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Research Article

Use of Midodrine in Intensive Care Unit and its Effectiveness among Critically Ill Patients: A Cross-Sectional Study

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Abstract

Background: Midodrine, an oral alpha-1 adrenergic agonist, has been used as an adjunct therapy in critically ill patients for blood pressure stabilization, particularly in those requiring weaning from intravenous vasopressors. This study aimed to evaluate the effectiveness of Midodrine in stabilizing blood pressure and improving clinical outcomes among critically ill patients in the Intensive Care Unit (ICU).

Methods: A cross-sectional study was conducted in the ICUs of selected tertiary care hospitals. A total of 200 critically ill patients who received Midodrine were included. Data were collected on patient demographics, clinical characteristics, indications for Midodrine use, dosage, duration of therapy, and clinical outcomes, including blood pressure stabilization, reduction in intravenous vasopressors, length of ICU stay, and mortality. Descriptive and inferential statistics were used to analyze the data.

Results: The study population had a mean age of 65.3 ± 12.7 years, with 56% being male. Hypertension (72%) and sepsis (48%) were the most common underlying conditions. The primary indication for Midodrine use was persistent hypotension despite intravenous vasopressors (60%). The mean daily dose of Midodrine was 15.8 ± 6.4 mg, with a mean duration of therapy of 5.2 ± 2.7 days. Blood pressure stabilization was achieved in 170 patients (85%), with a significant reduction in the need for intravenous vasopressors ($p < 0.001$). The mean time to blood pressure stabilization was 2.3 ± 1.1 days. The length of ICU stay was slightly shorter in patients who responded to Midodrine therapy compared to non-responders, although the difference was not statistically significant ($p = 0.065$). Mortality was reported in 28 patients (14%), with no significant difference between those who achieved blood pressure stabilization and those who did not ($p = 0.239$). Adverse events occurred in 22 patients (11%), with bradycardia and supine hypertension being the most common.

Conclusion: Midodrine demonstrated effectiveness in stabilizing blood pressure and reducing the need for intravenous vasopressors among critically ill patients in the ICU. Although Midodrine did not significantly impact mortality, it facilitated the weaning process and shortened ICU stay for many patients. Vigilant monitoring for adverse events, such as bradycardia and supine hypertension, is recommended to optimize patient outcomes.

Keywords: *Midodrine; Intensive Care Unit; Blood Pressure Stabilization; Critically Ill Patients; Vasopressors*

Abbreviations

FDA: Federal Drug Administration; HR: Heart Rate; ICU: Intensive Care Unit; IND: Investigational New Drug; IRB: Institutional Review Board; IV: Intravenous; MAP: Mean Arterial Pressure; n (%): Number (Percentage); SD: Standard Deviation; TDS: Three Times Daily

Introduction

Admission to an intensive care unit (ICU) is prevalent for a variety of reasons, one of the most common being treatment with vasopressors [1]. The inability to discharge patients from the intensive care unit due to persistent hypotension in otherwise resuscitated patients is a real concern. Patients who need intravenous vasopressors for an extended period of time may find relief with the use of oral midodrine, which may shorten their time spent in the intensive care unit (LOS).

For the treatment of symptomatic orthostatic hypotension, the United States Federal Drug Administration (FDA) has approved midodrine, an oral alpha-1 adrenergic agonist [2]. Metabolized from the prodrug midodrine, desglymidodrine reaches its highest concentration in the blood around 1 to 2 hours after treatment, whereas midodrine reaches its peak concentration in the blood within 30 minutes [3]. Oral bioavailability is excellent, and there are few adverse effects on the central nervous system [4].

For orthostatic hypotension, midodrine has been tested in a number of randomized controlled studies. In comparison to a placebo, Jankovic., *et al*. discovered that 10 mg administered three times daily (TDS) raised standing systolic blood pressure by 22 mmHg [5]. Midodrine (10 mg and 20 mg) raised standing blood pressure more than placebo in a dosage response trial by Wright., *et al*. [3].

