Incidence of and risk factors for delayed acute kidney injury in patients undergoing colorectal surgery

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A B S T R A C T

Background: The risk of delayed AKI (AKI development beyond the perioperative period) in patients undergoing colorectal surgery is greater than that in patients undergoing other major operations. However, the characteristics of and risk factors for delayed AKI are unclear.
Methods: We investigated 683 patients who underwent colorectal surgery with intestinal resection at a single tertiary hospital. All patients were followed-up for a year postoperatively. The primary outcome was the development of AKI during follow-up.
Results: AKI occurred in 177 (25.9%) during the first postoperative year. Patients who developed AKI were significantly older, showed a lower body mass index, and significantly lower preoperative hemoglobin and serum albumin levels. AKI occurred most commonly during the first 3 months postoperatively. However, AKI occurred persistently even after this initial period. Older age, lower preoperative serum albumin levels, and late ostomy closure were independently associated with a higher risk of delayed AKI.
Conclusion: AKI commonly occurs beyond the perioperative period. Careful risk stratification and modification of risk factors may prevent delayed AKI in patients undergoing colorectal cancer surgery.
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Introduction

Acute kidney injury (AKI) after major surgery is a common and serious complication.[1,2] AKI adversely affects patient outcomes by prolonging hospitalization with a consequent increase in health-care costs and morbidity and mortality rates.[3,4] The in-hospital mortality rate among patients undergoing colorectal surgery who develop AKI is approximately 10-fold higher than that in patients without AKI.[5]

Most previous studies evaluating the incidence of and risk factors for postoperative AKI have focused on the perioperative period. Perioperative AKI has primarily been reported in patients undergoing cardiac surgery or transplantation.[6–8] Perioperative AKI is primarily attributable to ischemia-reperfusion injury resulting from cardiopulmonary bypass and intraoperative hemodynamic alterations.[9,10] However, patients who undergo colorectal surgery with intestinal resection are also prone to AKI. The mechanism of AKI in patients undergoing colorectal surgery differs from that in patients undergoing other surgeries in that the former group shows chronic salt and water loss and malabsorption in addition to routine postoperative complications including bleeding, infection, and anastomotic site leakage.[11,12] These complications in patients undergoing colorectal surgery last far beyond the immediate postoperative period. Thus, the risk of delayed AKI in these patients is higher than that in those undergoing cardiac surgery.[13,14]

In spite of the possibility that patients undergoing colorectal surgery are more vulnerable to delayed AKI, limited data are available. Characterizing the risks associated with AKI in this group of patients could lead to improved outcomes by determining optimal clinical management strategies including risk modification and close surveillance of high-risk patients.

Therefore, the purpose of this study was to investigate the incidence of and risk factors for delayed AKI in patients undergoing colorectal surgery.

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Methods

Study population

A flow diagram showing the selection of patients is presented in Fig. 1. We evaluated 997 patients who underwent colorectal surgery with intestinal resection (for any indication) between November 2005 and December 2015 at Yonsei University Health System, Seoul, Korea. Exclusion criteria were: (i) patients aged < 18 or ≥ 80 years, (ii) patients with < 3-month follow-up, (iii) patients diagnosed with immediate post-renal AKI, (iv) patients with missing data and, (v) patients with preoperative chronic kidney disease or end-stage renal disease (ESRD). A total of 683 patients were included in the final analysis. These patients were divided into 2 groups based on the development of AKI within a year postoperatively. The study was approved by the Institutional Review Board of the Yonsei University Health System. Because the current study was a retrospective medical-record-based study and all patient data were de-identified, the board waived the need for written consent from patients.

Data collection

Electronic medical records of the included patients were retrospectively reviewed. Demographic and clinical data at the time of surgery including age, sex, body mass index (BMI), underlying disease (hypertension, diabetes, and cardiovascular disease), types of ostomy (none, colostomy, or ileostomy), timing of ostomy closure, and indications for surgery were recorded. An open ostomy for >3 months after ostomy creation was defined as late ostomy closure. Postoperative chemotherapy and the use of renin-angiotensin system (RAS) blockers were also investigated. Laboratory data including hemoglobin, albumin, total cholesterol, blood urea nitrogen, and creatinine levels, and the estimated glomerular filtration rate (eGFR) at the time of admission for surgery were recorded. The causes of delayed AKI were determined by the attending surgeon's or the consulting nephrologist's opinion at the time of AKI development documented in the medical records. In cases where there were no clear descriptions regarding the cause of AKI, the etiology was classified as unknown.

