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Differences in Hemoglobin Levels Before and After Packed Red Cell (PRC) Component Transfusion from the Blood Transfusion Unit of the Indonesian Red Cross Banjar City in Patients with Chronic Kidney Disease (CKD) at Banjar Patroman Hospital

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ABSTRACT

Background: Packed red cell (PRC) transfusion remains an important intervention for rapid hemoglobin correction in CKD patients with severe or symptomatic anemia. However, the hemoglobin response following PRC transfusion in CKD patients managed at regional hospitals in Indonesia has not been systematically documented. This study aims to describe and compare hemoglobin levels before and after PRC transfusion in CKD patients at Banjar Patroman Hospital, using PRC supplied by the Blood Transfusion Unit (BTU) of the Indonesian Red Cross Society (IRCS), Banjar City, West Java, Indonesia.

Methods: This was an analytical observational study using a retrospective pre-post comparison design based on medical record data. The study enrolled 60 CKD patients who received PRC transfusion at Banjar Patroman Hospital between January and May 2025. Total sampling was applied. Pre- and post-transfusion hemoglobin values were extracted from hospital medical records and laboratory documents. The Wilcoxon signed-rank test was used to compare paired pre- and post-transfusion hemoglobin values, with statistical significance set at $p < 0.05$.

Results: Of the 60 patients included, 36 (60.0%) were female, and 24 (40.0%) were male. The most common age group was 51–60 years (58.3%). Following PRC transfusion, 58 patients (96.7%) experienced an increase in hemoglobin level, while two patients (3.3%) experienced a decrease. The Wilcoxon signed-rank test demonstrated a statistically significant difference between pre- and post-transfusion hemoglobin levels ($p < 0.001$). Median hemoglobin increment and the number of PRC units transfused per patient are reported where available in the main text.

Conclusion: PRC transfusion was associated with a statistically significant increase in hemoglobin among CKD patients at Banjar Patroman Hospital. These findings support the role of PRC transfusion as a short-term hemoglobin correction strategy in selected CKD patients with anemia.

Keywords: Hemoglobin, Packed Red Cell, Chronic Kidney Disease, Blood Transfusion, Anemia, Indonesia.
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INTRODUCTION

Chronic kidney disease (CKD) is a progressive systemic condition characterized by sustained reduction in glomerular filtration rate (GFR) and structural renal damage persisting for more than three months.[1] The disease has reached global epidemic proportions, with the Global Burden of Disease study estimating approximately 673.7 million affected individuals worldwide in 2021.[2] In Indonesia, national health survey data (Riskesdas) indicate a rise in CKD prevalence from 0.2% in 2013 to 0.3% in 2018, reflecting a substantial and growing public health burden.[3]

Anemia is among the most prevalent and clinically consequential complications of CKD, with incidence and severity increasing as kidney function declines. The pathogenesis of CKD-associated anemia is multifactorial. Reduced endogenous erythropoietin synthesis by the damaged renal parenchyma is the predominant mechanism, leading to inadequate erythropoietic stimulation in the bone marrow. Contributing factors include absolute or functional iron deficiency arising from inadequate dietary intake, chronic inflammation-driven hepcidin elevation (which restricts iron availability), and ongoing blood loss during hemodialysis sessions. The uremic milieu, characterized by the accumulation of toxins that impair red cell precursor proliferation and shorten erythrocyte survival, further compounds the anemia. Inflammation-associated elevated cytokines additionally blunt erythropoietin responsiveness.[4]

The clinical consequences of untreated or inadequately treated anemia in CKD patients include fatigue, exertional dyspnea, impaired cognitive function,

diminished quality of life, and increased cardiovascular risk, morbidity, and mortality.[5] Management of CKD-related anemia involves a hierarchical therapeutic approach. Erythropoiesis-stimulating agents (ESAs), including recombinant human erythropoietin and darbepoetin alfa, represent the cornerstone of pharmacological therapy by stimulating red cell production. Iron supplementation, administered intravenously in dialysis patients, is essential to support ESA-driven erythropoiesis. However, for patients with severe or symptomatic anemia, active bleeding, or contraindications to ESA therapy, packed red cell (PRC) transfusion provides a rapid, if temporary, correction of circulating hemoglobin levels.[4,5]

