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Acute kidney injury following exploratory laparotomy and temporary abdominal closure

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Abstract

Background—Acute kidney injury (AKI) following exploratory laparotomy and temporary abdominal closure (TAC) is poorly understood but clinically significant. We hypothesized that the prevalence of AKI would be highest 96 hours following TAC, early hypoxemia would predict AKI, and that AKI would be an independent predictor of mortality.

Methods—We performed a retrospective analysis of 251 acute care surgery patients managed with TAC by negative pressure wound therapy (NPWT). Kidney Disease: Improving Global Outcomes (KDIGO) AKI stages were assessed on admission, initial TAC, and following TAC at 48h, 96h, and 7d. Multivariate regression was performed to identify risk factors for AKI and inpatient mortality.

Results—Fifty-seven percent of all patients developed AKI within seven days of laparotomy (stage 1: 14%, 2: 21%, 3: 22%). The prevalence of AKI peaked 48h following TAC, and stage correlated with inpatient mortality (stage 0: 7%, 1: 13%, 2: 19%, 3: 37%, $p < 0.001$). Overall mortality was 14%. Factors predictive of stage 2 or 3 AKI at 48h included age > 65 years (OR 2.6 [95% CI 1.4–4.9]), NPWT output > 30 ml/h from first TAC to 48h (2.0 [1.1–3.9]), and three parameters at initial laparotomy: mean arterial pressure < 60 mmHg (2.9 [1.0–8.5]), temperature $< 36^{\circ}\text{C}$ (2.1 [1.1–3.8]), and anion gap > 21 mEq/L (1.9 [1.0–3.7]). AKI was an independent predictor of inpatient mortality (5.5 [2.5–11.8]).

Conclusions—AKI is common following TAC, reaches greatest prevalence 48h after initial laparotomy, and is associated with increased mortality. NPWT fluid loss is risk factor for AKI that is unique to TAC patients.

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Keywords

acute kidney injury; exploratory laparotomy; acute care surgery; temporary abdominal closure; open abdomen; mortality

Introduction

Temporary abdominal closure (TAC) became prevalent during the advent of damage control surgery for trauma patients with exsanguinating hemorrhage necessitating early physiologic resuscitation in lieu of definitive surgical management and primary fascial closure (1–6). Accepted indications for TAC have expanded since that time (7). Among patients with intraabdominal sepsis, TAC may facilitate early diagnosis and treatment of residual infection, remove cytokine-rich peritoneal fluid, and defer anastomosis until physiologic optimization (4, 6, 8, 9). TAC is also employed for patients with intra-abdominal hemorrhage unrelated to traumatic injury and patients with abdominal compartment syndrome refractory to non-surgical management (7).

Acute renal failure in elderly patients and the requirement for renal replacement therapy have previously been associated with increased risk for inpatient mortality following TAC (10, 11), and we have previously described the prevalence of AKI among patients with TAC following laparotomy for trauma and abdominal sepsis (12). However, the prevalence, timing, and degree of acute kidney injury (AKI) following TAC have not been fully described, and risk factors for AKI among TAC patients are unknown. The purposes of this study were to characterize stages of AKI over time following TAC using consensus Kidney Disease: Improving Global Outcomes (KDIGO) definitions (13), identify predictors of AKI following TAC, and assess the association between AKI and mortality. We hypothesized that the prevalence of AKI would be greatest 96 hours following TAC, AKI would be predicted by global indicators of ischemia at initial laparotomy and 48 hours, and that AKI would be an independent predictor of inpatient mortality when controlling for age, American Society of Anesthesiologists (ASA) physical status classification (14), and Charlson comorbidity index score (15).

Methods

Study population

We performed a single-institution retrospective analysis of 251 consecutive patients managed with TAC during a four-year period ending June 2016. Institutional Review Board approval was obtained. Patients were identified by searching our institutional database for adult (age ≥ 18 years) patients who had CPT codes for relaparotomy (49002) or exploratory laparotomy (49000) with a planned or unplanned reoperation CPT modifier (48, 78) (Figure 1). Patients meeting these criteria were included if they underwent TAC with a negative pressure wound therapy (NPWT) dressing. Patients who survived for less than 96 hours following initial TAC were excluded because we hypothesized that the prevalence of AKI would be greatest 96 hours following TAC, and postulated that early deaths within 96 hours were likely attributable to refractory shock or other irreversible etiologies, rather than AKI.

