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ORIGINAL ARTICLE

Analysis of Blood Group Antigens as Risk Factors for Thrombosis in Polycythemia Vera Patients

Jay Ho Han [1], Hyung Suk Cho [3], Mi Jung Hwang [3], Taewon Kang [2], Howon Lee [4], Kwangsang Koh [2], Sung-Eun Lee [5], Hee-Je Kim [5], Dong Wook Jekarl [2, 6], Myungshin Kim [2], Yonggoo Kim [2]

[1] *Department of Laboratory Medicine, Uijeongbu St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Gyeonggi-do, Korea*
[2] *Department of Laboratory Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea*
[3] *Department of Laboratory Medicine, Apheresis Unit, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea*
[4] *Department of Laboratory Medicine, Yeouido St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea*
[5] *Department of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Korea*
[6] *Research and Development Institute for In Vitro Diagnostic Medical Devices, College of Medicine, The Catholic University of Korea, Seoul, Korea*

SUMMARY

Background: **Polycythemia vera (PV) is characterized by the abnormal proliferation of red blood cells (RBCs). Thrombosis and associated cardiovascular diseases are leading causes of mortality in patients with PV. This study aimed to investigate the association of Lutheran/BCAM (CD239) and other RBC antigens with thrombosis in PV.**
Materials and Methods: **This single-center, prospective study consecutively enrolled 50 PV patients, 39 with secondary polycythemia, and 20 healthy controls (HC) who visited the apheresis unit for phlebotomy between May 2022 and September 2023. The normalized expression levels of Lutheran/BCAM (CD239), Indian (CD44), LW/ICAM (CD242), and Rh-related integrin-associated protein (IAP, CD47) antigens were assessed by flow cytometry. *JAK2*V617F expression was quantified and coagulation parameters were analyzed. Laboratory and clinical data were retrieved from the medical records.**
Results: **PV patients exhibited significantly higher mean fluorescence intensity (MFI) for Lutheran/BCAM: 45.2 ± 32.8 vs. 33.0 ± 14.4, p = 0.047, Indian (CD44): 13.5 ± 18.4 vs. 8.6 ± 1.1, p = 0.195, and IAP (CD47): 604.8 ± 193.2 vs. 514.9 ± 63.2, p = 0.036 compared to HC. The Indian (CD44) antigen was identified as a risk factor for thrombosis with an odds ratio (OR) of 1.359 (95% confidence interval [CI]: 1.003 - 1.842) in a multivariable model. Positive *JAK2* measurable residual disease (*JAK2*-MRD) (expression was detected in 100% (25/25) of PV patients assessed, with a median variant allele frequency of 51.8% (95% CI: 45.4 - 65.0%).**
Conclusions: **Higher expression of Indian (CD44) MFI levels in RBCs were associated with thrombotic events in patients with PV. Assessing RBC Indian (CD44) expression may serve as a potential biomarker for thrombotic risk stratification and prevention in PV.**
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Correspondence:
Dong Wook Jekarl
Department of Laboratory Medicine
Seoul St. Mary's Hospital, College of Medicine
The Catholic University of Korea
222 Banpo-daero, Seocho-gu
Seoul, 06591
Republic of Korea
Phone: +82 2 22581643
Fax: +82 2 22581719
Email: bonokarl@catholic.ac.kr

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KEYWORDS

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INTRODUCTION

Polycythemia vera (PV) is a chronic myeloproliferative neoplasm characterized by abnormal proliferation of red blood cells (RBCs) due to the presence of the *JAK*2-V617F mutation in the intracellular portion of the eryth-

