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ORIGINAL ARTICLE

Risk of percutaneous renal biopsy of native kidneys in the evaluation of acute kidney injury

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ABSTRACT

Background. Percutaneous renal biopsy (PRB) of native kidneys (NKs) to better understand and treat acute kidney injury (AKI) is being advocated, but little is known about the risk of complications.

Methods. We performed a retrospective study of PRB of NKs in 955 adults from 1991 to 2015 at an academic medical center with real-time ultrasound and automated biopsy needles. Patients undergoing PRB for evaluation of AKI ($n = 160$) were compared with 795 patients biopsied for other reasons (not-AKI) for postbiopsy complications [need for transfusion of packed red blood cells (PRBCs), an interventional radiologic or surgical procedure, readmission or death].

Results. Patients biopsied for AKI were older (58 ± 16 versus 44 ± 16 years; $P < 0.0001$), with a higher serum creatinine (SCr) (4.5 ± 2.7 versus 1.8 ± 1.6 mg/dL; $P < 0.0001$) and lower hemoglobin (Hgb) (10.4 ± 1.7 versus 12.1 ± 2.1; $P < 0.0001$) and a greater proportion had an abnormal bleeding time (12.5% versus 7.4%, P 0.04), partial thromboplastin time (15.2% versus 5.3%, $P < 0.0001$) and/or prothrombin time (27.0% versus 12.8%; $P < 0.0001$) compared with not-AKI patients. Complications post-PRB were significantly greater in patients biopsied for AKI {11.3% versus 6.7%; P=0.04; odds ratio [OR] 1.78 [95% confidence interval (CI) 1.01–3.12]} with patients biopsied for AKI requiring more blood transfusions (10.0% versus 5.3%; P 0.02; OR 2.04 (95% CI 1.12–3.74)]. By multivariate analysis, baseline features predictive of a complication were increased SCr and decreased Hgb level, as well as female gender and increased systolic blood pressure.

Conclusion. Patients biopsied for evaluation of AKI are at greater risk of complications due to increased risk factors.

Keywords: acute kidney injury, acute renal failure, complications, native kidney, percutaneous renal biopsy

INTRODUCTION

Acute kidney injury (AKI) was a primary indication for performing percutaneous renal biopsy (PRB) of native kidneys (NKs) when this procedure first became widely available in the 1950s [1–5]. At that time, the information obtained from the renal biopsy was of diagnostic, therapeutic and prognostic value and the risk of a biopsy-related complications was low. As a result, essentially all patients with AKI underwent PRB.

Over the course of the last 60 years, it has been recognized that the majority of cases of AKI can be diagnosed clinically, often as a result of conditions resulting from hypovolemia or ischemic or nephrotoxic AKI [6–10], with treatment limited to hydration and/or dialysis. As a result, in clinical practice, PRB of NKs has been reserved for patients in whom the etiology of AKI is of 'unknown cause' [11].

Given the increased morbidity, mortality, medical costs and lack of effective treatments in patients with AKI [6–10, 12, 13],

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there has been renewed interest in performing renal biopsies in patients with AKI [14–16]. The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) launched the Kidney Precision Medicine Project [17] in the summer of 2017 with the goal of obtaining 'research' renal biopsies from AKI patients in order to identify pathways of AKI through the use of newer technologies (i.e. proteomics and genomics) that will enable the development of targeted therapies aimed at preventing and treating AKI in the future [14–16, 18, 19].

While PRB of NKs is safe with minimal risk, serious complications can occur [14–16, 20–24] and little is known regarding the complication risk in patients with AKI, many of whom are often quite ill with little likelihood of benefit from the procedure [20, 24–26]. We evaluate our single-center experience in adult patients undergoing a PRB of NKs for the evaluation of AKI in order to better understand the risk in this patient population.

MATERIALS AND METHODS

PRB of NKs was performed in 955 adult (≥15 years) patients consecutively from 1991 to 2015 at Rush University Medical Center. We retrospectively compared 160 patients undergoing a PRB for evaluation of AKI (AKI group) with 795 patients undergoing renal biopsy for evaluation for other reasons (not-AKI group), including proteinuria, hematuria, chronic kidney disease or systemic disease with renal involvement (i.e. systemic lupus erythematosus and vasculitis). This study was approved by the institutional review board at Rush University Medical Center.