Patients with cirrhosis and those who were on dialysis at the time of initial laparotomy were excluded to avoid the confounding effects of these conditions on the development of AKI. Patients with a pre-existing intestinal fistula were excluded because fistula formation was an outcome parameter. Patients with necrotizing pancreatitis were excluded due to significant differences in the pre and post-operative courses associated with this disease process.

Procedures and parameters

During the study period, strict postoperative resuscitation protocols were followed, as previously described (16–18). Specific operative techniques were at the discretion of the attending surgeon. All patients were managed with NPWT TAC with intention for planned relaparotomy and sequential fascial closure attempts at 24–48 hour intervals, favoring relaparotomy within 24 hours if possible (19). NPWT included primarily the use of individualized vacuum pack (i.e. Barker technique (20)), and use of commercial (ABThera system, KCI, San Antonio, TX) vacuum assisted closure dressings. During the study period, the degree of NPWT suction was at the discretion of the surgical team, and was typically maintained at 125 mmHg. Patients receiving open abdomen therapy remained intubated and lightly sedated to avoid disruption of the NPWT dressing and evisceration. If visceral edema and/or critical loss of abdominal domain precluded primary fascial closure during the index hospital admission, a planned ventral hernia was created with biologic mesh or polyglactin mesh with interval split thickness skin grafting. All clinicopathologic variables were derived from our institutional database or recorded by review of the electronic medical record. KDIGO stages of AKI (13) were assessed on admission, at the time of initial exploratory laparotomy with TAC, and at three intervals following TAC: 48 hours, 96 hours, and 7 days (stage 1: serum creatinine (sCr) 1.5–1.9× baseline or 0.3 mg/dl increase in sCr or urine output (UOP) < 0.5 ml/kg/h for 6–12 hours; stage 2: sCr 2.0–2.9× baseline or UOP < 0.5 ml/kg/h for 12 hours; stage 3: sCr 3× baseline or 4.0 mg/dl increase in sCr or initiation of renal replacement therapy or UOP < 3 ml/kg/h ×24 hours or anuria for 12 hours). Assessment of AKI on admission was based on comparisons between creatinine on admission versus creatinine prior to admission; this data was available for 125 subjects (50%). For patients without a documented creatinine level prior to admission, creatinine on admission was used as baseline creatinine. The primary outcomes were AKI and inpatient mortality. For patients surviving to discharge, readmissions within 90 days of discharge were assessed.

Statistical analysis

Statistical analysis was performed in SPSS (version 23, IBM, Armonk, NY). Continuous variables were compared by the Kruskal-Wallis test reported as median [interquartile range]. Discrete variables were compared by Fisher's Exact test and reported as n (%). Frequency distributions for stages of AKI were assessed to identify the time point at which TAC patients were most significantly affected by AKI. Multivariable logistic regression was performed to assess the capacity of AKI at the predetermined time point to predict inpatient mortality while accounting for the effects of age, ASA classification (14), and Charlson comorbidity index score (15). Logistic regression was also performed to identify risk factors for developing AKI at the predetermined time point for the entire study population and for two subgroups: trauma and emergency general surgery (EGS), based on previous work

demonstrating differences in AKI prevalence between these populations. Independent predictors were identified by selecting variables with significant bivariate association (odds ratio with a 95% confidence interval not including 1.0) to the outcome variable and no significant collinearity (Pearson's coefficient with $p < 0.05$) to other predictors. Optimal cutoff values for continuous variables were determined by calculating the point of maximal sensitivity and specificity by Youden's index (21). Selected variables were entered into the regression equation. Model efficacy was assessed by calculating area under the receiver operating characteristic curve (AUROC).

Results

Patient characteristics and outcomes

Two-hundred and fifty-one patients were included (Table 1). The study population was middle-aged (56 years), carried a low burden of chronic disease (Charlson comorbidity index 1), had a life-threatening acute disease (ASA class 4), and had normal baseline renal function (Cr 0.9). Intra-abdominal sepsis was the most common indication for TAC, composing 61% of the study population. One third of the study population was composed of severely injured trauma patients (ISS 33). The median number of abdominal operations per patient was three, with a one day interval between operations.