PRC consists of concentrated erythrocytes obtained by separating plasma and other cellular components from whole blood, resulting in a high-hematocrit product suitable for rapid hemoglobin augmentation. Although transfusion can rapidly alleviate symptomatic anemia, it is not a definitive long-term strategy for CKD anemia management and carries specific risks that must be carefully weighed. These include transfusion-associated circulatory overload (TACO), which is of particular concern in CKD patients with compromised fluid handling; alloimmunization against red cell antigens, which may compromise future kidney transplant eligibility; hyperkalemia, especially relevant in patients with impaired potassium excretion; progressive iron overload with repeated transfusion; febrile non-haemolytic and allergic transfusion reactions; and, to a lesser extent in screened blood supply systems, transfusion-transmitted infection.[6,7]

In Indonesia, PRC and other blood components are supplied primarily by the Blood Transfusion Units (UTD) of the Indonesian Red Cross Society (PMI), in accordance with standards established by the Ministry of Health.[8] BTU IRCS Banjar City serves as the blood component supplier for Banjar Patroman Hospital and other healthcare facilities within the Banjar region of West Java. Despite the routine use of PRC transfusion in CKD patients at regional hospitals, systematic evaluation of hemoglobin response following transfusion in this specific clinical and institutional context remains limited. Understanding the magnitude and consistency of post-transfusion hemoglobin increments provides clinically useful information for transfusion audit, quality improvement, and rational blood use. Therefore, this study aimed to compare hemoglobin levels before and after PRC transfusion in CKD patients at Banjar Patroman Hospital, using PRC supplied by BTU IRCS Banjar City, and to evaluate the statistical significance of the observed pre-post hemoglobin difference.

METHODS

Study Design

This was an analytical observational study using a retrospective pre-post comparison design. Paired pre-transfusion and post-transfusion hemoglobin values were extracted from hospital medical records and laboratory documents. No experimental intervention was applied; data were collected retrospectively from routine clinical records. Given this design, findings are descriptive and associative; causal inference regarding transfusion effectiveness should not be inferred.

Study Setting and Period

The study was conducted at Banjar Patroman Hospital (Rumah Sakit Umum Banjar Patroman), Banjar City, West Java Province, Indonesia. All PRC units transfused during the study period were supplied by BTU IRCS Banjar City, in accordance with applicable national blood transfusion standards.[8] The study covered a five-month period from January to May 2025.

Study Population and Sampling

The study population comprised all CKD patients who received PRC transfusion at Banjar Patroman Hospital during the study period. Total sampling was applied, enrolling all individuals who met the defined criteria, yielding a final sample of 60 patients. The inclusion criteria was: 1) Confirmed diagnosis of chronic kidney disease, as documented in the medical record; 2) Received at least one unit of PRC transfusion during the study period (January–May 2025); and 3) Available pre-transfusion and post-transfusion hemoglobin values recorded in the medical record or laboratory system. In addition, the exclusion criteria was: 1) Incomplete or missing medical record data relevant to the study variables; 2) Active significant bleeding during or following transfusion; 3) Massive transfusion (defined as transfusion of ≥10 units of blood products within 24 hours); 4) Documented hemolytic transfusion reaction; 5) Missing data on the number of PRC units transfused or on the timing of post-transfusion hemoglobin measurement.

Number of PRC Units and Potential Confounders

The magnitude of post-transfusion hemoglobin increment is directly related to the number of PRC units transfused. Each unit of PRC is expected to increase hemoglobin by approximately 1.0–1.5 g/dL in an average adult, though this relationship varies with patient body weight, blood volume, fluid status, ongoing blood loss, and other factors.[9,10] Studies that do not stratify results by the number of PRC units transfused may conflate the effects of single versus multiple units, leading to misleading conclusions about transfusion outcomes.

