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Acute Respiratory Distress Syndrome and Outcomes after Near-hanging

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Abstract

Purpose—Assess the case rate of Acute Respiratory Distress Syndrome (ARDS) after near-hanging, and the secondary outcomes of traumatic and/or anoxic brain injury, and death. Risk factors for the outcomes were assessed.

Method—Single-center, state-wide retrospective cohort study of consecutive patients admitted between August, 2002, and September, 2011, with a primary diagnosis of non-judicial "hanging injury".

Results—Of 56 patients, 73% were male. The median age was 31 (IQR: 16–56). Upon arrival, 9% (5/56) did not have a pulse, and 23% (13/56) patients were intubated. The median Glasgow

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Coma Scale (GCS) was 13 (IQR: 3–15); 14% (8/56) had a GCS=3. ARDS developed in 9% (5/56) of patients. Traumatic anoxic brain injury resulted in 9% (5/56) of patients. The in-hospital case fatality was 5% (3/56). Lower median GCS [3 (IQR: 3–7) vs. 14 (IQR: 3–15), $p=0.0003$] and intubation in field or in trauma resuscitation unit [100% (5/5) vs. 16% (8/51), $p=0.0003$] were associated with ARDS development. Risk factors of death were GCS=3 [100% (3/3) vs. 9% (5/53), $p=0.002$]; pulselessness upon arrival of emergency medical services [100% (3/3) vs. 4% (2/53), $p<0.001$]; and abnormal neurologic imaging [50% (1/2) vs. zero, $p=0.04$].

Conclusions—The ARDS case rate after near-hanging is similar to the general trauma population. Low GCS and intubation are associated with increased risk of ARDS development. The rate of traumatic and/or anoxic brain injury in this population is low.

Keywords

Wounds and injuries; Respiratory Distress Syndrome; Adult; Patient Outcome Assessment; Suicide; Attempted; Near-hanging

INTRODUCTION

Between 2000 and 2010, suicide rates in the US increased by 16%, and suicide by hanging or suffocation accounted for 52% of the increase[1]. Hanging or suffocation suicide case fatality rates ranged from 69% to 84%[1]. Injuries occurring after near-hanging include cervical spine and laryngotracheal fractures, anoxic brain injury, and retinal bleeding[2–4]. Previously, pulmonary complications such as aspiration pneumonia[3], and pulmonary edema[5–8] have been observed. However, the Acute Respiratory Distress Syndrome (ARDS), as defined by modern consensus definition, has not been evaluated as an outcome after near-hanging.

We hypothesized that patients suffering injury by near-hanging are at risk for ARDS development. In a cohort of consecutive near-hanging patients presenting to a state-wide trauma center, the association of near-hanging with ARDS was evaluated. Risk factors for death and traumatic and/or anoxic brain injury were also assessed.

METHODS

We conducted a single-center, state-wide retrospective cohort study of consecutive patients admitted with a primary diagnosis of non-judicial “hanging injury” between August, 2002, and September, 2011, to the R. Adams Cowley Shock Trauma Center (STC) at the University of Maryland Medical Center. The Shock Trauma Center is an independent, trauma center that is the main referral center for critically injured adults in the State of Maryland, from urban, suburban, and rural communities of over 6 million people for greater than 30 years[9].

For the assessment of ARDS development, the Berlin definition[10] was incorporated into a validated screening algorithm[11–13]. This algorithm was combined with administrative data for ventilator charges, in order to identify all mechanically ventilated patients at risk for ARDS. All patients presenting to STC are routinely assessed for Glasgow Coma Score

(GCS) upon arrival. Low GCS was defined as 3 and low Injury Severity Score (ISS) as less than 16. Neurologic outcome was assessed by Computed Tomography (CT) head/Computed Tomography Angiography (CTA) neck and/or clinical assessment by the neurology service. Patients' characteristics were presented as medians with standard deviations and compared using Student's *t*-test for independent samples with continuous variables, and comparison of proportions for categorical variables using Fishers test. We used the traditional threshold of $p < 0.05$ to determine statistical significance.