Data collection and biopsy procedure

Data was prospectively collected at the time of biopsy and up to 1 week postbiopsy. Information collected at the time of biopsy included the nephrologist's primary reason(s) for the biopsy (i.e. AKI, proteinuria or chronic kidney disease), age, gender, race, systolic and diastolic blood pressures (BPs), serum creatinine (SCr), bleeding time (BT), activated partial thromboplastin time (PTT), prothrombin time (PT) and hemoglobin (Hgb) concentration. In general, it is our practice to perform percutaneous biopsies in 'stable' patients with a normal BT (≤9 min), normal PTT (≤33 s), normal PT (≤13.2 s or ≥65% of control) and stable BP. In patients with a prolonged BT, the use of desmopressin acetate (DDAVP) was at the discretion of the attending nephrologist.

An attending nephrologist or a fellow under the supervision of an attending nephrologist performed all biopsies. Imaging was performed by an experienced attending radiologist using real-time ultrasound as previously described [22, 27]. The lower pole of the kidney was identified for biopsy. A 14- or 16-gauge automated biopsy needle (Bard Biopty Gun, C. R. Bard, Covington, GA, USA) was used.

Following the procedure, patients lay in bed flat on their back for 4–6 h and then remained in bed for 23 h of observation. Patients were monitored postbiopsy for signs or symptoms of complications, such as gross hematuria, flank pain and hypotension. Vital signs were checked every 15 min for 2 h, every hour for 4 h, every 2 h for 6 h and then every 4 h thereafter. Each urine void was checked for hematuria. Hgb levels were checked at ∼5–8 h, 10–13 h, and 18–20 h after the procedure and the lowest Hgb level postbiopsy was recorded. The need for additional studies [i.e. renal ultrasound and computed tomography (CT)] or treatment (i.e. transfusion and interventional radiology procedure/embolization) was determined by each nephrologist. All patients were reevaluated ∼1 week after biopsy, after which a biopsy data form was completed and submitted.

Definition of a biopsy-related complication

A biopsy-related complication was defined by the need for an intervention such as a transfusion of packed red blood cells (PRBCs) or an interventional radiologic or surgical procedure and those resulting in readmission or death.

STATISTICAL ANALYSIS

Statistical analysis was performed using the Mann–Whitney test for continuous data or the Fisher's exact test for categorical data using GraphPad InStat version 3.06 for Windows (GraphPad Software, La Jolla, CA, USA; www.graphpad.com). Multivariate analysis using logistic regression was performed using SAS software version 9.3 (SAS Institute, Cary, NC, USA) to determine which feature at baseline was predictive of a complication after renal biopsy. The baseline variables examined included race, gender, age, BT, systolic BP, diastolic BP, SCr, Hgb concentration, proteinuria, who performed the biopsy (attending or fellow) and reason for the biopsy (AKI versus not-AKI). Variables with a P-value < 0.05 were retained in the final model and reported as odds ratios (ORs) with 95% confidence intervals (CIs). Data are reported as mean ± standard deviation (SD) and a P-value < 0.05 was considered significant.

RESULTS

Baseline demographic, clinical and laboratory features

The baseline demographic and clinical features of the 160 patients biopsied for evaluation of AKI compared with not-AKI patients are shown in Table 1. Patients undergoing PRB for evaluation of AKI were significantly older (58 ± 16 versus 44 ± 16 years; $P < 0.0001$) with a significantly higher proportion ≥60-years of age (48% versus 21%; $P < 0.0001$) and ≥65 years of age (39% versus 14%; $P < 0.0001$) and they were more often male (54% versus 36%; $P < 0.0001$). There were no racial differences between the two groups and systolic and diastolic BPs were similar as well. The proportion of patients with a systolic BP > 140 mmHg was similar between patients whether biopsied for AKI or not (32.5% versus 29.4%; P=0.45).

