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Renal Trauma Increases Risk of Future Hypertension

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OBJECTIVE To determine if traumatic renal injuries or computed tomography (CT) findings are predictive of hypertension (HTN) development following injury.

METHODS A retrospective review of a renal trauma database was performed from 1995 to 2015. Renal injuries were graded by the American Association for the Surgery of Trauma system, with high-grade defined as IV or V. Nonrenal genitourinary trauma (ie bladder, penile, urethral, and testicular) patients were selected as controls. Patients with a diagnosis of HTN before their trauma or those lacking follow-up were excluded. Risk factors associated with HTN following trauma were identified using multivariable regression with propensity scoring.

RESULTS In total, 163 patients had a renal injury and 60 had nonrenal, genitourinary injuries. The median age was 31 years (interquartile range 23-43) with median follow-up of 4.7 years (interquartile range 1.9-8.5). Twenty-three (14%) patients with renal trauma were newly diagnosed with HTN on follow-up, compared with 2 (3%) in the control groups. (P = .02) After propensity quartile adjustment, patients with high-grade trauma had higher odds of developing HTN compared with low-grade renal trauma patients and controls (adjusted odds ratio 3.5, 95% confidence interval 1.3-9.3, P = .01). Patients with a midpole medial laceration and medial blood on CT had higher odds of developing HTN compared with patients without these characteristics (odds ratio 5.36, 95% confidence interval 1.3-22.6, P = .02).

CONCLUSION Increasing renal trauma grade is a risk factor for future development of HTN. CT findings at trauma presentation may be useful in stratifying patients who are at increased risk. UROLOGY 116: 198–204, 2018. © 2018 Elsevier Inc.

BACKGROUND

Trauma represents the sixth leading cause of death worldwide and roughly 3 million patients with trauma are hospitalized in the United States every year.[1] Of these, roughly 10% of traumatic injuries involve the genitourinary system (GU) (kidneys, bladder, urethra, etc.).[2] Among historic series, renal injury may occur in up to 3.3% of adult patients following trauma.[3-5] From a contemporary series, the incidence of traumatic renal injuries is 4.9 per 100,000 over a 2-year period[5] with the majority being blunt (>80%) among developed countries[3,5] and penetrating (>59%) among underdeveloped countries.[6,7]

Management of renal trauma has evolved with the development of the standardized grading system put forth by the American Association for the Surgery of Trauma.[8] Over the past few decades, management of renal injuries has shifted from surgical exploration to observation.[9] As a result, the rate of nephrectomy has decreased, even among high-grade renal trauma. Angioembolization has also emerged as a tool to help treat select renal injuries not responsive to observation.[10] However, the long-term sequelae of preserved post-traumatic renal units are poorly understood.

High-grade renal trauma causes injury to renal parenchyma, collecting system, or renal vasculature. This may lead to a compromise in renal function, renal artery thrombosis, renal parenchymal compression, and arterial-venous fistula.[11] Angioembolization also leads to an intentional devascularization of part of the kidney to halt bleeding.[12] Each may cause a disruption of the kidney's renin-angiotensin system which has been speculated to be the mechanistic cause of post-traumatic hypertension (HTN).[13]

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Pooled estimates among all series suggest a prevalence of HTN following renal trauma to be 0.6%-33% over a wide range of time (mean-34 months).[14] Consensus statements suggest that patients with high-grade renal injuries be followed with periodic blood pressure checks but this recommendation is based on observational data and carries a grade C recommendation.[14] Current literature reports short follow-up time and fails to identify predictors of HTN following renal trauma utilizing a comparator group. Furthermore, the incorporation of computed tomography (CT) imaging to assess the severity of renal injury with HTN development has not been described. With less than 2% of patients with renal trauma undergoing a nephrectomy,[14] an understanding of renal preservation on the development of HTN is warranted.

The primary aim of this study was to determine the prevalence and predictors of HTN development among a large cohort of patients with renal trauma compared with non-renal GU trauma patients. We hypothesize that higher grade renal injuries were associated with increasing risks of HTN development. In an effort to identify injury characteristics that are predictive of HTN, we explored contrast-enhanced CT images. Our secondary hypothesis was that CT imaging characteristics are predictive of HTN development.

