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ORIGINAL ARTICLE

The effects of smoking on adolescent trauma patients: a propensity-score-matched analysis

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Abstract

Purpose Cigarettes have been demonstrated to be toxic to the pulmonary connective tissue by impairing the lung's ability to clear debris, resulting in infection and acute respiratory distress syndrome (ARDS). Approximately 8% of adolescents are smokers. We hypothesized that adolescent trauma patients who smoke have a higher rate of ARDS and pneumonia when compared to non-smokers.

Methods The Trauma Quality Improvement Program (2014–2016) was queried for adolescent trauma patients aged 13–17 years. Adolescent smokers were 1:2 propensity-score-matched to non-smokers based on age, comorbidities, and injury type. Data were analyzed using chi square for categorical data and Mann–Whitney *U* test for continuous data.

Results From 32,610 adolescent patients, 997 (3.1%) were smokers. After matching, 459 smokers were compared to 918 non-smokers. There were no differences in matched characteristics. Compared to non-smokers, smokers had an increased rate of pneumonia (3.1% vs. 1.1%, $p=0.01$) but not ARDS (0.2% vs. 0%, $p=0.16$). Compared to the non-smoking group, the smokers had a longer median total hospital length-of-stay (3 vs. 2 days, $p=0.01$) and no difference in overall mortality (1.5% vs. 2.4%, $p=0.29$).

Conclusion Smoking is associated with an increased rate of pneumonia in adolescent trauma patients. Future research should target smoking cessation and/or interventions to mitigate the deleterious effects of smoking in this population.

Keywords Pediatric · Smokers · Trauma surgery · ARDS · Pneumonia · TQIP

Introduction

Tobacco consumption is currently the leading cause of preventable death worldwide, claiming approximately 5 million lives every year since 1990 [1, 2]. Although cigarette use among the youth has been declining since 2011, more than 300 adolescents each day become daily cigarette smokers in the United States [3]. Teenagers who smoke cigarettes are more likely to use other substances (i.e., alcohol and marijuana) and be involved in risky behaviors, such as violence, theft, and suicide [4, 5]; this behavior increases the risk of being involved in trauma [6].

There are over 7000 chemicals in tobacco smoke [7]. Acrolein, one of the cigarette's many carcinogens, has been demonstrated to increase lung vascular permeability by disrupting adherens junctions and actin fibers [8, 9]. Other chemicals in cigarette smoke are known to cause cellular insult, which increases the risk of bacterial pneumonia [10, 11]. Additionally, cigarette smoking causes an increase in reactive oxygen species and decreases surfactant production, both of which are underlying pathophysiological mechanisms of acute respiratory distress syndrome (ARDS) [12–17]. Approximately 200,000 patients in the United States develop ARDS each year, with up to 4.4% occurring in trauma patients [18, 19]. One retrospective cohort study suggested that 50% of ARDS cases were associated with cigarette smoking [20]. Similarly, a prospective study on blunt trauma patients found that cigarette smoking increased platelet aggregation and was associated with an increased

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risk of ARDS [21]. Additionally, there are studies that link smoking to the development of pneumonia [22, 23], and many studies show a high incidence of pneumonia in trauma patients [24–27]. However, to our knowledge, there are no studies evaluating the rate of pneumonia and ARDS in adolescent trauma smokers. We hypothesized that adolescent smokers with traumatic injury have a higher rate of ARDS and pneumonia compared to non-smokers. In addition, we also sought to provide a descriptive analysis of adolescent smokers involved in trauma.

Methods

This study was approved by the Institutional Review Board at the University of California, Irvine. A retrospective analysis of the Pediatric Trauma Quality Improvement Program (TQIP) was performed between 2014 and 2016 to identify adolescent trauma patients between the ages of 13 and 17. Patients who were current smokers were compared to non-smokers. This is listed as one of the 30-required comorbidities to be reported in TQIP and is defined by "A patient who reports smoking cigarettes every day or some days within the last 12 months. This excludes patients who smoke cigars or pipes or use smokeless tobacco (chewing tobacco or snuff)".

The primary outcomes were the development of ARDS or pneumonia. Other measured outcomes included total hospital length of stay (LOS), intensive care unit (ICU) LOS, ventilator days, in-hospital mortality, and other in-hospital complications including cardiac arrest, acute kidney injury (AKI), decubitus ulcer, deep site infection, compartment syndrome, deep vein thrombosis (DVT), pulmonary embolism, and cerebrovascular accident.

