



# Acute kidney injury after hip fracture surgery in patients over 80 years of age

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## Abstract

**Background** The aim of the study is to determine the rate of acute kidney injury (AKI) after hip fracture surgery over the age of 80 and to investigate the factors associated with AKI.

**Methods** From January 2015 to January 2020, 589 patients who underwent surgery of hip fractures at our institution were retrospectively reviewed. Serum creatinine (sCr) was analysed daily pre- and postoperatively during the hospital stay. Patients were divided into groups; AKI and non-AKI based on KDIGO (Kidney Disease Global Outcomes) criteria. The incidence, risk factors, and mortality of postoperative AKI were investigated.

**Results** Out of 589 patients, 58 developed an AKI (9.8%). Smoking ( $p: 0.004$ ), pre and postoperative low albumin level ( $p < 0.05$ ), pre- and postoperative high potassium level ( $p < 0.05$ ), pre- and postoperative high urea levels ( $p < 0.05$ ), high amount of intra-operative bleeding ( $p: 0.003$ ) and prolonged surgery time ( $p: 0.003$ ) were found to be risk factors associated with AKI. Although the mortality rate was higher in the AKI group, it was not statistically significant ( $p > 0.05$ ).

**Conclusion** AKI is a temporary but common complication following hip fracture surgery, which can also be predicted if risk factors are adequately observed. It typically increases the length of hospital stays, mortality and morbidity.

**Level of evidence** Level III evidence, Retrospective comparative study

**Keywords** Hip fracture · Surgery · Acute kidney injury · Incidence · Mortality

## Introduction

Advances in the medical field have contributed significantly to life expectancy, resulting in an ageing population prone to osteoporotic fractures, especially hip fractures [1]. Hip fractures in the elderly are a significant public health problem because they are associated with severe morbidity, mortality and significant socioeconomic burden [2–7]. Although excessive mortality following a hip fracture is well-defined in the literature [8, 9], its causes remain largely undefined.

Acute kidney injury (AKI), previously referred to as acute renal failure, has been widely studied among the critically ill [10, 11] and trauma patients [12, 13] and is known to reduce survival significantly. AKI is also a common perioperative complication in surgical patients [14, 15]. Several studies have cited dehydration, blood loss, nephrotoxic drugs [non-steroidal anti-inflammatory drugs (NSAIDs) and antibiotics], and patient comorbidities (such as hypertension, diabetes, and cerebrovascular disease) as significant risk factors for AKI [4, 16]. AKI in surgery can occur under

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two conditions: (1) hypovolemia due to acute tubular necrosis, pulmonary embolism, heart failure, acute myocardial infarction, sepsis and anaesthesia, while it may present as a prerenal AKI; and (2) renal artery occlusion (thrombus or embolism), drugs and pyelonephritis, while it may present as a renal AKI.

However, postoperative AKI is often unclear in aetiology and is multifactorial. While AKI has been well-studied in elective orthopaedic patients [17–20], fewer studies are available on low-energy hip fractures [21–23]. It is unclear whether current treatment strategies are adequate and effective for AKI, and current concepts emphasise prevention through close monitoring of risk factors for AKI [24, 25]. However, risk factors for AKI after hip fracture surgery remain unclear. Male gender, increased age, diabetes mellitus, pre-existing chronic kidney disease, acute or chronic heart failure, and surgery are risk factors common in patients over 80 years of age; may not be sensitive or specific to these populations [26, 27].

This study's primary aim was to determine the incidence of postoperative AKI and risk factors in patients over 80 years of age who were operated on for hip fracture. The study's secondary aim was to compare the postoperative mortality of patients with AKI and patients without AKI.

