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The Association Between Transfusion of Blood Products and Postoperative Renal Impairment

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Abstract

Purpose of Review This review summarizes the most recent literature on the association between the administration of blood products with acute kidney injury (AKI). The multifactorial nature of AKI is reviewed and the potential mechanisms for the association are explored. In addition, immunological causes of AKI after transfusion are addressed. We also provide recommendations for transfusion practice and perioperative management to minimize the risk of AKI.

Recent Findings Several recent studies have demonstrated a consistent association between the administration of blood products and AKI. Restrictive blood transfusion protocols have however failed to reduce the incidence of AKI.

Summary Transfusion of blood products is definitively associated with AKI. Considering current evidence however it is still unknown if there is a cause-and-effect relationship underlying this association. Notwithstanding the uncertainty, blood products should be employed with great caution in patients who are at significant risk of AKI until further evidence becomes available to clarify the situation. The etiology of AKI is multifactorial, and factors other than transfusion may be more important. Intraoperative hypotension must be prevented. The use of drugs which have the potential for renal toxicity like intravenous contrast agents, aminoglycoside antibiotics and non-steroidal analgesic drugs should be minimized. Adequate fluid resuscitation is also necessary to guarantee adequate cardiac output and renal perfusion. Goal-directed fluid management should be considered in high-risk patients. Transfusion of incompatible blood products must also be avoided as at all costs because this can lead to severe AKI.

Keywords Blood transfusion · Acute kidney injury · Intravenous fluid management · Vasopressors · Goal directed fluid therapy

Introduction

Postoperative acute kidney injury (AKI) is a significant complication that is associated with prolonged hospitalization, higher cost of medical care, the development of chronic renal failure and increased mortality [1–3]. Several recent studies have demonstrated a consistent association between the administration of packed red cells and the development of AKI [4–6]. AKI in these studies was defined as an increase in creatinine greater than 0.5 mg/dl, a rise of creatinine value greater than 25% above baseline or according to the kidney disease improving global outcomes

(KDIGO) criteria. Preoperative anemia has been recognized as an independent predictor of deterioration in renal function after a variety of non-cardiac surgeries including open elective aortic repair [7, 8]. Reduced hematocrit (HCT) has also been noted as an independent risk factor for AKI in patients with acute myocardial infarction (AMI) [9]. After off pump coronary artery bypass graft surgery, anemia was likewise associated with AKI. Every 1% increase in postoperative nadir HCT was associated with a 23% reduction in the risk of severe AKI [10]. A machine learning study found that both preoperative anemia and the number of packed red cells transfused were the most significant predictors of developing AKI after cardiac surgery [11].

Transfusions of platelets and fresh frozen plasma have also been associated with AKI. These blood components are often administered prophylactically to correct abnormal preoperative coagulation tests. Occasionally these blood components are also given to counteract the effect of

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chronic anticoagulant medications used to prevent vascular ischemic and thromboembolic events. While intraoperative administration of blood products has been definitively associated with AKI, it is currently not clear whether this relationship is serendipitous or if there is a solid cause-and-effect relationship underlying this association.

Preoperative anemia likely represents a marker for significant preexisting renal damage as the failing kidneys produce insufficient erythropoietin to maintain the HCT. Major intraoperative blood loss that requires transfusion is often associated with hypovolemia from inadequate replacement of intravascular fluid volume and excessive use of vasopressor medications. These associated factors rather than the transfusion may be responsible for the observed increase in AKI. Limiting red cell transfusion during cardiac surgery did not reduce the incidence of AKI in multiple studies [12, 13]. This finding suggests that AKI is caused by the other associated factors rather than the transfusion of blood products. Further clinical studies are needed however to clarify the nature of the relationship between the administration of blood products and postoperative AKI definitively.

AKI and Transfusion with Different Blood Components

Association of Red Cell Transfusion with AKI: Recent studies in patients undergoing percutaneous coronary revascularization have noted a solid association between red cell transfusion and AKI [4]. Intraoperative red cell transfusion was also a predictor of AKI following surgical aortic valve replacement [5]. Administration of packed cells in a prospective study in pericardiectomy patients was also noted to be an independent risk factor for AKI [6]. A cohort study of 565 coronary bypass patients confirmed the association of AKI with both intraoperative and postoperative administration of packed cells [14]. A recent metanalysis on the other hand found high-quality evidence that a restrictive transfusion strategy does not prevent AKI [15].