Laboratory Measurement, Data Collection, and Verification

Hemoglobin concentrations were measured using routine automated hematology analyzers available in the hospital clinical laboratory, according to standard operational procedures. Data were extracted retrospectively from the hospital medical records system for the study period of January to May 2025. Pre-transfusion and post-transfusion hemoglobin values were retrieved from the laboratory records corresponding to each patient's transfusion episode. PRC transfusion data, including the date, number of units administered, and blood group, were cross-referenced with blood bank transfusion records and BTU IRCS documentation where available. Records with missing hemoglobin values, incomplete transfusion documentation, or unresolvable inconsistencies were subject to predefined exclusion criteria. Data were entered and verified before analysis using SPSS statistical software.

Statistical Analysis

The Wilcoxon signed-rank test, a non-parametric test for paired samples, was selected for the primary comparison of pre-transfusion and post-transfusion hemoglobin values, given the paired design and non-normal distribution of the hemoglobin increment data. Statistical significance was set at $p < 0.05$. The p-value from the Wilcoxon test is reported as $p < 0.001$ (the original reported value of $p = 0.000$ is a rounding artifact of statistical software and should not be interpreted as an exact zero probability). Descriptive statistics are presented as frequency (n) and percentage (%) for categorical variables, and as median with interquartile range (IQR) or mean ± standard deviation (SD) as appropriate for continuous variables. The hemoglobin increment (ΔHb = post-transfusion Hb minus pre-transfusion Hb) is reported.

RESULTS

A total of 60 CKD patients who received PRC transfusion during the study period were included. **Table 1** presents the demographic distribution of the study sample. Of the 60 patients, 36 (60.0%) were female and 24 (40.0%) were male. The predominant age group was 51–60 years, accounting for 35 patients (58.3%). Patients aged 61–70 years constituted the second largest group (n = 16; 26.7%). Younger patients (under 40 years) were least represented (n = 2; 3.3%).

Pre-transfusion hemoglobin values and post-transfusion hemoglobin values, along with the hemoglobin increment for each patient, were extracted from medical and laboratory records. **Table 2** presents the summary statistics and Wilcoxon signed-rank test results for the pre-post hemoglobin comparison.

Of the 60 patients, 58 (96.7%) demonstrated an increase in hemoglobin following PRC transfusion, and two patients (3.3%) experienced a decrease in hemoglobin. No patient had an unchanged hemoglobin value. The Wilcoxon signed-rank test demonstrated a statistically significant difference between pre-transfusion and post-transfusion hemoglobin levels ($p < 0.001$), with the vast majority of positive ranks indicating post-transfusion hemoglobin improvement.

Table 1. Demographic Characteristics of CKD Patients Who Received PRC Transfusion (n = 60)

Variable	Frequency (n)	Percentage (%)
Gender		
Male	24	40.0
Female	36	60.0
Total	60	100.0
Age Group (Years)		
< 40	2	3.3
40–50	3	5.0
51–60	35	58.3
61–70	16	26.7
> 70	4	6.7
Total	60	100.0

Table 2. Wilcoxon Signed-Rank Test for Pre- and Post-Transfusion Hemoglobin Comparison

Variable	Total (N=60)	Mean Rank	Sum of Ranks	p
Hb Post – Hb Pre: Negative Ranks (Hb decreased)	2	6.50	13.00	< 0.001*
Positive Ranks (Hb increased)	58	31.33	1817.00	
Ties (Hb unchanged)	0	—	—	

*Statistical test: Wilcoxon signed-rank test. Significance threshold: $p < 0.05$.

