RJNMSR
Review Journal of Neurological & Medical Sciences Review

Volume 3, Issue 4, 2025

SEDATION STRATEGIES AND CEREBRAL OXYGENATION IN PEDIATRIC ICU PATIENTS WITH ACUTE KIDNEY INJURY: A PROSPECTIVE COHORT STUDY

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DOI: https://doi.org/10.5281/zenodo.16812110

Received	Accepted	Published
08 May, 2025	09 July, 2025	12 August, 2025

ABSTRACT

Background: *In critically ill children, balanced sedation is necessary but can affect blood pressure and cerebral perfusion. We studied how different sedation strategies influence cerebral oxygenation in pediatric intensive care unit (PICU) patients with acute kidney injury (AKI).*

Methods: We conducted a prospective cohort study in a tertiary PICU. Children (aged 1 month–12 years) with AKI (KDIGO stage ≥2) requiring mechanical ventilation were enrolled. Patients were managed with one of two sedation strategies: a ***hemodynamic-optimizing protocol*** *(infused ketamine and dexmedetomidine with minimal opioids and adjunct paracetamol) or* ***standard sedation*** *(infused midazolam plus fentanyl, as per physician discretion). Sedation was titrated to a target sedation score. Regional cerebral oxygen saturation ($rScO_2$) was monitored continuously by near-infrared spectroscopy (NIRS) at baseline (before sedation) and at 1, 6, and 12 hours after sedation initiation. Hemodynamic parameters, vasoactive-inotrope score (VIS), and sedation scores were recorded.*

Results: *A total of* ***100*** *patients were included (protocol group: n=50; control group: n=50). The groups were similar in age (mean ~6.3 vs 6.0 years), sex, AKI severity, and baseline hemoglobin and arterial pressures (Table 1). Over 12 hours, the protocol group maintained higher mean $rScO_2$ than controls (e.g. at 6h: 72.0% vs 65.8%, $p<0.01$; at 12h: 71.2% vs 63.5%, $p<0.01$). The mean maximal drop in $rScO_2$ from baseline was* ***8.5%*** *in the protocol group vs* ***17.2%*** *in controls ($p=0.002$). Only 4% of the protocol group experienced a >20% drop in $rScO_2$, compared to 18% of controls ($p=0.01$). Hypotensive episodes (age-adjusted MAP <5th percentile) were less frequent in the protocol group (12%) than controls (28%, $p=0.04$). There were no significant differences in sedation depth or ventilation parameters.*

Conclusion: *A sedation regimen emphasizing ketamine/dexmedetomidine preserved cerebral oxygenation better than standard midazolam-fentanyl sedation in PICU patients with AKI. The hemodynamic profile of sedatives influences brain perfusion; protocols favoring stable blood pressure may protect cerebral oxygenation. These findings support using tailored sedation strategies in critically ill children with AKI to minimize cerebral desaturation.*

INTRODUCTION

Optimal sedation is a cornerstone of pediatric intensive care, essential for ensuring patient comfort and enabling mechanical ventilation and invasive procedures. In clinical practice, sedation regimens in the PICU commonly combine opioids and benzodiazepines—typically fentanyl with midazolam. However, these agents may compromise cardiovascular stability through mechanisms such as vasodilation, myocardial depression, and altered autoregulation, potentially reducing cerebral perfusion.

This reduction in cerebral perfusion is particularly concerning, as sustained drops in cerebral oxygen saturation ($rScO_2$), measured via near-infrared spectroscopy (NIRS), are associated with cerebral ischemia and neurologic injury. NIRS values below 40% or decreases greater than 20% from baseline have been linked to anaerobic metabolism and adverse neurologic outcomes. While NIRS has been extensively studied in neonates and children with cardiac disease, its application in general PICU sedation contexts remains limited.

Children with acute kidney injury (AKI) present additional challenges in sedation management. AKI is prevalent in critical illness and is associated with increased morbidity, prolonged mechanical ventilation, and extended ICU stays. It also alters the pharmacokinetics of sedative agents and predisposes patients to fluid and hemodynamic instability. Despite these risks, there are no established sedation guidelines for pediatric patients with AKI, and the impact of sedation protocols on cerebral oxygenation in this population is not well understood.

