

Does Midodrine Matter in Obtaining Blood Pressure Goals for Traumatic Spinal Cord Injury? A Retrospective Cohort Study of Length of Stay and Clinical Outcomes

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Background: The clinical impact of traumatic spinal cord injuries (tSCI) is significant with prolonged hospital stays, rehabilitation, and loss of livelihood. Augmentation of mean arterial pressure (MAP) between 85-90 mmHg for seven days after injury to support perfusion of the spinal column is a common practice despite a lack of high-quality evidence. Achieving MAP augmentation often requires observation in the intensive care unit (ICU) and intravenous vasopressors, prolonging lengths of stay (LOS) and increasing healthcare costs. Midodrine, an oral α_1 -agonist, is often employed to maintain elevated MAP as a vasopressor-sparing agent. We hypothesized that midodrine use for tSCI patients with MAP goals would not be associated with shorter ICU LOS.

Methods: A retrospective cohort study of consecutive adult tSCI cases treated between January 1, 2021 and January 1, 2026 at a single Level 1 trauma center was performed. Participants were stratified by midodrine use. The primary outcome was ICU LOS. Secondary outcomes evaluated include hospital LOS, choice of IV vasopressor, duration of and adherence to MAP augmentation, use of invasive lines, and need for midodrine on discharge. Continuous variables are presented using median and IQR and compared using Wilcoxon rank-sum test. Categorical variables are presented as frequencies and percentages and compared using Chi-squared (χ^2) test.

Results: A total of 120 patients were enrolled in the study. The midodrine cohort included 81 patients (67.5%). The midodrine cohort's median ICU LOS was 10 days (IQR 7, 13) compared to 8 days (IQR 5, 13) in the vasopressor-only group ($p = 0.117$). The median proportion of time spent at or above MAP goals was 0.76 (IQR 0.68, 0.83) for the midodrine cohort and 0.82 (IQR 0.71, 0.87) for the vasopressor-only cohort. A statistically significant difference was observed in the achievement of MAP goals favoring the vasopressor-only cohort ($p = 0.047$). Hospital LOS was not different between groups, 16 days (IQR 12, 27) compared to 16 days (IQR 11, 27) in the midodrine versus vasopressor-only group, respectively ($p = 0.749$). Of the patients started on midodrine for MAP goals in the ICU, 31% ($n = 25$) were discharged on midodrine.

Conclusion: Midodrine administration was not associated with shorter ICU LOS. Midodrine was associated with less adherence to MAP goals suggesting less efficacy to IV pressors and may contribute to polypharmacy at discharge. Additional high-quality multi-center studies are needed to further define the role of midodrine in MAP augmentation in the ICU following tSCI.