E-ISSN 2250-0944

ISSN 2250-1150
doi: 10.48047/ijprt/15.02.367

Research Article

Renal, Urological, and Microbiological Perspectives on Acute Kidney Injury Following Colectomy

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Abstract

Acute kidney injury (AKI) is a common and serious complication following major colorectal surgery (colectomy), with multifactorial pathophysiology implicating renal perfusion disruption, urinary tract manipulations, and postoperative infections. This prospective observational study evaluated renal, urological and microbiological factors associated with postoperative AKI in adult patients undergoing elective colectomy. A total of 320 patients were enrolled and followed for 7 days post-surgery; AKI was defined by KDIGO criteria (increase in serum creatinine ≥0.3 mg/dL within 48 hours or ≥1.5× baseline within 7 days). The incidence of AKI was 12.8% (41/320). In multivariate logistic regression adjusting for age, baseline eGFR, surgical complexity, and intraoperative hypotension, independent predictors of AKI were prolonged operative time (>180 min) (adjusted OR 2.5; 95% CI 1.4–4.6; p=0.003), intraoperative hypotension (mean arterial pressure <65 mmHg for >20 min) (OR 3.1; 95% CI 1.7–5.8; p<0.001), urinary catheter reinsertion due to intra-operative urological issue (OR 2.8; 95% CI 1.3–6.1; p=0.008), and postoperative culture-positive urinary tract infection (UTI) (OR 4.2; 95% CI 2.0–8.9; p<0.001). Bacterial isolates from UTI-associated AKI cases were predominantly Enterococcus faecalis (37%) and Escherichia coli (29%) including extended-spectrum β-lactamase producers. Patients with AKI had longer hospital stay (median 11 vs 7 days; p<0.001) and higher 30-day morbidity (Clavien-Dindo ≥III, 29% vs 11%; p=0.002). Urinalysis evidence of proximal tubular injury (urinary neutrophil

gelatinase-associated lipocalin [uNGAL] >150 ng/mL) on postoperative day 1 was also significantly higher in AKI group (p=0.004). These findings underscore the interplay of renal perfusion, urinary tract injury/manipulation, and microbial factors in AKI after colectomy and highlight actionable risk factors for prevention and monitoring. Incorporation of intraoperative renal-protective protocols, urological evaluation of catheter management, and early microbiological surveillance may reduce AKI incidence and improve outcomes.

Keywords: acute kidney injury; colectomy; urinary tract infection; intraoperative hypotension; uNGAL.

Introduction

Acute kidney injury (AKI) is a frequent and clinically significant postoperative complication in major abdominal surgery, including colectomy for benign and malignant indications. The incidence of AKI following colorectal resection ranges from 10% to 20% in published cohorts, and is associated with increased morbidity, length of stay, progression to chronic kidney disease and long-term mortality. The pathophysiology of post-colectomy AKI is multifactorial, involving hemodynamic instability, abrupt shifts in renal perfusion, inflammatory mediators, nephrotoxic exposures and urinary tract perturbations. A nuanced understanding of renal, urological and microbiological perspectives is essential to developing effective prevention and management strategies.1-5

From a renal perspective, intraoperative factors are pivotal. Prolonged operative time, significant fluid shifts, hemorrhage, and episodes of hypotension all compromise renal perfusion and precipitate ischemic tubular injury. The use of vasopressor agents, variability in renal blood flow autoregulation, and transient reductions in glomerular filtration predispose to an environment wherein acute tubular necrosis may develop. Biomarkers of tubular injury—such as urinary neutrophil gelatinase-associated lipocalin (uNGAL), kidney injury molecule-1 and interleukin-18—have demonstrated potential to identify subclinical injury in this setting. However, the specific patterns of renal tubular injury after colectomy have been less extensively explored compared with cardiac or vascular surgery.6-8

From the urological perspective, colectomy often involves periprocedural urinary catheterization, manipulation of the bladder or ureters (especially in low anterior resections), ureteric stents, or inadvertent urinary tract trauma. Re-catheterization, obstruction, hematuria and urinary retention may thus influence renal function indirectly via obstruction, increased intravesical pressure and retrograde effects on the renal tract. Postoperative urinary tract infection (UTI) further complicates this scenario by adding an inflammatory and obstructive component, which may exacerbate renal injury. Studies examining AKI in abdominal surgery rarely separate purely renal perfusion issues from urological contributions, yet these may present additive risk.9-12

Microbiological factors are increasingly recognised as important contributors to postoperative AKI. UTIs, particularly in the setting of instrumentation, may lead to systemic inflammation, sepsis-associated renal dysfunction, urinary obstruction from fungal/ bacterial biofilms and direct tubular injury from urinary pathogens. The prevalence of catheter-associated UTIs in colectomy populations can approach 7–12%. The microbial flora encountered – including Enterococcus spp., Escherichia coli and even multi-drug-resistant organisms – may influence the severity and reversibility of associated renal insult. Understanding the microbiological profile of UTIs associated with AKI provides opportunities for targeted preventive strategies.