ropoietin (EPO) receptor [1-3]. Thrombosis is a major complication of PV and reduces patient survival [4]. The pathologic generation of thrombosis is characterized by microcirculatory disturbances [5,6] and may manifest as or result in either arterial or venous thrombotic events. Previous studies have examined both conventional and non-conventional risk factors for thrombosis [7-10]. Conventional risk factors for thrombosis include erythrocytosis and qualitative changes in RBCs, leukocytosis, and other variables, such as thrombotic event history, age, tobacco use, and comorbidity. Non-conventional risk factors include *JAK*2 allele burden, systemic inflammation, blood cell activation, serum microparticles, and coagulation system activation. Qualitative changes in RBCs, such as altered expression of adhesion molecules, may serve as conventional risk factors that influence thrombotic risk. Several known RBC adhesion molecules play a role in thrombosis, including Indian (CD44), Lutheran/BCAM (CD239), LW (CD-242), and Rh-related integrin-associated protein (Rh-related IAP) (CD47) [11,12]. Among these, Lutheran/BCAM (CD239) has been extensively studied for its role in various diseases [13,14]. Lutheran/BCAM (CD-239) phosphorylation, induced by *JAK*2V617F mutation, is known to increase RBC adhesion by forging a connection to laminin α5, which is present in the subendothelium and the apical surface of endothelial cells [15]. Despite evidence suggesting a role for RBC adhesion molecules in thrombosis, the roles of specific molecules such as Indian (CD44), LW/ICAM (CD242), and Rh-related IAP (CD47) in PV remain unexplored. This study aimed to analyze the expression of RBC adhesion molecules and other related factors to identify their potential roles as plausible causes or modifiers of thrombosis in patients with PV.

MATERIALS AND METHODS

Subjects

This single-center prospective study enrolled 50 patients with polycythemia vera (PV), 39 patients with secondary polycythemia, and 20 healthy controls (HCs). Participants were recruited among individuals visiting the apheresis unit for phlebotomy at Seoul St. Mary's Hospital between May 2022 and September 2023. Blood samples were obtained during the phlebotomy visits. The baseline demographic and clinical characteristics of the study population are summarized in Table 1 and Supplementary Table 1. The diagnosis of PV was made according to the World Health Organization (WHO) 2021 classification criteria, and all PV patients were confirmed to carry the *JAK2*V617F mutation [16]. This study was approved by the Institutional Review Board of Seoul St. Mary's Hospital, Seoul, South Korea (KC-22TISI0257).

Flow cytometry for analyzing expression of blood group antigens

Peripheral blood samples (10 μL) were diluted with 3 mL 0.9% normal saline (Dai Han Pharm. Co., Ltd., Seoul, Korea). A 50 μL aliquot of the diluted blood was incubated with 5 μL of fluorescently labeled monoclonal antibodies in separate tubes, including the following: Lutheran/BCAM (CD239; BV421 mouse anti-human BCAM, catalog no. 748007), Indian (CD44; FITC mouse anti-human CD44, catalog no. 555478), LW/ICAM (CD242; BV650 mouse anti-human ICAM-4, catalog no. 748056), and Rh-related IAP (CD47; PE mouse anti-human CD47, catalog no. 568089). Each tube was incubated for 60 minutes at room temperature, followed by four washes with 0.9% normal saline. After the final wash, 1 mL of 0.9% normal saline was added to each tube and the samples were centrifuged at 400 x g for 5 minutes. The supernatant was discarded and 200 μL of 0.9% normal saline was added to each tube for analysis. Flow cytometry analysis was performed using an LSRFortessa™ flow cytometer (Beckton-Dickinson, USA). The mean fluorescence intensity (MFI) values were normalized by dividing the MFI of each test sample by the MFI of the control, transforming the values to a common scale.

Molecular studies *(JAK2*V617F mutation, JAK2 measurable residual disease, whole-exome sequencing)

The *JAK*2V617F mutation was identified using a *JAK*2V617F kit (Biosewoom, Seoul, Korea), and real-time polymerase chain reaction (PCR) was performed using an ABI 7500 (Life Technology, Gaithersburg, MD, USA). Genomic DNA was analyzed for JAK2 measurable residual disease (*JAK2*-MRD) using the JAK2MutaQuant Kit (ipsogen, Luminy Biotech, France), following the manufacturer's protocol. Real-time PCR was performed using a LightCycler 480 instrument (Roche Diagnostics Corp., IN, USA). The variant allele frequency (VAF), expressed as the percentage of *JAK2*V617F copy numbers relative to the total JAK2 copy numbers (*JAK2*V617F and wild-type), was used to quantify *JAK2*-MRD [17]. Whole-exome sequencing (WES) was performed in a commercial laboratory to detect the Indian (CD44) mutation. The Human Core Exome kit (Twist Bioscience, San Francisco, CA, USA) was used for target capture, and sequencing was performed using NovaSeq X Plus (Illumina, San Diego, CA, USA). Data were processed as described previously [18]. The WES data quality acceptance metrics included a mean depth of coverage of > 50 x, with > 97% regions at 20 x.