## Patients and methods

A retrospective examination was made of patients hospitalised and treated surgically for proximal femur fractures at the University of Health Sciences, Kanuni Sultan Süleyman Training and Research Hospital Department of Orthopaedics and Traumatology Department, Istanbul, Turkey, between January 1, 2015, and January 1, 2020. All procedures were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as amended in 2008. Informed consent was waived due to its retrospective nature. The records of 614 patients over 80 years of age who underwent surgery for femoral neck fracture and intertrochanteric femur fracture were reviewed. Standard diagnosis and treatment were applied to all patients admitted for emergency services with hip fractures and operated on according to the local treatment protocol for various hip fractures under spinal anaesthesia or general anaesthesia.

All surgical procedures were performed in the same institution under a senior surgeon's supervision (C.E.). As a surgical procedure, cementless bipolar hip arthroplasty, proximal femoral nail, dynamic hip screw, external fixator and proximal femur plate were applied. Criteria for exclusion included a history of renal replacement therapy (RRT), AKI at admission and missing pre or postoperative laboratory data. As a result, 25 patients were excluded (three patients had kidney

transplants, 10 patients had AKI at the time of admission, and 12 patients did not have access to their data). Overall, 589 patients operated on for femoral neck fracture or intertrochanteric fracture of the hip joint were included in the final study cohort.

## Clinical and laboratory data

Demographic and medical history data of the patients were collected using the Hospital Information System Software, Panates, Istanbul, Turkey. Standard demographic, clinical and physiological data collected included age, sex, time from hospitalisation to surgery, length of hospital stay, primary diagnosis, ASA (American Society of Anesthesiologists) score, smoking status.

All patient medical comorbidities were recorded. These included hypertension, diabetes mellitus, chronic obstructive pulmonary disease, coronary artery disease, chronic renal failure, congestive heart failure, cerebrovascular disease, dementia and malignancy in the body. Reference to an existing disease in the records was considered an adequate definition and used to calculate the Charlson Comorbidity Index.

sCr (serum creatinine) was recorded for up to 7 days after surgery or while the patient was in the clinic. To determine risk factors for the onset of AKI, the following were evaluated:

- Type of femoral fracture (collum or intertrochanteric).
- Type of anaesthesia (general or spinal).
- Surgical treatment method.
- Length of surgery.
- Intra-operative blood loss (the amount of bleeding obtained by collecting the amount of blood collected in the aspirator during the operation and the amount of bleeding gauze).
- Amount of erythrocyte suspension and fresh frozen plasma applied during hospitalisation.
- Pre-operative and postoperative serum sodium, potassium, albumin, urea, BUN (blood urea nitrogen), haemoglobin, haematocrit, GFR (glomerular filtration rate), and creatinine levels.

Postoperative laboratory evaluations were conducted between 1 and 7 days after surgery. The patients were followed for an average of 23.62 (1–81) months; their mortality in the first 3 months, the first year and total time were compared.

## Definition of AKI

The primary goal of the study was to determine the incidence of and factors affecting postoperative AKI. We defined AKI as the change in SCr levels on postoperative days 1

to 7 compared to preoperative levels when admitted to the hospital. According to the criteria in the Improvement of Kidney Disease Global Outcomes (KDIGO), patients were diagnosed with AKI if any of the following conditions were present:  $\geq 0.3$  mg/dL, (26.5  $\mu\text{mol/L}$ ) increase in SCr within 48 h, or an increase in the SCr level of  $\geq 1.5$  times compared to the baseline value within postoperative 7 days.

## Statistics

When evaluating the study's findings, the IBM SPSS Statistics 22 for statistical analysis (SPSS IBM, Turkey) was used. While analysing the study data, the normal distribution parameters' suitability was evaluated with the Shapiro–Wilks test. In addition to the descriptive statistical methods (mean, standard deviation, frequency), the Student's *t*-test was used to compare normally distributed parameters between the two groups. The Mann–Whitney *U* test was used to compare abnormally distributed parameters between the two groups. The Chi-squared test was used to compare qualitative data. Fisher's exact test, the Fisher Freeman Halton test and Continuity (Yates) Correction were also used. A logistic regression analysis was used for multivariate analysis. The Kaplan–Meier analysis and Log Rank test were used for survival analysis. Finally, significance was evaluated at the  $p < 0.05$  level.