Follow-up and outcome

All patients were followed-up for a year postoperatively. The primary outcome was the occurrence of delayed AKI, defined as the development of AKI after discharge following colorectal surgery. AKI was diagnosed based on the Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease (RIFLE) criteria—Risk: > 1.5-fold increase in serum creatinine levels or a >25% decrease in baseline eGFR; Injury: > 2-fold increase in serum creatinine levels or a >50% decrease in baseline eGFR; Failure: > 2-fold increase in serum creatinine levels or a >50% decrease in baseline eGFR or serum creatinine levels ≥ 4.0 mg/dL with an acute rise of at least 0.5 mg/dL; Loss and ESRD: complete loss of renal function over > 4 weeks or initiation of renal replacement therapy. All patients who met the RIFLE criteria for stages higher than those classified under 'Risk' were considered to have AKI. Serum creatinine levels and eGFR readings recorded within the previous 3 months were considered baseline values.[15,16]

Statistical analysis

Continuous variables were expressed as means ± standard deviations, and categorical variables were expressed as absolute numbers with percentages. Intergroup comparisons were recorded using analysis of variance (ANOVA) or the Student's t-test for continuous variables and the chi-squared test or the Fisher exact test for categorical variables. The value for p for trend was calculated by means of polynomial trend analysis in a one-way ANOVA test. The Kolmogorov–Smirnov test was used to determine the normality of the distribution of parameters. The Univariate and multivariate Cox proportional hazard analysis were performed to identify the risk factors for AKI. Results were presented as hazard ratios (HRs) with 95% confidence intervals (CI). p values < 0.05 were considered statistically significant. All statistical analyses were performed using the SPSS software, version 23 (SPSS Inc., Chicago, IL, USA).
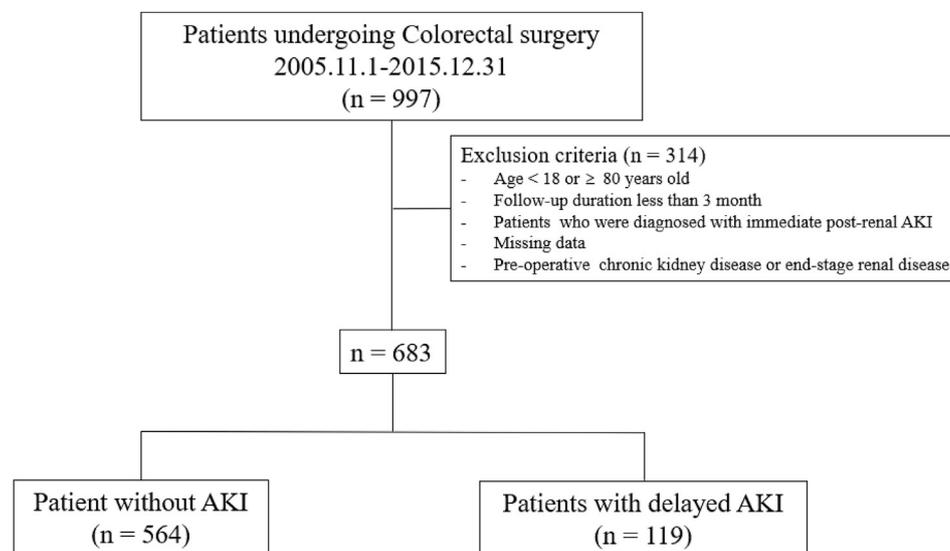
Fig. 1. Flow diagram showing patients included in the study and the study design
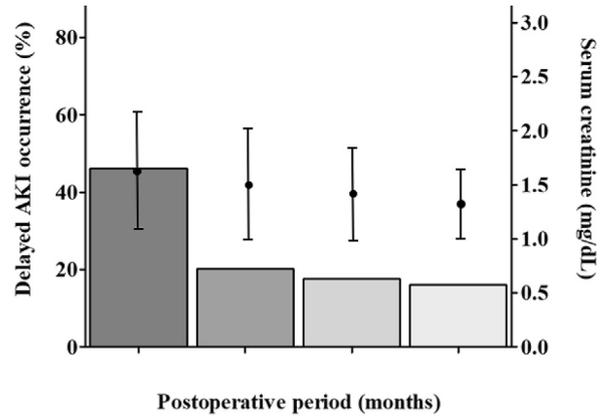AKI = acute kidney injury.