Data analysis

Continuous variables were presented as the median (range) and compared using the Mann-Whitney U test. An analysis of variance (ANOVA) was performed for the three groups (PV patients, secondary polycythemia patients, and healthy controls [HC]), followed by post

hoc Tukey's tests (Supplementary Table 2). Univariable and multivariable logistic regression models were constructed to predict thrombotic events in the PV and secondary polycythemia groups. All analyses were performed using R [19]. Between-group differences were considered statistically significant at $p < 0.05$.

RESULTS

Flow cytometry

The normalized MFI values for Lutheran/BCAM (CD-239) and Rh-related IAP (CD47) were significantly higher in PV patients than in HC (45.2 ± 32.8 vs. 33.0 ± 14.4; $p = 0.046$ and 604.8 ± 193.2 vs. 514.9 ± 63.2; $p = 0.023$, respectively). The MFI values for Indian (CD44) were also higher in PV patients than in HC; this result did not reach statistical significance ($p = 0.120$; Table 2). In contrast, MFI values for LW/ICAM (CD242) were higher in the HC than in the PV patients. The groups with and without thrombosis showed a statistically significant difference in the MFI values of Indian (CD44) and Lutheran/BCAM (CD239), respectively ($p = 0.059$, 0.013; Table 3).

Molecular studies

All patients with PV (25/25) were positive for *JAK*2-MRD, with a median VAF of 51.8% (95% CI: 45.4 - 65.0%). In contrast, *JAK2*-MRD in secondary polycythemia patients and healthy controls (HCs) was below the limit of detection (0.061%; Table 2)**.** When comparing patients with thrombosis to those without thrombosis, the thrombosis group exhibited higher VAF values; however, the correlation was not statistically significant ($p = 0.406$). WES was performed in 12 patients (eight PV patients and four secondary polycythemia patients) to identify mutations in the *CD44* gene. The detected variants are summarized in Supplementary Table 3. The single nucleotide variant (SNV) filtration process followed a phenotype-driven strategy. However, thrombosis-related mutations were not detected.

Univariable and multivariable regression model

Univariable and multivariable logistic regression models were constructed to evaluate factors associated with thrombotic events. Table 4 highlights the statistically significant factors and variables of interest. Among patients with PV, Indian (CD44) was a statistically significant factor distinguishing thrombotic from non-thrombotic groups. In patients with secondary polycythemia, age and Lutheran/BCAM (CD239) were significantly associated with thrombotic events in the univariable and multivariable models (Table 4).