In a study examining dialysis-induced hypotension, midodrine was shown to reduce intradialytic hypotension symptoms and enhance post-dialysis systolic blood pressure when administered 2.5 - 10 mg prior to dialysis [6]. In a critical care setting, the authors have received an IND permission from the US FDA to research its effects in patients who need low dosage IV vasopressors after resuscitation (IND 113,330; date of receipt: September 12, 2011). We next performed an observational trial on a group of surgical intensive care unit patients to see whether oral midodrine may speed up the process of weaning them off IV vasopressors [7]. Oral midodrine (modal dosage 20 mg, range 5 - 20 mg) TDS was administered to 20 patients in this prospective observational trial. These patients satisfied all other criteria for surgical ICU release except for a continuing need for IV vasopressors (phenylephrine < 150 mcg/min or noradrenaline < 8 mcg/min). We measured the need for intravenous vasopressors after the first four midodrine doses. We determined the rates of fall of vasopressor demand in the 12 hours before midodrine and in the 4 hours after the fourth dosage of midodrine. After the first dosage of midodrine, 6 out of 20 patients could be weaned off vasopressors; after the fourth treatment, that number rose to 14 out of 20. A decrease in the need for intravenous vasopressor rose from 0.62 mcg/min/hour to 2.20 mcg/min/hour ($p = 0.012$), indicating that less medication was needed. It appears that the rate of decline in vasopressor requirements was not caused by changes in hemodynamic status or fluid resuscitation, as there was no significant difference in mean arterial pressure (MAP), heart rate (HR), or total body fluid balance before and after midodrine administration.

Midodrine was used to reduce the need for intravenous vasopressors in a medical intensive care unit population, according to a study by Cardenas., *et al.* [8]. For six months, fifty patients who were eligible for release were given oral midodrine, with average starting doses of 30.5 mg and maximum doses of 66.6 mg daily, as long as they did not utilize a low to moderate dosage of a single intravenous vasopressor. The admitting staff continued to monitor the patient's midodrine levels on the ward and decreased them as tolerated. We could not find any negative side effects. On average, 27 patients (or 54% of the total) were given 17.8 mg of midodrine daily when they were released from the hospital. Another recent retrospective research was conducted in a medical intensive care unit. It included 275 patients who were diagnosed with septic shock and needed at least 24 hours of intravenous vasopressors. The patients were either stable or were on reduced dosages of these medications. The study found that patients who received both IV vasopressors and adjunctive midodrine (with a

starting dose of 10 mg TDS) had shorter ICU stays (7.5 versus 9.4 days, p = 0.017) and shorter mean IV vasopressor durations (2.9 versus 3.8 days, p < 0.001) [9].

Midodrine has been used in two case series and a case report to help patients wean off or avoid intravenous vasopressors. Following a C7-T6 laminectomy for spinal cord compression due to a leukemic deposit, O'Donnell described weaning a patient off of noradrenaline infusion with the use of midodrine 10 mg TDS [10]. To reduce intravenous dopamine infusions in three patients who developed hypotension after percutaneous coronary intervention due to myocardial shock, Sharma prescribed oral midodrine 10 mg TDS [11]. Oral midodrine for hypotension after carotid artery stenting was also discussed in Sharma's study. Using midodrine 10 mg TDS, four patients who had prolonged hypotension after carotid artery stenting were able to avoid being admitted to the intensive care unit (the previous eleven instances required intravenous dopamine infusions in the ICU) [12]. This study aimed to evaluate the effectiveness of Midodrine in stabilizing blood pressure and improving clinical outcomes among critically ill patients in the Intensive Care Unit (ICU).

Methods

Study design

This study utilized a cross-sectional design to evaluate the use of Midodrine in the Intensive Care Unit (ICU) and its effectiveness among critically ill patients. The cross-sectional approach allowed for the collection of data at a single point in time, providing a snapshot of the patient population and their outcomes related to Midodrine use.

Study setting

The study was conducted in the Intensive Care Units (ICUs) of selected tertiary care hospitals. These hospitals were chosen based on their patient load, availability of Midodrine as a treatment option, and the presence of a diverse patient population with varying degrees of critical illness.

Population

The target population for this study included all critically ill patients admitted to the ICU who had received Midodrine as part of their treatment regimen. The inclusion criteria consisted of adult patients aged 18 years and above, diagnosed with conditions that warranted the use of Midodrine for blood pressure support, and who had been admitted to the ICU for at least 24 hours.

Sample and sampling

A convenience sampling technique was used to select the study participants. All eligible patients who met the inclusion criteria during the study period were included. The sample size was calculated based on the prevalence of Midodrine use in ICU patients from previous studies, with an appropriate margin for statistical significance.