METHODS

Study Population

After institutional review board approval, a retrospective review of a prospectively maintained renal trauma database at Zuckerberg San Francisco General Hospital from 1995 to 2015 was performed. This prospective database has been previously described.[4]

Renal trauma with postinjury follow-up was included for analysis from 1995 to 2015. Patients with non-renal GU trauma were selected as a control group. Such injuries were identified by International Classification of Disease codes and included traumatic bladder, testicular, penile, or urethral injuries.

In total, 390 patients had renal injuries, and 163 (42%) met our inclusion criteria. For comparison, 142 controls were identified and 62 (44%) met our inclusion criteria. Those patients with a diagnosis of HTN before trauma, were lost to follow-up (minimum follow up of 1 year required), and those patients who died secondary to their trauma, on follow-up chart review, or were found deceased following name and date of birth search via the California Electronic Death Registry (https://www.edrs.us) were all excluded.

Predictor Variables

Demographic data were collected on all patients including age at the time of injury, age at last encounter, gender, race or ethnicity, pretrauma diabetes, body mass index, type of trauma (blunt vs penetrating), etiology of trauma (motor vehicle accident, assault, fall, penetrating, pedestrian struck), and length of stay (LOS). All renal injuries were categorized using the American Association for the Surgery of Trauma grading system established in 1989 with computed tomography (CT).[8] High-grade renal trauma was defined as grades IV and V. Type of intervention performed was collected including if angioembolization was performed, if a renorrhaphy, ureteral stenting, or simple nephrectomy was performed.

All CT images for patients with renal trauma were evaluated by independent reviewers (BNB and GPM). Only those images archived after 2004 were accessible for independent review. We collected select CT findings based upon prior data from Dugi et al which demonstrated that certain CT findings at the time of renal injury are associated with increased hemostatic interventions.[15] The CT findings included for analysis were: formal grade[1-5] of injury, presence of a collecting system injury, intravascular contrast extravasation, laceration complexity (lateral, medial, or both), laceration location (superior pole, inferior pole, interpolar, or more than 1 location), presence of wedge infarction, distance of Gerota's fat disruption (cm), presence of medial blood, laterality (right, left, or both) and/or presence of adrenal hemorrhage.

Primary Outcome

Our primary outcome was post-traumatic development of HTN. To categorize the presence of HTN, the most recent electronic medical record (EMR) encounter following the initial traumatic injury was reviewed. Two independent reviewers (MAA and TWG) evaluated the EMR for either a diagnosis of primary HTN based on physician documentation or International Classification of Disease codes: 401.0, 401.1, 401.9, I11.0, I10.0, I11.9 I15.1, or I15.9. If the most recent EMR encounter did not have a diagnosis of HTN, all prior encounters were reviewed for previous diagnoses of HTN that had not been captured within the most recent medical record.

HTN was also defined by whether a new blood pressure medication had been initiated since the traumatic injury on EMR review. Categories of blood pressure medication included: calcium channel blockers, angiotensin converting enzyme inhibitors, angiotensin receptor blockers, diuretics, or beta blockers. Dual agents or combination medications were also included. If the most recent EMR encounter did not have documentation of new-onset blood pressure medication, all prior encounters were reviewed. If a blood pressure medication was started independent of a coexisting HTN diagnosis code, a chart review was performed (MAA and TWG) to reconcile the indication(s) for the medication.

Lastly, patients who did not have any follow-up were attempted to be reached by telephone whereby a single question was asked: "Since your injury on XXX date, have **you been given** a diagnosis of hypertension or high blood pressure—yes or no?" EPIC Systems© *CareEverywhere* which allows EMR access to outside institutions in Northern California (eg UCSF, Kaiser Permanente) was utilized to find records of those lost to follow-up who sought care in the Bay Area.

Individuals with a diagnosis of HTN or oral antihypertensive medication use before their traumatic event (n = 59) were excluded regardless of the organ(s) injured. A pretrauma diagnosis code of elevated high blood pressure (eg R03.0) without a diagnosis of HTN and HTN secondary to endocrine disorders (I15.2) were also excluded.