## Results

Of the 589 patients included in the study, 58 (9.8%) developed AKI. Demographic and clinical characteristics, including laboratory parameters, are presented in Tables 1, 2 and 3.

The incidence of smoking in those patients with AKI (17.2%) was found to be statistically significantly higher than those without AKI (5.8%) ( $p: 0.004$ ;  $p < 0.05$ ) (Table 1).

Pre- and postoperative albumin levels in patients with AKI were statistically significantly lower than patients without AKI ( $p < 0.05$ ). Pre- and postoperative potassium levels were significantly higher in patients with AKI than those without AKI ( $p < 0.05$ ). In patients with AKI, pre- and postoperative urea values were substantially higher than those without AKI. In addition, the increase in pre- and postoperative urea and BUN levels in the group with AKI was statistically significant ( $p < 0.05$ ) (Table 3).

The pre-operative and postoperative changes in albumin ( $p: 0.000$ ;  $p < 0.05$ ), potassium ( $p: 0.014$ ;  $p < 0.05$ ), urea ( $p: 0.000$ ;  $p < 0.05$ ), BUN ( $p: 0.000$ ;  $p < 0.05$ ), creatinine ( $p: 0.000$ ;  $p < 0.05$ ) and GFR ( $p: 0.000$ ;  $p < 0.05$ ) values of the

patients with AKI were found to be statistically significantly higher than the patients without AKI (Table 3).

In patients with AKI, the amount of intraoperative bleeding ( $p: 0.003$ ;  $p < 0.05$ ), length of surgery ( $p: 0.003$ ;  $p < 0.05$ ) and length of hospitalisation ( $p: 0.000$ ;  $p < 0.05$ ) were found to be statistically significantly higher than patients without AKI (Table 2).

Using logistic regression analysis, we evaluated the effects of smoking, the amount of bleeding, duration of surgery, length of stay, pre–postoperative albumin, pre–postoperative potassium, pre–postoperative urea, postoperative creatinine and postoperative GFR parameters on AKI status. The model results were found to be significant ( $p: 0.000$ ;  $p < 0.05$ ); the Nagelkerke R square value was 0.731, and the explanatory coefficient of the model (95.2%) was found to be high. While the effect of surgery time was not significant in the model ( $p > 0.05$ ), the effects of postoperative albumin ( $p_1: 0.000$ ), postoperative potassium ( $p_2: 0.004$ ), pre-operative urea ( $p_3: 0.000$ ), postoperative urea ( $p_4: 0.000$ ) and postoperative GFR ( $p_5: 0.000$ ) were found to be statistically significant ( $p < 0.05$ ). The effects of surgery time (1.025-fold), postoperative albumin (0.019-fold), postoperative potassium (3.141-fold), pre-operative urea (0.910-fold), postoperative urea (1.055-fold), and postoperative GFR (0.891-fold) were found to have increasing effects on AKI. In the model, the effects of smoking, the amount of bleeding, hospitalisation time, pre-operative albumin, and pre-operative potassium on the incidence of AKI were not statistically significant ( $p > 0.05$ ) (Table 4).

In patients with AKI, 16 (27.6%) of the 58 cases died in the first 3 months (Table 2). The cumulative survival rate for the first-3 months was 72.4%, with a standard error of 5.9%. Of the 531 patients without AKI, 100 (18.8%) died in the first-3 months. For the first-3 months, the cumulative survival rate was 81.2%, with a standard error of 1.7%. When the groups' survival rates in the first-3 months were evaluated using the Log Rank test, no statistically significant difference was found ( $p: 0.098$ ;  $p > 0.05$ ) (Fig. 1).