Despite these inter-related domains, few studies have concurrently assessed renal hemodynamic/injury biomarkers, urological events (catheter manipulation, urinary retention) and microbiological data in a single post-colectomy population to identify combined predictors of AKI. Better characterisation of these variables could enable stratified risk-profiling, targeted intra- and post-operative surveillance, and tailored prophylactic interventions. Moreover, recognition of actionable urological and microbial factors may permit interventions beyond standard renal protective protocols (e.g., earlier catheter removal, antimicrobial prophylaxis, urinary flow monitoring).

Methodology

This prospective observational cohort study enrolled adult patients (aged ≥18 years) undergoing elective colectomy (open or laparoscopic) for benign or malignant indications at Central Park Medical College, Lahore over a 18-month period. Ethical approval was obtained and verbal informed consent secured from all participants. Patients with preoperative end-stage renal disease

(eGFR <30 mL/min/1.73 m² or on renal replacement therapy), known urinary tract obstruction, baseline AKI, or emergent surgery were excluded. Pre-operative baseline serum creatinine, eGFR (CKD-EPI), urinalysis and urinary neutrophil-gelatinase-associated lipocalin (uNGAL) were obtained. During surgery, intraoperative data were collected: operative time, estimated blood loss, fluid volumes, vasopressor use, recorded hypotensive episodes (mean arterial pressure <65 mmHg >20 minutes) and urinary catheterization events (insertion, reinsertion, intraoperative catheter change). Post- operatively, patients were followed for 7 days with daily serum creatinine, urine output monitoring and urinalysis. Urinary catheter was removed per standard protocol; any episodes of catheter re-insertion, retention, hematuria or signs of obstruction were recorded. Urinary cultures were performed promptly for suspected UTIs or catheter changes; positive cultures were recorded with organism and resistance profile. AKI was defined using KDIGO criteria (increase in serum creatinine ≥0.3 mg/dL within 48 h or ≥1.5× baseline within 7 d). Urinary tubular-injury biomarker uNGAL was measured on post-operative day 1. Length of hospital stay, Clavien-Dindo complication grade and 30-day readmission were recorded. After pilot data suggested AKI incidence ≈15%, sample size calculation using Epi Info (v7) for logistic regression (anticipated OR=3 for catheter re-insertion) with $\alpha=0.05$ and $\beta=0.80$ yielded requirement of 288 patients; enrollment target was increased to 320 to allow for drop-outs and missing microbiological data. Statistical analysis was conducted using SPSS v26: incidence reported as percentage with 95% CI; bivariate comparisons used chi-square for categorical variables and t-test or Mann-Whitney for continuous variables; multivariate logistic regression entered variables with $p<0.10$ in bivariate analysis and forced age/sex; urinary biomarker values were log-transformed prior to modelling; $p<0.05$ considered significant.

Results

Table 1. Baseline and intraoperative characteristics by AKI status (n = 320)

Characteristic	No AKI (n = 279)	AKI (n = 41)	p-value
Mean age (years)	62.3 ± 9.4	64.7 ± 8.8	0.11
Male sex — n (%)	164 (58.8%)	25 (61.0%)	0.78
Baseline eGFR (mL/min/1.73 m²)	82.4 ± 15.1	68.9 ± 13.4	<0.001

Characteristic	No AKI (n = 279)	AKI (n = 41)	p-value
Operative time >180 min — n (%)	115 (41.2%)	28 (68.3%)	0.001
Intraoperative hypotension — n (%)	46 (16.5%)	21 (51.2%)	<0.001
Catheter reinsertion — n (%)	23 (8.2%)	17 (41.5%)	<0.001

Brief explanation: Patients who developed AKI had lower baseline renal function, longer operative times, more intraoperative hypotension and higher rates of catheter reinsertion during or after surgery.

Table 2. Biomarker, urinary tract and microbiological findings

Parameter	No AKI	AKI	p-value
Post-op Day 1 uNGAL (ng/mL, median [IQR])	87 [55–130]	215 [162–310]	0.004
URINARY TRACT INFECTION (culture +) — n (%)	12 (4.3%)	19 (46.3%)	<0.001
Most common isolates	E. coli (42%), Entero faecalis (25%)	E. faecalis (37%), E. coli (29%)	—
ESBL/Group1 resistance — % among UTI isolates	17%	32%	0.04

Brief explanation: Urinary biomarker uNGAL was markedly higher in AKI cases. Culture-positive UTIs were significantly more frequent in the AKI group, with a shift toward more resistant organisms.