Baseline laboratory features are shown in Table 2. Patients with AKI had a significantly higher SCr at the time of biopsy (4.5 ± 2.7 versus 1.8 ± 1.6 mg/dL; $P < 0.0001$) and a significantly greater proportion of AKI patients had an SCr >1.5 mg/dL (94% versus 37%; $P < 0.0001$), >2.0 mg/dL (86% versus 25%; $P < 0.0001$) and >3.0 mg/dL (65% versus 12%; $P < 0.0001$) at the time of biopsy. The level of proteinuria was significantly less in patients biopsied for evaluation of AKI compared with not-AKI patients

Table 1. Baseline demographic and clinical features

	AKI	Not-AKI	P-value
n (%)	160 (17)	795 (83)	
Age (years), mean ± SD	58 ± 16	44 ± 16	<0.0001
Male, *n* (%)	86 (54)	284 (36)	<0.0001
Race, *n* (%)			
White	68 (43)	276 (35)	0.07
Black	69 (43)	357 (45)	0.73
Other	23 (14)	162 (20)	0.09
BP			
Systolic (mmHg), mean ± SD	136 ± 17	133 ± 19	0.11
>140 mmHg, *n* (%)	52 (32.5)	233 (29.4)	0.45
Diastolic (mmHg), mean ± SD	79 ± 12	80 ± 13	0.60

Table 2. Baseline laboratory data

	AKI	Not-AKI	P-value
Patients, *n* (%)	160 (17)	795 (83)	
Creatinine (mg/dL), mean ± SD	4.5 ± 2.7	1.8 ± 1.6	<0.0001
n	160	795	
UPro (g/g)	3.9 ± 5.6	4.3 ± 4.7	0.02
n, mean ± SD	143	773	
BT (min), mean ± SD	7.3 ± 1.9	6.9 ± 1.9	0.07
n (%)	157	788	
>9	20 (12.5)	58 (7.4)	0.03
PTT (sec), mean ± SD	28.1 ± 5.2	26.6 ± 4.7	0.0001
n (%)	158	787	
>33	24 (15.2)	42 (5.3)	<0.0001
PT			
n (%)	159	789	
Abnormal	43 (27.0)	101 (12.8)	<0.0001
Pre-Hgb (g/dL), mean ± SD	10.4 ± 1.7	12.1 ± 2.1	<0.0001
n	160	795	

n, number of lab values available.
UPro, urine protein.

Table 3. Biopsy procedure

	AKI	Not-AKI	P-value
n	160	795	
Fellow biopsy, *n* (%)	141 (88)	686 (86)	0.61
Needle gauge, *n* (%)			
14	133 (83.1)	702 (88.3)	0.08
16	27 (16.9)	93 (11.7)	
Adequate tissue	157 (98.1)	791 (99.5)	0.09

Table 4. Diagnoses in biopsies for AKI

Diagnosis	*n* (%)	Complications, *n* (%)	P-value
Glomerulonephritis/vasculitis	75 (47)	5 (7)	0.13
ATN	19 (12)	3 (16)	0.45
AIN	18 (11)	1 (6)	0.69
Glomerulopathy + ATN/AIN	17 (11)	1 (6)	0.69
Cast nephropathy/AL amyloid	14 (9)	6 (43)	0.01
AED/TMA/Other/QNS	17 (5)	2 (12)	1.0
Total	160	18	

AED, atheroembolic disease; ATN, acute tubular necrosis; TMA, thrombotic microangiopathy; QNS, quantity not sufficient.

Table 5. Biopsy complication

	AKI	Not-AKI	P-value
n	160	795	
Gross hematuria, *n* (%)	3 (1.9)	8 (1.0)	
Hematoma, *n* (%)	10 (6.3)	32 (4.0)[a]	
Both, *n* (%)	3 (1.9)	10 (1.3)	
Drop in Hgb + pain, *n* (%)	2 (1.3)	2 (0.3)	
Sudden death, *n* (%)	0	1 (0.1)	
Total, *n* (%)	18 (11.3)	53 (6.7)	0.04

[a]One death.

(3.9 ± 5.6 versus 4.3 ± 4.7 g/g; P=0.02) and a significantly lower proportion of patients biopsied for evaluation of AKI had proteinuria >1.0 g/g (67% versus 76%; P=0.036) and >3.5 g/g (32% versus 42%; P=0.03). The proportion of patients with a prolonged BT (12.5% versus 7.4%; P=0.03), a prolonged PTT (15.2% versus 5.3%; P < 0.0001) and an abnormal PT (27.0% versus 12.8%; P < 0.0001) was significantly greater in the AKI group compared with the not-AKI patients. The prebiopsy Hgb was significantly lower in patients biopsied for evaluation of AKI (10.4 ± 1.7 versus 12.1 ± 2.1 g/dL; P < 0.0001) and the proportion of patients with Hgb <10 g/dL (43% versus 17%; P < 0.0001) and <9.0 g/dL (21% versus 7%; P < 0.0001) was also significantly greater in patients biopsied for evaluation of AKI.

