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Research Article

Alcohol Intoxicated Trauma Patients: Hemodynamic Effects of General Anesthesia

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ABSTRACT

Purpose: The current work aims to determine whether patients presenting with Acute Alcohol Intoxicated (AAI) traumatic injuries are at higher risk for hemodynamic compromise after induction of general anesthesia.

Method: A retrospective chart review from January 1, 2008 to June 1, 2017 of patients aged ≥ 18 years of age undergoing emergent exploratory laparotomy within 4 hours of presentation were included in the study. Data collected included age, sex, weight, blood alcohol level, Systolic Blood Pressure (SBP), INR, injury severity score, base deficit, pre- and post-induction Mean Arterial Pressure (MAP), induction medication and dose, length of hypotension, and vasoactive medications administered within 20 minutes post-induction.

Results: Post-induction MAPs were lower for alcohol intoxicated patients than for non-intoxicated controls (p=0.018) despite both groups having similar admission SBP (0.295) and pre-induction MAPs (0.171). Alcohol intoxicated patients also have significant longer duration of hypotension following induction (p=0.016), however there was no significant difference in the requirement of vasopressor use within twenty minutes post-induction (p=0.279).

Conclusion: Our findings (lower MAPs and longer duration of hypotension than controls following induction of general anesthesia, as well as blood chemistry suggestive of volume contraction) support the notion of alcohol-induced hemodynamic dysregulation in intoxicated trauma patients with presumed intra-abdominal injury. Based on these results, it is essential for the anesthesiologist to be prepared to support blood pressures in order to maintain adequate perfusion in acutely intoxicated patients requiring general anesthesia.

Keywords: Trauma; General anesthesia; Alcohol

INTRODUCTION

Trauma is the leading cause of death among those 46 years of age and younger [1]. 1 It is responsible for causing more deaths before the age of 65 than that attributed to cancer, heart disease and HIV combined [2]. Multiple studies reveal that up to 40% of trauma victims have a positive Blood Alcohol Concentration (BAC) upon admission [3-7]. Acute alcohol intoxication (AAI) is not only a significant risk factor for traumatic injury [8-10], but is also related to an increased severity of traumatic injury [11]. Therefore, it can be inferred that surgeons across the United States and throughout the world are frequently preforming exploratory laparotomies on AAI patients who suffered traumatic injury.

Alcohol-induced hemodynamic dysregulation in the setting of acute hemorrhage has been demonstrated in both human and animal studies, where alcohol has been shown to aggravate injury-related hemodynamic instability [12-15]. Therefore, in practice, intoxicated patients undergoing general anesthesia are likely at increased risk of post-induction hypotension requiring vasopressor support. However, no studies could be found demonstrating this phenomenon.

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Thus, it is critical to analyze the dynamic instability enhanced by AAI for a significant portion of traumatically-injured patients. The purpose of this study was to determine whether patients presenting with AAI traumatic injuries require more intensive anesthesia management. Our hypothesis is intoxicated patients will have hemodynamic dysregulation which will manifest with (i) a more significant drop in blood pressure post-induction, and (ii) requirement of more vasopressor support post-induction compared to non-intoxicated controls.

METHODS

Data collection

A retrospective chart review was performed at an ACS-verified, level I trauma center registry from January 1, 2008 to June 1, 2017. Patients aged ≥ 18 years of age undergoing emergent exploratory laparotomy within 4 hours of presentation were included in the study. Approximately 320 charts were reviewed. Patients intubated in the field and patients with incomplete blood pressure records during intubation were excluded. Data from 202 adult trauma patients (44 study group, 158 control group) were analyzed. Indications for surgery included high index of suspicion for intra-abdominal, diaphragmatic injury or penetrating abdominal injury. Data collected included age, sex, weight, blood alcohol level, Systolic Blood Pressure (SBP), INR, injury severity score, base deficit, pre and post-induction Mean Arterial Pressure (MAP), induction medication and dose, length of hypotension, and vasoactive medications administered within 20 minutes post-induction.

Study group

Patients with a blood alcohol concentration of 0.8 g/dL or greater were defined as intoxicated and thus placed in the study group. Patients with a blood alcohol concentration of less than 0.8 g/dL were placed in the control group.

