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CASE SERIES

Renal Resistive Index in Early Detection of AKI after Neurosurgery: A Case Series

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Abstract

Postoperative acute kidney injury (AKI) is a frequent yet underrecognized complication in neurosurgical patients, often contributing to increased morbidity and mortality. Brain injury can trigger systemic effects, including sympathetic overactivation, inflammatory responses, and hemodynamic instability, all of which predispose patients to renal dysfunction. The Renal Resistive Index (RRI), obtained by Doppler ultrasonography, reflects intrarenal vascular resistance and has emerged as a promising early marker of AKI. This case series describes three patients undergoing decompressive craniectomy for intracranial hemorrhage who had normal preoperative renal function and subsequently developed stage 1 AKI according to Kidney Disease; Improving Global Outcome (KDIGO) guideline within 24 hours postoperatively. In each case, an elevated RRI (>0.7) measured during the early postoperative period preceded the rise in serum creatinine and the reduction in urine output. The consistent pattern across these patients highlights the potential utility of RRI as a noninvasive, bedside predictor of postoperative AKI in neurosurgical populations. The findings support the clinical relevance of integrating RRI into perioperative monitoring, particularly in high-risk patients where early detection of renal dysfunction may guide timely interventions to optimize hemodynamic stability and prevent further injury. Further prospective studies with larger cohorts are warranted to validate these observations and establish standardized thresholds for practice.

Keywords: Acute kidney injury; Intracranial hemorrhage; Neurosurgery; Postoperative care; Renal resistive index

Renal Resistive Index dalam Deteksi Dini AKI Pasca Bedah Saraf: Kasus Serial

Abstrak

Cedera ginjal akut pascaoperasi (*Acute Kidney Injury*/AKI) merupakan komplikasi yang sering terjadi namun kurang dikenali pada pasien bedah saraf. Hal ini berkontribusi pada peningkatan morbiditas serta mortalitas. Cedera otak dapat memicu efek sistemik, termasuk aktivasi simpatis berlebihan, respons inflamasi, dan ketidakstabilan hemodinamik, yang semuanya dapat meningkatkan risiko disfungsi ginjal. *Renal Resistive Index* (RRI), yang diperoleh melalui ultrasonografi Doppler, merefleksikan resistensi vaskular intrarenal dan muncul sebagai penanda awal yang menjanjikan untuk AKI. Seri kasus ini melaporkan tiga pasien yang menjalani kraniektomi dekompresi akibat perdarahan intrakranial dengan fungsi ginjal preoperatif normal. Namun, dalam 24 jam pascaoperasi berkembang menjadi AKI stadium 1 menurut kriteria *Kidney Disease; Improving Global Outcome* (KDIGO). Pada setiap kasus, peningkatan nilai RRI (>0,7) pada periode awal pascaoperasi mendahului kenaikan kadar kreatinin serum serta penurunan produksi urin. Pola konsisten yang ditemukan pada ketiga pasien ini menekankan potensi RRI sebagai prediktor noninvasif di samping tempat tidur untuk AKI pascaoperasi pada populasi bedah saraf. Temuan ini mendukung relevansi klinis integrasi pemantauan RRI dalam manajemen perioperatif, khususnya pada pasien berisiko tinggi di mana deteksi dini disfungsi ginjal dapat mengarahkan intervensi tepat waktu untuk mengoptimalkan stabilitas hemodinamik dan mencegah kerusakan lebih lanjut. Studi prospektif dengan jumlah sampel yang lebih besar masih diperlukan untuk memvalidasi temuan ini dan menetapkan ambang baku yang dapat diterapkan dalam praktik klinis.

Kata kunci: Cedera ginjal akut; Perdarahan intrakranial; Bedah saraf; Perawatan pascaoperasi; *Renal resistive index*

Introduction

Acute kidney injury (AKI) is among the most frequent complications after major surgery, associated with prolonged hospitalization and increased mortality.[1] A systematic review of 19 studies (82,514 patients) reported a pooled incidence of 13.4% after major abdominal surgery. Another meta-analysis estimated an overall postoperative AKI rate of 16%, with variations across surgical types. Gynecological procedures had the lowest incidence (6%), while digestive and mixed abdominal surgeries reached 15% and 19%, respectively.[1,2] In contrast, cardiac surgery showed wider variability (5–42%). At our hospital, AKI occurs in 7–30% of intensive care patients, contributing to longer stays, higher costs, and mortality.[3] Despite its importance, postoperative AKI following neurosurgery remains underreported, indicating a need for further research.

In neurosurgical patients, diagnosis is often delayed as attention focuses on neurological recovery. Yet, studies show cerebral injury may disrupt renal function through autonomic, inflammatory, and hemodynamic mechanisms.[4] Thus, monitoring kidney function in intracranial hemorrhage surgery is clinically relevant.