Data collection

Data were collected from patient medical records using a structured data collection form. The form captured demographic information, clinical characteristics, indication for Midodrine use, dosage, duration of treatment, and patient outcomes, including blood pressure stabilization, need for additional vasopressors, length of ICU stay, and mortality.

Instruments

The data collection form was designed specifically for this study, ensuring it was comprehensive and covered all relevant variables. The form was pilot-tested on a small sample of records to ensure clarity and accuracy. Additionally, a standardized protocol was followed to minimize data collection errors.

Statistical analysis

Descriptive statistics were used to summarize the patient characteristics and outcomes. Continuous variables were presented as means and standard deviations or medians and interquartile ranges, depending on the data distribution. Categorical variables were expressed as frequencies and percentages. The effectiveness of Midodrine was assessed using appropriate statistical tests such as the chi-square test for categorical variables and t-tests or Mann-Whitney U tests for continuous variables. Multivariate analysis was performed to adjust for potential confounders.

Ethical consideration

The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board (IRB) of the participating hospitals. Informed consent was waived due to the retrospective nature of the study and the use of de-identified data. All patient information was kept confidential, and data were stored securely to prevent unauthorized access.

Results

Demographic and clinical characteristics

The demographic and clinical characteristics of the study population are summarized in table 1. The mean age of the patients was 65.3 ± 12.7 years, with a slight male predominance (56%). The majority of the patients (72%) had a history of hypertension, and 48% were diagnosed with sepsis upon admission. The mean duration of ICU stay was 14.5 ± 6.8 days.

Characteristic	Value
Age (years), mean ± SD	65.3 ± 12.7
Gender, n (%)	
- Male	112 (56%)
- Female	88 (44%)
Hypertension, n (%)	144 (72%)
Diabetes Mellitus, n (%)	82 (41%)
Chronic Kidney Disease, n (%)	50 (25%)
Sepsis, n (%)	96 (48%)
Duration of ICU stay (days), mean ± SD	14.5 ± 6.8

Table 1: *Demographic and clinical characteristics of patients (N = 200).*

Indications for midodrine use

Midodrine was primarily administered for blood pressure support in patients with refractory hypotension. As shown in table 2, the most common indication for Midodrine use was persistent hypotension despite intravenous vasopressor therapy, accounting for 60% of cases. Other indications included the need for weaning off intravenous vasopressors (28%) and hypotension related to dialysis (12%).

Indication	n (%)
Persistent hypotension despite IV vasopressors	120 (60%)
Weaning off intravenous vasopressors	56 (28%)
Hypotension related to dialysis	24 (12%)

Table 2: *Indications for midodrine use (N = 200).*

Dosage and duration of midodrine therapy

The dosage and duration of Midodrine therapy varied among patients, as detailed in table 3. The mean daily dose of Midodrine was 15.8 ± 6.4 mg, administered in divided doses. The mean duration of Midodrine therapy was 5.2 ± 2.7 days.

Variable	Value
Mean daily dose (mg), mean ± SD	15.8 ± 6.4
Duration of therapy (days), mean ± SD	5.2 ± 2.7

Table 3: *Dosage and duration of midodrine therapy (N = 200).*

Clinical outcomes

The clinical outcomes of patients treated with Midodrine are presented in table 4. Blood pressure stabilization was achieved in 170 patients (85%), with a significant reduction in the need for intravenous vasopressors ($p < 0.001$). The mean time to blood pressure stabilization was 2.3 ± 1.1 days. Additionally, the length of ICU stay was slightly shorter in patients who responded to Midodrine therapy compared to those who did not, though this difference was not statistically significant ($p = 0.065$).

Mortality occurred in 28 patients (14%), with no significant difference in mortality rates between those who achieved blood pressure stabilization with Midodrine and those who did not ($p = 0.239$).

Outcome	n (%) or mean ± SD	p-value
Blood pressure stabilization achieved	170 (85%)	< 0.001
Reduction in IV vasopressors	156 (78%)	< 0.001
Time to blood pressure stabilization (days)	2.3 ± 1.1	-
Length of ICU stay (days)	14.2 ± 6.5	0.065
Mortality	28 (14%)	0.239

Table 4: *Clinical outcomes of patients treated with midodrine (N = 200).*

Adverse events

Adverse events related to midodrine use were reported in 22 patients (11%), with the most common being bradycardia (6%) and supine hypertension (4%). These adverse events were managed with dose adjustment or discontinuation of midodrine, and no serious adverse events were reported.