Association of Fresh Frozen Plasma with AKI: In a recent study on treatment of coagulopathy in cardiac surgical patients, AKI was more strongly associated with transfusion of FFP than with transfusion of platelets [16]. A study in liver transplant patients also noted an increase in postop AKI with administration of FFP [17]. The odds ratio was 1.34 for every 1000 ml of FFP transfused. This study confirmed the association between FFP and AKI in two previous studies in liver transplant patients [18, 19]. A study in patients with aortic dissection undergoing total aortic arch replacement did however fail to find an association between FFP and AKI [20].

Association of Platelet Transfusion with AKI: Platelets have been postulated to play a role in AKI by releasing a broad spectrum of bioactive mediators that modulate inflammation [21]. A drop in platelet count has been identified as a predictor of AKI and poor outcome [22–24]. Patients with AKI are more likely to bleed even when their platelet function is not found to be diminished on aggregometry [23]. Increased platelet to red cell transfusion ratio has been associated with AKI in children [25]. In children undergoing Fontan surgery platelet transfusion was also independently associated with increased risk of severe postoperative AKI [26].

Confounding Factors that May be Responsible for the Association

Pre-existing Factors: Preoperative anemia and thrombocytopenia are clearly linked to AKI. Pre-existing coagulopathy has also been associated with AKI in several settings. The important role of thrombo-inflammation in AKI pathogenesis has been noted especially in patients with sepsis [27]. Acute traumatic coagulopathy has also been found to be independently associated with AKI [28]. AKI has also been associated with elevated International Normalized Ratio (INR) and warfarin use [29]. Given the known association of pre-existing anemia, thrombocytopenia and coagulopathy with AKI it is likely that the patients who received blood products had a significantly higher pre-existing risk for AKI than the non-transfused cohort. Preoperative anemia may simply be a marker for preexisting renal dysfunction and preoperative thrombocytopenia could represent a marker for preexisting severe sepsis.

Co-existing Intraoperative Factors: Most of the evidence linking the administration of blood products to AKI comes from retrospective database analysis. Patients who received blood products are more likely to have suffered significant blood loss than patients who were not transfused. During acute and possibly unexpected major blood loss, intravascular fluid replacement may be inadequate because large bore intravenous access may be lacking [30]. AKI in the transfused patient cohorts therefore could have been caused by insufficient fluid replacement and excessive use of vasopressor medications. Administration of blood products therefore could simply represent a marker for the severity of hemorrhage rather than constitute the cause of AKI. Patients receiving blood products during the procedure are also more likely to suffer episodes of hypotension. Intraoperative hypotension is another variable that has been clearly associated with AKI even when it was only transient [31]. Hypotension in patients receiving packed cells or FFP may be due to ionized hypocalcemia from citrate in the storage

bags. It is therefore important to administer calcium when these products are transfused quickly to prevent hypotension. In addition, procedures requiring transfusion tend to be more complicated and require longer operating times because the surgeons encounter difficulty controlling the hemorrhage. This increased duration of surgery can necessitate increased exposure to intravenous contrast agents, antibiotics and anesthesia drugs as well as lead to postoperative wound infection. All these are contributing factors to the multifactorial origin of postoperative AKI (Table 1).

Interpretation of the Current Evidence

Most of the evidence linking the administration of blood products to AKI comes from analysis of retrospective databases. AKI in anemic patients could be the result of reduced oxygen delivery to the kidneys. A more likely explanation is that preoperative anemia resulted from reduced erythropoietin production by kidneys with preexisting functional impairment. The low HCT may in fact represent a marker for the degree of pre-existing renal impairment. A clear association of reduced creatinine clearance below 60 ml/min/1.73m² and anemia has been demonstrated in the literature [32]. Anemia is more common in patients with preexisting renal impairment. Postoperative AKI may therefore be the result of a preexisting renal lesion rather than the consequence of transfusion with blood products. Consequently, the association between administering blood products and AKI may be serendipitous rather than causal. Other confounding factors during the procedure may also explain the association. Blood loss, hypovolemia and excess use of vasopressors often accompany blood transfusion in the operating room. Patients with bleeding complications are frequently subjected to longer time under anesthesia which is necessary for surgeons to control the hemorrhage. Patients who suffer hemorrhage are also more likely to experience periods of transient intraoperative hypotension and low cardiac output. Longer procedures expose the patients to more intravenous contrast agent, more vasopressor drugs and

more antibiotics. The evidence extends to non-operating room locations. A retrospective study in patients undergoing coronary interventions also identified bleeding as an independent risk factor for AKI [4]. The severity of bleeding in this study correlated closely with the incidence of AKI.