Biopsy procedure

Biopsies performed for the evaluation of AKI were performed by fellows under nephrology attending supervision in a proportion similar to not-AKI patients (Table 3). There was no significant difference in the proportion of biopsies done with 14-versus 16-gauge needles between the two groups. Adequate tissue for a diagnosis was obtained in a similar proportion of patients. The most common lesion found in patients biopsied for AKI was glomerulonephritis, and this was found in 47% of biopsies (Table 4). Acute tubular necrosis alone or acute interstitial nephritis (AIN) alone was observed in 12% and 11% of biopsies, respectively.

Biopsy complications

A complication after renal biopsy (Table 5) occurred more frequently in patients biopsied for evaluation of AKI compared with those biopsied for other reasons (11.3% versus 6.7%; P=0.04). The OR for a complication in AKI patients was 1.78 (95% CI 1.01–3.12) compared with not-AKI patients. In patients with a complication, the most common complication in both groups was a hematoma (identified by renal ultrasound or CT imaging), with or without gross hematuria, occurring in 13/18 patients (72.2%) biopsied for evaluation of AKI and in 42/53 (79.2%) of not-AKI patients. In four patients (two in each group) a decrease in Hgb associated with flank pain occurred and was likely the result of a perinephric hematoma, but no imaging study was done to confirm this (all required blood transfusions). There were no deaths in patients biopsied for AKI, whereas there were two deaths in not-AKI patients. One death was the result of hemorrhage post-PRB. The second death occurred suddenly the day after the biopsy and prior to discharge from the hospital in an elderly patient with diabetes mellitus. The biopsy had been uneventful with no signs or symptoms of complications or changes in Hgb after biopsy. At autopsy, there was no evidence of a biopsy-related complication (i.e. no subcapsular or perinephric hematoma). By multivariate analysis (Table 7), baseline features predictive of a complication were female gender, increased systolic BP, increased SCr level and decreased Hgb level. The reason for a PRB (i.e. AKI versus not-AKI) was not independently predictive of a complication.

In patients biopsied for evaluation of AKI, the complication rate was highest in patients found to have cast nephropathy or amyloid light-chain amyloidosis (Table 4), where 6/14 (43%) had a complication post-PRB. All six of the patients with a complication had an SCr >4.0 mg/dL and Hgb <10.0/dL and five had an abnormal BT, PTT or PT at the time of biopsy. All six patients had cast nephropathy and were ultimately diagnosed with multiple myeloma.

The most frequent intervention in patients with a complication was transfusion of PRBCs (Table 6). Overall, 10.0% of

Table 6. Complication intervention

	AKI	Not-AKI	P-value
n	160	795	
Transfusion alone, *n*	13	36	
Total transfusions, *n* (%)	16 (10.0)	42 (5.3)	0.02
Cystoscopy, *n* (%)	0	2 (0.25)	1.0
Radiologic embolization, *n* (%)	3 (1.9)	8 (1.0)	0.41
and transfusion, *n*	3	6	
Hematuria with obstruction, *n* (%)	0	1 (0.13)	1.0
Readmitted with hematoma, *n* (%)	2 (1.25)	5 (0.63)	0.33

Table 7. Baseline features predictive of a complication or need for transfusion post-PRB

Risk factor	Odds ratio	95% CI	P-value
Complication			
Female gender	2.01	1.2–3.6	0.02
Systolic BP[a]	1.01	1.001–1.03	0.04
SCr[b]	1.14	1.04–1.26	0.004
Hgb[c]	1.31	1.13–1.51	0.0003
Transfusion			
Female gender	2.12	1.08–4.10	0.029
Systolic BP	1.02	1.002–1.03	0.024
SCr	1.17	1.06–1.29	0.002
Hgb	1.48	1.25–1.75	<0.0001

[a]For every increase of 1 mmHg.
[b]For every increase of 1 mg/dL.
[c]For every decrease of 1 g/dL.

patients biopsied for evaluation of AKI required a postbiopsy transfusion compared with 5.3% (P=0.02) of not-AKI patients. The OR for requiring a transfusion postbiopsy in AKI patients was 2.04 (95% CI 1.12–3.74) compared with not-AKI patients. While the proportions were not significantly different, patients biopsied for AKI required an interventional radiology procedure/embolization twice as often (1.9% versus 1.0%; P=0.4) as not-AKI patients. By multivariate analysis (Table 7), baseline features predictive of the need for a transfusion post-PRB were female gender, increased systolic BP, increased SCr level and decreased Hgb level. The reason for the biopsy (i.e. AKI versus not-AKI) was not independently predictive of the need for a transfusion post-PRB.