The Renal Resistive Index (RRI), assessed by Doppler ultrasonography, is a simple noninvasive indicator of intrarenal vascular resistance. Values above 0.7 suggest early renal impairment.[5] Unlike serum creatinine, which increases only after significant damage, RRI detects early perfusion changes, making it useful for perioperative monitoring.[6]

This case series presents three patients with intracranial hemorrhage undergoing emergency craniectomy who developed postoperative AKI despite normal baseline renal function. Increased RRI consistently preceded biochemical AKI markers, emphasizing its potential as an early predictor of renal dysfunction in neurosurgical cases.

Case Presentation

Case I

A 59-year-old male with no chronic kidney disease presented with sudden severe headache and decreased consciousness (GCS 7, BP 160/95 mmHg, HR 96 bpm). Baseline renal function was normal (creatinine 1.03 mg/dL; clearance 71 mL/min). CT revealed a large intracerebral and epidural hematoma with midline shift, and emergency decompressive craniectomy was performed. With balanced fluids and intermitten vasopressor support. At 12 hours postoperatively, RRI was 0.81 and urine output 0.44 mL/kg/h. At 24 hours, creatinine rose to 1.51 mg/dL (clearance 48.4 mL/min), fulfilling stage 1 AKI. Conservative management with fluid optimization and monitoring was applied.

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Case II

A 60-year-old hypertensive female presented with altered consciousness after a traumatic fall (GCS 7, BP 150/90 mmHg, HR 88 bpm). Baseline renal function was normal (serum creatinine 1 mg/dL; clearance 66.7 mL/min). Head CT revealed a large intracerebral and intraventricular hemorrhage with significant mass effect, prompting emergency craniectomy. Postoperatively, hemodynamics remained stable under standard anesthetic and intensive care management. At 12 hours, Doppler ultrasound showed an RRI of 0.763 with urine output 0.34 mL/kg/h; at 24 hours, serum creatinine increased to 2.22 mg/dL (clearance 3.03 mL/min), fulfilling stage 2 AKI criteria. Neurological status remained stable, and conservative management effectively prevented additional complications.

Case III

A 47-year-old hypertensive male presented with sudden loss of consciousness (GCS 9, BP 160/90 mmHg, HR 102 bpm). Preoperative renal function was normal (serum creatinine 1.05 mg/dL; clearance 104.5 mL/min). CT scan showed spontaneous intracerebral hemorrhage, and urgent decompressive craniectomy was performed. The intraoperative course was stable with adequate fluid and vasopressor support. At 12 hours postoperatively, RRI was 0.77 with urine output 0.31 mL/kg/h; at 24 hours, serum creatinine increased to 1.4 mg/dL, consistent with stage 1 AKI. Conservative renoprotective management restored urine output to 1.5 mL/kg/h without complications.

Table 1. Demographic and Clinical Characteristics of Patients

Charateristics	Case I	Case II	Case III
Gender	Male	Male	Male
Age (years)	59	60	47
Diagnosis	Moderate TBI + tEDH R Temporal + tICH R Temporoparietal + tSAH R FrontoTemporal + IVH R Lateral Ventricle	sICH R FrontoTemporal + sIVH	sICH L External Capsule ec Rupture MCA Bifurcation Aneurysm + sIVH L Lateral Ventricle + sSAH + Acute Non Communicating Hydrocephalus
Type of Surgery	Decompressive Craniectomy	Decompressive Craniectomy	Decompressive Craniectomy + EVD
Co-morbidities	None	Hypertension	Hypertension
Baseline Scr (mg/dL)			
Intraoperative	1.03	1.00	1.05
Postoperative (24 hr)	1.51	2.22	1.40
Baseline BUN (mg/dL)			
Intraoperative	12.8	10.8	11.1
Postoperative (24 hr)	19.2	16.2	16.6
Baseline CCT (mg/dL)			
Intraoperative	71.0	66.6	104.5
Postoperative (24 hr)	48.4	30.3	78.4
Urine Output (mL/kgBB/hr)			
Intraoperative	2.08	0.41	0.93
Postoperative (12 hr)	0.44	0.34	0.31
Postoperative (24 hr)	0.58	0.57	1.50
KDIGO Stage	I	II	1
RRI Postoperative (12 hr)	0.810	0.763	0.770
Outcome	Improved after optimization	Improved after optimization	Improved after optimization

