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# Perioperative Blood Transfusion Is a Predictor of Acute and Chronic Renal Function Deterioration after Partial and Radical Nephrectomy for Renal Cell Carcinoma

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**Keywords**
Blood transfusion · Nephrectomy · Renal cell carcinoma · Renal function

**Abstract**
***Introduction:*** The aim was to evaluate the impact of perioperative blood transfusions (PBTs) on renal function after surgery for renal cell carcinoma (RCC). ***Methods:*** Consecutive patients with RCC who underwent partial nephrectomy or radical nephrectomy between 2005 and 2015 at a tertiary care center were included. Minimum follow-up period was 6 months. A PBT was defined as the transfusion of packed erythrocyte concentrate (EC) within 7 days before until 30 days after surgery. The multivariable analyses were carried out by Cox regression. ***Results:*** The overall cohort included 851 patients, of whom 93 (10.9%) received a PBT. The median follow-up was 46 months (range 28–72). In case of a PBT, a median of 2 EC was transfused. PBT patients were older and had a poorer performance status and more comorbidities as well as locally more advanced or metastatic tumors. In the multivariable analyses, PBT was an independent prognostic factor for acute as well as chronic renal impairment (hazard ratio (HR) 2.72, 95% confidence interval (95% CI) 1.45–5.10, $p = 0.002$ and HR 2.23, 95% CI 1.26–3.70, $p = 0.007$). ***Conclusion:*** PBT is associated with acute and chronic deterioration of kidney function after surgery for RCC. Thus, it may be used to identify patients requiring close nephrological monitoring.


## Introduction

Renal cell carcinoma (RCC) is a common malignancy. According to the American Cancer Society, 73,750 new RCCs are diagnosed annually, and the incidence is currently increasing [1]. The gold standard for the therapy of localized RCC is renal tumor surgery using partial nephrectomy (PN) or radical nephrectomy (RN) in case of advanced tumors or if a nephron sparing approach is not feasible. Despite the high standardization, PN and RN still need to be considered as major surgery, which poses a rather significant risk of perioperative complications [2]. Since acute and delayed hemorrhage are among the most common procedure-related complications, the re-

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quirement for perioperative blood transfusion (PBT) is still high and is usually estimated with rates between 10 and 20% in current studies [3]. Recently, PBTs have been evaluated as a potential risk factor for impaired oncological outcome in patients undergoing surgery for different malignancies, including colorectal, bladder, and prostate cancer [4–6]. Regarding RCC, a recent meta-analysis found a significant association between PBT and disease recurrence and cancer-specific and all-cause mortality for patients undergoing surgery for RCC [3]. To date, the available studies in the field of renal tumor surgery focus exclusively on the oncological outcome, whereas the influence on the functional outcome has not been investigated.

Therefore, the aim of this study was to examine the influence of PBT on postoperative short- and long-term renal function.

## Materials and Methods

*Study Design and Outcome Measurement*

The study was approved by the local ethical committee. The ethical approval number for data acquisition is 2014-811R-MA with amendment from August 1, 2016. Consecutive patients with RCC who received a PN or RN between January 1, 2005, and December 31, 2015, were retrospectively assessed. Patients with a follow-up of less than 6 months, an age of less than 18 years, missing entries in the register of transfusion medicine, Mannheim, and patients with unclear histopathology or surgery for recurrent RCC were excluded from the study.

A PBT was defined as the transfusion of packed leukocyte-depleted erythrocyte concentrate within 7 days before surgery until 30 days after surgery. Indications for the application of a PBT were hemoglobin levels below 6 g/dL, hemoglobin levels below 8 g/dL in case of additional risk factors (coronary heart disease, heart failure, and cerebrovascular insufficiency), and hemoglobin levels below 10 g/dL in case of evidence of an anemic hypoxia (tachycardia, lactic acidosis, and ischemia signs on the ECG).

End points included an acute renal failure, which was defined as the perioperative occurrence of an acute kidney injury (AKI) stages 2–3 according to the Acute Kidney Injury Network (AKIN) criteria and a new onset Chronic Kidney Disease (CKD), defined as the deterioration of a preoperative CKD stage from 1 to 2 preoperatively to 3–5 postoperatively [7]. For CKD assessment, the most recent estimated glomerular filtration rate value was regarded.