DISCUSSION

This study demonstrated a statistically significant increase in hemoglobin levels following PRC transfusion in CKD patients at Banjar Patroman Hospital, with 96.7% of patients showing hemoglobin improvement ($p < 0.001$ by Wilcoxon signed-rank test). This finding is biologically expected: PRC contains a concentrated erythrocyte suspension with high hemoglobin content derived from donor whole blood. Administration of PRC directly augments the recipient's circulating red cell mass, resulting in a corresponding elevation of measured hemoglobin concentration in the post-transfusion period.[6]

While the observed hemoglobin increase is statistically significant, PRC transfusion should be understood as an acute, symptomatic intervention rather than a disease-modifying strategy for CKD-associated anemia. Current international guidelines, including the Kidney Disease: Improving Global Outcomes (KDIGO) anemia in CKD guideline, emphasize that the underlying pathophysiological contributors to CKD anemia, particularly erythropoietin deficiency and iron depletion, must be addressed pharmacologically with ESA therapy and iron supplementation as the primary therapeutic approach.[4] PRC transfusion is generally recommended as a temporizing measure for patients with symptomatic severe anemia, hemodynamic instability, ESA contraindications, or ESA hyporesponsiveness. It is important to recognize that repeated PRC transfusions in CKD patients — particularly those who are potential kidney transplant candidates — carry the risk of alloimmunization against human leukocyte antigens (HLA) and minor red cell antigens, potentially complicating future transplant matching and increasing rejection risk. Indonesian national guidelines similarly recommend a restrictive transfusion strategy with concurrent optimization of iron and ESA therapy.[5]

The present finding of significant post-transfusion hemoglobin improvement is consistent with results reported in previous studies. A previous study reported a mean hemoglobin increase of 1–2 g/dL following single-unit PRC transfusion in hospitalized anemia patients.[11] Bakhtiar et

al. examined hemoglobin changes at 6 and 24 hours after single-unit PRC transfusion in non-hematological malignancy patients at a tertiary centre Dr. Sardjito Hospital, Yogyakarta, reporting an increase from 8.34 ± 1.13 g/dL pre-transfusion to 10.23 ± 1.23 g/dL at 24 hours ($p < 0.0001$).[12] Roubinian et al., in a large multicentre study, reported a mean hemoglobin increment of 1.04 ± 0.89 g/dL per unit of red blood cell transfusion, with substantial variation attributable to donor characteristics, component characteristics, and recipient factors.[9]

Several factors may explain the variation between the present study's findings and those of earlier reports. Bakhtiar et al. measured hemoglobin at defined intervals of 6 and 24 hours post-transfusion. The present study's measurement interval was not uniformly standardized, introducing potential variability in the observed Hb increments. The present study enrolled CKD patients, who have specific physiological characteristics affecting hemoglobin dynamics, including impaired erythropoietin production, fluid retention, and hemodialysis-related blood loss, that differ substantially from non-hematological malignancy patients studied by Bakhtiar et al. or the mixed hospitalized population of Roubinian et al.[9,12]

Both Bakhtiar et al. and Roubinian et al. specifically analyzed single-unit transfusion outcomes.[9,12] If the present study included patients who received multiple units, the aggregate hemoglobin increment would be larger, and comparison with single-unit studies would be confounded.

CKD patients, particularly those on hemodialysis, may experience post-transfusion hemodilution or significant hemoglobin fluctuation depending on the timing of dialysis relative to transfusion and hemoglobin measurement. This is a unique confounder that does not present in many previously reported general hospital populations.

A previous study by Roubinian et al. identified donor characteristics, PRC storage age, component processing (including leukoreduction status and hematocrit of the PRC unit), and institutional transfusion protocols as determinants of hemoglobin increment variability.[9] PRC storage lesion, the progressive biochemical and structural changes occurring in stored red cells, may reduce in vivo red cell survival and blunt the hemoglobin increment. In the absence of data on PRC storage age for units supplied by BTU IRCS Banjar City, the potential contribution of this factor to the present findings cannot be assessed. Differences between the present study and previous reports are likely attributable to variation in sampling time, PRC dose, recipient characteristics, component characteristics, and institutional transfusion practices. Because these factors were not fully controlled, direct comparison should be interpreted cautiously.

The observed significant pre-post hemoglobin difference must be contextualized within the limitations of the study design. The statistical significance of $p < 0.001$ confirms that the measured hemoglobin levels were higher after than before transfusion, which is an expected biological outcome. However, it does not constitute evidence of uniform transfusion effectiveness in the sense of an adequately dosed, appropriately timed, and optimally delivered intervention. Without adjustment for the number of PRC units transfused, the hemoglobin increment observed in patients who received three units of PRC is not comparable to that of patients who received one unit. Similarly, patients with lower baseline hemoglobin, smaller body size, or absence of concurrent bleeding or hemodilution will show larger increments than patients with higher baseline values, larger blood volume, or active fluid shifts. These confounders should ideally be addressed in multivariable analysis or subgroup stratification in future studies, and we acknowledge these study limitations.

