Pilot study to evaluate the difference between opioid use in trauma patients in motorized vehicle collisions with elevated blood alcohol levels versus those without evidence of intoxication

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BACKGROUND

- The mechanism of injury for over 25% of patients within the national trauma data base (NTDB) is motor vehicle crash (MVC)[9]

- 13% of NTDB patients test positive for alcohol[9]

- 1 in 3 Trauma Alert Activation criteria test positive for alcohol and /or illicit drugs [3]

- Alcohol positive patients utilize more resources than their counter parts and have increase odds of ICU admission[12]

- Alcohol has neurobiological interactions that affect the production, secretion and binding of opioids to receptors [6]

OBJECTIVE

Determine if there is a correlation between the intoxicated trauma patient and the amount of opioids they consume during the recovery phase of a traumatic injury compared to their counterparts who are not intoxicated

METHODS

Study design: Single-center, retrospective evaluation

Dates: January 1, 2014 through December 31, 2015

Inclusion criteria:

- Age ≥ 18 years
- Inclusion criteria met for NTDB inclusion criteria
- Mechanism of injury documented as MVC

Exclusion criteria:

- Discharge <48 hours after arrival
- Glasgow Coma Scale (GCS) score <13
- Injury Severity Score (ISS) >20
- Presence of any other illicit substance on toxicology screening
- Any patient started on alcohol withdraw treatment

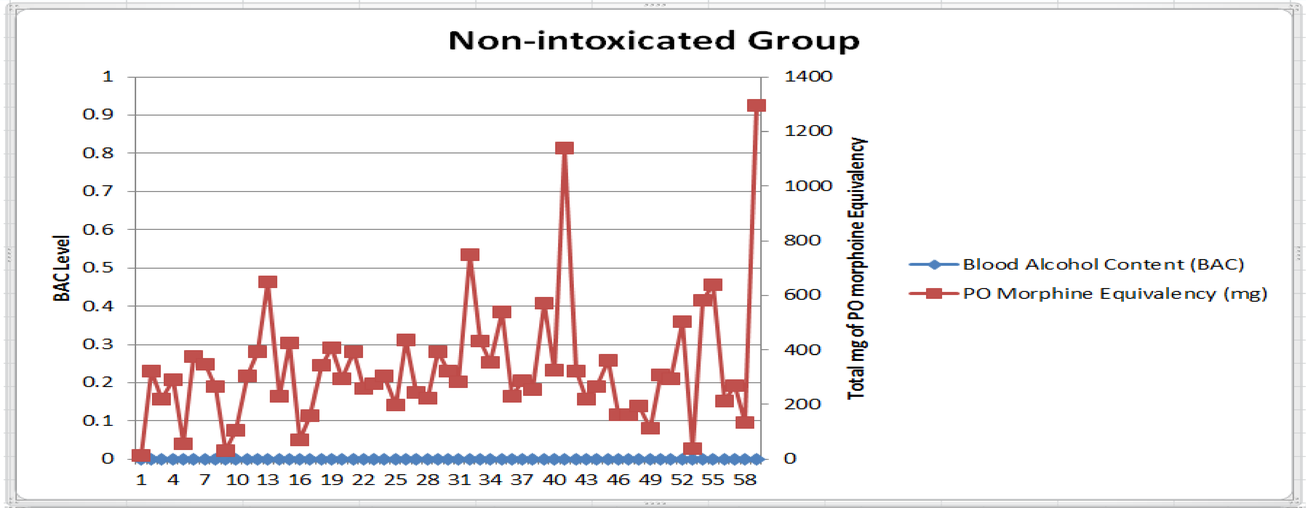
Methods: Divided into 2 groups: Alcohol Intoxicated Group (AIG) and Non-Alcohol Intoxicated Group (NIG)