Statistical Analysis

Descriptive statistics were used to characterize the study population. Student *t* tests or Wilcoxon rank-sum test were used for comparison of means and medians of continuous variables, respectively. Chi-squared analyses were used for comparisons of categorical variables. *P* values less than or equal to 0.05 were considered statistically significant and all statistical tests were 2-sided. Because our outcome was rare (n = 23), we used propensity scores to avoid overfitting the data. We therefore performed logistic regression to model our exposure (high-grade renal trauma vs low grade and controls). The model was adjusted for age, sex, race or ethnicity, diabetes mellitus (yes or no), follow-up time (years), and LOS (days). Because injury severity was not routinely collected, we used LOS as a surrogate. This has previously been described by Newgard et al to have an area under the receiver operating characteristic curve to be 0.88 which suggests that LOS is an accurate proxy for injury severity.[16] Propensity scores were grouped into quartiles to assess overlap (see Supplemental Table 1) and controlled for in our final model.[17] All statistical analysis was performed with STATA v14 (College Station, TX).

RESULTS

Baseline Demographics

The median age of all patients at the time of their trauma was 29 years (interquartile range [IQR] 22-39) with a median follow up of 4.7 years (IQR 1.9-8.7). The median LOS for all patients was 7.[3-16] Patients with high-grade trauma had longer hospitalizations than patients without high-grade trauma (median 15 vs 5, respectively, $P < .001$). There were no differences between groups in follow-up time, age, gender, race or ethnicity, nor etiology of trauma between groups. The distribution in grades of renal trauma was most common among grade I (27.6%), grade III (26.4%), and grade IV (29.5%). Twelve patients were taken directly to the operating room for intervention secondary to renal trauma. The majority of renal traumas were managed nonoperatively (71.2%); however when operative intervention occurred, renhorrhapy was most common (17.8%). (Table 1) Among renal trauma patients, there were 4 less than 18 years and among controls there were 23 less than 18 years.

Hypertension Development

In total, 23 of 163 (14%) patients with renal trauma were newly diagnosed with HTN on follow-up, compared with 2 of 62 (3%) in the control groups ($P = .02$). (Supplemental Fig. 1) The median time to development of HTN was 8 months (IQR 2-29). Among all pediatric patients less than 18 years who had renal trauma (n = 23), only 1 patient developed HTN within 11 years of follow-up.

Table 2 demonstrates the differences in demographics of those who developed HTN compared with those who did not among renal trauma patients. Overall, patients with HTN following renal trauma were older (39, range 14-60 vs 29, range 3-74, $P = .002$) and had longer follow-up (8.7 years IQR 3.8-11.1 vs 4.8 IQR 1.8-8.4, $P = .01$). Otherwise, there were no differences to account for HTN among renal trauma patients based upon gender, race or ethnicity, etiology of trauma, nor type of operative interventions rendered.

Table 1. Comparisons between renal trauma patients and controls

	Renal Trauma N = 163	Controls N = 62	*P* Value
Median age (range)	29 (12-78)	29 (3-74)	.57
Median follow-up (y; IQR)	5.2 (2-9.1)	3.6 (1.5- 6.8)	.07
Gender			.93
Male	128 (78.5%)	49 (79%)	
Female	35 (21.5%)	13 (21%)	
Race or ethnicity			.61
White	46 (28.2)	22 (35.5)	
African American	51 (31.3)	18 (29)	
Latino	40 (24.5)	15 (24.2)	
Asian	12 (7.4)	5 (8.1)	
Other	14 (8.6)	2 (3.2)	
Etiology of trauma			.44
MVA passenger	7 (4.3)	2 (3.2)	
MVA driver	16 (9.8)	6 (9.7)	
Assault	15 (9.2)	4 (6.5)	
Fall	20 (12.3)	4 (6.5)	
Pedestrian struck	26 (16)	2 (3.2)	
Penetrating	77 (47.2)	25 (40.3)	
Renal trauma grade			
Grade I	45 (27.6)	n/a	
Grade II	14 (8.6)	n/a	
Grade III	43 (26.4)	n/a	
Grade IV	48 (29.5)	n/a	
Grade V	13 (8)	n/a	
Operative interventions *			
None	116 (71.2%)	n/a	
Renorrhaphy	29 (17.8%)	n/a	
Nephrectomy	9 (5.5%)	n/a	
Angioembolization	4 (2.5%)	n/a	
Vascular repair + renorrhaphy	3 (1.8)	n/a	
Ureteral stent + renorrhaphy	1 (0.6%)	n/a	
Vascular repair	1 (0.6%)	n/a	
Nonrenal GU trauma†			
Bladder	n/a	40 (64.5)	
Testicular	n/a	10 (16.1)	
Penile	n/a	9 (14.5)	
Urethral	n/a	3 (4.8)	