Primary fascial closure was achieved in 75% of all patients (Table 2). Eleven percent of the study population started on dialysis following TAC; 8% remained on dialysis until discharge. Patients who were already on dialysis at the time of initial laparotomy were excluded to ensure that analysis of AKI was not confounded by preoperative renal replacement therapy. During admission, patients with AKI received significantly more blood products compared to patients without AKI, including red blood cells (7.0 [3.0–19.5] vs. 4.0 [1.0–10.0], $p = 0.004$), plasma (4.0 [0.0–14.3] vs. 2.0 [0.0–6.0], $p = 0.029$), cryoprecipitate (0.0 [0.0–3.0] vs. 0.0 [0.0–0.0], $p = 0.002$), and platelets (0.0 [0.0–5.3] vs. 0.0 [0.0–1.0], $p = 0.001$). On average, patients spent 22 days in the hospital, 14 days in the intensive care unit, and 9 days on mechanical ventilation. Only twenty-eight percent of all patients were discharged to their home. Overall inpatient mortality was 14%. Of the 35 inpatient mortalities, there were five cases non-survivable traumatic brain injury, four cases of massive cerebrovascular accident without hope for meaningful recovery, two cases of unresectable bowel ischemia, one case of multiple organ failure following postoperative hemorrhage, one refractory pulseless electrical activity arrest, one gastrointestinal bleed in a patient who refused blood transfusion, and 21 cases of multiple organ failure and withdraw of care. Thirty-five percent of all patients were readmitted to the hospital within 90 days; 21% were readmitted with a postoperative complication.

Acute kidney injury

Fifty-seven percent of all patients had AKI by KDIGO criteria within seven days of laparotomy. Fourteen percent had stage 1 AKI, 21% had stage 2 AKI, and 22% had stage 3 AKI. The prevalence of AKI peaked 48 hours following TAC (Figure 2). There was a direct correlation between KDIGO score at 48 hours and inpatient mortality (Figure 3). Patients

with no AKI or stage 1 AKI each had lower than average mortality, while patients with stage 2 or 3 AKI had significantly higher mortality rates ($p < 0.001$).

Salient variables were assessed for the capacity to predict stage 2 or 3 AKI 48 hours after TAC (Table 3). Optimal cutoff values were determined by selecting the point with optimal sensitivity and specificity on the receiver operating characteristic curve, and entered into a multivariate logistic regression equation. Factors predictive of stage 2 or 3 AKI 48 hours after TAC included age > 65 years, NPWT output $> 30\text{ml/h}$ from first TAC to 48h, and three parameters measured at the time of initial laparotomy: MAP $< 60\text{ mmHg}$, temperature $< 36^\circ\text{C}$, and an anion gap $> 21\text{ mEq/L}$ (Table 3). This model had AUROC 0.716 (0.645–0.787).

Mortality

Multivariate logistic regression was performed to determine if Stage 2 or 3 AKI 48 hours after TAC was predictive of inpatient mortality while accounting for ASA classification, which also demonstrated a significant bivariate association with mortality (Table 3). Stage 2 or 3 AKI was a stronger predictor of inpatient mortality with OR 5.5 (95% CI 2.5–11.8), followed by ASA class with OR 3.2 (95% CI 1.3–8.0) (Table 4). This model had AUROC 0.709 (0.604–0.814).

Subgroup Analysis

The prevalence of stage 2 or 3 AKI was higher among EGS patients ($n=166$) than trauma patients ($n=85$) on admission (11% vs. 0%, $p = 0.001$), at initial laparotomy (17% vs. 0%, $p < 0.001$), and 48 hours after initial laparotomy (33% vs. 19%, $p = 0.026$), but not at 96 hours (28% vs. 19%, $p = 0.124$) or 7 days (24% vs. 13%, $p = 0.080$) (Figure 4). For trauma patients, independent predictors of stage 2 or 3 AKI at 48 hours included temperature $< 36^\circ\text{C}$ at initial laparotomy (OR 4.9, 95% CI 1.2–19.7) and NPWT output $> 40\text{ ml/hr}$ (OR 4.8, 95% CI 1.4–16.4). Together, these variables composed a model with AUROC 0.779 (95% CI 0.659–0.900). For EGS patients, independent predictors of stage 2 or 3 AKI at 48 hours included Charlson comorbidity index > 3 (OR 2.4, 95% CI 1.1–5.3), vasopressor requirement at the time of initial laparotomy (OR 2.3, 95% CI 1.1–4.6), and temperature $< 36^\circ\text{C}$ at initial laparotomy (OR 2.3, 95% CI 1.1–4.7). Together, these variables composed a model with AUROC 0.697 (0.610–0.784).