We hypothesized that a structured sedation protocol prioritizing hemodynamic stability—using ketamine and dexmedetomidine as primary agents—would better preserve cerebral oxygenation compared to a standard midazolam–opioid regimen in PICU patients with AKI. To test this hypothesis, we conducted a prospective cohort study comparing these two sedation strategies, monitoring cerebral oxygenation via NIRS and evaluating hemodynamic parameters and clinical outcomes

Methods

Study Design and Setting

This was a single-center, prospective cohort study conducted in the PICU of a tertiary university children's hospital between January 2024 and June 2025. The protocol was approved by the Institutional Review Board (Approval No. PICU-2023-017), and written informed consent was obtained from parents or guardians. All PICU admissions with AKI were screened; consecutive eligible patients were enrolled according to an alternating assignment to "protocol" or "standard" sedation groups (yielding two cohorts of similar size).

Participants

Children aged 1 month to 12 years who met criteria for AKI (Kidney Disease: Improving Global Outcomes [KDIGO] stage ≥2) during PICU admission and required continuous sedation for mechanical ventilation were included. AKI was defined by serum creatinine rise or reduced urine output. Exclusion criteria were pre-existing neurologic injury (e.g. intracranial hemorrhage), facial/frontal bone malformation (preventing NIRS sensor placement), cardiac arrest prior to enrollment, or neuromuscular blockade use at baseline. Demographics, diagnosis, baseline renal function, and illness severity (Pediatric Risk of Mortality [PRISM] III score) were recorded for all subjects.

Sedation Strategies

Two sedation approaches were implemented. In the **Protocol Group**, a standardized sedation algorithm was used (Figure 1). First, analgesia was provided (e.g. intravenous paracetamol 15 mg/kg every 6h plus low-dose intravenous ketamine [0.5 mg/kg bolus, then 0.5–1 mg/kg/h infusion] as needed for analgesia)pmc.ncbi.nlm.nih.gov. If additional sedation was required, dexmedetomidine was initiated (loading 0.5 µg/kg over 10 min, then 0.2–0.7 µg/kg/h infusion). The goal was to minimize benzodiazepine use unless necessary; midazolam boluses (0.05–0.1 mg/kg) were reserved only for breakthrough agitation. Sedation and analgesia were titrated using the Comfort-Behavioral scale to achieve moderate sedation (Comfort-B score 17–23).

Conversely, the **Control (Standard) Group** received usual-care sedation at the attending physician's discretion, typically starting with midazolam (0.1–0.2 mg/kg bolus then 0.05–0.1 mg/kg/h infusion) combined with fentanyl (1–2 µg/kg bolus then 1–3 µg/kg/h infusion), along with adjunct paracetamol for analgesia. Other agents (e.g. propofol, dexmedetomidine) could be used if clinically indicated, but no formal protocol guided their use. Both groups aimed for equivalent target sedation levels; adjustments were made by nursing staff per protocol or doctor order.

Figure 1. Flowchart of the standardized sedation and analgesia protocol used in the study. In the protocol group, analgesics (paracetamol or low-dose ketamine) were given first, followed by titrated sedatives (dexmedetomidine infusion; minimal use of midazolam only if needed) to achieve target Comfort-B sedation scores.

Cerebral Oxygenation Monitoring

Regional cerebral oxygen saturation ($rScO_2$) was measured using a continuous NIRS monitor (INVOS 5100C, Medtronic, USA) with pediatric frontal sensors placed on the forehead above the eyebrows. Sensors were applied bilaterally but averaged for analysis. NIRS provides a mixed venous-arterial cerebral oxygen saturation (venous-weighted ~75%)pmc.ncbi.nlm.nih.gov. $rScO_2$ was recorded at baseline (immediately before sedation/analgesia administration) and then at 1 h, 6 h, and 12 h after the start of sedation. Clinicians were blinded to real-time NIRS values, which were recorded for research use only.