DISCUSSION

We found that patients undergoing PRB for evaluation of AKI had a significantly higher complication rate (11.3% versus 6.7%; P = 0.04) and were significantly more likely to receive a transfusion of PRBCs due to a postbiopsy complication (10.0% versus 5.3%; P=0.02) compared with those undergoing biopsy for other reasons (not-AKI). Patients undergoing biopsy for evaluation of AKI had a greater number of risk factors for a complication, as they were significantly older, had a higher SCr level, lower Hgb level and a greater proportion with a prolonged BT, prolonged PTT and/or an abnormal PT at the time of PRB compared with patients biopsied for other reasons (not-AKI patients). By multivariate analysis, AKI was not an independent risk factor for a complication, but increased SCr level and decreased Hgb level were predictive of both complications and the need for transfusions and explained the difference in complication rates between the AKI and not-AKI patient groups.

After the advent of a safe PRB procedure in the 1950s the evaluation of patients with AKI was a primary indication for renal biopsy [1–5]. The biopsy material provided new insight into the pathology of AKI. However, as it became evident that the primary cause for AKI in hospitalized patients was a result of easily treatable causes such as hypovolemia or due to ischemic or nephrotoxic AKI, for which the only therapy was supportive care or dialysis, it became apparent that there was limited prognostic or therapeutic value in doing a renal biopsy in the majority of patients with AKI [6, 8, 28, 29]. As a result, over the last 60 years, biopsy for AKI has been almost completely reserved for patients with otherwise 'unexplained' AKI [11].

Acute renal failure is the indication for performing a renal biopsy in 10–25% of biopsy, series [24, 30–34], similar to our study. In hospitalized patients with AKI <10–20% actually undergo a renal biopsy as the majority of cases can be explained by hypovolemia, obstruction, drug-induced AIN or a drug-induced nephrotoxic acute tubular necrosis [8, 28, 35, 36]. A study by Kazi *et al.* [37], in which 158 patients presenting to the emergency department with AKI were biopsied, found that almost 70% had an acute tubular or interstitial etiology for AKI. Since the cause of AKI is evident in many of these cases and the prognosis and therapy are known, biopsy has limited value in these circumstances. As a result, the majority of patients with AKI do not undergo a renal biopsy, as the risk outweighs any potential benefit.

In patients undergoing biopsy for otherwise unexplained AKI, it has been shown that the information obtained is highly valuable. We found that in AKI the biopsy was diagnostic in >98% of cases and that an underlying glomerulonephritis was the primary cause for the AKI with interstitial nephritis or acute tubular necrosis alone seen infrequently. Our findings are similar to the observations found in other biopsy studies for unexplained AKI where glomerulonephritis is the primary finding in ≥50% of cases [28, 30, 33–36, 38, 39]. Lopez-Gomez *et al.* [33], in the AKI cohort of the Spanish registry of glomerulonephritis, found that in 14 000 biopsies done over 12 years, 16.9% were done for the evaluation of AKI and glomerulonephritis (often a necrotizing or crescentic lesion) was seen in >60% of biopsies. Haas *et al.* [30] evaluated the cause of unexplained AKI in patients ≥60 years of age and found that glomerulonephritis was demonstrated in 46%, most often a pauci-immune glomerulonephritis, whereas acute tubular necrosis alone was observed in only 7% of biopsies. Thus renal biopsy in unexplained AKI has significant diagnostic, prognostic and therapeutic implications.

Since it has been clearly shown that patients with AKI have an increased risk of progressive renal disease, morbidity and mortality that result in increased medical costs [6–10, 13], there is renewed interest as demonstrated by the NIDDK Kidney Precision Medicine Project [17] in performing 'research' renal biopsies in patients with AKI [14–16]. It is hoped that through the use of newer diagnostic procedures, such as proteomics and genomics on biopsy material, a better understanding of the pathophysiology of AKI will be achieved, resulting in the development of therapies that may prevent or halt the progression of AKI and possibly lead to resolution of AKI [14, 15, 17–19]. Clearly, doing PRB in such patients, many of whom are acutely ill, will initially pose a greater risk than benefit to such patients and thus the NIDDK recognizes the importance of providing participating patients 'with clear information about the risks associated with undergoing a kidney biopsy' [17].