Results

Baseline characteristics of patients

Baseline characteristics and laboratory findings of the study population are shown in Table 1. Among the 683 patients who underwent colorectal surgery with intestinal resection, delayed AKI occurred in 177 (25.9%) within the first postoperative year. The mean age of patients was 57.9 ± 13.5 years at the time of surgery, and 415 patients (60.8%) were men. Patients who developed AKI were significantly older ($p < 0.001$), showed a higher prevalence of hypertension ($p = 0.01$) and diabetes ($p = 0.02$), and a higher postoperative prescription rate of RAS blockers ($p = 0.04$). No intergroup differences were observed in the presence of cardiovascular disease. Additionally, no differences were observed between the AKI and the non-AKI groups in terms of the percentage of patients who underwent postoperative chemotherapy. Preoperative hemoglobin and serum albumin levels in patients who developed AKI were significantly lower than those in patients without AKI. The preoperative eGFR was comparable between groups. Colorectal cancer was the most common surgical indication in both groups. Surgery for urogynecological cancer ($p < 0.001$), and panperitonitis ($p = 0.04$) was performed more frequently in patients with AKI than in patients without AKI. An ostomy was created in 427 patients (62.5%). AKI occurred more commonly among patients with an ostomy. Among the patients with AKI, a colostomy procedure (62.1%) was more common than an ileostomy (29.4%). Late ostomy closure was more common in those with delayed AKI than in those without AKI ($p < 0.001$).

Incidence and causes of delayed acute kidney injury after colorectal surgery

During the first postoperative year, delayed AKI occurred in 177 patients (25.9%). The incidence of AKI was highest during the first 3 months postoperatively and showed a gradual decline thereafter. None of the patients received renal replacement due to the development of delayed AKI in this study, and renal function was recovered in all of the cases. However, when the serum creatinine levels were compared, the mean creatinine levels in those who developed delayed AKI was highest during the first 3 months and showed a tendency to decrease thereafter (p for trend $= 0.029$) (Fig. 2). Most cases of AKI were attributable to unknown etiology (45.2%), followed by dehydration (38.4%), and the use of nephrotoxic medications (9.6%) (Table 2).

Fig. 2. Incidence and severity of delayed acute kidney injury during the first postoperative year.
Boxes indicate delayed AKI incidence rate. Dots indicate mean serum creatinine level of delayed AKI patients. Error bars show the standard deviation of serum creatinine. AKI = acute kidney injury.

Table 1
Baseline characteristics of patients.