DISCUSSION

RBC antigens perform various functions, including maintaining membrane structural integrity, facilitating molecular transport, and serving as receptors for extracellular ligands, adhesion molecules, complement components and regulators [20]. As adhesion molecules, many RBC antigens have the potential to influence both normal human physiology and disease development [21]. Despite their biological importance, studies on RBC antigens remain limited, except for research on Lutheran/BCAM (CD239) [15,22-24]. In this study, we primarily investigated blood group antigens that may contribute to thrombotic events in patients with PV. Utilizing Pearson correlation coefficients (Supplementary Table 4), we aimed to elucidate the contribution of these antigens to the pathophysiology of thrombosis in PV. Indian (CD44) functions as a multifunctional cell-surface receptor by mediating cell adhesion and migration in a variety of physiological and pathological processes. In this study, Indian (CD44) showed a statistically significant association with thrombosis in patients with PV using multivariable and univariable regression models. The odds ratio (OR) was 1.359 (95% CI, 1.003 - 1.842) in the quantitative analysis**.** When analyzed qualitatively using an optical threshold derived from a receiver operating characteristic (ROC) curve, the OR was 10.500 (95% CI, 1.765 - 62.439). For qualitative analysis, MFI values were categorized into high and low groups based on the ROC-derived threshold, with large MFI values set to "1" and small values set to "0". These results suggest that high expression levels of Indian (CD44) increase the risk of thrombosis. Specifically, quantitative analysis revealed that the risk of thrombosis increased 1.359 times with high expression, while qualitative analysis showed an even greater increase of 10.500 times. Although the function of Indian (CD44) in RBCs is not yet fully understood [25], our findings suggest that Indian (CD44) may act as an adhesion molecule. Previous studies are likely to provide supporting evidence by demonstrating an interaction between the Indian (CD44) isoforms and hyaluronan [26-29]. These studies highlight the presence of Indian (CD44) isoforms generated through alternative splicing, which are expressed on transformed epithelial cells. These isoforms have the capacity to bind hyaluronan [30-33], and we hypothesize that this interaction enhances RBC adhesion. To verify the presence of CD44 isoforms, WES was performed to investigate the association between Indian (CD44) and thrombosis in patients with PV. Among the 12 patients (8 PV patients and 4 secondary patients), two missense mutations were identified within exons. However, based on the NCBI SNP database, no association with thrombosis was found for the variants rs9666607 and rs1467558 (Supplementary Table 3). To detect Indian (CD44) variant isoforms, a larger number of samples needs to be analyzed. This study had several limitations. First, the number of participants was relatively small. Specifically, *JAK2*-MRD and WES ana-

Table 1. Characteristics of the polycythemia vera study patients and controls.

	PV patients (n = 50)	Secondary polycythemia patients (n = 37)	Controls (n = 20)	p-value
Gender, male/female	33/17	33/4	14/6	0.006 *
Age (years)	56.3 ± 13.1	50.7 ± 11.0	32.5 ± 4.2	< 0.001 *
Diagnosis				
PV with genetic mutation	50	0	0	-
Secondary polycythemia	0	37	0	-
Diagnosis to sampling (yr)	5.3 ± 4.6	-	-	
Palpable splenomegaly [a]	24 (68.5)	2 (5.4)	-	< 0.001 *
Comorbidities, n (%)				< 0.001 *
Hypertension	12 (24.0)	13 (35.1)	0 (0)	-
Metabolic disease	13 (26.0)	10 (27.0)	0 (0)	-
Others				-
DCMP	1 (2.8)	0 (0)	0 (0)	-
COPD	0 (0)	3 (8.1)	0 (0)	-
Asthma	0 (0)	1 (4.2)	0 (0)	-
Thrombosis, n (%)				0.009 *
Arterial	7 (14.0)	2 (2.2)	0 (0)	-
Venous	1 (2.0)	0 (0)	0 (0)	-
Therapy, n (%)				< 0.001 *
Phlebotomy	36 (100)	36 (97.2)	0 (0)	-
Medication (ASA, HU)	49 (98.0)	24 (64.8)	0 (0)	-
Medication (ASA, HU)	49 (98.0)	24 (64.8)	0 (0)	-

PV polycythemia vera, DM diabetes mellitus, DCMP dilated cardiomyopathy, COPD chronic obstructive pulmonary disease, ASA aspirin, HU hydroxyurea.
Values are expressed as the number of patients (n) or n (%) or the mean ± SD.
[a] Data available for 35 of 50 patients with PV and 23 of 37 patients with secondary polycythemia.
[*] Statistically significant (p < 0.005).

Table 2. Laboratory characteristics and statistics of polycythemia vera study patients and controls.

	PV patients (n = 50)	Secondary polycythemia patients (n = 37)	Controls (n = 20)	p-value
Hemoglobin (g/L)	154.6 ± 14.5	165.9 ± 17.7	141.7 ± 12.8	< 0.001 *
Hematocrit (%)	46.3 ± 4.5	49.1 ± 5.4	43.0 ± 3.7	< 0.001 *
WBC (10^9/L)	10.6 ± 3.5	7.8 ± 2.6	5.3 ± 1.1	< 0.001 *
Platelet counts (10^9/L)	417 ± 167	281 ± 169	247 ± 50	< 0.001 *
EPO (mIU/mL) [a]	2.9 ± 3.2	10.0 ± 7.5	NT	< 0.001 [b] *
JAK2-MRD (%)	51.8[c]	0 [d]	0 [e]	< 0.001 [f] *
Cytogenetics [g]				
Normal	34 (91.8)	32 (100)		0.251
Abnormal [h]	3 (8.1)	0 (0)		
RBC molecule (MFI) [i]				
Lutheran/BCAM (CD239)	45.2 ± 32.8	47.5 ± 24.4	33.0 ± 14.4	0.046 *
LW/ICAM (CD242)	3.5 ± 2.2	3.5 ± 1.0	4.2 ± 1.2	0.715