When the patients' long-term survival was evaluated, 37 (63.8%) of 58 patients with AKI died (Table 2). The last death occurred in the 64th month; the cumulative survival rate in that period was 32%, with a standard error of 6.6%. The mean life expectancy was  $34.67 \pm 4.39$ , and the median was 22 months. Of the 531 patients without AKI, 285 (53.7%) died. The last death occurred in the 64th month, and the cumulative survival rate for the period was 33%, with a standard error of 3.9%. The mean life expectancy was  $39.5 \pm 1.66$ , and the median was 31 months. When the groups' survival rates were evaluated using the Log Rank test, no statistically significant difference was found ( $p: 0.282$ ;  $p > 0.05$ ) (Fig. 2).

**Table 1** Distribution of the general characteristics of the patients according to groups

	AKI (min–max)–(mean ± SD)	Non-AKI (min–max)–(mean ± SD)	<i>p</i>
Age	(80–97)–(85.74±4.19)	(80–99)–(85.8 ± 4.14)	<sup>1</sup> <b>0.918</b>
Charlson comorbidity index	(3–10)–(4.6±1.4 (4))	(3–10)–(4.63 ± 1.29 (4))	<sup>2</sup> <b>0.787</b>
	<i>n</i> (%)	<i>n</i> (%)	
Gender			
Male	15 (%25.9)	154 (%29)	<sup>3</sup> <b>0.727</b>
Female	43 (%74.1)	377 (%71)	
Comorbidities			
HT	32 (%74.4)	283 (%75.1)	<sup>3</sup> <b>1.000</b>
DM	12 (%27.9)	111 (%29.4)	<sup>3</sup> <b>0.974</b>
COPD	12 (%27.9)	72 (%19.1)	<sup>3</sup> <b>0.243</b>
CAD	3 (%7)	36 (%9.5)	<sup>4</sup> <b>0.415</b>
CRF	3 (%7)	20 (%5.3)	<sup>4</sup> <b>0.425</b>
CHF	9 (%20.9)	46 (%12.2)	<sup>3</sup> <b>0.171</b>
CVD	1 (%2.3)	19 (%5)	–
Dementia	7 (%16.3)	66 (%17.5)	<sup>3</sup> <b>1.000</b>
Malignancy	1 (%2.3)	8 (%2.1)	–
Presence of comorbidity			
No	15 (%25.9)	154 (%29)	<sup>3</sup> <b>0.727</b>
Yes	43 (%74.1)	377 (%71)	
ASA			
2	18 (%31)	186 (%35)	<sup>5</sup> <b>0.522</b>
3	34 (%58.6)	310 (%58.4)	
4	6 (%10.3)	35 (%6.6)	
Smoking			
No	48 (%82.8)	500 (%94.2)	<sup>4</sup> <b>0.004*</b>
Yes	10 (%17.2)	31 (%5.8)	

HT hypertension, DM diabetes mellitus, COPD chronic obstructive pulmonary disease, CAD coronary artery disease, CRF chronic renal failure, CHF congestive heart failure, CVD cerebrovascular disease

<sup>1</sup>Student's *t* test, <sup>2</sup>Mann–Whitney *U* test, <sup>3</sup>Continuity (Yates) correction, <sup>4</sup>Fisher's exact test, <sup>5</sup>Chi-squared test, \**p* < 0.05

## Discussion

In this study, the rate of AKI was 9.8%. In one of the most extensive studies in the literature on AKI incidence following hip fracture surgery, an incidence of 12.7% was found in 13,529 patients in Denmark [28]. The literature concludes that the AKI rate in high-energy traumas varies between 15 and 40% [12–14]; in hip fractures, this rate is between 15 and 24% [4, 21]. We believe the lower rate of AKI in our study compared to the literature is due to fluid supplementation starting in the emergency department, especially in elderly patients, and the severe avoidance of nephrotoxic drugs. In addition, this study observed that the waiting times from trauma to surgery were longer than those in the literature [23, 29]. In our institution, surgery is performed after optimal conditions are achieved for advanced elderly patients; this may contribute to a lower rate of AKI.

