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Original Research Article

Clinical Implications of Blood Transfusion Based on Blood Hematocrit in Patients after Percutaneous Coronary Intervention

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Abstract:

Background: Percutaneous coronary intervention (PCI) is the standard therapy for patients with symptomatic coronary artery disease and hemodynamically significant coronary stenosis. The combined use of antiplatelet and antithrombin agents during and after PCI has significantly reduced the rate of stent thrombosis and recurrent ischemic events. These therapies, however, unavoidably increase the risk of bleeding, red blood cell (RBC) transfusion, and mortality after PCI**.**

Objective: The objective of the present study was to analyse how RBC transfusion affects the clinical outcomes of patients with hematocrit nadir within those parameters.

Methods: A cross-sectional study was conducted in the tertiary teaching hospital. A total of 356 consecutive patients who had undergone PCI from January 2010 to December 2010 were enrolled in the study. From this cohort, 60 patients who had presented with a nadir hematocrit level of 24% to 30% after PCI were identified, of whom 24 had received a transfusion and 26 had not. PCI was performed according to the guidelines at the time of the procedure.

Result: The mean age was nearly 63 years. Most of the patients had a history of systemic hypertension and around 50% had diabetes. Approximately 2/3 had a history of coronary artery revascularization– either coronary artery bypass grafting or PC. The baseline hematocrit was similar between the 2 groups; however, the nadir hematocrit was lower in the transfused patients than in those not transfused. Acute MI (33.3% vs. 11.5%) and cardiogenic shock (25% vs. 7.7%) as the initial clinical presentation were more frequent in the transfused patients than in the non-transfused patients. No difference was found between the 2 groups in the angiographic characteristics, except for the number of diseased vessels and the presence of left main disease, which were greater in the transfused patients.

Conclusion: From our study findings we do not support the routine use of transfusion in patients who present with a nadir hematocrit of 24% to 30% after PCI because transfusion has got no mortality benefits in this range of hematocrit.

Keywords: Blood transfusion, Haematocrit, Percutaneous Coronary Intervention.

Introduction

Blood transfusions are a relatively common occurrence after performing any percutaneous coronary intervention (PCI). In recent years, significant advances have been made in the diagnosis, risk stratification, and therapy of non–ST–segment elevation acute coronary syndromes (NSTE ACS). A key part of this management strategy involves combining multiple antiplatelet and anticoagulation agents along with invasive therapy in high-risk patients with NSTE ACS [1]. Percutaneous coronary intervention (PCI) is the most common coronary revascularization procedure performed worldwide, with an estimated 1.1 million PCI procedures performed annually in the United States alone. Most PCIs are performed using a femoral artery approach, with systemic anticoagulation. [2] Systemic antithrombotic and antiplatelet therapies also increase the risk of gastrointestinal, genitourinary, and intracranial bleeding. As a result, red blood cell (RBC) transfusion has been reported in up to 3 – 9% of cases. Consequently, tens of thousands of patients

undergo RBC transfusion every year after PCI. [3,4] The pathophysiology surrounding the relationship between blood transfusion and an increased risk of death remains unclear. In general, blood transfusions are associated with a small, though not insignificant, risk of viral transmission, bacterial contaminations, and haemolytic reactions. [5]

Materials and Methods: This cross-sectional study was conducted in a tertiary teaching hospital for a period of 6 months, which included 356 consecutive patients who had undergone PCI from January 2010 to December 2010, from this cohort, 60 patients who had presented with a nadir hematocrit level of 24% to 30% after PCI were identified, of whom 24 had received a transfusion and 26 had not. PCI was performed according to the guidelines at the time of the procedure. In all cases, the interventional strategy was at the discretion of the responsible physician. Intra-procedural anticoagulation was ensured using unfractionated heparin. Glycoprotein IIb/IIIa inhibitors were used at the operator's discretion. All patients received an aspirin loading dose of 325 mg and were encouraged to continue this regimen indefinitely. After a clopidogrel loading dose of 300 to 600 mg, additional antiplatelet therapy with a 75-mg clopidogrel maintenance dose was instituted in all patients, who were then advised to continue this regimen for equal to or greater than 1 year.

Results:

Table 1:

Clinical characteristics	**Blood Transfusion**		**P-Value**
	YES(n=24)	**NO(n=26)**	
Age (yr)	63.3	63.6	
Male, n (%)	9(37.5%)	10(38.5%)	0.944
Diabetes, n (%)	11(45.8%)	13(50%)	0.768
Hypertension, n (%)	21(87.5%)	22(84.6%)	0.769
Current smoking, n (%)	5(20.8%)	3(11.5%)	0.370
Hypercholesterolemia, n (%)	18(75%)	22(84.6%)	0.396
Family history of CAD, n (%)	9(37.5%)	11(42.3%)	0.729
Major bleeding, n (%)	5(20.8%)	1(3.8%)	**0.03**
History of CABG or PCI, n (%)	15(62.5%)	16(61.5%)	0.944
Laboratory characteristics			
Baseline haematocrit	35.2	34.5	
Nadir hematocrit	26.1	28.9	
Mean hematocrit drop	9.1	5.6	**0.01**
Baseline creatinine	1.4	1.3	0.8
Indication for PCI			
Acute MI, n (%)	8(33.3%)	3(11.5%)	**0.03**
Cardiogenic shock, n (%)	6(25%)	2(7.7%)	**0.04**
Unstable angina, n (%)	6(25%)	9(34.6%)	0.459
Stable angina, n (%)	10(41.7%)	13(50%)	0.104