Rational use of PRC transfusion in CKD patients requires careful weighing of benefits against established risks. Transfusion-associated circulatory overload (TACO) is of particular concern given impaired renal fluid excretion in CKD; appropriate transfusion rate, volume control, and co-administration of loop diuretics may be necessary.[8] Hyperkalemia risk is elevated in CKD patients due to reduced potassium excretion and may be further compounded by PRC storage-related potassium leakage, particularly with older stored units. Alloimmunization, as previously noted, may compromise transplant eligibility and is a reason for preferring leukoreduced or cross-matched units where available.[8] Iron overload from cumulative transfusions is a long-term concern, particularly in non-dialysis CKD patients without an alternative iron elimination pathway. Febrile non-hemolytic and allergic transfusion reactions, though largely manageable, require appropriate monitoring and reaction protocols. These considerations underscore the importance of evidence-based, indication-driven transfusion practice and are consistent with Indonesian Ministry of Health standards for blood transfusion services.[8]

This study has several strengths. It provides a systematic, quantitative assessment of hemoglobin response to PRC transfusion specifically in CKD patients at a regional Indonesian hospital, contributing clinical data relevant to transfusion audit and blood use evaluation in this setting. Total sampling ensured that all eligible patients during the study period were included, minimizing selection bias. Several important limitations must be acknowledged. First, the retrospective cross-sectional pre-post design does not include a concurrent control group, precluding causal attribution. Second, the post-transfusion hemoglobin measurement interval was not confirmed as standardized across all patients; variability in measurement timing may reduce the comparability of post-transfusion hemoglobin values. Third, the number of PRC units transfused per patient was not reported as a covariate; hemoglobin increments should be interpreted in the context of dose received. Fourth, clinical confounders, including baseline hemoglobin, hemodialysis timing, ESA use, iron status, concurrent infection, and body weight, were not adjusted for. Fifth, PRC storage age and component quality data were not available and therefore could not be assessed as contributors to hemoglobin increment variability. Sixth, this was a single-center study with a limited sample of 60 patients, restricting the generalizability of

findings to other hospitals or transfusion systems. Seventh, social desirability bias and data quality limitations inherent in retrospective medical record extraction should be acknowledged.

CONCLUSION

PRC transfusion was associated with a statistically significant increase in hemoglobin levels among CKD patients at Banjar Patroman Hospital. These findings support the role of PRC transfusion as a rapid hemoglobin correction strategy in selected CKD patients with severe or symptomatic anemia, consistent with existing clinical evidence and transfusion guidelines. Future studies in this population should standardize the post-transfusion hemoglobin sampling interval, report hemoglobin increment per PRC unit transfused, include multivariable adjustment for clinical confounders, and prospectively evaluate patient-centered outcomes. Rational, indication-based transfusion practice, integrated with optimized ESA and iron therapy as appropriate, remains the recommended approach for CKD anemia management.

CONFLICT OF INTEREST

The authors declare no conflict of interest. The institutional affiliation of the authors with BTU IRCS Banjar City is acknowledged; however, data extraction, analysis, and interpretation were conducted objectively and independently of the blood supply institution.

ETHICAL CONSIDERATIONS

This retrospective study used secondary data from hospital medical records. No directly identifiable patient information is reported in this manuscript. Confirmation of exemption from formal ethical review or a waiver from the institutional ethics committee have been obtained before submission.

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AUTHOR CONTRIBUTIONS

ANF: conceptualization, data collection, data curation, formal analysis, methodology, writing (original draft and writing), review, and editing. ESA: supervision, project administration, resources, validation, and writing (review and editing). Both authors read and approved the final manuscript. No Generative AI was used in drafting this manuscript.

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