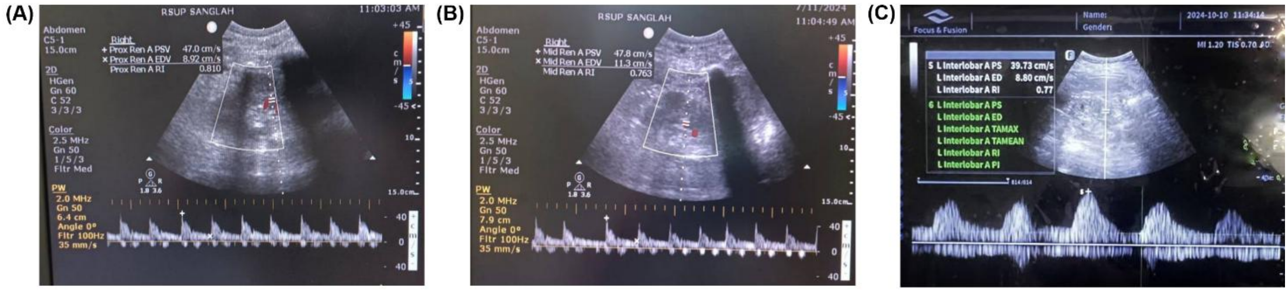

Figure 1. Postoperative Doppler Assessment of RRI

Discussion

RRI measurement is most valuable after achieving hemodynamic stability, typically within 12–24 hours post-surgery, when the transition from intraoperative stress to recovery occurs. A persistently elevated RRI (>0.7) during this phase may indicate subclinical renal hypoperfusion, even when serum creatinine and urine output remain normal. Detection of a high RRI should prompt reassessment of mean arterial pressure, fluid status, and vasoactive drug use.[2]

This case series highlights the utility of RRI as an early indicator of postoperative AKI in neurosurgical patients. Despite stable perioperative hemodynamics and normal baseline renal function, all three cases developed stage 1–2 AKI within 24 hours of craniectomy. Notably, RRI values >0.7 consistently preceded biochemical changes, suggesting that RRI may allow earlier identification of renal impairment than conventional laboratory parameters.

The brain–kidney interaction has gained increasing attention in neurosurgical care, as AKI occurs in 3–20% of patients and contributes to poorer outcomes.[7,8] In those undergoing intracranial hemorrhage surgery, systemic complications often extend beyond the central nervous system, with AKI representing an underrecognized contributor to morbidity and mortality.[9]

Several mechanisms explain the brain–kidney interplay (Figure 2). Neurohumoral activation following brain injury triggers sympathetic overactivity and stimulation of the renin–angiotensin–aldosterone system (RAAS), leading to vasoconstriction, sodium retention, and renal hypoperfusion.[4,10] Systemic inflammation driven by cytokines such as interleukin-6 and TNF-α further impairs endothelial and microvascular function.[10,11] Oxidative stress and mitochondrial dysfunction add to tubular injury and reduce renal adaptive capacity, making the kidneys more vulnerable to perioperative insults.[12]

Moreover, neurosurgical procedures themselves pose hemodynamic challenges. Tight blood-pressure control, osmotic diuretics, vasopressors, and anesthetic agents can compromise renal perfusion. Combined with systemic inflammatory responses and surgical stress, these factors create a “perfect storm” predisposing patients to postoperative AKI.[4]

In this case series, all patients displayed a consistent pattern: early RRI elevation followed by AKI within 24 hours, underscoring RRI’s potential to bridge neurological and renal monitoring. RRI reflects renal vascular resistance and perfusion in real time.[1] Brain injury induces sympathetic activation, inflammation, and circulatory disturbances that can impair renal perfusion before creatinine elevation or oliguria occur.[7,8] As a bedside Doppler parameter, RRI enables early detection of these changes. Prior studies in intensive care and surgical populations have similarly linked elevated RRI to AKI, particularly in cardiac and abdominal procedures, suggesting comparable mechanisms in neurosurgical settings where renal monitoring is often secondary.[1,5]

Nonetheless, RRI interpretation requires caution. It is influenced by systemic factors such as arterial compliance, intra-abdominal pressure, and vasoactive drug use.[5] The limited number of cases precludes statistical inference, and operator expertise may affect reproducibility, highlighting the need for standardized measurement protocols. In this series, certified anesthesiologists performed RRI assessment 12 hours postoperatively using portable Doppler ultrasonography to detect early renal perfusion changes. AKI was defined by using both creatinine and urine output, ensuring diagnostic accuracy and alignment with international recommendations.