Discussion

In a single-center retrospective cohort study of patients undergoing TAC we have demonstrated that AKI is a common complication with peak occurrence at 48h after initial laparotomy. Our results confirm strong independent association between AKI and hospital mortality. Notably, stage I AKI was not associated with increased risk for mortality. This may represent a type II error due to small sample size. However, for the purposes of this study, analysis focused on stage 2 and 3 AKI. Along with advanced age, hypotension, hypothermia, and acidosis, high NPWT output was an independent predictor of stage 2 or 3 AKI at 48h, suggesting that factors unique to TAC may contribute to AKI pathophysiology in this patient population. Replacing NPWT fluid losses with intravenous fluids in a

standardized fashion (e.g. 1:1 ratio) may be appropriate to avoid hypovolemia in patients with high NPWT output while avoiding large resuscitation volumes, which have been associated with adverse outcomes (22). High NPWT output may have represented a surrogate for ongoing intra-abdominal bleeding, though it is difficult to determine the relative proportions of blood and ascites in NPWT output retrospectively. Notably, the NPWT output cutoff with optimal sensitivity and specificity for predicting AKI was higher in the trauma subgroup than the entire study population, and it seems likely that NPWT output among trauma patients would contain more blood and less ascites relative to EGS patients.

Seternes et al. (10) reported that renal failure requiring dialysis was associated with increased risk for inpatient mortality among TAC patients in Norway (OR 3.9, 95% CI 1.4–11.1). In this retrospective study, 23% of all TAC patients required renal replacement therapy, compared to 11% in our study. However, patients who were already on dialysis at the time of initial laparotomy were excluded from our study, which may account for this difference. In addition, patients undergoing laparotomy for abdominal aortic aneurysm composed 38% of the sample analyzed by Seternes and colleagues, whereas our study population was most commonly affected by abdominal sepsis and traumatic injury. Juxtarenal aneurysmal disease may have influenced rates of preoperative and postoperative renal failure in the Norwegian study. Arhinful, Zielinski, et al. (23) retrospectively reviewed 67 TAC patients age 80 years at the Mayo Clinic in Rochester, and found that 36% developed acute renal failure per RIFLE criteria (24), corresponding to KDIGO stage 3 AKI, which occurred in 22% of our population. The higher prevalence of acute renal failure among octogenarians represented in the Arhinful study is also consistent with our finding that advanced age was an independent predictor of developing AKI. When Arhinful and colleagues performed multivariate regression, acute renal failure was the strongest predictor of inpatient mortality (OR 11.8, 95% CI 2.0–69.1), as in our study. Finally, acute renal failure occurred in 18% of 572 patients enrolled in a prospective observational study by the American Association for the Surgery of Trauma Open Abdomen Study Group, representing a broad population of TAC patients managed at some of the most prominent academic institutions in the United States (25). Therefore, our results are consistent with previous work performed in a variety of settings, all demonstrating that kidney injury among TAC patients is common and associated with adverse outcomes.

The major limitations of this study are its single-institution retrospective design, small sample size (n=251), and lack of long-term follow up. Selection bias inherent to retrospective analyses was limited by including all consecutive cases meeting criteria. Despite a small sample of patients, our relatively broad inclusion criteria allow these findings to be generalized to most TAC patients. Although data from a single institution may not represent nationwide practice patterns, this design allowed for standardization of resuscitation practices, a critical element for the study of AKI. Future studies should evaluate the long-term effects of AKI following TAC, and assess the utility of replacing NPWT fluid losses with intravenous fluids in a standardized fashion.

Conclusions

The development of AKI is common following TAC, reaches greatest prevalence 48h after initial laparotomy, and is an independent predictor of increased mortality. Stage I AKI was not associated with increased mortality. Risk factors for stage 2 or 3 AKI at 48h included advanced age, hypotension, hypothermia, and acidosis. High NPWT output was also an independent predictor of AKI, suggesting that TAC patients may have unique AKI pathophysiology, and that standardized replacement of NPWT losses with intravenous fluids should be considered. Future studies should assess the long-term effects of AKI following TAC.

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Figure 1.
Inclusion and exclusion criteria for the study population.

Figure 2.

The prevalence of acute kidney injury (13) peaked 48 hours after exploratory laparotomy and temporary abdominal closure (TAC).

Figure 3.

There was a direct correlation between inpatient mortality and acute kidney injury (AKI) stage (13) 48 hours after exploratory laparotomy and temporary abdominal closure.

Figure 4.