Reported risk factors for AKI in hip fracture patients are advanced age, male gender, peripheral vascular diseases, vascular occlusive disease, hypertension, diabetes, chronic obstructive pulmonary disease, chronic kidney disease, congestive heart disease, emergency surgery, and pre-illness use of nephrotoxic drugs [4, 21]. However, the evidence on this subject is not sufficient. In this study, we observed that gender, comorbid diseases, ASA score, and length of emergency surgery were not statistically related to the onset of AKI. However, the rate of smoking was found to be statistically higher in patients with AKI. Still, when evaluated using regression analysis, the effect of tobacco on AKI was not found to be statistically significant. Ritz et al concluded in their study that smoking increased renovascular resistance by 11% and, consequently, there was a 15% reduction in GFR [30].

Ulucay et al found that the amount of intraoperative bleeding is a significant risk factor for the onset of AKI

**Table 2** Distribution of working parameters according to groups

	AKI (min–max)–(mean $\pm$ SD (median))	Non-AKI (min–max)–(mean $\pm$ SD (median))	<i>p</i>
Intraoperative blood loss (ml)	(50–650)–(390.52 $\pm$ 206.15 (450))	(50–650)–(328.53 $\pm$ 180.67 (400))	<sup>1</sup> <b>0.003*</b>
Length of surgery (min)	(30–110)–(68.09 $\pm$ 17.9 (70))	(25–120)–(61.1 $\pm$ 17.31 (60))	<sup>1</sup> <b>0.003*</b>
Length of hospital stay (day)	(6–36)–(11.12 $\pm$ 4.74 (10))	(3–23)–(8.99 $\pm$ 3.12 (9))	<sup>1</sup> <b>0.000*</b>
Time from hospitalisation to surgery (day)	(1–31)–(5.64 $\pm$ 4.17 (5))	(1–18)–(5.1 $\pm$ 2.72 (5))	<sup>1</sup> <b>0.372</b>
Erythrocyte suspension transfusion	(0–9)–(1.76 $\pm$ 2 (1))	(0–11)–(1.54 $\pm$ 1.73 (1))	<sup>1</sup> <b>0.572</b>
Fresh frozen plasma transfusion	(0–5)–(0.43 $\pm$ 0.99 (0))	(0–13)–(0.45 $\pm$ 1.04 (0))	<sup>1</sup> <b>0.646</b>
Mortality time (month)	(1–42)–(12.59 $\pm$ 12.35 (9))	(1–64)–(12.04 $\pm$ 13.58 (6))	<sup>1</sup> <b>0.997</b>
Follow-up time (month)	(1–79)–(24.29 $\pm$ 21.14(20.5))	(1–81)–(23.54 $\pm$ 19.37 (20))	<sup>1</sup> <b>0.917</b>
	<i>n</i> (%)	<i>n</i> (%)	
Fracture type			
Femoral neck	19 (%32.8)	133 (%25)	<sup>2</sup> <b>0.264</b>
Intertrochanteric	39 (%67.2)	398 (%75)	
Operation type			
Dynamic hip screw	3 (%5.2)	19 (%3.6)	<sup>3</sup> <b>0.094</b>
External fixator	3 (%5.2)	6 (%1.1)	
Proximal femoral nail	17 (%29.3)	211 (%39.7)	
Proximal femur plate	0 (%0)	5 (%0.9)	
Hemiarthroplasty	35 (%60.3)	290 (%54.6)	
Mortality time ( <i>n</i> = 322)			
First-3 months	16 (%43.2)	100 (%35.1)	<sup>4</sup> <b>0.158</b>
4–12 months	6 (%16.2)	90 (%31.6)	
13 months and above	15 (%40.5)	95 (%33.3)	
90-day mortality			
Ex	16 (%27.6)	100 (%18.8)	<sup>2</sup> <b>0.156</b>
Not-ex	42 (%72.4)	431 (%81.2)	
1-year mortality			
Ex	22 (%37.9)	190 (%35.8)	<sup>4</sup> <b>0.746</b>
Not-ex	36 (%62.1)	341 (%64.2)	
Total mortality			
Ex	37 (%63.8)	285 (%53.7)	<sup>2</sup> <b>0.183</b>
Not-ex	21 (%36.2)	246 (%46.3)	
Anaesthesia type			
General	16 (%27.6)	150 (%28.2)	<sup>2</sup> <b>1.000</b>
Spinal	42 (%72.4)	381 (%71.8)	