Frequency statistics were performed for all groups. A Mann–Whitney *U* test was used to compare continuous variables and chi-square was used to compare categorical variables for bivariate analysis. Categorical data were reported as percentages, and continuous data were reported as medians with interquartile range or as means with standard deviations. Due to the observed imbalance in the sample size between the two groups, smokers were matched with non-smokers using a 1:2 propensity score model based on age, gender, hypotension on admission (systolic blood pressure < 90 mmHg), injury severity score (ISS), as well as injuries to the brain, spine, upper and lower extremity, lung, heart, stomach, small intestine, colorectal, pancreas, liver, spleen, and kidney. Injuries were defined by appropriate international-classification of diseases version-9 (ICD-9) diagnosis codes. We included in our analysis only those cases that were within 0.001 of the estimated logit. This technique of defining the closeness of a matched case is termed caliper matching and is a validated method of emulating randomization in observational studies [28]. Once propensity scores were calculated for each case, one smoker and two non-smoking-matched patients were identified from the sample. If a patient that smoked did not have a close match available, they were excluded from any further analysis. All *p* values were two sided, with a statistical significance level of < 0.05. All analyses were performed with IBM SPSS Statistics for Windows (Version 24, IBM Corp., Armonk, NY).

Results

Demographics and injury types

From 32,610 adolescent trauma patients, 997 (3.1%) were smokers. Of the smokers, 759 (76.1%) were male with a median age of 17 years and a median ISS of 9. After propensity matching, 459 smokers were compared to 918 non-smokers. There were 538 patients who did not fit into our propensity-matched model, and thus, were excluded from the analysis. There were no differences among the smokers and non-smokers with respect to median age (16 vs. 16 years, $p = 0.99$), male gender (75.6% vs. 73.7%, $p = 0.51$), hypotension on admission (1.3% vs. 1.9%, $p = 0.46$), and median ISS (9 vs. 9, $p = 0.20$) (Table 1). The injury profile was similar

Table 1 Demographics of adolescent trauma patients stratified by smoking status

Characteristic	All smokers ($n = 997$)	Non-smokers ($n = 918$)	Smokers ($n = 459$)	*p* value
Age, year, median (IQR)	17.0 (16, 17)	16.0 (16, 17)	16.0 (16, 17)	0.99
Male, *n* (%)	759 (76.1%)	677 (73.7%)	346 (75.4%)	0.51
ISS, median (IQR)	9.0 (6, 12)	9 (5, 12)	9 (5, 13)	0.20
Hypotensive on admission, *n* (%)	14 (1.4%)	17 (1.9%)	6 (1.3%)	0.46
Comorbidities, *n* (%)				
Diabetes	3 (0.3%)	0	1 (0.2%)	0.16
Hypertension	4 (0.4%)	2 (0.2%)	2 (0.4%)	0.48
COPD	19 (1.9%)	15 (1.6%)	12 (2.6%)	0.22

ISS injury severity score, *IQR* interquartile range, *COPD* chronic obstructive pulmonary disease

for both groups, with most patients sustaining blunt trauma (86.1% vs. 89.2%, $p=0.10$), and presenting with a traumatic brain injury (38.6% vs. 39.0%, $p=0.88$) (Table 2).

Primary outcomes and hospital outcomes/complications

Compared to non-smokers, smokers had an increased rate of pneumonia (3.1% vs. 1.1%, $p=0.01$). There was no difference in the rate of all other in-hospital complications including ARDS (0.2% vs. 0%, $p=0.16$), DVT (0.2% vs. 1.0%, $p=0.12$), and AKI (0% vs. 0.1%, $p=0.48$).

Smokers had a longer median hospital LOS (3 vs. 2 days, $p=0.01$) compared to non-smokers. There was no difference in mortality (1.5% vs. 2.4%, $p=0.29$), ICU LOS (3 vs. 2 days, $p=0.55$), or ventilator days (3 vs. 2 days, $p=0.75$) between the two groups (Table 3).

Discussion

While there is existing literature on the effects of smoking on adult trauma patient outcomes, to our knowledge, this is the first study to evaluate the effects of smoking on outcomes in the adolescent trauma patient population. Our retrospective analysis reported that 3.1% of adolescent trauma patients were cigarette smokers. Compared to non-smoking trauma patients, smokers were more likely to develop pneumonia and have longer LOS, but there was no difference between the groups with respect to mortality or ARDS, the latter being an extremely rare complication in this population.