Outcome measures

Admission SBP, pre-induction MAP and post-induction MAP, patients who had a duration of hypotension >10 minutes post-induction were compared between study and control group to assess hemodynamic stability. Patients in both groups that required vasopressors within 20 minutes post-induction were used to assess requirement of vasopressor support. Other data collected were used to identify baseline characteristics between the groups of patients presenting with traumatic injury.

Data analysis

Groups were compared using the two-sample t-test (normally distributed continuous variables; reported as mean ± SD) and the Wilcoxon rank-sum test (continuous data not normally distributed; reported as median and interquartile range [Q1,Q3]). Exact versions of the Pearson chi-square and Mantel-Haenszel chi-square test were used to compare groups for binary and ordinal variables, respectively.

Penn State Health Institutional Review Board approved this retrospective study.

RESULTS

Demographic variables

Inclusion criteria were comprised of 202 adult trauma patients, 44 with acute alcohol intoxication and 158 without acute alcohol intoxication. Indications for surgery, which included a high index of suspicion for intra-abdominal or diaphragmatic injury, and any penetrating abdominal injury, were the same for both groups.

Pre-operative results

The mean injury severity score was 21.05 ± 16.20 and 20.06 ± 13.40 for the acute alcohol intoxicated patients and control group, respectively (p=0.998). The incidence of intra-abdominal organ laceration was 52.27% and 57.59% for the acute alcohol intoxicated patients and control groups, respectively (p=0.607). The mean admission SBP was 121.95 mmHg ± 29.94 and 126.94 mmHg ± 27.28 for the acute alcohol intoxicated patients and control groups, respectively (p=0.295). The mean pre-induction MAP was 95.20 mmHg ± 17 and 99.82 mmHg ± 20.26 for the acute alcohol intoxicated patients and control groups, respectively (p=0.171). The mean hematocrit was 39.13% ± 5.13 and 35.53% ± 6.28 for the acute alcohol intoxicated patients and control groups, respectively (p<0.001). The mean INR was 1.11 ± 0.14 and 1.21 ± 0.31 for the acute alcohol intoxicated patients and control groups, respectively (p=0.027). Base deficit was 8.57 mmol/L ± 4.57 and 5.30 mmol/L ± 3.79 for the acute alcohol intoxicated patients and control groups, respectively (p<0.001) (Table 1).

Intra-operative results

No significant difference was observed between the primary induction type (p=0.255) that the patients were receiving (Table 2). Post-induction MAPs were lower for alcohol intoxicated patients than for non-intoxicated controls (62.77 mmHg vs. 69.75 mm Hg, respectively, p=0.018) (Table 1). When comparing duration of hypotension, 41.73% of alcohol intoxicated patients and 28.48% of the control group had a duration >10 minutes post-induction, p=0.016. There was no statistically significant difference for requirement of vasopressor use within twenty minutes post-induction for each group (p=0.279) (Table 3).

	AAI Group	Control Group	p-Value
Injury severity score (mean ± SD)	21.05 ± 16.20	20.06 ± 13.40	0.998
Baseline deficit (mean mmol/L ± SD)	8.57 ± 4.57	5.30 ± 3.79	<0.001
Intra-abdominal organ laceration (mean %)	52.27	57.59	0.607

Admission hematocrit (mean % ± SD)	39.13 ± 5.13	35.53 ± 6.28	<0.001
Admission INR (mean ± SD)	1.11 ± 0.14	1.21 ± 0.31	0.027
Admission SBP (mean mmHg ± SD)	121.95 ± 29.94	126.94 ± 27.28	0.295
Pre-induction MAP (mean mmHg ± SD)	95.20 ± 17	99.82 ± 20.26	0.171
Post-induction MAP (mean mmHg ± SD)	62.77 ± 9.48	69.75 ± 18.70	0.018

Table 1: Pre- and Intraoperative Results: The mean values of acute alcohol intoxicated patients and non-intoxicated control group data from both admission and intraoperative as well as their associated p-values are reported.