Emergency general surgery (EGS) patients (n = 166) had significantly higher prevalence of stage 2 or 3 AKI than trauma patients (n = 85) on admission and at the time of initial laparotomy and temporary abdominal closure (TAC).

Table 1

Characteristics for patients who developed acute kidney injury[†] and those that did not.

Parameter	All patients n = 251	AKI [†] n = 70	No AKI [†] n = 181	<i>P</i>
Age (years)	56 [43–69]	63 [56–74]	53 [41–66]	<0.001
Male	133 (53%)	37 (53%)	96 (53%)	>0.999
ASA physical status classification	4.0 [3.0–4.0]	4.0 [4.0–4.0]	4.0 [3.0–4.0]	0.133
Charlson comorbidity index	1.0 [0.0–3.0]	2.0 [0.8–4.0]	1.0 [0.0–2.0]	<0.001
Body mass index	28 [23–33]	30 [24–40]	26 [22–32]	0.001
Creatinine (mg/dL) prior to admission	0.9 [0.7–1.0]	0.9 [0.8–1.3]	0.9 [0.7–1.0]	0.276
Reason for laparotomy				
Sepsis	153 (61%)	53 (76%)	100 (55%)	0.002
Trauma	85 (34%)	16 (23%)	69 (38%)	0.026
Injury severity score	33 [24–44]	44 [32–49]	32 [23–40]	0.008
Non-trauma hemorrhage	9 (4%)	8 (4%)	1 (1%)	0.451
Abdominal compartment syndrome	4 (2%)	4 (2%)	0	0.579
At initial laparotomy				
Temperature (°C)	36.9 [36.3–37.3]	36.6 [36.1–37.0]	36.9 [36.5–37.4]	0.001
Mean arterial pressure (mmHg)	81 [70–97]	74 [64–88]	86 [73–116]	<0.001
Base deficit (mEq/L)	5.1 [2.6–8.8]	6.1 [2.9–9.4]	4.9 [2.4–7.9]	0.292
Anion gap (mEq/L)	18.5 [15.8–22.5]	19.4 [16.8–23.5]	18.2 [15.6–21.2]	0.018
Solid organ resection or repair	69 (27%)	9 (13%)	44 (24%)	0.057
Hollow viscous resection or repair	155 (62%)	46 (66%)	100 (55%)	0.154
Bowel resection or repair	141 (56%)	40 (57%)	92 (51%)	0.400
Washout/lysis of adhesions only	38 (15%)	10 (14%)	19 (11%)	0.387

ASA: American Society of Anesthesiologists.

[†]Stage 2 or 3 acute kidney injury 48 hours following exploratory laparotomy and temporary abdominal closure per consensus Kidney Disease: Improving Global Outcomes (KDIGO) definitions (13). Data are presented as median [interquartile range] or n (%).

Table 2

Outcomes for patients who developed acute kidney injury[†] and those that did not.

Parameter	All patients n = 251	AKI [†] n = 70	No AKI [†] n = 181	P
Abdominal operations	3.0 [2.0–4.0]	3.0 [2.0–4.0]	2.0 [2.0–4.0]	0.229
Primary fascial closure	187 (75%)	45 (64%)	142 (79%)	0.024
Days to primary fascial closure	2.0 [1.0–3.0]	2.0 [1.0–4.0]	2.0 [1.0–3.0]	0.095
Complications				
Abdominal compartment syndrome	2 (1%)	1 (1%)	1 (1%)	0.481
Fascial dehiscence	12 (5%)	4 (6%)	8 (4%)	0.743
Intestinal fistula formation	14 (6%)	3 (4%)	11 (6%)	0.763
Dialysis during admission [#]	27 (11%)	22 (31%)	5 (3%)	<0.001
Discharged on dialysis	21 (8%)	17 (24%)	4 (2%)	<0.001
Hospital length of stay (days)	22 [11–34]	23 [12–33]	22 [11–33]	0.751
ICU length of stay (days)	14 [6–23]	18 [7–28]	12 [5–22]	0.020
ICU-free days	6 [2–12]	3 [0–9]	7 [3–13]	<0.001
Days on mechanical ventilation	9 [4–17]	11 [6–18]	8 [3–16]	0.026
Discharge disposition				
Home	71 (28%)	11 (16%)	60 (33%)	0.008
Subacute nursing facility	47 (19%)	9 (13%)	38 (21%)	0.153
Long term care facility	43 (17%)	13 (19%)	30 (17%)	0.711
Inpatient rehabilitation	29 (12%)	4 (6%)	25 (14%)	0.081
Another hospital	15 (6%)	7 (10%)	8 (4%)	0.134
Hospice	11 (4%)	5 (7%)	6 (3%)	0.187
Inpatient mortality	35 (14%)	21 (30%)	14 (8%)	<0.001
Readmission within 90 days	88 (35%)	22 (31%)	66 (37%)	0.555
With postoperative complication	52 (21%)	12 (17%)	40 (22%)	0.488

ICU: intensive care unit.