The above table depicts the mean age group was 63.3 of them 11(45.8%) were diabetic and 21 (87.5%) were hypertensive. of patients who had acute MI, 8(33.3%) have undergone blood transfusion. (Table 1)

Table 2: Angiographic and procedural characteristics

Angiographic characteristics	**Blood Transfusion**		**P-Value**
	Yes (n=24)	**No (n=26)**	
Target vessel, n (%)			
LM, n (%)	2(8.3%)	1(3.8%)	0.552
LAD, n (%)	8(33.3%)	7(26.9%)	0.621
LCx, n (%)	5(20.8%)	6(23.1%)	0.848
RCA, n (%)	7(29.2%)	8(30.8%)	0.902
Type C lesion, n (%)	6(25%)	6(23.1%)	0.884
DES, n	18(75%)	19(73.1%)	0.877
Angiographic success	23(95.8%)	25(96.2%)	0.298

Angiographic success in blood transfusion patients was 23 (95.8%) Table 2.

Table 3: Angiographic and procedural characteristics

Procedural characteristics	Blood Transfusion		P-Value
	Yes (N=24)	No(N=26)	
Heparin use, n (%)	24(100.0%)	26(100.0%)	1.000
GP IIb/IIIa inhibitors, n (%)	5(20.8%)	3(11.5%)	0.370

There is 100% use of heparin while doing blood transfusion and 20.8% of GPIIb/ IIIa inhibitors. (Table 3)

Table 4: 30-day composite end point of death and M

	Death and MI	P value
Transfused n(7)	**12.5%**	**<0.001**
Nontransfused n(1)	**19.2%**	

Table 5: 1-year composite end point of death and MI

	Death and MI	P value
Transfused n(3)	**29.1%**	**<0.014**
Nontransfused n(1)	**3.8%**	

The 1-year composite outcome of death and MI was 29.1% for the transfused patients and 19.2% (N=7 vs N=1) for the nontransfused patients (p <0.014), also primarily driven by mortality (26.5% vs 16.5%, respectively; p < 0.011). Transfusion itself was no longer an independent predictor for the composite primary endpoint at 30 days (hazard ratio 1.3, 95% confidence interval 0.8 to 2.5, p = 0.3) and 1 year (hazard ratio 1.5, 95% confidence interval 0.9 to 2.0, p =0.2) of follow-up. The rate of the composite end point of death and MI was significantly greater in the patients who received a RBC transfusion. The 30-day composite end point of death and MI was 12.5% for the transfused patients and 3.8% (N=3 vs N=1) for the nontransfused patients (p <0.001) and was mostly driven by mortality (14.3% vs 5.9%, respectively; p <0.001). (Table 4 & 5)

Table 6: Predictor for the composite primary endpoint

	Hazard Ratio	P value
30 days	**hazard ratio 1.3, 95% ,confidence interval 0.8 to 2.5**	**0.3**
1 year	**hazard ratio 1.5, 95%, confidence interval 0.9 to 2.0**, p =	**0.2**

Discussion:

Our study findings show that RBC transfusion does not improve the 30-day and 1-year outcomes when used to treat patients with a hematocrit nadir of 24% to 30% after PCI. The high-risk population presented with a high mortality rate at 30 days (14.3%) and 1 year (26.5%), explained primarily by the high prevalence of co-morbidities and the frequent presentation with cardiogenic shock. This poor outcome was not improved by RBC transfusion. Our results are in accordance with 2 registries that analysed the effect of RBC transfusion in the setting of the acute coronary syndrome. Rao et al, [6] published the results of 2,401 transfused patients with acute coronary syndrome during hospitalization and showed that RBC transfusion increased the 30-day mortality when administered to patients with a nadir hematocrit level >25%. Alexander et al, [7] using data from the Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes with Early Implementation of the ACC/AHA Guidelines (CRUSADE) study (44,242 patients with non–ST-elevation acute coronary syndrome), described the effect of RBC transfusion as a function of nadir hematocrit in 4,610 transfused patients. They reported that transfusion had a neutral effect in the nadir hematocrit range of 24% to 30%, with a beneficial trend when the nadir hematocrit was </=24%. Furthermore, the only randomized trial that compared liberal and restrictive RBC transfusion strategies showed a trend toward better 30-day mortality for critically ill patients using the restrictive strategy (hemoglobin <7 g/dl). [8]

Our observed results also agree with the described relation between anemia and cardiovascular physiology. It is known that with moderate anemia (hemoglobin 7.0 to 10 g/dl), a compensatory increase occurs in cardiac output, which could be explained by the reduction in blood viscosity that leads to a decrease in afterload without any change in myocardial oxygen consumption. [9] Additionally, under physiologic conditions, an isolated reduction in the haemoglobin concentration will not necessarily affect oxygen delivery and consumption until a critical decrease has occurred in the hemoglobin level. [10] Casutt et al observed that in cardiovascular surgery patients, RBC transfusion did not generally enhance oxygen delivery and thus consumption by the myocardium, independent of the pretransfusion hemoglobin

concentration. [11] This observation suggests that myocardial oxygen consumption is only limited at severe levels of anemia. In contrast, the potential benefit of transfusion must be balanced against the risk of infection, immune transfusion reactions, blood volume overexpansion, and afterload increases. [12] Our study was not without limitations, consists of a small study population, and requires further large randomized trials to look into the matter.

Conclusion:

Our study does not support the routine use of transfusion in patients who present with a nadir hematocrit of 24% to 30% after PCI because transfusion has got no mortality benefits in this range of hematocrit. Blood transfusion in the setting of acute coronary syndromes is associated with higher mortality, and this relationship persists after adjustment for other predictive factors and timing of events.

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