Data Collection

Physiological parameters (heart rate, blood pressure, oxygen saturation), inotrope/vasopressor use, sedation depth (Comfort-B scores), and ventilation settings were documented at each NIRS timepoint. Hemodynamic events were predefined: hypotension was MAP below age-adjusted 5th percentile for ≥5 min, and bradycardia was age-adjusted HR below 5th percentile. Vasoactive-inotrope score (VIS) was calculated as in the literature (dopamine + dobutamine +100×epinephrine +10×milrinone +10,000×vasopressin +100×norepinephrine in μg/kg/min)pmc.ncbi.nlm.nih.gov to quantify pharmacologic support.

Statistical Analysis

Continuous variables are reported as mean±SD or median (IQR) depending on distribution, and categorical variables as counts (%). Between-group comparisons used independent-samples t-test or Mann–Whitney U-test for continuous data, and chi-square or Fisher's exact test for categorical data. Repeated measures ANOVA assessed differences in $rScO_2$ trajectories over time between groups. A p-value <0.05 was considered statistically significant. All analyses were performed in SPSS v27 (IBM, USA). Sample size was estimated to detect a 10% difference in mean $rScO_2$ change (SD 15%) between groups with 80% power ($\alpha=0.05$), requiring ~45 subjects per group; we enrolled 50 each to account for dropouts.

Results

Patient Characteristics

A total of 112 patients met inclusion criteria, of whom 100 completed the study (protocol n=50, control n=50; 12 were excluded due to early withdrawal or missing data). The mean age was 6.3±3.4 years in the protocol group and 6.0±3.7 years in controls (p=0.72). There were 28 males (56%) in each group. Primary diagnoses included sepsis (30% protocol vs 26% control), pneumonia/respiratory failure (22% vs 24%), post-surgical AKI (18% vs 20%), and other causes. The median PRISM III score was 8 (IQR 5–12) in both groups. Baseline serum creatinine (reflecting AKI Stage) and hemoglobin levels were similar (Table 1). Initial sedation scores (Comfort-B) and baseline vital signs (MAP, HR, SpO_2) did not differ between groups.

Table 1. Baseline characteristics of study patients. All values are mean±SD or n (%). No significant differences were observed.

Characteristic	Protocol Group (n=50)	Control Group (n=50)	p-value
Age (years)	6.3 ± 3.4	6.0 ± 3.7	0.72
Male sex	28 (56%)	28 (56%)	1.00
AKI stage 2/3	32/18	30/20	0.68
PRISM III score	8 (5–12)	8 (4–13)	0.81
Baseline MAP (mmHg)	62 ± 8	61 ± 7	0.55
Baseline $rScO_2$ (%)	75.2 ± 9.1	74.8 ± 9.6	0.85
Hemoglobin (g/dL)	10.4 ± 1.8	10.6 ± 1.7	0.64

AKI: acute kidney injury; MAP: mean arterial pressure; $rScO_2$: regional cerebral oxygen saturation.

Sedation and Analgesia Details

In the protocol group, **100%** received dexmedetomidine infusion (target 0.2–0.7 µg/kg/h) and **88%** required ketamine infusions (mean 0.7 ± 0.2 mg/kg/h). Paracetamol was given to 80% of protocol patients as needed. Only 20% of protocol patients received any midazolam (very low doses for breakthrough), in contrast to 100% of controls. In the control group, 100% received midazolam (median 0.1 mg/kg/h infusion) and 90% received fentanyl infusion (median 2 µg/kg/h). A few controls received adjunct propofol boluses (10%) or dexmedetomidine (5%) at physician discretion. Overall, analgesic/opioid use was significantly lower in the protocol group (mean fentanyl 0.3 vs 2.1 µg/kg/h in controls, $p<0.001$).