Table 2. Laboratory characteristics and statistics of polycythemia vera study patients and controls (continued).

	PV patients (n = 50)	Secondary polycythemia patients (n = 37)	Controls (n = 20)	p-value
Indian (CD44)	13.5 ± 18.4	10.2 ± 2.4	8.6 ± 1.1	0.120
Rh-related IAP (CD47)	604.8 ± 193.2	559.2 ± 131.4	514.9 ± 63.2	0.023 *
Coagulation factor				
PT-INR	1.0 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	< 0.001 *
aPTT (s)	28.1 ± 2.5	26.3 ± 2.2	27.5 ± 2.2	< 0.001 *
Fibrinogen (mg/dL)	264.3 ± 84.1	255.3 ± 52.5	269.3 ± 62.1	0.756
Antithrombin III (%)	102.6 ± 12.7	99.1 ± 14.4	103.7 ± 7.6	0.238
FDP (mcg/dL)	1.0 ± 1.3	1.0 ± 1.1	0.8 ± 0.5	0.333
Factor VIII (%)	61.9 ± 43.0	66.4 ± 50.7	78.6 ± 23.5	0.350
Protein S (%)	80.5 ± 30.0	101.9 ± 33.9	101.9 ± 27.4	< 0.001 *
Protein C (%)	95.2 ± 25.7	98.5 ± 19.2	103.1 ± 15.3	0.395
Lupus anticoagulant	1.0 ± 0.1	1.1 ± 0.1	1.1 ± 0.1	0.006 *

PV polycythemia vera, MRD minimal residual disease. Unless otherwise specified, all values are expressed as mean ± SD.
[a] Initial value when the patient was diagnosed.
[b f] Analyzed by the Mann-Whitney U test.
[c] 25 of 50 PV patients were tested.
[d] 9 of 37 secondary polycythemia patients were tested.
[e] 6 of 20 healthy controls were tested.
[g] Data available for 37 of 50 patients with PV and 32 of 37 patients with secondary polycythemia.
[h] 46,XX,der(20)add(20)(p13)add(20)(q13.1)[16]/46,idem,add(8)(p23)[2]/46,XX[2], 47,XY,+9[2] and 46,XY,t(5;8)(q31;q24.1)[6]/46,idem,der(18)t(9;18)(p13;p11.2)[14], no relationship to PV, known pathogenesis of PV is at chromosome band 9p24.1.
[i] Normalized MFI value.
* Statistically significant (p < 0.005).

Table 3. Demographics and laboratory characteristics and statistics of polycythemia vera study patients with and without thrombosis.

	Thrombosis (8/50)	Without thrombosis (42/50)	p-value
Gender, male/female	5/3	28/14	
Age (years)	65.5 ± 10.1	54.6 ± 13.1	0.041 *
Hemoglobin (g/L)	156.3 ± 12.8	154.6 ± 16.1	0.426
Hematocrit (%)	46.5 ± 4.6	46.3 ± 4.5	0.843
WBC (10^9/L)	8.7 ± 1.6	11.0 ± 4.4	0.294
Platelet counts (10^9/L)	320 ± 181	445 ± 161	0.028 *
EPO (mIU/mL) [a]	2.9 ± 3.8	2.8 ± 3.1	0.358
JAK2-MRD (%)	60.6 [b]	40.1 [c]	0.406
RBC molecule (MFI) [d]			
Lutheran/BCAM (CD239)	31.1 ± 15.1	54.2 ± 29.2	0.013 *
LW/ICAM (CD242)	5.2 ± 3.3	3.9 ± 1.3	0.519
Indian (CD44)	14.4 ± 4.2	11.2 ± 2.9	0.059
Rh-related IAP (CD47)	651.3 ± 240.0	620.4 ± 158.0	0.448
Coagulation factor			
PT-INR	1.0 ± 0.1	1.0 ± 0.1	0.585
aPTT (s)	28.5 ± 2.4	27.9 ± 2.5	0.862
Fibrinogen (mg/dL)	293.5 ± 137.4	258.7 ± 68.0	0.637

Table 3. Demographics and laboratory characteristics and statistics of polycythemia vera study patients with and without thrombosis (continued).