*Data Collection*

The data underlying this study was retrospectively acquired from a comprehensive database of all patients undergoing surgery for RCC maintained by the urological department of the University Medical Center Mannheim. Collected data included demographic patient-related data, such as age, sex, and ECOG performance status (PS), as well as comorbidities such as obesity (BMI ≥30 kg/m$^2$), arterial hypertension, diabetes mellitus, CKD stage, and anemia. The presence of anemia was defined according to the WHO criteria [8]. Collected tumor-specific parameters included the histological subtype, a sarcomatoid differentiation, the tumor diameter, the TNM stage according to UICC 2009, surgical margins, and grading. With regard to tumor grading, tumors from 2005 to 2013 were classified according to the Fuhrman grading system and after 2013 according to the WHO/ISUP classification [9]. Regarding the surgical parameters, data on the type of surgery (RN vs. PN) and the estimated intraoperative blood loss were assessed.

*Statistical Analyses*

The statistical analyses were carried out using the statistical software JMP (version 13.0.0, SAS Institute, Cary, NC, USA) and SPSS (version 26, IBM, Armonk, NY, USA). The demographic factors were described using median and interquartile range for continuous variables and using the absolute and valid percentage frequencies for categorical variables. Group comparisons between PBT and no PBT patients were based on the *t* test for independent samples for continuous variables and on the $\chi^2$ test for categorical variables.

In order to examine the prognostic value of the variables for the application of a PBT, binary logistic regression was used. Uni- and multivariable Cox regression was carried out for the analysis of the acute and chronic renal function deterioration. In each case, the hazard ratio (HR) or odds ratio (OR) and the associated 95% confidence interval (95% CI), as well as the *p* value were calculated. *p* values <0.05 were described as significant.

## Results

*Comparison of Patients with PBT versus without PBT and Risk Factors for a PBT*

Totally, 851 patients were included in this study (Table 1). In case of a PBT ($n$ = 758, 89.1%), a median of two erythrocyte concentrate was applied (interquartile range 2–5, range 1–55). Patients receiving a PBT were significantly older (median age: 72 vs. 63 years, $p < 0.001$) and had a worse PS (ECOG ≥ 1: 21.4 vs. 6.6%, $p < 0.001$), a significantly higher rate of arterial hypertension (86.4 vs. 65.3%, $p < 0.001$), diabetes mellitus (31.8 vs. 17.0%, $p = 0.001$), and anemia (61.3 vs. 16.0%, $p < 0.001$), as well as locally advanced and distant metastatic tumors. In addition, PBT patients showed a much higher percentage of preoperatively impaired kidney function with 59.1% compared to 22.7% of the patients having a CKD stage ≥3 ($p < 0.001$). Binary logistic regression identified a poorer ECOG-PS ≥ 1 (HR 2.30, 95% CI 1.10–4.77, $p = 0.026$), a higher tumor stage ≥pT3 (HR 2.01, 95% CI 1.06–3.83, $p = 0.034$), a preoperatively CKD stage ≥3 (HR 2.55, 95% CI 1.46–4.45, $p = 0.001$), preoperative anemia (HR 5.63, 95% CI 3.23–9.81, $p < 0.001$), and the presence of distant metastases (HR 2.58, 95% CI 1.04–6.38, $p = 0.041$) as in-