Cigarette smoking impedes the lung's healing ability and predisposes it to injury following an inciting event. Previous studies on adult smokers have demonstrated increased rates of ARDS secondary to lung injury when compared to non-smokers in both critically ill and trauma patients [21, 29–32]. In a study of 74 patients by Panzer et al., critically ill adult trauma smokers had an altered respiratory bacterial community including enrichment of *Enterobacteriaceae* associated with an increased incidence of ARDS [29]. There is a progressive increase in incidence of ARDS following trauma with increasing age, with a peak incidence in patients aged 60–69 years [33]. Our results demonstrated a low incidence (0.3%) of ARDS in the adolescent trauma population, with all cases occurring in smokers; however, this was not statistically significant in comparison to the non-smokers. Our findings are consistent with other pediatric studies, which report a low incidence of ARDS in non-trauma patients. Furthermore, the rate of ARDS has previously been shown to be low specifically in pediatric trauma patients with Roulet et al. finding an incidence of 0.5% of all pediatric trauma admissions and Killen et al. demonstrating an incidence of 1.8% of all pediatric patients admitted to an ICU [34, 35]. Lee et al. postulated the lower rate of ARDS and associated mortality in younger patients is due to greater capacity for repair, and altered cytokine burden including interleukin-1 and -8, and tissue necrosis factor alpha [36]. In addition, there is also likely a dose/duration association with smoking that may have not reached a critical threshold for these adolescents [37]. Future investigations regarding the pathophysiology that protects adolescents from developing ARDS appears warranted.

Smoking increases the risk of inflammatory and infectious complications in trauma patients. Bagaitkar et al. demonstrated that adult patients who smoke cigarettes have a 2.6-fold increased risk of developing pneumonia [22].

Table 2 Injury type in adolescent trauma patients stratified by smoking status

Injury type, *n* (%)	All smokers ($n=997$)	Non-smoker ($n=918$)	Smoker ($n=459$)	*p* value
Blunt trauma	871 (87.4%)	794 (89.2%)	384 (86.1%)	0.10
TBI	238 (23.9%)	358 (39.0%)	177 (38.6%)	0.88
Spine	125 (12.5%)	163 (17.8%)	83 (18.1%)	0.88
Upper extremity	139 (13.9%)	177 (19.3%)	98 (21.4%)	0.37
Lower extremity	132 (13.2%)	170 (18.5%)	96 (20.9%)	0.29
Esophagus	0	1 (0.1%)	0	0.48
Colorectal	13 (1.3%)	12 (1.3%)	6 (1.3%)	1.00
Pancreas	2 (0.2%)	1 (0.1%)	1 (0.2%)	0.62
Stomach	6 (0.6%)	1 (0.1%)	0	0.48
Liver	35 (3.5%)	42 (4.6%)	15 (3.3%)	0.25
Small intestine	14 (1.4%)	9 (1.0%)	8 (1.7%)	0.23
Spleen	33 (3.3%)	32 (3.5%)	22 (4.8%)	0.24
Kidney	25 (2.5%)	21 (2.3%)	10 (2.2%)	0.90
Lung	137 (13.7%)	145 (15.8%)	73 (15.9%)	0.96
Heart	2 (0.2%)	2 (0.2%)	2 (0.4%)	0.48

TBI traumatic brain injury

Table 3 Clinical outcomes in adolescent trauma patients stratified by smoking status

Outcome	All smokers ($n=997$)	Non-smokers ($n=918$)	Smokers ($n=459$)	p value
LOS, days, median (IQR)	3.0 (2, 4)	2.0 (2, 4)	3.0 (2, 5)	0.01
ICU, days, median (IQR)	3.0 (1, 4)	2.0 (1, 4)	3.0 (2, 5)	0.55
Ventilator, days, median (IQR)	2.0 (1, 4)	2.0 (1, 4)	3.0 (1, 10.5)	0.75
Complications, *n* (%)				
Unplanned ICU, *n* (%)	7 (0.7%)	8 (0.9%)	3 (0.7%)	0.67
Unplanned intubation, *n* (%)	2 (0.2%)	2.0 (0.2%)	1.0 (0.2%)	1.00
Unplanned return to OR, *n* (%)	6 (0.6%)	3.0 (0.3%)	6.0 (1.3%)	0.03
Acute respiratory distress syndrome	3 (0.3%)	0	1 (0.2%)	0.16
Acute kidney injury	2 (0.2%)	1 (0.1%)	0	0.48
Cardiac arrest	2 (0.2%)	4 (0.4%)	1 (0.2%)	0.53
Decubitus ulcer	2 (0.2%)	3 (0.3%)	1 (0.2%)	0.72
Deep site infection	2 (0.2%)	1 (0.1%)	1 (0.2%)	0.62
Compartment syndrome	1 (0.1%)	0	1 (0.2%)	0.16
Deep vein thrombosis	4 (0.4%)	9 (1.0%)	1 (0.2%)	0.12
Graft failure	2 (0.2%)	2 (0.2%)	2 (0.4%)	0.48
Pneumonia	14 (1.4%)	10 (1.1%)	14 (3.1%)	0.01
Pulmonary embolism	0	1 (0.1%)	0	0.48
Cerebrovascular accident	2 (0.2%)	1 (0.1%)	1 (0.2%)	0.62
Superficial infection	4 (0.4%)	1 (0.1%)	3 (0.7%)	0.08
Urinary tract infection	2 (0.2%)	2 (0.2%)	2 (0.4%)	0.48
CRBSI	0	1 (0.1%)	0	0.48
Severe sepsis	1 (0.1%)	1 (0.1%)	0	0.48
Other	66 (6.6%)	78 (8.5%)	33 (7.2%)	0.40
Mortality, *n* (%)	9 (0.9%)	22 (2.4%)	7 (1.5%)	0.29