	Thrombosis (8/50)	Without thrombosis (42/50)	p-value
Antithrombin III (%)	96.7 ± 4.5	103.7 ± 13.5	0.534
FDP (mcg/dL)	1.0 ± 1.2	1.1 ± 1.3	0.999
Factor VIII (%)	52.4 ± 40.1	63.7 ± 43.2	0.283
Protein S (%)	78.0 ± 13.2	80.9 ± 32.2	0.637
Protein C (%)	90.5 ± 15.8	96.1 ± 27.1	0.843
Lupus anticoagulant	0.9 ± 0.1	1.1 ± 0.1	0.318

PV polycythemia vera, MRD, minimal residual disease.
Unless otherwise specified, all values are expressed as the mean ± SD.
[a] Initial value when the patient was diagnosed.
[b] 3 of 8 PV patients in thrombosis group were tested.
[c] 22 of 42 PV patients in the without-thrombosis group were tested.
[d] Normalized MFI value.
[*] Statistically significant ($p < 0.005$).

Table 4. Univariable followed by multivariable logistic regression analysis.

	Univariable				Multivariable			
Disease group	OR	LCI	UCI	p-value	OR	LCI	UCI	p-value
Polycythemia vera								
Age (years)				NS				NS
Platelet counts (10^9/L)				NS				NS
RBC molecule (MFI) [a] Lutheran/BCAM (CD239)	0.942	0.891	0.997	0.039				NS
LW/ICAM (CD242)				NS				NS
Indian (CD44) (threshold 9.12) [b]	1.367 10.500	1.037 1.765	1.502 62.439	0.027	1.359	1.003	1.842	0.048
Rh-related IAP (CD47)				NS				NS
Secondary polycythemia								
Age (years)	1.142	1.001	1.303	0.049	1.339	1.047	1.713	0.02
Platelet counts (10^9/L)				NS				NS
RBC molecule (MFI) [a] Lutheran/BCAM (CD239)	1.084	1.004	1.171	0.039	1.084	1.004	1.171	0.039
LW/ICAM (CD242)				NS				NS
Indian (CD44)				NS				NS
Rh-related IAP (CD47)				NS				NS

OR odds ratio, LCI lower 95% confidence interval, UCI upper 95% confidence interval, NS non-specific.
[a] Normalized MFI value.
[b] Threshold MFI value of normalized Indian (CD44) by ROC curve analysis.

lyses were performed in a limited number of patients, which may not provide sufficient data to explain thrombosis in patients with PV. Second, patients with PV were undergoing treatment with phlebotomy and medi - cations, which could have influenced the interpretation of the results. Third, additional functional studies are re-

quired to confirm the role of the blood group antigens in thrombosis. Although we hypothesized that CD44 (Indian) may contribute to thrombus formation via its isoforms, this requires experimental validation. Despite these limitations, our study suggests that Indian (CD44) may be a potential risk factor for thrombosis in patients with PV. Given that thrombotic events are the leading cause of mortality in PV [34], implementing effective risk stratification strategies is critical for the proper management of these patients. We analyzed the risk factors for thrombosis in patients with PV. The analysis indicated that the expression of RBC antigens was significantly different between the PV patients and the HC group. Correlation analysis identified higher Indian (CD 44) MFI levels as a significant risk factor for thrombosis.

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There is no sponsor involved in this study.

Ethical Consideration:
This study was approved by the Institutional Review Board of Seoul St. Mary's Hospital (KC22TISI0257), and informed consent was obtained from all the patients.

Declaration of Interest:
The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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