	AAI group	Control group	p-Value
Etomidate, n (%)	19 (43.18%)	73 (46.20%)	0.255
Ketamine, n (%)	14 (31.82%)	30 (18.99%)	
Methohexital, n (%)	1 (2.27%)	3 (1.90%)	
Propofol, n (%)	10 (22.73%)	52 (32.91%)	

Table 2: Induction Medication and Dose: The number and percent of patients given various primary induction medications as well as the primary induction dose in both the acute alcohol intoxication group and the control group are reported.

	AAI group, n (%)	Control group, n (%)	Chi square	p-Value
<10 minutes of hypotension	23 (52.27%)	113 (71.52%)	5.7952	0.016
10-20 minutes of hypotension	21 (47.73%)	66 (28.48%)		
Received vasopressors	8 (3.81%)	48 (5.68%)	1.1714	0.279
Did not receive vasopressors	202 (96.19%)	797 (94.32%)		

Table 3: Minutes of Hypotension and Requirement of Vasopressors: The number and percent of patients who were hypotensive for <10 minutes and 10-20 minutes as well as those that did and did not receive vasopressors within 20 minutes post induction in the acute alcohol intoxicated group as well as the control group.

DISCUSSION

Our study demonstrated that post-induction MAPs were lower for alcohol intoxicated patients than for non-intoxicated controls despite both groups having similar admission SBP and pre-induction MAPs. There was also a greater number of alcohol intoxicated patients who had duration of hypotension >10 minutes post induction. Additionally, there was a higher hematocrit in the acute alcohol intoxication group than the control group, which can be explained by mild dehydration in the alcohol intoxicated group leading to relative increased levels of hematocrit [16]. These findings support the notion of alcohol-induced hemodynamic dysregulation in intoxicated trauma patients with presumed intra-abdominal injury.

Our data reveal that one must be prepared to support blood pressure in order to maintain adequate perfusion in acutely intoxicated patients requiring general anesthesia. The patients analyzed in this study underwent an exploratory laparotomy due to high index of suspicion for intraabdominal injuries, and therefore had an increased risk of pathophysiologic response to injury due to alcohol intoxication. Pathophysiologic responses of the hemodynamic system can lead to poor surgical outcomes, delayed recovery, and increased mortality [13]. Use of high doses of vasopressors in severely injured patients can lead to increased morbidity and mortality [17]. Knowledge and awareness on the impact of alcohol induced post-induction hypotension allows for preventative measures such as adequate volume resuscitation and appropriate vasopressor use to avoid adverse perioperative outcomes [12-18].

Other studies have shown the influence of alcohol intoxication on physiologic parameters in severely injured patients. One study in particular measured the influence of alcohol intoxication with the use of Glasgow Coma Scale (GCS) and Computed Tomography (CT) scan findings in patients with traumatic brain injuries. The alcohol intoxicated group showed lower GCS scores but there were no significant changes in CT findings or time to response [19]. This study provides further evidence on the effect alcohol has on the body's physiology.

Another study observed the effect of alcohol on coagulation, showing impairment in clot formation and decreased fibrinolysis in alcohol-intoxicated trauma patients [20]. Our study demonstrated slightly lower INR values in the alcohol intoxicated group compared to the control group. Although there have been numerous studies on the hemodynamic instability after AAI traumatic injury in mouse models [13-15] and the pathophysiology has been explained theoretically, to our

knowledge, no literature has been published on these effects in humans undergoing anesthesia.

There are limitations to our study. This data was gathered retrospectively from a single ACS-verified, level I trauma center registry. Therefore, additional prospective, multicenter studies should be conducted to determine generalizability of these results.

CONCLUSION

To our knowledge, this is the first published retrospective chart analysis addressing the impact of AAI on hemodynamic instability after induction of general anesthesia in severely injured patients. Our findings demonstrated that alcohol-intoxicated patients undergoing general anesthesia are at increased risk of post-induction hypotension, thus requiring vasopressor support. This study will help to understand the influence of AAI on anesthesia induced hemodynamically instability and help to prepare for preventative measures in severely injured intoxicated patients.