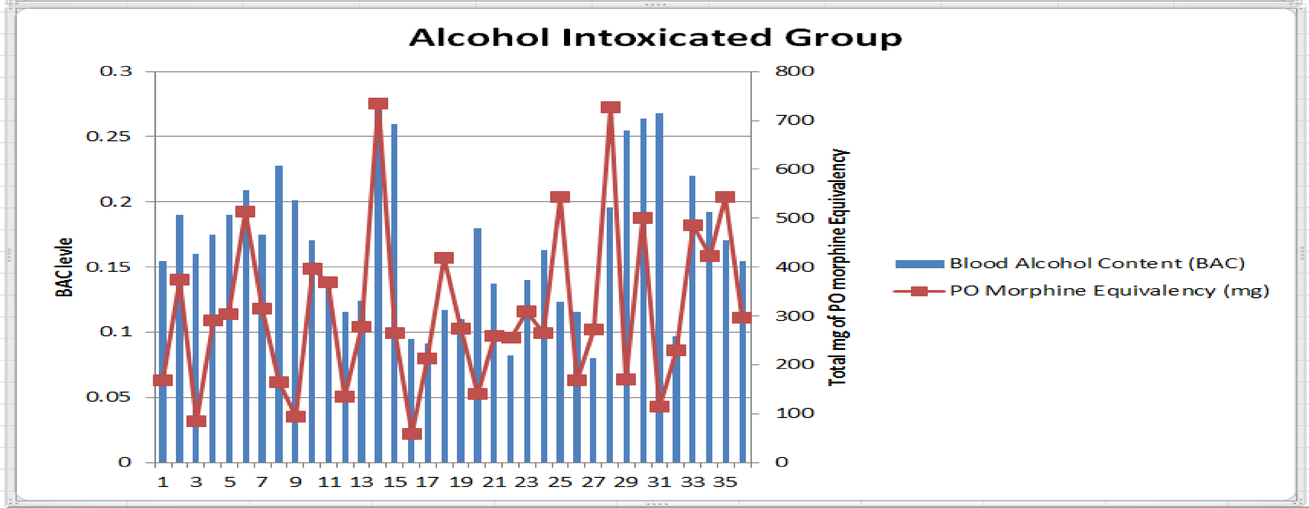
Outcome: Total morphine equivalents consumed within the initial 72 hours of both the AIG and NIG groups

EQUIANALGESIC DOSING TABLE

Drugs	Equianalgesic parenteral dose (mg)	Equivalent Oral dose (mg)	Parenteral:Oral dosing ratio (same drug)	Equianalgesic dose ratio with Morphine (approximations)
Morphine (MOR)	10	30	1:3	
Hydromorphone (DIL)	1.5-2	7.5-8	1:5	IV MOR: IV DIL 7:1 PO MOR: PO DIL 5:1 PO DIL: PO MOR 3.5:1
Oxycodone (OXY)	-	20	n/a	PO MOR: PO OXY 1.5-2:1
Hydrocodone (HCD)	-	20	n/a	PO MOR: PO HCD 1:1
Codeine (COD)	60-130	130 – 200	1:1.5 – 2	IV MOR: IV COD 1:7.5 PO MOR: PO COD 1:4 [10]
Fentanyl (FEN)	0.1 – 0.2	-	n/a	

RESULTS





Median Morphine Equivalents

AIG
- Median 276 mg
- First Quartile 170 mg
- Third Quartile 407 mg
- IQR 238mg

NIG
- Median 294 mg
- First Quartile 220 mg
- Third Quartile 392 mg
- IQR 173 mg

NIG utilized more total morphine equivalents during the first 72 hours of admission

p <0.5

(Mann Whitney U Test)

DISCUSSION

- Alcohol stimulates dopaminergic receptors in the brain and opioids stimulate the opioidergic receptors

- Evidence supports interactions between opioidergic and dopaminergic neurotransmissions[8]

- Alcohol may modulate endogenous opioid systems by disrupting opioid receptor signalling; low concentrations of ethanol slightly potentiate μ-opioid receptor binding[1]

- Alcohol has been shown to increase endogenous opioid release in the nucleus accumbens and the ventral tegmental area (reward system activation) and facilitate dopaminergic transmission activity[6]

- High concentrations of alcohol (0.2) have been shown to cause increased opioid binding after 18 to 24 hours[2]

CONCLUSION

- The AIG utilized less opioids possibly indicating there is, in addition to opioid medications, some endogenous opioid effect already at work when the patient presents after traumatic injury [6]

- During the period of alcohol withdrawal, dopaminergic transmission is increased [4], thus the AIG could have greater dopaminergic activity leading to decreased use of opioids for pain control after traumatic injury

- Most recent research done to evaluate these links have been geared towards the treatment of alcohol withdrawal symptoms with opioid antagonists rather than the agonist effects of opioid and dopamine on each other

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DISCLOSURES

Nothing to disclose noted for authors