<sup>1</sup>Mann–Whitney *U* test, <sup>2</sup>Continuity (Yates) correction, <sup>3</sup>Fisher Freeman Halton test, <sup>4</sup>Chi-squared test, \**p* < 0.05

[21]. In this study, we observed that the amount of bleeding and length of surgery, which are among the intraoperative factors, were significantly higher in patients with AKI. However, when this finding was evaluated using regression analysis, the effect of bleeding on the incidence of AKI was not statistically significant. Because the two groups in this study were similar in the amount of perioperative transfusion, we concluded that postoperative AKI might occur in patients with massive bleeding if appropriate blood transfusion is not performed.

The literature has shown that postoperative hypoalbuminemia is an independent risk factor for the onset of

postoperative AKI [23, 29]. Available data indicate that albumin maintains colloid osmotic pressure, increases adequate circulating volume, promotes increased renal blood flow, and preserves renal function [31]. Albumin also maintains renal perfusion, glomerular filtration, and medullary fluid reabsorption [31]. In addition, Ulucay et al found that postoperative potassium levels in patients with AKI were higher than in patients without AKI [21]. In this study, when patients with AKI were evaluated using regression analysis, the effects of postoperative albumin, postoperative potassium, pre-operative urea, postoperative

**Table 3** Assessment of pre and postoperative laboratory parameters between and within groups

	AKI (min–max)–(mean ± SD)	Non-AKI (min–max)–(mean ± SD)	<i>p</i> <sup>1</sup>
Haemoglobin (g/dl)			
Pre-operative	(6.5–15.16)–(11.27 ± 1.8)	(4.9–18)–(11.25 ± 2.01)	<sup>1a</sup> <b>0.952</b>
Postoperative	(6.8–12.7)–(9.38 ± 1.22)	(4.1–14.7)–(9.57 ± 1.57)	<sup>1a</sup> <b>0.366</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.000*	0.000*	
Albumin (g/dl)			
Pre-operative	(2.2–4.4)–(3.55 ± 0.46)	(2–5.8)–(3.73 ± 0.44)	<sup>1a</sup> <b>0.005*</b>
Postoperative	(1.7–4)–(2.64 ± 0.35)	(1.7–5.2)–(3.11 ± 0.42)	<sup>1a</sup> <b>0.000*</b>
Preop–postop <i>p</i> <sup>2a</sup>	0.000*	0.000*	
Sodium (mmol/l)			
Pre-operative	(131–146)–(139.21 ± 3.28)	(115–155)–(139.24 ± 3.95)	<sup>1a</sup> <b>0.949</b>
Postoperative	(128–151)–(140.28 ± 4.76)	(122–158)–(139.53 ± 3.9)	<sup>1a</sup> <b>0.177</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.067	0.062	
Potassium (mmol/l)			
