, hemothorax, or pleural effusion.

Deep Venous Thrombosis and Pulmonary Embolism

The incidence of Deep venous thrombosis (DVT) in neurosurgical patients varies between 18% and 50%.[15,16] The rate of pulmonary embolism (PE) in untreated neurosurgical patients has been reported to range from 0.5% to 25% with a mortality rate of nearly 60%.[15-17] Tumors (benign or malignant), spinal cord and head trauma, hemorrhagic or ischemic stroke, long duration of surgery, and decreased mobility are risk factors for venous thromboembolism in neurosurgical patients.[16,18] Certain tumors like astrocytomas can induce a hypercoagulable state.[19]

The diagnosis of PTE should be considered in any neurosurgical patient developing acute onset dyspnea, hypoxemia, or hypotension. Significant PE can lead to sudden cardiovascular collapse. Clinical prediction rules can help in assessment of a low or high likelihood of DVT/PE.[20] In patients with low or intermediate risk of DVT/PE, initial screening can be done by d-dimer estimation. If the value is below threshold, diagnosis is ruled out. In case the value is above threshold, lower limb compression ultrasound or multidetector CT (MDCT) angiography should be performed, which are the initial investigations of choice if there is a high probability for DVT/PE. If compression ultrasound or MDCT is positive, diagnosis is ruled in or else ruled out. In patients who are hemodynamically unstable, bedside echocardiography is an important investigation which can point toward the presence of hemodynamically significant PE. Both mechanical and pharmacological thromboprophylactic methods have been shown to reduce DVT formation. Mechanical prophylaxis methods include early ambulation, intermittent pneumatic compression, and sequential compression devices which have usually been the standards of care in neurosurgical patients in view of reduction in haemorrhagic complications. Pharmacological methods like low-dose subcutaneous unfractionated heparin (5000 U every 8–12 h) and low-molecular-weight heparins (such as enoxaparin) have demonstrated efficacy in reduction in venous thromboembolism rates without significant increases in bleeding complications. Warfarin, aspirin, other heparinoids, and thrombin inhibitors (danaparoid, hirudin, fondaparinux, ximelagatran) have also been used for this purpose. Thromboprophylaxis is an extremely important intervention which should be considered in all neurosurgical patients.[18]

Acute Lung Injury/Acute Respiratory Distress Syndrome

ARDS is a pathophysiological syndrome defined as acute onset, bilateral lung infiltrates, no evidence of elevated left atrial pressure, and arterial oxygen tension to inspired oxygen fraction (PaO_2/FiO_2) less than 200. The condition is defined as Acute lung injury (ALI) when PaO_2/FiO_2 is between 200 and 300 along with fulfilment of rest of the criteria. ARDS is a syndrome associated with multiple diagnoses like pneumonia, gastric aspiration, alveolar haemorrhage, and is not a disease in itself, and occurs frequently in neurosurgical patients.[21] Neurogenic pulmonary edema is a specific cause of ARDS which is seen in neurological and neurosurgical patients.

The only method of mechanical ventilation that has proven to reduce the mortality rate in the management of patients with ARDS is low tidal volume ventilation (i.e. 6 mL/kg predicted body weight) with the maintenance of plateau pressures of <30 cm H_2O, whenever possible. Other methods such as high-PEEP, recruitment manoeuvres, high-frequency ventilation and prone positioning may be used as rescue therapy in life-threatening hypoxemia but their routine use cannot be recommended as they have not been shown to confer mortality benefit in a broad population of patients. In addition, careful attention must also be given to supportive treatment strategies like avoidance or minimization of sedation and neuromuscular blockade and using a conservative fluid strategy. In patients with refractory hypoxemia, extra corporeal membrane oxygenation can be used if facilities permit.

Strategies for Prevention of Pulmonary Complications

It is prudent to be aware of and make certain simple strategies a part of the routine protocol of care in ICUs as well as ward patients so as to reduce the various pulmonary complications in the postoperative period. Pulmonary embolism is a killer. It is imperative that neurosurgical patients receive thromboprophylaxis by either mechanical or pharmacological means, through their nonambulatory period. Most VTE occurs early (within the first week) after a neurosurgical intervention, hence this is the period in which prophylactic strategies are likely to have the greatest benefit.[18] In 2006, ACP guidelines were published on "Risk Assessment for and Strategies To Reduce Perioperative Pulmonary Complications for Patients Undergoing Noncardiothoracic Surgery" and evidence grades were assigned to various strategies to reduce risk for postoperative pulmonary complications based on a systematic review of the literature.[22,23] Postoperative lung expansion modalities, which comprise incentive spirometry, deep breathing exercises, intermittent positive-pressure breathing, and continuous positive airway pressure were the only strategy that were supported by good evidence for reduction in postoperative pulmonary complications. Fair evidence supported selective postoperative use of nasogastric tubes and use of short-acting neuromuscular blockade.[22,23] Orotracheal intubation is preferable for urgent intubation in neurosurgical patients in the presence of increased ICP, hypoxemia, and/or hemodynamic instability. Nasotracheal intubation has minimal role in neurosurgical patients as it can be associated with risk of sinusitis and pneumonia. Nasotracheal intubation is absolutely contraindicated in the presence of a basilar skull fracture. High cervical spinal cord injury is an absolute indication for tracheostomy. In patients with coexistent severe facial trauma, emergency tracheotomy or cricothyroidotomy could be required as it can hamper bag-and-mask ventilation. The advantages of tracheostomy include decrease in the anatomical dead space, reduced work of breathing, ease of suctioning, improved patient comfort, and reduced sedation requirements. Bedside percutaneous dilatational tracheostomy has increasingly become the technique of choice for tracheostomy in neurosurgical patients.[24] Early tracheostomy should be considered in patients who are likely to require a prolonged duration of mechanical ventilation.

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