REFERENCES

1. Trauma Statistics & Facts, National Trauma Institute.
2. Rhee P, Joseph B, Pandit V, Aziz H, Vercruysse G, Kulvatunyou N, et al. Increasing trauma deaths in the United States. Ann Surg. 2014;260(1): 13-21.
3. Cherpitel CJ. Alcohol and casualties: A comparison of emergency room and coroner data. Alcohol Alcohol. 1994;29(2): 211-218.
4. Cherpitel CJ, Ye Y, Bond J, Borges G. The causal attribution of injury to alcohol consumption: A cross-national meta-analysis from the emergency room collaborative alcohol analysis project. Alcohol Clin Exp Res. 2003;27(11): 1805-1812.
5. Cherpitel CJ, Bond J, Ye Y, Borges G, MacDonald S, Stockwell T, et al. Alcohol-related injury in the ER: A cross-national meta-analysis from the Emergency Room Collaborative Alcohol Analysis Project (ERCAAP). J Stud Alcohol. 2003;64(5): 641-649.
6. Madan AK, Yu K, Beech DJ. Alcohol and drug use in victims of life-threatening trauma. J Trauma. 1999;47(3): 568-571.
7. Pories SE, Gamelli RL, Vacek P, Goodwin G, Shinozaki T, Harris F. Intoxication and injury. J Trauma. 1992;32(1): 60-64.
8. Scheenen ME, de Koning ME, van der Horn HJ, Roks G, Yilmaz T, van der Naalt J, et al. Acute alcohol intoxication in patients with mild traumatic brain injury: Characteristics, recovery, and outcome. J Neurotrauma. 2016;33(4): 339-345.
9. Valdez C, Radomski M, Renne C, Amdur R, Dunne J, Sarani B. Alcohol level and injury severity: Is the floppy patient myth true? J Surg Res. 2016;200(2): 664-668.
10. Nunn J, Erdogan M, Green RS. The prevalence of alcohol-related trauma recidivism: A systematic review. Injury. 2016;47(3): 551-558.
11. Molina PE, Katz PS, Souza-Smith F, Ford SM, Teng SX, Dodd TY, et al. Alcohol's Burden on Immunity Following Burn, Hemorrhagic Shock, or Traumatic Brain Injury. Alcohol Res. 2015;37(2): 263-278.
12. Molina PE, Sulzer JK, Whitaker AM. Alcohol abuse and the injured host: Dysregulation of counterregulatory mechanisms review. Shock. 2013;39(3): 240-249.
13. Molina PE, Zambell KL, Norenberg K, Eason J, Phelan H, Zhang P, et al. Consequences of alcohol-induced early dysregulation of responses to trauma/hemorrhage. Alcohol. 2004;33(3): 217-227.
14. Phelan H, Stahls P, Hunt J, Bagby GJ, Molina PE. Impact of alcohol intoxication on hemodynamic, metabolic, and cytokine responses to hemorrhagic shock. J Trauma. 2002;52(4): 675-682.
15. Paloczi J, Matyas C, Cinar R, Varga ZV, Hasko G, Schindler TH, et al. Alcohol binge-induced cardiovascular dysfunction involves endocannabinoid-CB1-R signaling. JACC Basic Transl Sci. 2019;4(5): 625-637.
16. Malisova O, Athanasatou A, Pepa A, Husemann M, Domnik K, Braun H, et al. Water intake and hydration indices in healthy european adults: The European Hydration Research Study (EHRS). Nutrients. 2016;8(4): 204.
17. Plurad DS, Talving P, Lam L, Inaba K, Green D, Demetriades D. Early vasopressor use in critical injury is associated with mortality independent from volume status. J Trauma. 2011;71(3): 565-570.
18. Hassani V, Movaseghi G, Safaeeyan R, Masghati S, Ghorbani Yekta B, Farahmand Rad R. Comparison of ephedrine vs. norepinephrine in treating anesthesia-induced hypotension in hypertensive patients: Randomized double-blinded study. Anesth Pain Med. 2018;8: e79626.
19. Grzelczak AC, Ceccon A, Guetter CR, Pimentel SK. Evaluation of traumatic brain injury patients with signs of alcohol intoxication. Rev Col Bras Cir. 2019;46: e20192272
20. Howard BM, Kornblith LZ, Redick BJ, Conroy AS, Nelson MF, Calfee CS, et al. Exposing the bidirectional effects of alcohol on coagulation in trauma: Impaired clot formation and decreased fibrinolysis in rotational thromboelastometry. J Trauma Acute Care Surg. 2018;84(1): 97-103.