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The effect of smoking status on burn inhalation injury mortality

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ABSTRACT

Introduction: Three factors that effect burn mortality are age, total body surface of burn (TBSA), and inhalation injury. Of the three, inhalation injury is the strongest predictor of mortality thus its inclusion in the revised Baux score (age+TBSA+17* (inhalation injury, 1=yes, 0=no)). However, the weighted contribution of specific comorbidities such as smoker status on mortality has traditionally not been accounted for nor studied in this subset of burn patients. We therefore sought to examine the impact of current tobacco and/or marijuana smoking in patients with inhalation injury.

Methods: A retrospective analysis of patients admitted to a regional burn center from 2002 to 2012. Independent variables analyzed included basic demographics, burn mechanism, presence of inhalation injury, TBSA, pre-existing comorbidities, and smoker status. Bivariate analysis was performed and logistic regression modeling using significant variables was utilized to estimate odds of mortality.

Results: There were a total of 7640 patients over the study period. 7% (n=580) of the burn cohort with inhalation injury were included in this study. In-hospital burn mortality for inhalation injury patients was 23%. Current smokers (20%) included cigarette smokers and marijuana users, 19% and 3%, respectively. Preexisting respiratory disease (17%) was present in 36% of smokers compared to 13% of non-smokers ($p < 0.001$). Smokers had significantly lower mortality rate (9%) compared to non-smokers (26%, $p < 0.01$). The logistic regression model for mortality outcomes identified statistically four significant variables: age, TBSA, ethnicity, and smoker status (OR=0.41, 95% CI=0.18-0.93). Presence of comorbidities, including preexisting respiratory disease, was not significant.

Conclusion: In the sub group of burn patients with inhalation injury, the odds of mortality significantly decreased in pre-existing smokers after adjusting for significant covariates. We postulate that an immune tolerance mechanism that modulates and diminishes the pro-inflammatory response confers a survival advantage in smokers after exposure to acute smoke inhalation injury. Future prospective studies in human and/or animal models are needed to confirm these findings.

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1. Introduction

Burn is one of the most devastating traumatic injuries affecting an estimated 486,000 people in the United States in 2015 [1]. Advancements over the past three decades in burn care such as measured fluid resuscitation, improved critical care management, and early excision and grafting have resulted in improved burn outcomes. Despite these advances, the three major determinants of increased burn mortality include age >60 years old, % total body surface area (%TBSA) >40%, and presence of inhalation injury [2–5]. Inhalation injury is considered to be the strongest predictor of burn mortality [2,4]. To better prognosticate burn outcomes, the Baux score was created [6,7], however, to account for the weighted contribution of inhalational injury to burn mortality, it was later revised to include this predictor variable (age + percent burn + 17*) (inhalation injury, 1=yes, 0=no) [8,9]. More recently, with the recognition of increased longevity of the US population and its health related sequelae, it is paramount that future burn mortality prediction models account for pre-existing comorbidities in the prognostication of injury outcomes. Specifically the role of pre-existing respiratory disease markers such as smoking in the subset of burn patients with inhalational injury.

Smoking is the single largest preventable cause of death and disease in the United States [10]. It was estimated in 2014, that 16.8% (40 million) adults in the United States are current smokers of tobacco or marijuana. Of which 30.7 million smoked every day. Majority of smokers range between 18 and 64 years of age. There are a variety of harmful substances in tobacco smoke and marijuana that impair mucociliary clearance, damage the cell lining of the trachea, bronchus and bronchioles, and kill cells in the lungs that are responsible for removing dust and bacteria leading to more mucus production [11–15]. Toxins liberated during smoking can cause damage to lung airways and alveoli leading to emphysema, and chronic bronchitis [16,17]. Marijuana also can suppress the immune system that could lead to increased risk of lower respiratory tract infection in these smokers [16].

There have been no previous studies examining the independent effect of smoking on burn mortality in patients with inhalation injury. As the pathologic pulmonary manifestation of smoking is akin to chronic inhalational injury, we hypothesize that there will be an increased mortality in burn patients with inhalational injury that are current smokers at the time of the injury as compared to non-smokers.

2. Methods

This is a retrospective study of all burn patients admitted to the University of North Carolina Jaycee Burn Center from 2001 to 2012. The North Carolina Jaycee Burn Center at UNC was established in 1981 and averages more than 1200 acute admissions per year. The burn center is a single unit, 36-bed facility that has been verified by the American Burn Association for pediatric and adult care.

The medical records of subjects identified by the UNC Burn database query were reviewed to verify baseline demographic

data, injury characteristics, and provide detailed information and associated preexisting comorbidities. Injury characteristics of interest included burn etiology, %TBSA burn, presence of inhalation injury, and intubation status on admission to the burn center. Inhalation injury diagnosis is based on history and confirmed by fiberoptic bronchoscopy examination per our clinical protocol. Per National Burn Data Standards (NBDS) created by the American Burn Association, inhalation injury is recorded in the burn registry as either absent or present with and without cutaneous burn based on medical records. Other useful modalities such as chest-computed tomography (CT), radionuclide imaging with ¹³³Xenon, carbon monoxide levels and pulmonary function testing results are not part of our inhalational injury protocol.

Preexisting comorbidities were weighted using the Charlson Comorbidity Index (CCI) score [18,19]. The score is the weighted sum of comorbid conditions. There are 17 comorbid conditions included in the score and each is assigned a weight from 1 to 6 points (Table 1). Pre-existing comorbidities were identified utilizing the National Trauma Data Bank (NTDB) from the American College of Surgeons Committee on Trauma. Current smoking status was obtained from medical records and recorded into the burn registry. Smoking status is reported from patient, family member, or persons living with the patient or with intimate knowledge of patient's smoking habits upon admission to the burn center. In addition to tobacco smoke, marijuana use information was enquired.

Our outcome of interest is in-hospital mortality in patients with inhalation injury. Baseline patient and injury characteristics were compared between groups for mortality and smoker status using Analysis of Variance for continuous variables and chi-squared for categorical variables. Kruskal-Wallis test was used to compare medians. We employed both bivariate analyses to determine the relative influence of smoking on mortality among covariates. To determine odds of mortality, we used a multivariate logistic regression model controlling for pertinent confounders (age, TBSA, ethnicity,

Table 1 – Charlson Comorbidity Index score system.

Comorbidity	Score
Myocardial infarction	1
Congestive heart failure	1
Peripheral vascular disease	1
Cerebrovascular disease	1
Dementia	1
Chronic pulmonary disease	1
Rheumatologic disease	1
Peptic ulcer disease	1
Mild liver disease	1
Diabetes without chronic complications	1
Diabetes with chronic complications	2
Hemiplegia or paraplegia	2
Renal disease	2
Any malignancy, including leukemia and lymphoma	2
Moderate or severe liver disease	3
AIDS/HIV	6
Metastatic solid tumor	6
Maximum comorbidity score	33

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