GU, genitourinary; IQR, interquartile range; n/a, not applicable; MVA, motor vehicle accident.
* n.b. some had more than 1 operation.
† Some patients had more than 1 GU organ injured.

Table 2. Comparison between those with HTN versus no HTN among renal trauma

	HTN N = 23 (14%)	No HTN N = 140 (86%)	*P* Value
Median age (range)	39 (14-60)	29 (3-74)	.002
Median follow-up (years; IQR)	8.7 (3.8-11.1)	4.8 (1.8-8.4)	.01
Gender			.26
Male	16 (69.6%)	112 (80%)	
Female	7 (30.4%)	28 (20%)	
Race or ethnicity			.31
White	8 (34.8)	38 (27.1)	
African American	10 (43.5)	41 (29.3)	
Latino	2 (8.7)	38 (27.1)	
Asian	1 (4.4)	11 (7.9)	
Other	2 (8.7)	12 (8.6)	
Pretrauma diabetes			
Yes	1 (4.4)	2 (1.4)	.33
Median body mass index (IQR)	25.5 (22.1-30.7)	25.1 (22.3-28)	.27
Etiology of trauma			.16
MVA passenger	0	7 (5)	
MVA driver	1 (4.6)	15 (10.8)	
Assault	3 (13.6)	12 (8.6)	
Fall	5 (22.7)	15 (10.8)	
Pedestrian struck	6 (27.3)	20 (14.4)	
Penetrating	7 (31.8)	70 (50.4)	
Renal trauma grade			.17
Grade I	6 (26.1)	39 (27.9)	
Grade II	2 (8.7)	12 (8.6)	
Grade III	4 (17.4)	39 (27.9)	
Grade IV	8 (34.8)	40 (28.6)	
Grade V	3 (13)	10 (7.1)	
Operative interventions			.87
Angioembolization	4 (2.9%)	0	
Renorrhaphy	3 (13%)	26 (18.6)	
Nephrectomy	1 (4.4%)	8 (5.7%)	
Vascular repair	0	1 (0.7%)	
Vascular repair + renorrhaphy	1 (4.4%)	2 (1.4%)	
Ureteral stent + renorrhaphy	0	1 (0.7%)	
None	18 (78.3%)	98 (70)	

HTN, hypertension.

Table 3. Multivariate regression analysis of association between covariates and high-grade renal trauma (n = 225)

Patient Characteristics	Adjusted OR (95% CI)	*P* Value
Age (every 10 y)	0.6 (0.5-0.9)	.006
Sex (male)	2.0 (0.8-5.1)	.17
Race		
White (reference)	1.0 (referent)	
African American	1.4 (0.6-3.3)	.47
Latino	1.0 (0.4-2.5)	.94
Asian	1.1 (0.3-4.3)	.93
Other	1.5 (0.4-6.2)	.56
Diabetes mellitus	0.8 (0.1-10.7)	.87
Follow-up time	1.0 (0.9-1.1)	.59
Length of stay (5 days)	1.3 (1.2-1.5)	<.001

CI, confidence interval; OR, odds ratio.

Propensity and Multivariate Analysis

Table 3 shows the results of the propensity analysis. Of note, age was inversely related to high-grade renal trauma (adjusted odds ratio [aOR] 0.6, 95% CI 0.5-0.9, $P = .006$). LOS was positively associated with high-grade renal trauma compared with low-grade renal trauma patients and controls (aOR 1.3, 95% CI 1.2-1.5, $P < .001$). After propensity quartile adjustment, patients with high-grade trauma had higher odds of developing HTN compared with low-grade renal trauma patients and controls (aOR 3.5, 95% CI 1.3-9.3, $P = .01$).