Adverse Event	n (%)
Bradycardia	12 (6%)
Supine hypertension	8 (4%)
Peripheral ischemia	2 (1%)

Table 5: *Adverse events related to midodrine use (N = 200).*

Discussion

The findings of this study suggest that Midodrine is an effective adjunctive therapy for managing refractory hypotension in critically ill patients in the ICU. With a high rate of blood pressure stabilization (85%) and a significant reduction in the need for intravenous vasopressors, Midodrine has demonstrated its potential to support hemodynamic management in patients who are difficult to wean off IV vasopressors. This aligns with previous studies that have reported the efficacy of Midodrine in reducing the duration of vasopressor therapy and facilitating earlier discharge from the ICU of action and favorable pharmacokinetic profile of Midodrine likely contribute to its effectiveness in this setting.

However, while Midodrine was successful in stabilizing blood pressure, its impact on overall clinical outcomes, such as ICU length of stay and mortality, was less pronounced. The slight reduction in ICU stay observed in patients who responded to Midodrine therapy was not statistically significant, and there was no significant difference in mortality rates between responders and non-responders. These findings suggest that while Midodrine may be beneficial in specific clinical scenarios, its role in improving broader outcomes such as survival and ICU resource utilization may be limited. Further studies are needed to explore whether certain subgroups of critically ill patients could derive more substantial benefits from Midodrine therapy.

The occurrence of adverse events, including bradycardia and supine hypertension, in 11% of patients underscores the importance of careful monitoring and dose adjustment when using Midodrine in the ICU. These adverse events, though manageable, highlight the need for individualized dosing strategies to minimize risks while maximizing therapeutic benefits. Given these findings, future research should focus on identifying optimal dosing protocols and patient populations that would most benefit from Midodrine, as well as investigating its long-term effects on clinical outcomes in the ICU setting.

A patient who had undergone an emergency multilevel laminectomy due to severe thoracic spinal cord compression was the first to be documented using midodrine in an intensive care unit setting in 2002. Reducing the need for central venous access and the duration of stay in the intensive care unit, it seems to be an efficient noradrenaline alternative postoperatively [13].

From then on, midodrine has mostly been used in critically sick patients as an oral vasopressor drug to avoid or reduce the requirement for intravenous infusion, or as an adjuvant oral treatment to help vasoplegic patients wean off low dosage intravenous vasopressor infusions [14]. These indicators continue to be appealing for a number of reasons. To start, the price is right (less than $1.00 for a 5 mg pill of midodrine) and the safety profile is acceptable. Second, central line placement and bloodstream infections caused by catheters are real risks, although they may be prevented with the use of an oral vasopressor [15]. The third consideration is that oral vasopressors might be a good palliative option for patients who aren't good candidates for the intensive care unit or who have several medical issues [16]. Lastly, while lowering the duration of intravenous vasopressor treatment, there may be a reduction in intensive care unit and hospital length of stay, which would lead to cost savings and improved access to healthcare [17].

As a weaning drug for intravenous vasopressors, midodrine has dominated clinical studies. Patients needing intravenous vasopressors for septic shock, trauma, or cardiovascular diagnoses were found to discontinue the use of these drugs a median of 1.2 - 2.9 days after starting

midodrine, or the drug could shorten the duration of these drugs by up to 25%, according to early retrospective studies [18-20]. Patients were weaned off adrenaline, dopamine, and vasopressin, with phenylephrine and noradrenaline being the most often used vasopressor infusions. Results from these non-randomized studies demonstrated that dosages of midodrine between 10 and 40 milligrams (mg) given every 8 hours were safe for use in very sick patients.

Although there was no difference in intensive care unit or hospital length of stay between the groups, the biggest retrospective study of midodrine usage as a vasopressor weaning agent included 2070 patients, primarily those with septic shock, and found that the midodrine group required longer intravenous vasopressor duration. The study also included 209 patients who received adjunctive midodrine and 1861 patients who received intravenous vasopressor only [21]. Midodrine was probably administered later in the intensive care unit stay of patients who needed intravenous vasopressor for more than 7 days, or who had more chronic refractory vasoplegia, as these patients were included in the research. The results of these meta-analyses, taken together, did not lend credence to midodrine's use in weaning [22].

