CLINICAL VIGNETTE

Ventilator Associated Pneumonia (VAP) in Burn Patients

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Background

In early 2015 UCLA pulmonary/critical physicians were asked to provide critical care services to the critically ill burn patients in Grossman Burn Center. Critically ill burn patients have unique needs that are different from other critically ill patients, and their care demands a better understanding of burn physiology and its complications.

Below is a brief clinical review of one of the most common clinical problems we encounter in Burn ICUs.

Clinical Problem

Critically ill burn patients are uniquely susceptible to infectious complications, as significant burn injuries induce a state of immunosuppression. Nosocomial pneumonia, including Ventilator Associated Pneumonia (VAP), remains an important cause of morbidity and mortality in these patients. Reports from single center studies estimate the incidence of VAP as 22 to 26 per 1000 ventilator days in burn patients. This is more than double than in medical ICUs where VAP rates ranges from 1.2 to 8.5 per 1000 ventilator days. Burn patients with inhalational injury are the most vulnerable for developing VAP as it causes destruction of the airway lining resulting in loss of protective function. In addition, the necrotic endothelium of the airways becomes an excellent growth medium for microorganisms. Inhalational injury is also an important predictor of prolonged ventilator dependence that also increases the risk of VAP. Pulmonary complications are also common in burn patients without inhalational injury as reduced lung expansion associated with chest and abdominal burns makes them susceptible to atelectasis and hydrostatic pneumonia. These patients are also at high risk for aspiration due to high dose narcotics for pain control.

Diagnosis

Despite its frequent occurrence, no clinical “gold standard” exists for the diagnosis of VAP. Fevers, purulent sputum, leukocytosis, decreased oxygenation, and abnormal chest radiographs can commonly occur in burn patients due to non-infectious causes, making diagnosis of VAP particularly challenging. Scoring systems such as CPIS and CDC criteria have not been validated in burn victims and presence of overwhelming systemic inflammation in these patients limit its application in clinical setting. Sputum cultures and tracheal aspirates do not accurately reflect the presence or absence of infectious organisms in the lower airways because of the rapid colonization of the upper airways and endotracheal tubes. American Burn Association (ABA) recommends that if VAP is clinically suspected, then quantitative cultures from the lower airways using BAL and/or protected specimen brushings be obtained, whenever possible, to confirm VAP diagnosis. Greater than 100,000 cfu/mL for BAL and 10,000 cfu/mL for protected specimen brush is considered the threshold for positivity in these patients.

Treatment

Several studies indicated that a delay in initiating appropriate antibiotics may result in increased morbidity and mortality. Early onset pneumonia, within the first 4 days, usually carries a better prognosis and is more likely community acquired, most notably Streptococcus pneumoniae and Haemophilus influenzae. Later onset VAPs are more likely a consequence of MRSA and gram negative organisms like Pseudomonas aeruginosa, E. coli, Klebsiella pneumoniae, and Acinetobacter baumannii. Broad spectrum antibiotics should be replaced with more specific treatment, preferably within 48 hours, based on quantitative cultures and sensitivity. Monotherapy is preferred. The duration of antibiotic therapy should be limited to 8 days for VAP, unless caused by MRSA or non-lactose fermenting gram negative rods, in which case, be continued for 15 days.

VAP Prevention Strategies

Although considerable research has been devoted to VAP prevention, few studies have focused specifically on burn patients. Therefore, strategies to prevent VAP in burn patients have largely been adapted from studies on other patient groups.

Here is the list of preventive strategies recommended by ABA:

1. Avoid unnecessary intubation and re-intubation (class 1). (See Table)

   Intubation and mechanical ventilation increases the risk of VAP by 6-21 fold. This is because intubation neutralizes many of the normal respiratory tract defenses against infection by bypassing the glottic barrier, allowing leakage of contaminated secretions around the endotracheal cuff and pooling into the lower airways. ABA recommends that attempts should be made to avoid unnecessary intubations and necessary intubations should be performed under
controlled conditions as much as possible (Grade A recommendation).

2. Implement ventilator weaning and sedation protocols (class 1).
   Daily interruption of sedation and weaning protocols has become a fundamental strategy to reduce the duration of mechanical ventilation in ICU patients. Although very few burn patients were included in these studies, centers that have adopted the practice of daily spontaneous breathing trials report an improvement in extubation rates without an increase in reintubation rates.12,13

3. Use of specialized endotracheal tubes (class 1).
   Use of specialized endotracheal tubes has been investigated in several studies in an attempt to reduce the incidence of VAP. The NASCENT trial14 showed a 35.9% risk reduction when a silver impregnated endotracheal tube was used. Techniques to continuously aspirate subglottic secretions have also been shown to be effective.

4. Reducing aspiration From Gastrointestinal Tract sources.
   Apart from maintaining mechanically ventilated burn patients in the semirecumbent position, especially when enteraly fed, the ABA also suggests post-pyloric feeding, whenever possible. Three randomized controlled trials comparing gastric with post-pyloric feeding found an 8-15% absolute reduction in VAP with post-pyloric feeding.15 However, they do not recommend delaying enteral feedings while awaiting placement of post-pyloric feeding tubes.

5. Topical oral antiseptics (e.g., chlorhexidine) (Class 1).
   Chlorhexidine mouthwash is cost-effective and has been shown in many studies to be an effective strategy for reducing VAP.

6. Stress ulcer prophylaxis and early enteral nutrition (Class 1).
   These are now well-established practices in both burn and adult ICUs. However, there have been concerns that raising pH increases gastric bacterial colonization, which predisposes to VAP. Gastritis is rare in the well resuscitated burn victim who is started on early enteral nutrition. Sucralfate may be a better alternative to other agents that raise gastric pH.

7. Avoid unnecessary red blood cell transfusions (Class 1).
   Large burn injury induces a state of immunosuppression, and blood transfusions add to the immune compromised state.6,17 The American Burn Association Burn Multicenter Trials group examined the effect of blood transfusion in 666 patients at 21 burn centers with acute burn injuries greater than 20% TBSA (Total Body Surface Area) and found a 13% increased risk of infection with each unit of blood transfused. VAP occurred in 42% of those transfused and only in 6% of those who did not receive a blood transfusion.18 Hence, ABA recommends a restrictive transfusion policy in mechanically ventilated burn patients.

Conclusion
Ventilated Associated Pneumonia is common in mechanically ventilated burn patients, and inhalation injury is a unique risk factor in this patient population. With our experience in Burn ICU, we have recognized that prevention, accurate diagnosis, and treatment of VAP are important components of the care of the mechanically ventilated burn patients.

| Relation Between Levels of Evidence and Grades of Recommendations19 |
|-------------------|-------------------|
| **Level of Evidence** | **Grade of Recommendations** |
| Level I: Large randomized trials with clear-cut results (and low-risk of error) | A |
| Level II: Small randomized trials with uncertain results (and moderate- to high-risk of error) | B |
| Level III: Nonrandomized, contemporaneous controls | |
| Level IV: Nonrandomized, historical controls | |
| Level V: No controls, case-series only | C |

REFERENCES


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