[†]Stage 2 or 3 acute kidney injury 48 hours following exploratory laparotomy and temporary abdominal closure per consensus Kidney Disease: Improving Global Outcomes (KDIGO) definitions (13).

[#]Patients who were on dialysis at the time of initial laparotomy were excluded from the study. Data are presented as n (%) or median [interquartile range].

Table 3

Bivariate associations between patient characteristics, acute kidney injury[†], and inpatient mortality.

Outcome Factors	OR	95% CI	<i>p</i>
Acute kidney injury[†]			
Age ^a	1.03	1.02–1.05	<0.001
ASA classification	1.25	0.88–1.77	0.220
Charlson comorbidity index ^b	1.32	1.14–1.53	<0.001
Body mass index ^b	1.05	1.02–1.08	0.002
Sepsis ^c	2.53	1.36–4.69	0.003
Trauma ^c	0.48	0.26–0.91	0.024
Injury Severity Score ^c	1.07	1.02–1.13	0.011
At initial laparotomy			
Mean arterial pressure (mmHg) ^a	0.97	0.95–0.98	<0.001
Temperature (°C) ^a	0.66	0.50–0.88	0.004
Anion gap (mEq/L) ^a	1.08	1.03–1.13	0.001
NPWT output (initial laparotomy-48h) ^a	1.01	1.00–1.02	0.004
Mortality			
Acute kidney injury ^{†a}	5.11	2.42–10.80	<0.001
Age	1.00	0.98–1.03	0.758
ASA physical status classification ^a	1.78	1.07–2.96	0.026
Charlson comorbidity index	1.10	0.92–1.31	0.287
Body mass index	0.99	0.95–1.03	0.565
Sepsis	0.83	0.40–1.72	0.618
Trauma	1.18	0.56–2.48	0.659
Injury Severity Score ^c	1.14	1.05–1.23	0.001
At initial laparotomy			
Mean arterial pressure (mmHg)	0.98	0.96–1.00	0.076
Temperature (°C) ^b	0.55	0.39–0.79	0.001
Anion gap (mEq/L) ^b	1.08	1.02–1.14	0.007
NPWT output (initial laparotomy-48h) ^b	1.02	1.01–1.03	<0.001

OR: odds ratio, CI: confidence interval, ASA: American Society of Anesthesiologists, NPWT: negative pressure wound therapy.

[†] Stage 2 or 3 acute kidney injury 48 hours following exploratory laparotomy and temporary abdominal closure per consensus Kidney Disease: Improving Global Outcomes (KDIGO) definitions (13).

^a Included in the multivariate model,

^b excluded from the multivariate model due to collinearity to other variables,

^c excluded from the multivariate model because the variable represents a subpopulation.

Table 4

Multivariate logistic regression models for predicting acute kidney injury[†] and inpatient mortality.

Outcome Factors	OR	95% CI	<i>p</i>
Acute kidney injury[†]			
Mean arterial pressure < 60 mmHg at initial laparotomy	2.94	1.01–8.51	0.048
Age > 65 years	2.58	1.37–4.87	0.003
Temperature < 36°C at initial laparotomy	2.09	1.14–3.81	0.016
NPWT output > 30ml/h from initial laparotomy-48h	2.04	1.06–3.93	0.033
Anion gap > 21 mEq/L at initial laparotomy	1.92	1.01–3.66	0.047
Mortality			
Acute kidney injury [†]	5.48	2.54–11.81	<0.001
ASA physical status classification > 4	3.24	1.31–7.97	0.011

OR: odds ratio, CI: confidence interval, NPWT: negative pressure wound therapy, ASA: American Society of Anesthesiologists.

[†] Stage 2 or 3 acute kidney injury 48 hours following exploratory laparotomy and temporary abdominal closure per consensus Kidney Disease: Improving Global Outcomes (KDIGO) definitions (13). Area under the receiver operating characteristic curve for the acute kidney injury and mortality models were 0.716 (0.645–0.787) and 0.709 (0.604–0.814), respectively.