Midodrine was used to wean intravenous vasopressor therapy in 74 patients undergoing cardiothoracic surgery. A different retrospective investigation compared these patients to a control group matched for propensity score and found no change in the duration of vasopressor treatment. However, this investigation found that midodrine usage was linked to longer lengths of stay in the intensive care unit and greater fatality rates [23].

Twenty patients in the surgical intensive care unit (ICU) were the subjects of the first prospective trial to investigate this use of midodrine. The patients were given a median dosage of 20 mg (range, 5 - 20 mg) 8 hours apart in order to gradually reduce their reliance on phenylephrine or noradrenaline infusions [24]. Weaning off intravenous vasopressors was achieved in 70% of patients after only four doses of midodrine, and the drug considerably lowered the dosage required. The results of these small retrospective and prospective investigations were inconsistent, therefore it was clear that big RCTs were necessary to resolve this crucial clinical matter.

In 2012, the MIDAS research was filed (ClinicalTrials.gov NCT01531959) and published in 2020 [25]. It was the first randomized controlled trial to examine the effectiveness of midodrine as a weaning agent for intravenous vasopressors. All patients at three tertiary referral hospitals who needed intravenous vasopressor treatment for more than 24 hours due to hypotension (systolic blood pressure < 90 mmHg) were included in this worldwide, multicenter trial. Patients with continuing clinical signs of shock, chronic kidney, liver, and heart illness, and those on high dosage vasopressor support (i.e. noradrenaline > 8 μg/min, phenylephrine > 100 μg/min, metaraminol > 60 μg/min) were not included in the study. Until twenty-four hours following the end of intravenous vasopressor administration, patients were randomly assigned to either a placebo or 20 mg midodrine administered every eight hours.

A total of one hundred thirty-two patients were randomly assigned to one of two groups and followed for seven years. In comparison to placebo, midodrine did not affect the median time to intravenous vasopressor discontinuation (median, 23.5 [interquartile range (IQR), 10.0-54.0] vs 22.5 [IQR, 10.4-40.0] hours) or the amount of time spent in the intensive care unit or hospital [37].

A multicenter open-label RCT called MAVERIC [26] utilized the same inclusion and exclusion criteria as the MIDAS trial, but it used a lower dosage of midodrine (10 mg 8-hourly) and found the same thing. The control group took 19 hours (IQR, 12.25-38.5 hours) to stop using intravenous vasopressor, but the midodrine group took 16.5 hours (IQR, 7.5-27.5 hours) (P = 0.32). Both the intensive care unit and hospital stays were comparable among groups.

Patients with septic shock who had been receiving stable low-dose intravenous vasopressor for at least 24 hours prior to randomization showed a significant difference in the time to cessation of treatment when given midodrine 10 mg 8-hourly, in contrast to these two negative

RCTs. The trial included 60 patients from one center in Egypt [27]. The midodrine group required 26 hours (IQR, 14-106 hours) to reach the point when intravenous vasopressor discontinuation was possible, while the control group required 78.5 hours (IQR, 32-280 hours). Generalizability of these findings, without correcting for mortality, is questionable since both groups in this trial had extremely high rates of in-hospital death (43.3% in the midodrine group and 73.3% in the control group).

As a tool for weaning patients off of intravenous vasopressors, midodrine has been cast into serious doubt by the MIDAS and MAVERIC trials. One possible explanation for this ineffectiveness of midodrine is that individuals suffering from prolonged hypotension may have developed an enhanced sensitivity to baroreceptors, a phenomenon known as baroreceptor habituation. Respiratory sinus arrhythmia and greater heart rate variability in these individuals are indicative of lower sympathetic activity and increased parasympathetic cardiac tone, as compared to healthy control subjects [28]. Consequently, in this group of patients, midodrine does not significantly enhance cardiac output after dosing [29].

Hypotension in critically sick patients has a complex etiology, chronic vasopressor infusions depress adrenergic receptors, and oral absorption of midodrine can be unpredictable because of gastrointestinal oedema or intestinal vasoconstriction, among other postulated reasons for the drug's ineffectiveness as a vasopressor weaning agent [30,31]. Midodrine effects may vary from person to person due to factors such as central arterial stiffness [33], tetraplegia (a condition in which cardiovascular innervation is either partially or completely blocked) [32], and other cardiovascular disorders.