**Table 1.** Comparison of patients with PBT versus without PBT

| | Total cohort ($n$ = 851) | | PBT ($n$ = 93, 10.9%) | | No PBT ($n$ = 758, 89.1%) | | $p$ value[a] |
|---|---|---|---|---|---|---|---|
| | Median | IQR | Median | IQR | Median | IQR | |
| Age, years | 64 | 56–72 | 72 | 63–76 | 63 | 55–71 | <0.001[b] |
| | *N* | % | *n* | % | *n* | % | |
| Gender | | | | | | | |
| Female | 241 | 28.3 | 29 | 31.2 | 212 | 28.0 | 0.516c |
| Male | 610 | 71.7 | 64 | 68.8 | 546 | 72.0 | |
| ECOG | | | | | | | |
| 0 | 698 | 91.7 | 66 | 78.6 | 632 | 93.4 | <0.001[c] |
| ≥1 | 63 | 8.3 | 18 | 21.4 | 45 | 6.6 | |
| | 63 | 8.3 | 18 | 21.4 | 45 | 6.6 | |
| Obesity | 203 | 25.9 | 21 | 23.6 | 182 | 26.2 | 0.599[c] |
| AH | 531 | 67.6 | 76 | 86.4 | 455 | 65.3 | <0.001[c] |
| DM | 146 | 18.7 | 28 | 31.8 | 118 | 17.0 | 0.001[c] |
| Anemia | 178 | 21.0 | 57 | 61.3 | 121 | 16.0 | <0.001[c] |
| CKD | | | | | | | |
| 1–2 | 621 | 73.3 | 38 | 40.9 | 583 | 77.3 | <0.001[c] |
| 3–5 | 226 | 26.7 | 55 | 59.1 | 171 | 22.7 | |
| | Median | IQR | Median | IQR | Median | IQR | |
| Diameter, cm | 4.2 | 2.6–6.5 | 4.5 | 2.7–7.2 | 4.2 | 2.5–6.4 | 0.063[b] |
| | *N* | % | *n* | % | *n* | % | |
| Histology | | | | | | | |
| Cc | 604 | 71.5 | 71 | 76.3 | 533 | 70.9 | 0.271[c] |
| Ncc | 241 | 28.5 | 22 | 23.7 | 219 | 29.1 | |
| TNM | | | | | | | |
| pT1–2 | 665 | 78.5 | 58 | 62.4 | 607 | 80.5 | <0.001[c] |
| pT3–4 | 182 | 21.5 | 35 | 37.6 | 147 | 19.5 | |
| N1 | 42 | 5.2 | 10 | 10.9 | 32 | 4.5 | 0.009[c] |
| M1 | 51 | 6.1 | 12 | 13.0 | 39 | 5.2 | 0.003[c] |
| R1–2 | 30 | 3.6 | 7 | 7.8 | 23 | 3.1 | 0.023[c] |
| Grading | | | | | | | |
| 1–2 | 741 | 90.0 | 74 | 79.6 | 667 | 91.4 | <0.001[c] |
| 3–4 | 82 | 10.0 | 19 | 20.4 | 63 | 8.6 | |
| Sarcomatoid | 11 | 1.4 | 4 | 4.5 | 7 | 1.0 | 0.008[c] |
| Operation | | | | | | | |
| RN | 338 | 39.7 | 44 | 47.3 | 294 | 38.8 | 0.117[c] |
| PN | 513 | 60.3 | 49 | 52.7 | 464 | 61.2 | |
| Approach | | | | | | | |
| Open | 751 | 88.5 | 86 | 92.5 | 665 | 88.0 | 0.199[c] |
| Lap./rob. | 98 | 11.5 | 7 | 7.5 | 91 | 12.0 | |
| | Median | IQR | Median | IQR | Median | IQR | |
| Blood loss, mL | 200 | 100–400 | 500 | 100–700 | 200 | 100–375 | 0.029[b] |

PBT, perioperative blood transfusion; IQR, interquartile range; AH, arterial hypertension; DM, diabetes mellitus; cc, clear cell; ncc, non-clear cell; PN, partial nephrectomy; RN, radical nephrectomy; lap., laparoscopic; rob., robotic assisted. [a] PBT versus no PBT. [b] $t$ test. [c] $\chi^2$ test.

**Table 2.** Multivariable analyses of acute and chronic deterioration of renal function

| | Acute deterioration of renal function | | | Chronic deterioration of renal function | | |
|---|---|---|---|---|---|---|
| | HR | 95% CI | *p* value[a] | HR | 95% CI | *p* value[a] |
| Age (>65 vs. ≤65 years) | 0.82 | 0.51–1.31 | 0.408 | 1.77 | 1.28–2.46 | 0.001 |
| ECOG (≥1 vs. 0) | 2.01 | 1.01–3.99 | 0.047 | 1.72 | 0.93–2.95 | 0.083 |
| Obesity (BMI ≥30 vs. <30) | 0.92 | 0.55–1.53 | 0.743 | 1.00 | 0.69–1.42 | 0.988 |
| Diabetes mellitus | 1.21 | 0.70–2.09 | 0.488 | 1.34 | 0.90–1.87 | 0.149 |
| Arterial hypertension | 1.99 | 1.15–3.46 | 0.015 | 1.12 | 0.78–1.62 | 0.546 |
| Anemia | 0.73 | 0.40–1.31 | 0.287 | 1.06 | 0.69–1.59 | 0.783 |
| CKD (3–5 vs. 1–2) | 1.11 | 0.66–1.87 | 0.703 | | | |
| AKI (2–3 vs. 0–1) | | | | 1.74 | 1.18–2.53 | 0.006 |
| T stadium (T3–4 vs. T1–2) | 0.62 | 0.35–1.11 | 0.109 | 1.52 | 1.04–2.18 | 0.031 |
| Type of operation (RN vs. PN) | 1.85 | 1.14–2.99 | 0.012 | 2.25 | 1.59–3.20 | <0.001 |
| PBT | 2.72 | 1.45–5.10 | 0.002 | 2.23 | 1.26–3.70 | 0.007 |