Pre-operative	(2.7–6.6)–(4.66 ± 0.76)	(2.5–7.5)–(4.38 ± 0.6)	<sup>1a</sup> <b>0.009*</b>
Postoperative	(3.4–7)–(5.01 ± 0.85)	(2.4–6.4)–(4.42 ± 0.6)	<sup>1a</sup> <b>0.000*</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.005*	0.158	
Urea (mg/dl) <sub>(median)</sub>			
Pre-operative	(28–218)–(61.86 ± 29.67 (56))	(15–218)–(55.41 ± 25.91 (50))	<sup>1b</sup> <b>0.039*</b>
Postoperative	(40–223)–(96.81 ± 43.47 (88))	(15–205)–(55.73 ± 23.51 (51))	<sup>1b</sup> <b>0.000*</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.000*	0.280	
BUN (mg/dl) <sub>(median)</sub>			
Pre-operative	(13.08–101.87)–(28.53 ± 13.67 (25.9))	(7.01–101.87)–(25.91 ± 12.08 (23.4))	<sup>1b</sup> <b>0.068</b>
Postoperative	(16.45–104.21)–(44.83 ± 20.56 (40))	(7.01–95.79)–(26.04 ± 10.97 (23.4))	<sup>1b</sup> <b>0.000*</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.000*	0.302	
Haematocrit (%)			
Pre-operative	(21.1–44.71)–(34.65 ± 4.82)	(15.5–51.84)–(34.26 ± 5.84)	<sup>1a</sup> <b>0.630</b>
Postoperative	(18–40.39)–(28.98 ± 5.26)	(12.96–44.28)–(29.99 ± 5.04)	<sup>1a</sup> <b>0.150</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.000*	0.000*	
Creatinine (mg/dl) <sub>(median)</sub>			
Pre-operative	(0.48–6.85)–(1.27 ± 1.13 (1))	(0.35–5.87)–(1.02 ± 0.51 (0.9))	<sup>1b</sup> <b>0.261</b>
Postoperative	(0.79–7.19)–(1.86 ± 1.22 (1.5))	(0.32–4.06)–(0.91 ± 0.41 (0.8))	<sup>1b</sup> <b>0.000*</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.000*	0.000*	
GFR (ml/min)			
Pre-operative	(6.84–95)–(54.46 ± 20.39)	(6.15–102.22)–(59.22 ± 19.66)	<sup>1a</sup> <b>0.082</b>
Postoperative	(3.5–78)–(33.5 ± 14.47)	(9.6–112.58)–(64.81 ± 19.24)	<sup>1a</sup> <b>0.000*</b>
Pre-op–postop <i>p</i> <sup>2a</sup>	0.000*	0.000*	

<sup>1a</sup>Student's *t* test, <sup>1b</sup>Mann–Whitney *U* test, <sup>2a</sup>Paired samples *t* test, <sup>2b</sup>Wilcoxon sign test, \**p* < 0.05

urea and postoperative GFR parameters were found to be statistically significant.

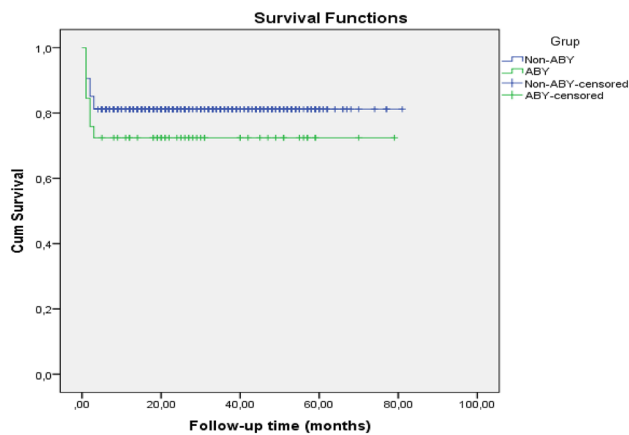
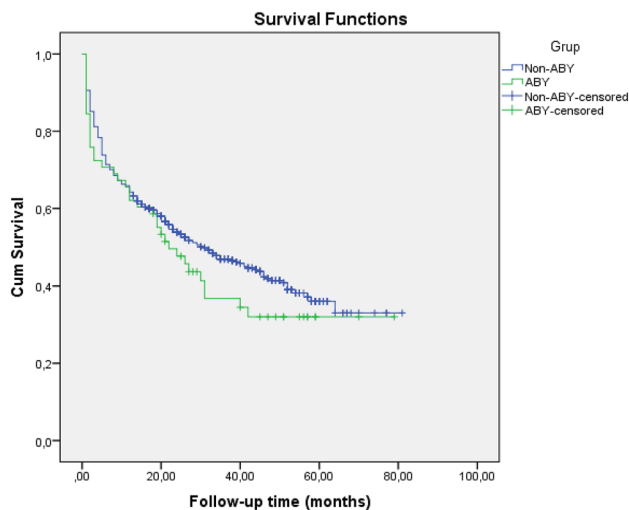
Many studies in the literature evaluated early mortality after AKI. Pedersen et al. found that 1717 (12.7%) of 13,529 patients had AKI within 5 days of surgery; the mortality rate in those patients increased 2.8 times in the first-3 months, and the mortality rate beyond 3 months rose by a factor of 1.3 [28]. Rantalaiho et al reported a 3-month mortality rate of 35% in patients with AKI and 12.7% in those without AKI [22]. In this study, although the mortality rate in the