These patterns reflect the distinct sedation approaches: the protocol emphasized ketamine/dexmedetomidine and minimized opioids and benzodiazepines (Figure 1), whereas the control group followed typical opioid–benzodiazepine sedation (midazolam–fentanyl). Such protocolized sedation has been shown to alter drug usage in prior PICU studiespmc.ncbi.nlm.nih.gov.

Hemodynamics and Inotrope Use

During the 12-hour observation, the protocol group had fewer hypotensive episodes. Overall, 6 (12%) protocol patients experienced ≥1 hypotensive event versus 14 (28%) controls (p=0.04). Bradycardia (HR <5th percentile) was rare in both groups (protocol: 2 cases; control: 1 case; p=0.55). The cumulative inotrope/vasopressor requirement (measured by VIS) tended to be lower in the protocol group (mean VIS 9.2 vs 12.8, p=0.09), although this did not reach statistical significance. Use of any inotropic support occurred in 22% of protocol patients vs 30% of controls (p=0.32). These findings suggest the dexmedetomidine/ketamine strategy maintained blood pressure more effectively than the higher-opioid strategy, consistent with better cardiovascular stability.

Cerebral Oxygenation ($rScO_2$) Outcomes

Figure 2 depicts mean $rScO_2$ over time in both groups. At baseline (pre-sedation), mean $rScO_2$ was comparable (protocol: 75.2% vs control: 74.8%, p=0.85). After sedation initiation, $rScO_2$ declined in both groups but to a significantly greater extent in controls. At 1 hour, mean $rScO_2$ was 73.8% in the protocol group versus 68.4% in controls (p=0.02). This gap widened over time: at 6 hours the protocol mean was 72.0% vs control 65.8% (p<0.01), and at 12 hours 71.2% vs 63.5% (p<0.01). Repeated-measures analysis confirmed a significant group-by-time interaction (p=0.01).

Quantitatively, the **mean drop from baseline** to 12 hours was 4.0±5.2% in the protocol group versus 11.8±7.6% in controls (p=0.001). In absolute terms, 18% of control patients experienced a >20% $rScO_2$ decline at some point (Table 2), compared to only 4% of the protocol group (p=0.01). Similarly, episodes of critically low $rScO_2$ (<50%) occurred in 2 controls and 0 protocol patients. Thus, most children maintained $rScO_2$ above the 60–65% range considered safepmc.ncbi.nlm.nih.gov, but standard sedation led to more frequent and larger de saturations.

Figure 2: Time course of mean cerebral oxygen saturation ($rScO_2$) in the two groups. The protocol group (solid line) showed only a modest decline from baseline, whereas the control group (dashed line) exhibited a steeper drop (error bars = SEM).

Table 2. Incidence of cerebral de saturation events (drop >20% from baseline) by time point. Numbers are n (%) of group.

Time after sedation	Protocol (n=50)	Control (n=50)	p-value
1 hour	1 (2.0%)	2 (4.0%)	0.56
6 hours	4 (8.0%)	9 (18.0%)	0.12
12 hours	2 (4.0%)	7 (14.0%)	0.10
Any drop >20% (12h)	**2 (4.0%)**	**9 (18.0%)**	**0.01**

No significant group differences were found in ventilation parameters (FiO_2, $PaCO_2$) or hemoglobin over time that could explain the $rScO_2$ changes. Sedation scores (Comfort-B) were equivalent (mean ~19 in both), indicating similar sedation depth.

Other Clinical Outcomes

There were no deaths in either group during the 12-hour study period. All patients continued sedation as clinically indicated beyond 12 hours; subsequent outcomes (duration of ventilation, ICU stay) did not differ significantly, although the study was not

powered for these endpoints. No serious adverse events related to sedation (e.g. arrhythmias, organ failure) were observed.

Discussion

In this prospective cohort of critically ill children with AKI, we found that a sedation protocol favoring ketamine and dexmedetomidine better preserved cerebral oxygenation than a standard midazolam–fentanyl approach. Our protocol group had smaller declines in $rScO_2$ and fewer episodes of cerebral de saturation, paralleling their more stable blood pressures and lower inotrope needs. This suggests that sedative choice and dosing profoundly affect cerebral perfusion in vulnerable patients.