RESULTS

Between August, 2002, and September, 2011, 56 patients were admitted with primary diagnosis of hanging injury (Table 1). Of these, 73% (41/56) were male. The median age was 31 (IQR: 16–56). The median GCS was 13 (IQR: 3–15); 14% (8/56) had a GCS equal to 3 (unresponsive). Upon arrival, 9% (5/56) were pulseless. Low ISS (ISS<16) was noted in 98% (55/56), blood alcohol content (BAC) was positive in 20% (11/56), and urine toxicology screen was positive in 48% (27/56) and comprised of the following: benzodiazepine (16/56), barbituates (9/56), opiates (6/56), marijuana (6/56), PCP (1/56), amphetamines (1/56), and tricyclics (1/56).

Among the cohort, 23% (13/56) patients were intubated, either in the field or in the trauma resuscitation unit. ARDS developed in 9% (5/56) of patients (Table 2). Low GCS and younger age was associated with ARDS development. An association with high ISS was also found but this did not meet our definition of significance ($p=0.09$). After near-hanging, 3% (2/56) of patients were transferred to acute care rehabilitation facilities, 57% (32/56) transferred to inpatient psychiatric facilities, 30% (17/56) were discharged to home, and 3% (2/56) transferred to prison. Traumatic anoxic brain injury occurred in 9% (5/56) of patients. Of the patients receiving mechanical ventilation, 38% (5/13) patients developed ARDS. Those with ARDS and receiving mechanical ventilation had lower median 28-day ventilator-free days than patients receiving mechanical ventilation without ARDS (Table 3). Lower median GCS [3 (IQR: 3–7) vs. 14 (IQR: 3–15), $p=0.0003$] and mechanical ventilation [100% (5/5) vs. 16% (8/51), $p=0.0003$] were associated with the ARDS development (Table 3).

The in-hospital case fatality rate was 5% (3/56) (Table 4). Risk factors for death were GCS equal to 3 [100% (3/3) vs. 9% (5/53), $p=0.002$]; pulselessness upon arrival of emergency medical services [100% (3/3) vs. 4% (2/53), $p<0.001$]; and abnormal neurologic imaging [50% (1/2) vs. 0/50, $p=0.04$].

DISCUSSION

ARDS is an important outcome after near-hanging. Low GCS and mechanical ventilation were associated with ARDS development. Higher case fatality rates were observed in patients with low GCS, pulselessness upon arrival of EMS, and abnormal neurologic imaging. Interestingly, despite the mechanism of injury, anoxic brain injury was relatively uncommon.

ARDS development after suicidal hanging has previously been reported in literature[14; 15]. Disruption of alveolar-capillary membrane results in exudation of fluid, and pulmonary congestion leading to decreased lung compliance[16; 17]. ARDS may develop after near-hanging by several mechanisms, including aspiration from a low GCS. Depressed sensorium may predispose patients to aspiration, leading to pneumonitis or pneumonia, and ARDS development[18; 19]. Neurologic injury associated with near-hanging may be another potential etiology. Neurogenic pulmonary edema and acute lung injury (ALI) can develop after neurologic injury[20; 21]. Cerebral hypoxia induced in animals has been shown to lead to pulmonary changes consistent with ARDS development[22–24]. During hanging attempts, acute negative intrathoracic pressure developing after upper airway obstruction may cause disruption of the alveolar-capillary membrane[25]; which is termed negative pressure pulmonary edema (NPPE)[7; 8; 26; 27] or post-obstructive pulmonary edema (POPE)[6; 28]. While the pathophysiology of NPPE involves hydrostatic pressure, this is not left atrial hydrostatic pressure, thus defining it as ARDS[25]. Carotid compression leading to cerebral anoxia, rather than tracheal obstruction, may be essential to the development of ARDS as ARDS development has been observed in patients who hang themselves above the tracheotomy site[29; 30].