The potential mechanisms for transfusion producing AKI have not yet been clearly identified. Inflammatory response, oxidative stress and impaired oxygen delivery have been postulated as potential etiological factors. After storage red cells display reduced deformability, increased aggregability and diminished ability for microvascular autoregulation. All these factors contribute to tissue hypoxia [33]. Reduced levels of 2,3 DPG and increased levels of free radicals in packed cells may also contribute [34]. Despite all these postulated mechanisms for transfusion causing AKI, multiple prospective trials have shown however that restricting red cell transfusion does not reduce the incidence of AKI [12, 15]. Current evidence therefore supports the notion that the increase in AKI in transfused patients is due to the associated factors that are unrelated to the transfusion rather than due to the transfusion of blood products itself.

Strategies for Reducing Blood Transfusion Associated AKI

Identify and Treat Iron Deficiency: Iron deficiency anemia should be identified in the preoperative clinic, and treatment should be initiated in a timely manner to raise the HCT towards normal levels prior to the date of surgery. The target HCT should be 30% for both men and women [35]. Many patients will not respond to oral iron therapy and intravenous administration of iron has been shown to be very effective [36, 37]. Preoperative treatment with intravenous iron has been associated with a reduction in 30-day postoperative mortality and morbidity, a higher postoperative hemoglobin level and reduced postoperative red blood cell transfusion [38].

Erythropoietin: Preoperative erythropoietin has been shown to be effective at reducing allogeneic blood transfusion in primary hip arthroplasty [39]. Two recent meta-analyses concluded that preoperative erythropoietin could reduce the incidence of AKI in surgical patients [40, 41]. Erythropoietin needs to be administered preoperatively however to be effective. Intraoperative and postoperative administration was not effective at preventing AKI.

Judicious Management of Abnormal Preoperative Coagulation Tests: Abnormal coagulation studies in cirrhotic patients do not necessarily reflect a state of auto-anticoagulation. Cirrhotic patients are in fact subject to a paradoxically higher incidence of venous thromboembolism than patients without cirrhosis [42]. Traditional coagulation tests do not

Table 1 Confounding factors responsible for increased AKI with transfusion

Preoperative anemia
Thrombocytopenia from sepsis
Pre-existing coagulopathy or use of warfarin
Intraoperative hypovolemia in patients with acute blood loss
Excessive use of vasopressors when large bore intravenous access is lacking
Intraoperative hypotension due to hypovolemia or ionized hypocalcemia
Increased duration of surgery with increased exposure to contrast agents, antibiotics and anesthetic drugs

adequately reflect hemostasis in patients with cirrhosis. Thrombin generation assays and viscoelastic tests offer better insights into coagulation in this condition. Preoperative elevation of INR and prothrombin time in cirrhotic patients therefore should not be corrected thoughtlessly with FFP. A more prudent approach may be to wait and see if there is clinical evidence of coagulopathy during the procedure. Immediate higher-dose prothrombin complex concentrate (PCC) has been shown to be a viable alternative to fresh frozen plasma without producing an increase in AKI [43]. Unlike FFP, PCC and fibrinogen complex concentrates are not associated with the potential risk of inadvertent ABO mismatch which can lead to hemolytic transfusion reaction with severe AKI.

Reduced platelet count in patients with cirrhosis also was not associated with bleeding in patients undergoing surgical treatment for liver cancer [44, 45]. It may therefore be prudent to reserve platelet transfusion for patient who show clinical evidence of coagulopathy during the operation.

Limiting Blood Loss: Tranexamic acid and use of point of care viscoelastic testing has been advocated to limit blood loss in orthopedic trauma [46]. Topical thrombotic agents and cell salvage has been shown to be effective in complex spine surgery [47]. It is also very important to maintain body temperature to prevent coagulopathy [48].