	Total (n = 683)	Delayed AKI (n = 177)	Non-AKI (n = 506)	p
Demographic data				
Age (years)	57.9 ± 13.5	60.9 ± 13.0	56.8 ± 13.6	<0.001
Male sex (%)	415 (60.8)	104 (58.8)	311 (61.5)	0.29
BMI (kg/m^2)	22.2 ± 3.6	21.6 ± 3.5	22.4 ± 3.6	<0.01
Medication history (%)				
Postoperative RAS blocker use	82 (12.0)	28 (15.8)	54 (10.7)	0.04
Postoperative chemotherapy	378 (55.3)	98 (55.4)	280 (55.3)	0.53
Underlying diseases (%)				
Hypertension	214 (31.3)	68 (38.4)	146 (28.9)	0.01
Diabetes	116 (17.0)	40 (22.6)	76 (15.0)	0.02
Cardiovascular disease	40 (5.9)	12 (6.8)	28 (5.5)	0.33
Laboratory data				
Hemoglobin (g/dL)	11.7 ± 2.0	11.1 ± 1.9	12.0 ± 2.0	<0.001
Albumin (g/dL)	3.6 ± 0.7	3.3 ± 0.7	3.7 ± 0.7	<0.001
BUN (mg/dL)	12.6 ± 7.1	12.8 ± 6.8	12.6 ± 7.3	0.74
Creatinine (mg/dL)	0.79 ± 0.19	0.77 ± 0.20	0.80 ± 0.19	0.09
eGFR (mL/min/1.73 m^2)	99.2 ± 24.1	100.2 ± 18.3	98.8 ± 25.8	0.44
Indications for surgery (%)				
Colorectal cancer	462 (67.6)	110 (62.1)	352 (69.6)	0.04
Urogynecological cancer	36 (5.3)	24 (13.6)	12 (2.4)	<0.001
Inflammatory bowel disease	43 (6.3)	7 (4.0)	36 (7.1)	0.09
Panperitonitis	22 (3.2)	10 (5.6)	12 (2.4)	0.04
Other benign conditions	79 (11.6)	15 (8.5)	64 (12.6)	0.09
Other malignant conditions	41 (6.0)	11 (6.2)	30 (5.9)	0.51
Ostomy status, n (%)				
Ostomy opening	427 (62.5)	162 (91.5)	265 (52.4)	<0.001
Colostomy	244 (35.7)	110 (62.1)	134 (26.5)	<0.001
Ileostomy	183 (26.8)	52 (29.4)	131 (25.9)	<0.001
Non-ostomy opening	256 (37.5)	15 (8.5)	241 (47.6)	<0.001
Late ostomy closure	385 (56.4)	158 (41.0)	227 (59.0)	<0.001

AKI = acute kidney injury; BMI = body mass index; RAS = renin angiotensin system; BUN = blood urea nitrogen; eGFR = estimated glomerular filtration rate.

Table 2
Causes of delayed acute kidney injury during the first postoperative year.

	Patients with AKI (n = 177)
Unknown (%)	80 (45.2)
Dehydration (%)	68 (38.4)
Nephrotoxic medications (%)	17 (9.6)
Sepsis (%)	12 (6.8)

Nephrotoxic medications include antibiotics, contrast agents, and nonsteroidal anti-inflammatory drugs (NSAIDs). AKI = acute kidney injury.

Factors associated with the development of delayed acute kidney injury

Univariate Cox regression analysis revealed that AKI was significantly associated with age at the time of surgery (HR 1.02, CI 1.01–1.02, p < 0.001), BMI (HR 0.95, CI 0.91–0.99, p = 0.01), hypertension (HR 1.48, CI 1.09–2.0, p = 0.01) and diabetes (HR 1.51, CI 1.06–2.15, p = 0.02), preoperative hemoglobin (HR 0.82, CI 0.77–0.89, p < 0.001) and albumin levels (HR 0.52, CI 0.42–0.63, p < 0.001), and late ostomy closure (HR 8.04, CI 4.99–12.94, p < 0.001). After adjustments for confounders, older age at the time of surgery, lower preoperative serum albumin levels, and late ostomy closure were independently associated with a higher incidence of AKI during the first year of colorectal surgery. Notably, late ostomy closure was the most powerful risk factor for AKI. The risk of AKI in patients with an open ostomy over >3 months was 6.94–fold higher than that in patients who underwent early closure or those without an ostomy (Table 3).

Discussion

This study showed that AKI was prevalent in patients undergoing colorectal surgery during their 1-year postoperative follow-up. AKI occurred most commonly during the first 3 months postoperatively. Although the frequency of AKI gradually decreased after the first 3 months postoperatively, incident AKI was observed throughout the follow-up. Old age, low preoperative serum albumin levels, and the duration of ostomy maintenance were significantly associated with delayed AKI after colorectal surgery.