Previous studies using non-standardized definitions of pulmonary edema found case rates varying widely between 4.2%[31]and 56%[5–8; 31–37], contextualizing our mechanical ventilation case rate of 23%. The ARDS case rate after near-hanging in our cohort (9%) is similar to that of the general trauma population[38–40], and that of our institution[13]. Other pulmonary complications after near hanging include aspiration pneumonia[3; 41], chest injury[42], and respiratory distress[43].

We found a case fatality rate of 5%, comparable to some studies[4; 5] but not all. Two case series found case-fatality rates between 33%[42] and 76%[31]. In general, while judicial hanging has a high mortality rate, non-judicial hanging attempts result in a high probability of survival for those transported to a trauma center. Consistent with prior studies, low GCS was a risk factor for death[2; 4; 5; 31; 42]. However, low GCS was not associated with anoxic brain injury[2; 4; 41; 42]. In our cohort, the majority of patients with low GCS survived without traumatic anoxic brain injury, and were discharged to inpatient psychiatry units. Given that non-judicial hanging can cause jugular venous and arterial obstruction and airway obstruction[2; 4], this low rate of anoxic brain injury was surprising. However, this finding is consistent with prior studies in which the rate of significant neurologic impairment ranged from 0–5%[2–4].

Our study benefits from a state-wide referral center as its data source and the use of consecutive near-hanging admission. While these factors make the results generalizable, this is a single-center study. The lack of data regarding mode of suspension (complete vs. incomplete), height of fall, and duration of hanging limit our understanding of the neurologic consequences after near-hanging. As the number of hanging attempts resulting in death before hospital admission is unknown, these outcome case-rates apply only to patients presenting for appropriate care.

CONCLUSION

Near-hanging patients are at risk for ARDS development, lower median GCS and younger age being risk factors. Near-hanging patients with ARDS were more likely to receive mechanical ventilation. Low GCS and mechanical ventilation are also associated with higher case fatality rates. The rate of traumatic or anoxic brain injury in this population was low.

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Table 1

Patient Characteristics

Total Patients (n)	56
Gender	
Male (%)	73% (41/56)
Female (%)	27% (15/56)
Age (yrs) \pm SD, Median (IQR)	32 \pm 10, 31 (16–56)
ISS ^a <16 (%)	98% (55/56)
GCS ^b	
Avg (\pm SD), median (IQR)	11 \pm 4, 13 (3–15)
GCS ^b =3 (%)	14% (8/56)
Fatalities among patients with GCS ^b =3 (%)	37% (3/8)
Pulselessness upon EMS ^c arrival (%)	9% (5/56)
Positive blood alcohol level (%)	20% (11/56)
Positive urine toxicology screen (%)	48% (27/56)

^aInjury Severity Score;

^bGlasgow Coma Score;

^cEmergency Medical Services

Table 2

Patient Outcomes

Total Patients (n)	56
In-hospital death(%)	5% (3/56)
ARDS ^a (%)	9% (5/56)
Mechanical Ventilation (%)	23% (13/56)
Abnormal CT ^b head (%)	2% (1/52)
Abnormal CT ^b neck/CTA ^c neck (%)	2% (1/53)
TBI ^d and/or anoxic brain injury (%)	9% (5/56)
Disposition	
Home (including original place of residence) (%)	30% (17/56)
Inpatient psychiatric unit (%)	57% (32/56)
Acute care rehabilitation facility (%)	3% (2/56)
Expired (%)	5% (3/56)
Prison (%)	3% (2/56)

^a Acute Respiratory Distress Syndrome;

^b Computed Tomography;

^c Computed Tomography Angiography;