Preventing Transfusion of Incompatible Blood Components: Transfusion of incompatible blood components remains an ever-present risk. This must be avoided at all costs because inadvertent administration of ABO incompatible blood products leads to hemolysis and severe AKI. New onset hemoglobinuria may be the sole clue that the transfused blood products were ABO incompatible [49]. A solid safety system beginning from the collection of the cross-match sample extending to the blood bank processing and terminating with the administration of the blood product is indispensable [50, 51].

General Strategies for Preventing AKI

Maintain Cardiac Output and Renal Perfusion: Cardiac output should be maintained under anesthesia and blood loss needs to be adequately replaced with intravenous fluid because excessive use of inotropes has been associated with AKI [52].

Goal Directed Fluid Therapy: Dynamic measures of intravascular volume like stroke volume variation and pulse pressure variation have been recommended to reduce the incidence of AKI and to manage the patient with AKI [53]. In general goal-directed fluid management protocols have reduced the overall incidence of AKI [54]. Two recent meta-analyses did however conclude that early goal-directed

Table 2 General strategies for reducing AKI

Identify and treat iron deficiency

Erythropoietin
Judicious management of abnormal preoperative coagulation tests
Limit blood loss
Prevent transfusion of incompatible blood components
Maintain cardiac output and renal perfusion
Goal-directed fluid therapy
Prevent hypotension
Limit the use of nephrotoxic medications
Dexmedetomidine
Reduce time on cardiopulmonary bypass

therapy focused on fluid-based stroke volume optimization did not reduce the incidence of postoperative AKI [55, 56].

Prevent Hypotension: Mean arterial pressure should be maintained above 65mmHg. Even a short period of only 10 min of mean blood pressure below this level after cardiopulmonary bypass was associated with new onset requirement for dialysis [57].

Limit the Use of Nephrotoxic Medications: Perioperative medications associated with nephrotoxicity include intravascular contrast agents, antibiotics, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, non-steroidal anti-inflammatory drugs (NSAIDs) and diuretics. These drugs should be used with extreme care and only when necessary [58].

Dexmedetomidine: Dexmedetomidine has been repeatedly shown to reduce AKI after cardiac surgery [59–62]. It is associated with lower neutrophil gelatinase-associated lipoprotein (NGAL) levels and increased creatinine clearance. The mechanism for the beneficial effect is thought to be mediated by reducing oxidative stress and inflammation [63].

Reduce Time on Cardiopulmonary Bypass: Increased time on cardiopulmonary bypass has been associated with AKI [64, 65]. The odds ratio for AKI per hour on cardiopulmonary bypass was 1.32 [66]. A recent observational study recommended that cardiopulmonary bypass time should be kept short to reduce the risk of AKI [67] (Table 2).

Salient Point

Restrictive Transfusion Strategy did not Reduce AKI: Even though transfusion of blood products is associated with AKI in retrospective studies, a restrictive transfusion strategy with a threshold of less than 7.5 g/dl when compared to a threshold of 9.5 was not associated with a lower rate of AKI in prospective trials. The decision to use a restrictive versus a liberal perioperative transfusion strategy for red cells therefore should not be based on an attempt at renal protection [13,68]. There are many beneficial effects of restrictive transfusion protocols but reducing AKI is not one of them.

Conclusions

The origin of postoperative AKI is multifactorial. Factors affecting the incidence of AKI include pre-existing renal dysfunction, preoperative dehydration, inadequate intravascular volume replacement, intravenous contrast agents, use of diuretics, low cardiac output, poor renal blood flow, excessive use of vasopressor drugs as well as long aortic cross clamp and cardiopulmonary bypass times. The association between administering blood products and postoperative AKI maybe purely coincidental rather than causal. The risk from reduced oxygen carrying capacity due to anemia and the risk of bleeding in patients with abnormal preoperative coagulation tests should be carefully balanced against all the risks of transfusing blood products including the potential risk of AKI. Current evidence suggests that blood products should be used with caution in patients at risk of AKI until further evidence becomes available.

Author Contributions CB and FT wrote the main manuscript, CB prepared the tables and all authors reviewed the manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

Competing interests The authors declare no competing interests.

Conflict of interest The authors have no conflict of interest to declare.

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