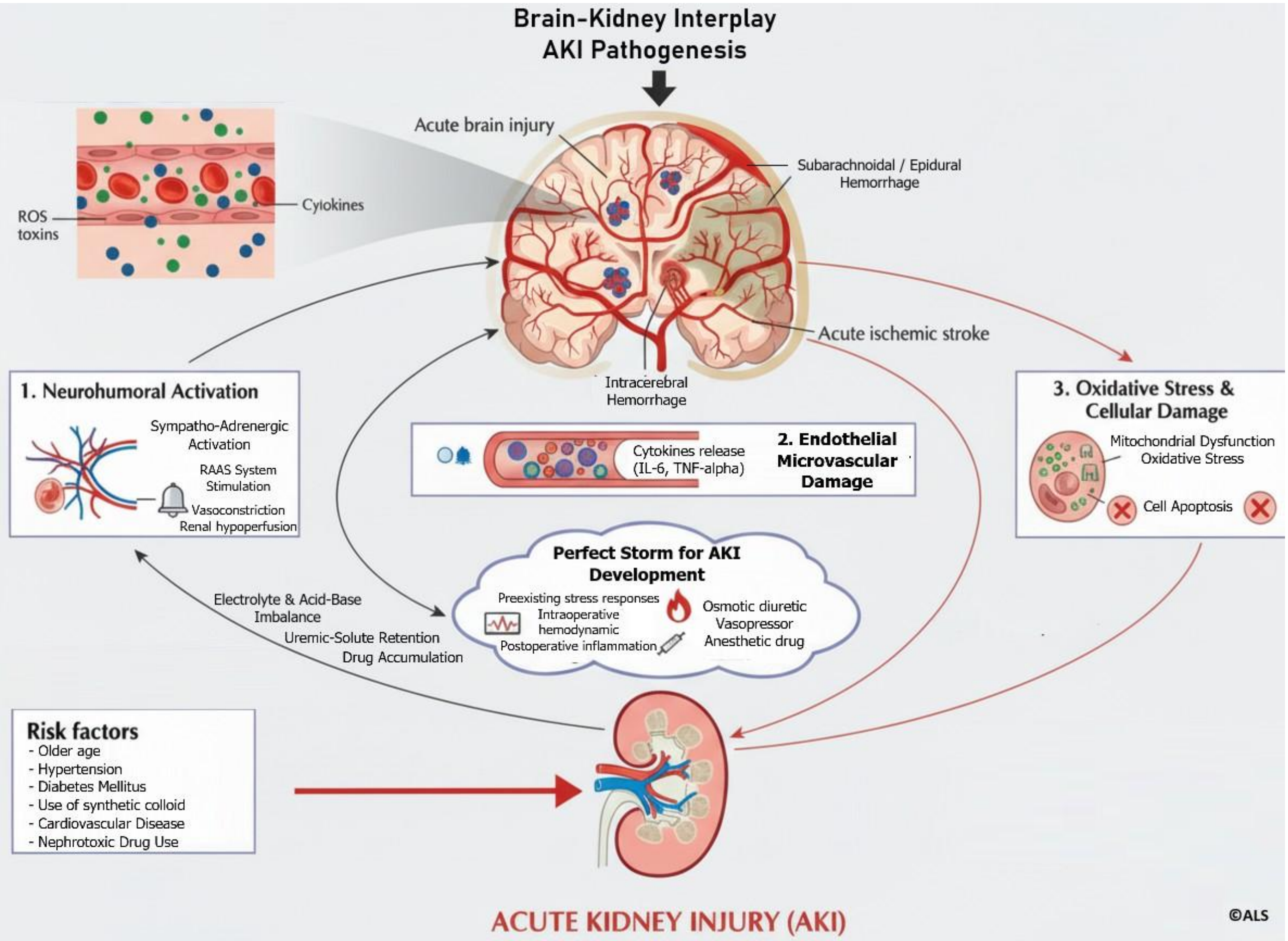

Figure 2. Schematic of Brain–Kidney Interplay in Postoperative AKI [4,10–12]

All patients received careful fluid management, hemodynamic optimization, and avoidance of nephrotoxins.[2] Although AKI was not prevented, early identification enabled intensified monitoring and supportive interventions that may have mitigated progression. Neurosurgical patients should thus be monitored not only for intracranial stability but also for early renal vulnerability.[4,8] Larger, prospective studies are needed to determine whether RRI-guided management improves renal outcomes in this population.

This report remains limited by its small sample size, lack of a control group, and absence of long-term renal follow-up. Consequently, the prognostic value of early postoperative RRI elevation requires confirmation through larger, controlled studies employing standardized ultrasonography protocols and defined RRI thresholds for intervention.

Acknowledgement

Nil.

Declaration of Patient Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published, and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of Interest

The authors report no conflict of interest.

Data Availability Statement

De-identified patient data from this case report will be made available upon reasonable request to the corresponding author following publication, subject to institutional data-sharing policies and ethics approval.

Author's Contributions

All authors contributed significantly to the conception and design of the study, data collection, analysis, and interpretation of the results. All authors participated in writing and critically revising the manuscript for important intellectual content, approved the final version to be published, and are accountable for all aspects of the research.

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