Table 3. Outcomes and multivariate predictors of AKI

Predictor	Adjusted OR (95% CI)	p-value
Operative time >180 min	2.5 (1.4–4.6)	0.003
Intraoperative hypotension	3.1 (1.7–5.8)	<0.001

Predictor	Adjusted OR (95% CI)	p-value
Catheter reinsertion	2.8 (1.3–6.1)	0.008
Culture-positive UTI	4.2 (2.0–8.9)	<0.001

Postoperative length of stay was longer in the AKI group (median 11 vs 7 days; p<0.001). Thirty-day Clavien-Dindo grade ≥III complications occurred in 29% of AKI versus 11% of no-AKI (p=0.002). Thirty-day readmission rate was 14.6% in AKI group vs 4.3% (p=0.01).

Intraoperative, urological and microbiological variables emerged as independent predictors of AKI. AKI was associated with substantially worse postoperative outcomes including longer hospital stay, higher complication and readmission rates.

Discussion

This prospective study demonstrates an AKI incidence of 12.8% following elective colectomy in a tertiary surgical setting and identifies a triad of predictive domains: renal perfusion/hemodynamic derangements, urinary tract instrumentation/infection, and early tubular injury biomarker elevation. The findings reinforce the concept that AKI in major abdominal surgery is not purely a matter of renal perfusion but often involves urological and microbiological pathways that are modifiable.13-15

Prolonged operative time and intraoperative hypotension surfaced as prominent renal perfusion–based predictors, consistent with literature on abdominal and vascular surgical populations. These findings underscore the critical importance of surgical efficiency, intraoperative monitoring of MAP, and optimisation of fluid/vasopressor management for renal protection. Moreover, the strong association between urinary catheter reinsertion/trauma and AKI emphasises the urological dimension: intraoperative or postoperative manipulations, catheter dislodgement or retention may precipitate intravesical hypertension or facilitate ascending infection, thereby contributing to renal insult beyond pure perfusion issues.16-18

The microbiological findings are particularly noteworthy: nearly half of AKI cases had culture-positive UTIs, compared with only 4.3% of non-AKI cases, and resistance rates were higher in the

AKI group. This suggests that catheter-related infection plays a potent role in precipitating or amplifying renal injury in the postoperative setting. The dominance of Enterococcus faecalis and Escherichia coli aligns with known urinary device-related pathogens, but the elevated rate of extended-spectrum resistance signals a greater challenge for prevention and therapy in this surgical population.19-20

The marked elevation of urinary NGAL on post-operative day 1 among AKI patients supports the utility of early tubular injury biomarkers for risk stratification. uNGAL may identify subclinical injury before overt creatinine rise, allowing earlier intervention. Incorporation of such biomarkers into postoperative monitoring protocols could enable targeted nephrology consults, tighter fluid balance surveillance and earlier urinary tract assessment in high-risk patients.

Clinically, the implication is that a bundled strategy addressing perfusion, urology and infection may reduce AKI incidence. Practical steps include minimising catheter dwell time, enforcing aseptic care and early removal, intraoperative fluid and hemodynamic optimisation, and prompt urinary culture and targeted antibiotic therapy in suspected infections. Such a multifaceted approach may yield better outcomes than renal-only protocols.

Limitations include a single-centre design, potential residual confounding from unmeasured comorbidities (e.g., subclinical nephropathy), and absence of long-term renal follow-up to assess progression to chronic kidney disease. Future multicentre investigations should evaluate cost-effectiveness of uNGAL monitoring and intervention bundles and consider randomised trials of urinary catheter removal timing or antimicrobial prophylaxis in colectomy populations.

In conclusion, this study advances understanding of post-colectomy AKI by integrating renal, urological and microbiological perspectives, identifying actionable predictors and supporting a holistic prevention paradigm. Adoption of such an integrated framework may lead to improved renal outcomes and reduced surgical morbidity.

Conclusion

AKI occurs in approximately one-in-eight patients undergoing elective colectomy and is predicted by prolonged operative time, intraoperative hypotension, urinary catheter reinsertion and culture-

positive UTI. Early monitoring with tubular-injury biomarkers and a preventive bundle addressing renal perfusion, urinary tract management and microbiology may reduce AKI incidence and improve surgical outcomes.

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