Patients undergoing colorectal surgery differ from those undergoing other major surgeries in that the risk of development of AKI persists in this group far beyond the perioperative period. These patients show a higher risk of in-hospital perioperative AKI,[5,12,17] and the risk of AKI continues beyond the perioperative period owing to complications related to ostomy maintenance, malnutrition, underlying intestinal disease, and exposure to nephrotoxic agents (chemotherapeutic and radiocontrast agents).[18–21] Our study showed that the incidence of AKI during the first postoperative year was 37.9% in patients with ostomy formation and 5.8% in patients without ostomy formation. These are considerably higher incidences compared to the AKI development rate of the general population who had not gone through surgery (0.2 per year to 0.4% per year).[22,23] In addition, the delayed AKI rate after colorectal surgery in those with or without ostomy formation was even higher than the AKI development rate of high risk patients such as those with diabetes (0.2% per year)[24] or over 80 years of age who did not undergo surgery (2.8% per year).[25] Although approximately 50% of patients developed AKI within the first 3 months of surgery, incident AKI was continuously detected throughout the 1-year postoperative follow-up. Interestingly, > 15% of the AKI cases that occurred during the 1-year postoperative period occurred after 9 months of surgery. Therefore, close monitoring and risk surveillance for the development of AKI are warranted even after patients are discharged and in the absence of perioperative renal complications after colorectal surgery.

Advanced age and low preoperative serum albumin levels were identified as significant risk factors associated with delayed AKI in patients undergoing colorectal surgery. Several previous studies have shown advanced age to be closely related to a higher risk for perioperative AKI.[26,27] Age-related changes in renal structure and functional decline predispose elderly patients to renal insult and AKI.[26] Additionally, elderly patients present with several comorbidities including hypertension and diabetes, which are known to exacerbate renal damage. This factor would also play a role in the close association between age and delayed AKI post colorectal surgery. Previous studies have consistently shown a close association between preoperative serum albumin levels and the increased risk of postoperative morbidity and mortality rates.[28,29] Renoprotective effects of serum albumin can be attributed to its ability to maintain renal perfusion, prevent oxidative damage, inhibit apoptosis, and promote the proliferation of renal tubular cells.[30–32] Therefore, preoperative evaluation and improvement of nutritional status could decrease the risk of delayed AKI after colorectal surgery. However, further clinical investigations are warranted to conclusively establish the role of improved serum albumin levels in preventing delayed AKI.

The creation of a temporary or permanent ostomy is a common procedure accompanying colorectal surgery.[33,34] Maintaining an ostomy offers various advantages and is necessary in certain situations.[35,36] However, an ostomy is often associated with several complications that increase the morbidity risk.[37,38] Although several previous studies have reported ostomy-related local complications, its long-term effects on renal function have not been widely studied.[39,40] The risk of delayed AKI in patients with an ostomy maintained over >3 months was 7-fold higher than that in patients without an ostomy or those who underwent early ostomy closure. Moreover, among patients who developed AKI, 88.7% had a

Table 3
Factors associated with the development of delayed acute kidney injury.

Variable	Univariate analysis	p	Multivariate analysis	p
	HR (95% CI)		HR (95% CI)	
Age (years)	1.02 (1.01–1.02)	<0.001	1.01 (1.00–1.03)	0.02
Male sex	1.12 (0.83–1.52)	0.44	–	–
BMI (kg/m²)	0.95 (0.91–0.99)	0.01	0.99 (0.94–1.04)	0.61
RAS blocker use	1.45 (0.97–2.17)	0.07	–	–
Hypertension	1.48 (1.09–2.00)	0.01	1.31 (0.93–1.84)	0.13
Diabetes	1.51 (1.06–2.15)	0.02	1.35 (0.93–1.96)	0.12
Hemoglobin (mg/dL)	0.82 (0.77–0.89)	<0.001	0.92 (0.85–1.00)	0.08
Albumin (g/dL)	0.52 (0.42–0.63)	<0.001	0.65 (0.51–0.83)	0.01
eGFR (mL/min/1.73 m²)	1.00 (0.99–1.01)	0.49	–	–
Late ostomy closure	8.04 (4.99–12.94)	<0.001	6.94 (4.29–11.23)	<0.001

HR = hazard ratio; CI = confidential interval; BMI = body mass index; RAS = renin-angiotensin system; eGFR = estimated glomerular filtration rate.

functioning ostomy at the time of AKI occurrence. The increased risk of AKI in patients with an open ostomy is attributed to volume depletion. The creation of an ostomy involves varying degrees of colonic resection, thereby reducing the surface area available for fluid absorption. Additionally, uncontrolled fecal discharge following the creation of the ostomy causes dehydration.[41–43] Although excessive ostomy output is common only during the immediate postoperative period and gradually improves over time, prolonged excessive ostomy output has been reported.[37,44] Dehydration was recognized as the most common known cause of delayed AKI in this study. This finding supports the possibility that ostomy-related fluid loss increases the risk of delayed AKI in patients undergoing colorectal surgery.