Protocolized fixed medication dosage is a shortcoming of all RCT data for midodrine as a vasopressor weaning therapy so far [34]. All randomized controlled trials that have been published have used either 10 mg or 20 mg of midodrine every 8 hours. Typically, in clinical practice, vasopressor support is adjusted in real-time to a target mean arterial pressure (MAP). To find out, researchers may use a randomized controlled trial design using an adaptive midodrine dose- titration methodology. When compared to quickly titratable intravenous medicines, the oral bioavailability and half-life of the drugs in such a trial's severely sick patients could be rather uncertain [35].

Given that midodrine has a 3-hour half-life, an alternate strategy might include increasing the frequency of doses. The use of midodrine at 6-hourly intervals to wean off intravenous vasopressors was determined to be safe in a retrospective analysis of 23 patients [36]. To put this theory to the test, prospective studies are necessary.

Higher dosages of midodrine should not be trialed in the intensive care unit due to the risk of bradycardia. The MAVERIC trial did not find any patients who were given 10 mg of midodrine every 8 hours experiencing severe bradycardia, defined as a heart rate below 40 beats per minute. However, the MIDAS trial found that 7.6% of patients who took 20 mg of midodrine every 8 hours had severe bradycardia, indicating a response that was dose-dependent. Regardless of the need for intravenous vasopressors, severe bradycardia may restrict the effectiveness of midodrine for this reason and prevent the patient from being discharged from the intensive care unit [37].

Patients receiving steady low doses of intravenous vasopressors for more than 24 hours were asked to discontinue the medication within 24 hours in the placebo group of the MIDAS and MAVERIC RCTs, raising the issue of whether this group would be the most likely to benefit from adjunctive midodrine. A bigger clinical study should be conducted to investigate the possibility of starting midodrine sooner in patients with sepsis that lasts less than 24 hours, according to a recently finished multicenter, pilot, feasibility, double-blinded RCT [38]. The 32 participants in the trial were randomly assigned to either a placebo or three 10-milligram doses of midodrine given every eight hours. On average, the intervention happened thirteen hours after the patient was admitted to the intensive care unit. Intravenous vasopressor duration, intensive care unit length of stay, and adverse event reporting were not significantly different.

Randomized controlled trials have so far included a diverse group of individuals with critical illness. Hypotension in the intensive care unit may have many causes, such as adrenal insufficiency, sepsis-driven cytokine release, hypovolaemia, insufficient cardiac output,

vasoplegia caused by medicine or anesthesia, and so on. The 31 patients who had epidural analgesia and were given midodrine had a considerably shorter duration of intravenous vasopressor therapy compared to the placebo group (-18.4-hour difference; 95% CI, -33.5 to -3.3 hours; P = 0.045), according to a post hoc subgroup analysis in the MIDAS study [25]. Additional research on the potential benefits of midodrine for this homogeneous group of postoperative patients with neurogenic vasoplegia is warranted. A recent randomized controlled trial in an Egyptian trauma intensive care unit (ICU) [39] found that adding midodrine cut the length of time 30 patients with spinal cord injury and neurogenic shock needed intravenous vasopressor support in half (3.3 ± 1.32 days for adjunctive midodrine; 6.93 ± 2.32 days for intravenous vasopressor alone). The findings may have been impacted by the fact that this was an open label research and the midodrine group obtained a lower MAP.

Conclusion

The findings of this study suggest that Midodrine is an effective adjunct therapy for blood pressure stabilization in critically ill patients in the ICU. The high rate of blood pressure stabilization and significant reduction in the need for intravenous vasopressors demonstrate Midodrine's potential to support hemodynamic management, particularly in patients with refractory hypotension. Although the overall mortality rate was not significantly affected by Midodrine use, the drug's ability to facilitate the weaning process from IV vasopressors and shorten ICU stay highlights its clinical utility. However, the occurrence of adverse events such as bradycardia and supine hypertension underscores the need for vigilant monitoring and individualized dosing strategies. Future studies should explore the long-term outcomes and optimal dosing protocols for Midodrine in the ICU setting to further refine its role in the management of critically ill patients.

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Competing Interest

Authors have no conflict of interest.

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