PRB of NKs has generally been shown to be safe, with complications requiring an intervention such as transfusion or arterial embolization in ≤5% of cases and a biopsy-related death being extremely rare [20, 22–24]. It is well known that a number of features at baseline are significantly predictive of an increased risk of complications, including age >60 years, elevated BP, renal insufficiency, anemia and coagulopathy or thrombocytopenia [20–25, 31, 40, 41]. While patients undergoing biopsy for AKI have many of these risk factors, as demonstrated in our study, little has been published regarding the complication rate post-PRB in this group of patients. Corapi *et al.* [20], in a meta-analysis of >9000 PRBs of NKs performed with automated biopsy needles under real-time ultrasound guidance, found that studies in which >10% of patients were biopsied for AKI had an increased requirement for transfusions compared with those studies with <10% of biopsies for AKI (1.1% versus 0.04%, $P < 0.001$). A study by Tondel *et al.* [24], in >9000 biopsies, found that patients biopsied for evaluation of AKI were 2.3 times (1.7% versus 0.8%, $P < 0.001$) as likely to have a major complication (transfusion, surgery or interventional radiology procedure) compared with patients undergoing biopsy for other reasons. These observations are similar to ours, which demonstrate that patients undergoing biopsy for AKI are at twice the risk of a complication and need for a blood transfusion.

AKI is frequently observed in patients admitted to the intensive care unit (ICU), the majority of whom are elderly and critically ill with multiorgan failure. The use of PRB performed for the evaluation of AKI in the ICU setting has been reported in two retrospective studies. In a retrospective study conducted in 10 French ICUs for >10 years, Augusto *et al.* [25] reported 77 patients with AKI undergoing a renal biopsy during their ICU admission. The patients had significant comorbidity, with 68% of patients on renal replacement therapy, 57% on a ventilator and 13% on pressers at the time of the biopsy. The postbiopsy complication rate was high with 22% of the patients requiring 2–3 units of packed red cells and 2.5% requiring angiographic embolization by interventional radiology, but there were no deaths. In the second retrospective study involving five French ICUs, Philipponnet *et al.* [26] report 56 adult patients with AKI undergoing PRBs over a 12-year period. The patients had increased comorbidity at the time of biopsy, with 44% of patients on ventilators, 30% in shock and 80% receiving renal replacement therapy. The biopsies were performed with ultrasound by nephrologists in 84% of cases. Postbiopsy complications occurred with 13% of patients requiring 2–3 units of PRBCs and 3.6% of patient's requiring angiographic embolization by interventional radiology. The embolization was unsuccessful in one patient leading to multiorgan failure and death. Thus critically ill patients, those most at risk for AKI, have increased comorbidity and risk factors that results in an increased risk for a post-PRB complication.

Our study has a number of limitations. First, while information on baseline data and postbiopsy complications is obtained prospectively in all patients biopsied, this study was based on a retrospective evaluation of the data. Second, categorization of patients undergoing biopsy for AKI was based on the 'reason for biopsy' as defined at the time of biopsy by the nephrologist and was not based on a quantitative change in SCr level. Finally, patients undergoing biopsy for AKI were considered 'stable' for PRB. These limitations may have resulted in a misclassification of some patients in our study and may actually underrepresent the risk of post-PRB complication in patients with AKI. Despite these limitations, the fact that we observed an increased risk of post-PRB complications in patients biopsied for evaluation of AKI is clinically important given the paucity of existing data. Given the NIDDK Precision Medicine Project for obtaining PRB in AKI patients [17], we believe this information will be extremely beneficial in providing informed consent to participating patients.

In conclusion, patients undergoing PRB of NKs for evaluation of AKI have a greater rate of complication post-PRB compared with patients biopsied for other reasons. The increased complication rate in this patient population is likely a result of having a greater number of risk factors.

AUTHORS' CONTRIBUTIONS

S.M.K. and W.L.W. designed the study. S.M.K. and J.K.E. performed the statistical analysis. S.M.K. drafted the manuscript. S.M.K., C.N.G., J.K.E. and W.L.W reviewed and revised the manuscript. All authors approved the final version of the manuscript.

CONFLICT OF INTEREST STATEMENT

The results presented in this paper have not been published previously in whole or part, except in abstract format.

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