PBT, perioperative blood transfusion; 95% CI, 95% confidence interval; HR, hazard ratio; PN, partial nephrectomy; RN, radical nephrectomy; AKI, acute kidney injury. [a] Cox regression.

dependent risk factors to obtain a PBT (see online suppl. Table 1; see www.karger.com/doi/10.1159/000509206 for all online suppl. material).

*Acute Deterioration of Renal Function*

Multivariable analyses of acute and chronic deterioration of renal function are shown in Table 2 (univariable analyses are shown in online suppl. Table 2). PBT patients were significantly more likely to experience an AKI in the univariable analysis with 28% ($n$ = 26) of PBT patients and only 13% ($n$ = 95) of non-transfused patients having a postoperative AKIN stage ≥2 ($p < 0.001$). In addition, a PBT was an independent predictor for the postoperative appearance of a higher grade AKI in the multivariable analysis (HR 2.72, 95% CI 1.45–5.10, $p = 0.002$). Besides a PBT, an arterial hypertension (HR 1.99, 95% CI 1.15–3.46, $p = 0.015$), a preoperative ECOG-PS ≥1 (vs. <1) (HR 2.01, 95% CI 1.01–3.99, $p = 0.047$), and RN (vs. PN) (HR 1.85, 95% CI 1.14–2.99, $p = 0.012$) were significant factors in the multivariable analysis for the occurrence of an AKIN stage ≥2.

*Chronic Deterioration of Renal Function*

60% ($n$ = 18) of the PBT patients had a new-onset CKD in the long-term follow-up compared to only 35% ($n$ = 186) of the patients without a PBT ($p = 0.010$). PBT showed a significant influence in the multivariable analysis (HR 2.23, 95% CI 1.26–3.70, $p = 0.007$). Apart from PBT, age >65 years (vs. ≤65 years) (HR 1.77, 95% CI 1.28–2.46, $p = 0.001$), RN (vs. PN) (HR 2.25, 95% CI 1.59–3.20, $p < 0.001$), and a postoperative AKIN stage ≥2 (vs. <2) (HR 1.74, 95% CI 1.18–2.53, $p = 0.006$) were independent prognostic factors for the long-term worsening of the kidney function.

*Oncological Outcome*

PBT showed no significant influence on the progression-free (HR 0.77, 95% CI 0.39–1.42, $p = 0.415$) and cancer-specific survival (HR 0.98, 95% CI 0.58–3.50, $p = 0.967$) in the multivariable analyses of the oncological outcome.

## Discussion

Despite the more frequent diagnosis of RCC in earlier stages and the increasing distribution of minimally invasive procedures in the last 2 decades, perioperative transfusion rates in renal tumor surgery remain high with reported rates of 10–20% [10]. Recently, various studies have examined the influence of PBT on the oncological outcome of patients after surgery for RCC [3]. This study is the first to investigate a possible link of PBT to kidney function after renal tumor surgery. Our analyses revealed a close association between these two.

In our analysis, patients who received a PBT presented a significantly more unfavorable starting situation: they were older and had a poorer PS and a higher rate of comorbidities (arterial hypertension, diabetes mellitus, anemia, and CKD) as well as locally advanced and distant metastatic tumors. Of these observed differences between

PBT and non-PBT patients, the presence of a preoperatively impaired renal function was, in addition to PS, tumor stage, distant metastases, and anemia, an independent risk factor to obtain a PBT in the multivariable analysis (HR 2.55, 95% CI 1.46–4.45, $p = 0.001$). Other authors found that the odds of a PBT was associated with age, gender, PS, tumor stage and localization, surgical volume and experience, open surgery, a preoperative anemia, and cumulative comorbidity scores, but they did not investigate the relationship between a preoperative CKD alone with the risk of applying a PBT [3, 11, 12]. Therefore, our study is the first that reveals patients with a CKD are a priori at risk to obtain a PBT, which represents the first link between a PBT and renal function in our study.