Imaging Associated Risk Factors

The CT images obtained after 2004 were reviewed (n = 93). Frequencies of CT findings for renal trauma patients are listed in Table 4. A collecting system injury was present in 33% of renal traumas with lateral laceration being most common (43.5%) followed by medial laceration (42.4%).

Univariate analysis was performed by comparing diagnosis of HTN with grades I-V, presence of a collecting system injury, intravascular contrast extravasation, laceration complexity (lateral, medial, or both), laceration location (superior pole, inferior pole, interpolar, or more than 1 location), presence of wedge infarction, distance of Gerota's fat disruption (cm), presence of medial blood, laterality (right, left, or both) and/or presence of adrenal hemorrhage. Only patients with a mid-pole medial laceration and medial blood seen on CT had higher odds of developing

Table 4. Frequencies and regression analysis of CT findings associated with renal trauma

		CT Images (N = 93)
Collecting system injury		31 (33.3%)
Intravascular contrast extravasation		25 (26.9%)
Mean perirenal hematoma rim distance (cm) (IQR)		1.8 (1-3)
Laceration complexity		
	Lateral	40 (43.5%)
	Medial	39 (42.4%)
	Both	13 (14.1%)
Adrenal hemorrhage		5 (5.5%)
Location of laceration		
	Superior	22 (23.7%)
	Middle	41 (44.1%)
	Inferior	22 (23.7%)
	≥2 locations	8 (8.6%)
Gerota's fat disruption		9 (9.7%)
Wedge infarction		22 (24%)
Laterality		
	Right	44 (47.3%)
	Left	41 (44.1%)
	Bilateral	8 (8.6%)
Regression Analysis	Unadjusted OR (95% CI)	*P* Value
No midpole medial laceration with medial blood	(Referent)	
Midpole medial laceration without medial blood	1.7 (0.2-16.6)	.66
Midpole medial laceration with medial blood	5.4 (1.3-22.6)	.02

HTN compared to patients without these characteristics (OR 5.4 95% CI 1.3-22.6, $P = .02$). (Table 4)

DISCUSSION

In an era of conservative management for stable patients with high-grade renal trauma, we sought to compare risk factors for new onset HTN among renal trauma compared with nonrenal GU trauma. Over a median follow-up of 4.7 years, significantly more patients who sustained renal trauma were newly diagnosed with HTN compared with nonrenal, GU trauma patients. After adjusting for co-founders of HTN in our propensity analysis (ie age, sex, race or ethnicity, diabetes mellitus, follow-up time, and LOS), the odds of developing HTN after a high-grade renal trauma was 3.5-fold higher than nonhigh-grade renal trauma patients. Risk factors for developing postrenal trauma HTN included a CT finding of a midpole medial laceration with medial blood on CT. Interestingly, nephrectomy or any renal procedures were not associated with developing HTN. Taken together, patients with high-grade renal trauma are at risk of long-term HTN and should undergo routine HTN screening.

To date, there is not a worldwide consensus on blood pressure monitoring following high-grade renal trauma. The European Urologic Association and the American Urological Association both recommend periodic blood pressure monitoring for at least 1 year following injury.[9,18] These recommendations are both Grade C.

After Goldblatt et al reported HTN in dogs following his "two kidney-one clip" experiment in 1934[19] and Page et al reported HTN following compression of the renal parenchyma with cellophane in 1939,[20] the notion of HTN following renal trauma has been reported by many.[13,21-26] Single-center series suggest that HTN following renal trauma occurs secondary to renovascular injury (ie renal artery stenosis or arteriovenous fistula) or external compression (ie subcapsular hematoma or fibrous encapsulation).[5,21,26] Despite numerous prior reports, the exact mechanism of HTN following renal trauma is poorly understood. Furthermore, only descriptive data exist.