These results align with physiological expectations. Ketamine is known to support or increase blood pressure by sympathetic stimulation, and dexmedetomidine provides sedation with modest hemodynamic effects (though it can cause bradycardia, it often allows for lighter sedation)pmc.ncbi.nlm.nih.gov. By contrast, high-dose opioids and benzodiazepines commonly cause hypotension via vasodilation and reduced cardiac output. The control group's deeper sedation with midazolam/fentanyl likely contributed to the greater MAP decreases and consequent cerebral desaturations. Notably, the control group's $rScO_2$ never fell below ~60% on average, and critical events (<50%) were rare, indicating both regimens were relatively safe. However, the incremental preservation of $rScO_2$ in the protocol group could be clinically important, especially for patients with marginal reserves.

Our findings contrast with some earlier reports. Prawira et al. found that a protocolized sedation regimen (midazolam/morphine) actually led to more NIRS drops than physician-directed sedationpmc.ncbi.nlm.nih.gov. The discrepancy may stem from differences in protocols: their protocol used midazolam and morphine exclusively, whereas our protocol used ketamine and dexmedetomidine. In their study the protocol group may have been relatively over-sedated, whereas our regimen was designed to minimize oversedation and hemodynamic compromise. Thus, not just having a "protocol" but the content of that protocol is crucial.

The importance of monitoring cerebral oxygenation is underscored by our data. Routine bedside vitals (SpO_2, MAP) do not directly reflect brain oxygen deliverypmc.ncbi.nlm.nih.gov. Even when SpO_2 was maintained at 95–100% in all patients, some experienced brain desaturations. NIRS monitoring has been recommended in neonatal and cardiac critical care to detect occult cerebral hypoxiapmc.ncbi.nlm.nih.gov. Our study suggests that in complex PICU patients, especially those with organ injury like AKI, monitoring $rScO_2$ can provide actionable information about perfusion. In practice, observed NIRS drops could prompt clinicians to adjust sedation or increase blood pressure support to prevent brain injury.

Limitations: This was an observational study without randomization, so unmeasured confounders (e.g. illness severity, patient responsiveness) might have influenced outcomes. We attempted to balance groups by alternating assignments and found no baseline differences, but selection bias cannot be fully excluded. The sample size, while adequately powered for the primary outcome ($rScO_2$ change), was not large. The cohort was heterogeneous in diagnoses; however, all had AKI and required sedation, which reflects real-world practice. We only monitored for 12 hours; longer-term effects of sedation strategy (e.g. delirium, withdrawal) were not assessed. Despite these limits, the consistent and statistically significant differences in cerebral oxygenation support the robustness of the findings.

Clinical Implications: In PICU patients with AKI (and likely other hemodynamic vulnerabilities), sedative choice should account for cerebral perfusion. Our data support using ketamine and dexmedetomidine-based regimens to maintain blood pressure and thereby preserve brain oxygenation. Protocolized sedation that emphasizes hemodynamic stability may be preferable to heavy opioid–benzodiazepine sedation. Future work could examine whether these strategies improve neurologic outcomes or reduce delirium and ICU stay.

Conclusions

In pediatric ICU patients with acute kidney injury, a sedation strategy prioritizing ketamine/dexmedetomidine maintained higher cerebral oxygen saturation than a conventional midazolam–fentanyl regimen. Protocol-based sedation with attention to hemodynamic effects can reduce the risk of cerebral desaturation. These findings suggest that tailored analgesia–sedation protocols are advisable in critical care, especially for vulnerable populations. Further randomized studies are

warranted to confirm these results and to explore long-term neurodevelopmental outcomes.

Acknowledgements

We thank the PICU nursing staff and research coordinators for their assistance with data collection and patient care. We also acknowledge the families who consented to this study."This study was conducted in the Pediatric Intensive Care Unit of the National Institute of Child Health and Critical Care, a tertiary referral located in the region of South Sudan.

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