The second link between a PBT and renal function which we discovered was that a PBT showed a significant association with an acute and chronic deterioration in postoperative renal function (HR 2.72, 95% CI 1.45–5.10, $p = 0.002$ and HR 2.23, 95% CI 1.26–3.70, $p = 0.007$). Especially in cardiac surgery, a possible association of a PBT with kidney function has already been investigated. Freelander et al. [13] showed that patients with normal preoperative kidney function had a high risk (HR 5.25, $p = 0.005$) of developing an AKI in case of a PBT within the first 14 days after surgery. Accordingly, an impact of PBT on impaired kidney function was found in other operative fields [14]. Corresponding analyses in the field of renal tumor surgery are missing.

We know that the preservation of renal function plays a major role in the achievement of a better health-related and overall quality of life after renal tumor surgery [15]. Furthermore, worsening of the renal function has an important value for the long-term overall survival of patients, as it presents a risk factor for the occurrence of cardiac events [16]. Remarkably, regarding the increasing number of small renal masses and their inherent favorable prognosis, it seems to have more influence on the long-term survival than the tumor itself [17]. Indeed, patients with T1 RCC are more likely to die as a consequence of an impaired renal function than for oncological reasons [17]. Therefore, these facts underline the need for renal function preservation during and after renal tumor surgery.

The deterioration of renal function associated with PBT may arise by different mechanisms. First, clinical circumstances as an anemic hypoxia or cardiovascular instability, which require a PBT because of a decreased tolerance to blood loss, may lead to a priori renal tissue changes and consecutively to kidney damage. Therefore, a PBT may be not the reason, but rather a marker of other factors causing renal function deterioration here. The second is the transfusion-induced immune modulation, in which residual donor leukocytes, antigens, and transfused growth factors are suspected to contribute to immunosuppressive, pro-inflammatory, and pro-angiogenic effects causing damage in several organs and thereby affecting the kidney function [18]. As a third mechanism, the possible occurrence of transfusion-related complications caused by hemolysis or infection must be considered. Despite being rare, hemolytic transfusion reactions with intravascular hemolysis can lead to complement-induced endothelial injury, hemodynamic instability, and disseminated intravascular coagulation and with substantial end-organ injury, including AKI [19]. Furthermore, free hemoglobin released during intravascular hemolysis can directly induce kidney injury [19].

In renal tumor surgery, lower transfusion rates were found by using minimally invasive surgery compared to open surgery [10]. In addition, nephron-sparing surgery may have a positive effect on the frequency of PBT [10, 20]. There are currently no clear guidelines on how preoperative circumstances can be improved for patients regarding comorbidities, which lead to a reduced tolerance for blood loss and, therefore, require an increased need for PBT.

In summary, our study was the first in the field of renal tumor surgery to examine an association of PBT with the functional outcome. We observed that a preoperatively impaired renal function was an independent risk factor to obtain a PBT and that PBT was associated with an acute and long-term deterioration of kidney function.

Limitations of this study are mainly due to its retrospective nature and should be considered. As patients with PBT presented an initially unfavorable clinical starting situation, confounding especially through differences in terms of the existing comorbidities and tumor-and surgical-related factors should be considered, and results should be interpreted with caution. In addition, some clinical and surgical data, such as the RENAL scores of the tumors, the need and duration for intraoperative ischemia, and the surgeons' volume, could not be properly assessed retrospectively for our cohort and could, therefore, not be taken into account for the analyses.

## Conclusion

PBT may be associated with acute and chronic deterioration of renal function after PN and RN. However, due to the retrospective design and potential confounding of this study, a causative relationship remains unclear. Nevertheless, our results underline the need for a thorough nephrological consultation and a close follow-up of patients receiving PBT in the course of renal tumor surgery.

## Statement of Ethics

The study was approved by the local ethical committee. Ethical approval number for data acquisition is 2014-811R-MA with amendment from August 1, 2016.

## Conflict of Interest Statement

The authors have no conflicts of interest to declare.


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## Author Contributions

Julia Mühlbauer: protocol/project development, manuscript writing/editing, and data analysis. Johannes de Gilde: data collection and analysis. Michael Mueller-Steinhardt: critical revision and scientific input. Stefan Porubsky: critical revision and scientific input. Margarete Walach: critical revision and scientific input. Philipp Nuhn: critical revision and scientific input. Harald Klüter: critical revision and scientific input. Nina Wagener: critical revision and scientific input. Maximilian C. Kriegmair: protocol/project development, manuscript writing/editing, and data analysis.

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