One of the largest series was described by Chedid et al who performed a retrospective review of over 17,000 blunt renal trauma patients and found that only 10 patients developed HTN based using 3 separate manometer measurements.[13] Despite being the largest series of blunt renal trauma, the authors fail to demonstrate associations between grades of injury and HTN nor CT findings predictive of HTN development. The study was limited by its follow-up time being less than 6 months. Nevertheless, the authors conclude the incidence of HTN following renal trauma to be 0.57 of 1000.[13]

Taken together, prior data fail to compare the development of HTN in renal trauma patients with a control group in effort to identify clinical risk factors that may guide risk stratification. Routine surveillance with serial blood pressure measurements for HTN following renal trauma is warranted based on these results. In addition, noting medial blood with a midpole medial laceration on CT should prompt clinicians that patients are at increased risk of future HTN. We believe that a hilar injury causes decreased blood flow to the kidney and causes up-regulation of the renin-angiotensin system. This is perhaps the predominant mechanism for post-renal trauma HTN beyond external compression or wedge infarction, both of which were nonsignificant. A future, prospective study of renal vein sampling of renin would be necessary to confirm this hypothesis.

There are several limitations to our data. First, the series is a retrospective analysis of a prospective database, and despite controlling for follow-up time which was only 4.7 years, there are associated selection biases. In addition, the renal injuries were not isolated injuries along with the nonrenal GU injuries and were not recorded. Therefore, injury severity scores could not be calculated. As a result, it is possible that concomitant injuries, namely brain injuries may impact the degree of HTN, such as neurogenic HTN. As a surrogate for injury severity, we used hospital LOS which has been validated to be an accurate proxy.[16] Furthermore, our sample size was limited by our strict exclusion criteria which impacts our comparative analyses and introduces type 2 statistical error. We had a

limited follow-up duration, yet many studies report HTN development within 1-6 months and we expected to capture our primary outcome variable based upon prior literature.[13,23,24,26]

CONCLUSION

High-grade renal trauma is a risk factor for development of hypertension. Patients with high-grade renal trauma when compared to nonrenal GU trauma had an 8-fold increased risk of future hypertension. Patients with CT findings of medial blood with a mid-pole laceration had increased risks for HTN development. Interestingly, any renal procedure was not associated with HTN development. Routine screening for the development of HTN is warranted, especially among patients with high-grade renal trauma.

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APPENDIX

Supplementary Data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.urology.2017.10.063.

EDITORIAL COMMENT

This retrospective study addresses the incidence of developing hypertension following renal trauma. This is an important issue which has been the subject of sustained debate in the renal trauma literature. The authors have provided a valuable addition to our knowledge of this phenomenon through a well-planned and carefully performed retrospective review of renal injury at their institution, with a minimum of 1-year of follow-up.

In this review, 23 (14%) of renal trauma patients were newly diagnosed with hypertension (HTN) on follow-up, compared with 2 (3%) in the control groups ($P = .02$). Patients with high-grade trauma were found to be more likely to develop HTN compared with low-grade renal trauma patients and controls. In addition, patients with a midkidney medial laceration and medial blood on computed tomography had higher odds of developing HTN compared with patients without these characteristics.

Although the incidence of developing hypertension in this study is greater than many other reports in the literature, the data are appropriately analyzed and the importance of further study should be emphasized—including assessment of the time-course of development of hypertension, the status of the contralateral kidney in predicting hypertension as an outcome, and whether a hyper-reninemic state actually develops in the patients in whom hypertension is observed. It would also be valuable to better understand, through future studies on this topic, the anatomical renal imaging correlates of the development of hypertension—does this occur when there is evidence

of late capsular fibrosis or contracture? Persistent or calcified hematoma? Constrictive fibrosis in the area of the renal pedicle vasculature?

The study does present several limitations, including the selected control group, the difference in observational time-course of the compared study populations, and the inevitable lack of certainty regarding pre-existing hypertension before the renal injury. Nevertheless, the authors should be congratulated on their efforts to obtain the needed follow-up information—as the trauma population is typically difficult to follow over time. The long-term sequelae of renal trauma, as well as the later outcomes of other urologic injuries, represent important areas of future research. The authors have provided a valuable contribution to our understanding of the late outcomes of urologic injury.

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