One of the major functions of colon is to reabsorb water, therefore, resection of colon may aggravate dehydration depending on the extent of resected colon. Therefore, the incidences of delayed AKI according to the surgery type details were also evaluated. When delayed AKI incidences were compared among those with right hemicolectomy, left colectomy, and total colectomy, the delayed AKI occurrence rate was highest in those who underwent left colectomy (Supplemental Table 1). Ostomy formation status may have played a role on this different delayed AKI incidence depending on colectomy type. Most of the patients who underwent left colectomy had ostomy formations (90.7%). Among them, 60.0% had ileostomy formation. On the other hand, most of the right hemicolectomy patients underwent primary resection with anastomosis (83.2%). Therefore, reduced fluid reabsorption capability resulting from lack of colon passage, due to proximal ostomy formations, could have increased the rate of delayed AKI in those with left colectomy procedures. This possibility is supported by the finding that delayed AKI incidence was comparable among those who underwent right hemicolectomy, left colectomy, and total colectomy without ostomy opening (Supplemental Table 2). The delayed AKI incidences were also compared between those with and without ileocecal valve preservation. Delayed AKI development risk was significantly higher in those whose ileocecal valve was conserved (Supplemental Table 1). Ostomy formation being more common in those with conserved ileocecal valve procedures could be a reason for this finding (86.3% vs. 35.4%). The fact that the difference in delayed AKI risk depending on ileocecal valve conservation was lost in those without ostomy opening or those with colostomy formation strengthens this possibility (Supplemental Tables 2 and 3). Interestingly, when the delayed AKI occurrence rate was compared among types of ostomy, delayed AKI was more prevalent in those who had colostomy (45.1%) compared to ileostomy formations (28.4%). The rate of late ostomy closure was higher among those with colostomy compared to those with ileostomy formation (97.1% vs. 80.9%). Although ileostomy formations would result in decreased colon function, the duration of ostomy maintenance seems to have had a stronger effect on the occurrence of delayed AKI. Comparing the delayed AKI rate between colostomy and ileostomy formers who had similar ostomy maintenance duration was not possible due to the limited number of patients. Further investigations would be needed to clarify this relationship.

This study has several Limitations. First, the retrospective observational design serves as a drawback. Etiological factors associated with AKI could only be identified from medical records, which resulted in a high rate of unknown causes. Additionally, the causal relationship could not be conclusively established. Nonetheless, potential risk factors including medication and laboratory data were included in the evaluation and adjustments were made to investigate independent factors affecting the development of delayed AKI. Second, a large number of patients included in the study were diagnosed with cancer (78.9%). Colorectal surgery related to malignancy are known to be more often accompanied

with stoma formation than other causes.[45] Therefore, the stoma formation rate of recent study was somewhat higher than previous reports. Furthermore, cancer patients tend to show a higher risk of AKI secondary to poor nutritional status and chemotherapy. Therefore, the results of this study may not be applicable to patients who undergo colorectal surgery for non-cancerous indications. Third, we did not evaluate perioperative factors including anastomotic leakage, ventilator care, or infection, which could affect the occurrence of delayed AKI. Further studies investigating the role of these factors are warranted.

In conclusion, AKI occurred frequently during 1-year follow-up after colorectal surgery. Advanced age, low preoperative serum albumin levels, and late ostomy closure were independently associated with delayed AKI. Careful risk stratification and improvement of modifiable factors in high-risk patients may prevent the development of delayed AKI in patients undergoing colorectal surgery.

Conflicts of interest

The authors have no conflicts of interests to declare.

Acknowledgements

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.amjsurg.2019.03.027.

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