^d Traumatic Brain Injury

Table 3Patient Characteristics and Outcomes by ARDS^a status

	ARDS ^a (5)	No ARDS ^a (51)	P Value
Total Patients (%)	9% (5/56)	91% (51/56)	
Male (%)	60% (3/5)	75% (38/51)	0.60 ^b
Female (%)	40% (2/5)	25% (13/51)	
Age (yrs) ± SD, Median (range)	26±2, 27 (23–29)	31±10, 31 (19–56)	0.004 ^c
In-hospital death (%)	20% (1/5)	4% (2/51)	0.25 ^a
ISS ^d <16 (%)	80% (4/5)	100% (51/51)	0.09 ^b
ISS ^d >16 (%)	20% (1/5)	0	
GCS ^e			
Avg (±SD), Median (IQR)	4±2, 3 (3–7)	11.5±4, 14 (3–15)	0.0003 ^c
Total Number of patients with GCS ^e equal to 3 (%)	60% (3/5)	10% (5/51)	0.02 ^b
Absence of pulse upon arrival of EMS ^f (%)	20% (1/5)	8% (4/51)	0.39 ^b
Positive Blood Alcohol Content (%)	40% (2/5)	18% (9/51)	0.25 ^b
Urine toxicology screen positive (%)	40% (2/5)	49% (25/51)	1 ^b
Mechanical ventilation (%)	100% (5/5)	16% (8/51)	0.0003 ^b
Ventilator-free days Median (IQR)	19 (0–27)	27 (21–26)	
Abnormal CT ^g head (%)	20% (1/5)	0 (0/47)	0.10 ^b
Abnormal CT ^g neck/CTA ^h neck (%)	0 (0/5)	2% (1/49)	1 ^b
Disposition			0.14 ^b
Home (including original place of residence) (%)	20% (1/5)	31% (16/51)	
Inpatient psychiatric unit (%)	40% (2/5)	59% (30/51)	
Acute care rehabilitation facility (%)	20% (1/5)	2% (1/51)	
Expired (%)	20% (1/5)	4% (2/51)	
Prison (%)	0 (0/5)	4% (2/51)	

Data presented as medians with interquartile range or as proportions

^a Acute Respiratory Distress Syndrome;^b Fisher's Exact test;^c Student's t-test;^d Injury Severity Score;^e Glasgow Coma Score;^f Emergency Medical Services;^g Computed Tomography;^h Computed Tomography Angiography

Table 4

Patient characteristics and death

	Died (3)	Survived (53)	P value
Total Patients (%)	5% (3/56)	95% (53/56)	
Male (%)	67% (2/3)	74% (39/53)	1 ^a
Female (%)	33% (1/3)	26% (14/53)	
Age (yrs) ± SD, Median (range)	33±10, 33 (23–43)	32±10, 31 (25–37)	0.83 ^b
ISS ^c <16 (%)	66% (2/3)	100% (53/53)	0.0536 ^a
ISS ^c >16 (%)	33% (1/3)	0 (0/53)	
GCS ^d			
Avg(±SD), Median (IQR)	3, 3 (3)	11±4, 14 (3–15)	<0.001 ^b
Total Number of patients with GCS ^d equal to 3 (%)	100% (3/3)	9% (5/53)	0.002 ^a
Absence of pulse upon arrival of EMS ^e (%)	100% (3/3)	4% (2/53)	<0.001 ^a
Positive Blood Alcohol Content	33% (1/3)	19% (10/53)	0.49 ^a
Positive urine toxicology screen positive (%)	0 (0/3)	51% (27/53)	0.23 ^a
Mechanical Ventilation (%)	33% (1/3)	23% (12/53)	0.55 ^a
ARDS ^f Development (%)	33% (1/3)	8% (4/53)	0.25 ^a
Abnormal CT ^g head (%)	50% (1/2)	0(0/50)	0.04 ^a
Abnormal CT ^g neck/CTA ^h neck (%)	0 (0/2)	2% (1/52)	1 ^a
Neurological intervention performed (%)	0 (0/3)	0 (0/51)	n/a

Data presented as medians with interquartile range or as proportions;

^a Fisher's Exact test;

^b Student's t-test;

^c Injury Severity Score;

^d Glasgow Coma Score;

^e Emergency Medical Services;

^f Acute Respiratory Distress Syndrome;

^g Computed Tomography;

^h Computed Tomography Angiography