first-3 months and long-term was found to be higher in patients with AKI than those without AKI, the difference was not statistically significant.

There are several limitations to this study. The study was a retrospective observational study conducted at a single institution. AKI was defined according to increments in the sCr level only, not in urine output, which correlates with renal function [4, 32]. Due to insufficient analyses, patients with AKI could not be further divided into prerenal, renal, and postrenal groups. Although the drugs used

**Table 4** Evaluation of factors affecting AKI status by logistic regression analysis

	OR	95% CI	<i>p</i>
Length of surgery	1.025	1.000–1.052	0.052
Postoperative albumin	0.019	0.005–0.073	0.000*
Postoperative potassium	3.141	1.453–6.788	0.004*
Pre-operative urea	0.910	0.881–0.940	0.000*
Postoperative urea	1.055	1.030–1.080	0.000*
Postoperative GFR	0.891	0.857–0.927	0.000*
Constant	9738.224		0.002*

\**p* < 0.05**Fig. 1** 3-month survival chart by groups**Fig. 2** Overall survival chart by groups

after hospitalisation were prescribed according to their renal functions, and if nephrotoxic drugs, especially NSAIDs, are avoided starting from hospitalisation in this age group, the

nephrotoxic effects of the drugs used daily and the drugs prescribed for anaesthesia could not be examined due to the lack of homogeneity. In fact, polypharmacy involving nephrotoxic drugs such as ACE inhibitors, aminoglycoside and NSAIDs were frequently observed in the patient population enrolled in the study. However, due to the multiplicity of drug combinations, it was impossible to determine the effects of these drugs alone. In addition, the cost of AKI to the healthcare system was not evaluated in this study. However, it is not difficult to predict that costs will increase because AKI prolongs the length of stay and creates additional treatment burdens. It is also known that sCr and GFR levels depend on muscle mass. Decrease in muscle mass, especially in elderly patients, has an undeniable effect on kidney functions. Another limitation of the study is that muscle mass in patients is not taken into account. In addition, since most of the patients were referred to our center from an external center, detailed information about the pre-operative functional capacities of the patients could not be provided.

## Conclusion

AKI is a temporary but common complication following hip fracture surgery. It typically increases the length of hospital stays, mortality and morbidity. In this study, we determined that basal kidney functions, postoperative albumin and potassium levels are independent factors contributing to the onset of AKI. In addition, smoking, lengthy surgery time and excessive intra-operative bleeding may lead to AKI. Surgeons should be alert to the development of AKI and minimise preventable risk factors. Nephrological consultation should be requested for patients in high-risk groups.

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## Declarations

**Conflict of interest** The authors declare that they have no conflict of interest.

**Ethical approval** This article does not contain any studies with human participants or animals performed by any of the authors.

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