LOS length of stay, *IQR* interquartile range, *OR* operating room, *ICU* intensive care unit, *CRBSI* catheter-related bloodstream infection

However, Ferro et al.'s retrospective analysis of adult trauma patients demonstrated no difference in the rate of pneumonia in smokers compared to non-smokers [38]. Our data are the first to suggest an increased incidence of pneumonia in adolescent trauma patients who smoke. One explanation for the difference in results is that unlike the study by Ferro et al., we propensity-matched smokers to similar non-smokers based on comorbidities and injuries. In addition to the morbidity associated with pneumonia, treatment costs $6042 on average for younger patients with the majority of the cost due to the hospital LOS [39, 40]. Our data corroborate this finding as adolescent smoking trauma patients were demonstrated to have a longer LOS. While there is certainly a dose–response increase in respiratory complications caused by cigarette smoking, this study demonstrates an increased rate of pneumonia and hospital LOS despite only minimal cumulative exposure. Our results suggest there is no safe smoking age, and by understanding this risk, clinicians can counsel their adolescent patients on the dangers of both short- and long-term cigarette smoking.

Trauma patients who smoke do not consistently have worse outcomes compared to non-smokers [41]. In fact, several studies have demonstrated a lower mortality in adult smokers following a traumatic injury [38, 42, 43]. This phenomenon is termed the "smoker's paradox," and was originally described in adult patients who sustained acute myocardial infarction [44–46]. One proposed mechanism is that smoking increases coagulability, which may be beneficial during an acute hemorrhagic injury [47]. Another possible mechanism is that exposure to cigarette smoke causes adaptation to hypoxemia such that periods of acute ischemia associated with hemorrhagic shock from trauma have less of a deleterious effect [48]. There are currently no studies demonstrating the "smoker's paradox" in the adolescent population regardless of the disease process. Our data demonstrated that smoking had no effect on risk of survival in adolescent trauma patients. In fact, the only two complications that were increased in this population were pneumonia and an unplanned return to the operating room. This may suggest that the protective mechanism of cigarette smoking has not yet developed in adolescent smokers who likely have not smoked for as long as adults who exhibit the smoker's paradox. Future combined clinical and basic science research to evaluate this is needed.

As a large retrospective database study, there are inherent limitations including reporting bias, coding errors, and missing data. A significant limitation is that 538 patients did not fit into our propensity-matched model and thus, were excluded from the analysis. Alternatively, we could have decreased the number of controls we utilized in our propensity matching; however, this may have resulted in a biased matching system. Furthermore, there was no difference in baseline characteristics and outcomes in the total smokers compared to the smokers we utilized after propensity matching. In addition, missing pertinent data variables include the timing of pneumonia and whether it was community acquired or nosocomial, the bacterial pathogen, and means of diagnosis of pneumonia (i.e., clinical vs. culture). Urine markers indicating recent cigarette smoke exposure, as well as pulmonary functional status (i.e., baseline incentive spirometer or pulmonary function testing) are not available within the TQIP database. Also, given that smoking is a self-reported variable, it is highly likely the incidence is significantly underreported, especially in an adolescent population that may be apprehensive to disclose illegal conduct. In addition, limitations of the database include the lack of granularity regarding the smoking history including the number of cigarettes smoked daily as well as how many years of smoking previously occurred. Although the non-smoking group had not smoked within the past 12 months, the percentage of ex-smokers in this group is not available within the database. Finally, there are no data within TQIP regarding other forms of smoking such as vaping. Despite these limitations, we are the first to evaluate adolescent smokers using a large trauma database and our study is strengthened by its generalizability.

Conclusion

In this 3-year review of the TQIP database, 3.1% of adolescent trauma patients were found to be smokers. In support of our hypothesis, there was nearly triple the rate of pneumonia among the smokers, compared to propensity-matched non-smokers. However, there was no difference in the rate of ARDS with only three cases of an adolescent trauma patient developing this complication. Future research is needed to target this at-risk adolescent trauma population for smoking cessation counseling to help decrease the risk of pneumonia and reduce hospital costs. In addition, trauma providers should be aware of the significantly increased rate of pneumonia in this population to either mitigate the risk or diagnose and treat this population early in the hopes of reducing associated morbidity like the increased LOS observed in this study.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Ethical approval This research involved humans. However, since this retrospective study was performed using a national database with deidentified patients, risk to participants is minimal.

Informed consent There is no consent required.

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