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ORIGINAL ARTICLE

Renal Dysfunction as a Predictor of Slow-Flow/No-Reflow Phenomenon and Impaired ST-Segment Resolution after Percutaneous Coronary Intervention in ST- Elevation Myocardial Infarction. A Retrospective Analysis

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ABSTRACT

Objective: The research aimed to examine the relationship between kidney dysfunction and the occurrence of slow blood flow or no-reflow phenomenon and insufficient ST-segment resolution after the percutaneous coronary intervention procedure in individuals suffering from ST-elevation myocardial infarction.

Methods: In a retrospective analysis conducted at Hayatabad Medical Complex Peshawar, for six months, 210 consecutive patients have undergone percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI). The term slow-flow/no-reflow referred to an angiogram result of TIMI Grade <3 after the implantation of a stent, despite a residual stenosis of less than 50%, and with no major damage or visible blood clots.

Results: The study included 210 patients, with 185 (88.0%) having normal flow and 25 (11.9%) having slow flow/no-reflow after PCI. Comparison between patients with normal flow and those with slow flow or no-reflow. Male sex was more common in the LVEF ≥50% group compared to the LVEF <50% group (84.2% vs. 74.0%, p=0.043).

Practical Implication: Overall, the practical implications of this study can positively impact the community by enhancing risk assessment, patient management, treatment approaches, communication, and future research in the context of renal dysfunction and PCI outcomes in STEMI patients.

Conclusion: The study found that patients with STEMI who have renal dysfunction are more likely to experience SFR and ISR after undergoing PCI. We should regard renal dysfunction as a significant factor that increases the risk of these complications. Treatment may need to be more intensive to achieve better results in these patients.

Keywords: Renal dysfunction, slow-flow/no-reflow phenomenon, inadequate ST-segment resolution,

INTRODUCTION

A leading cause of death worldwide, cardiovascular disease (CVD) is a common condition. China is experiencing an increase in CVD prevalence, according to recent reports.[1] Approximately three million deaths occur every year due to CVD in China, which accounts for 41% of all deaths in the country. China is estimated to have 230 million people with CVD.[2] Myocardial infarction mortality rates can be reduced through early diagnosis and effective treatment. It is essential to start coronary reperfusion therapy early in the treatment process.[2] From 2001 to 2011, more people in China went to the hospital with STEMI, but the death rate stayed the same. STEMI is the most serious form of Acute Coronary Syndrome.[3] Acute myocardial infarction (AMI) mortality rates are expected to reduce with direct percutaneous coronary intervention (PCI).[4] It is possible for the infarction-related artery (IRA) to remain blocked despite successful percutaneous coronary intervention (PCI) during ST-segment elevation myocardial infarction (STEMI).[5] The exact cause of no-reflow is currently unknown, but it may be related to capillary bed embolism, ischemic injury, endothelial dysfunction, oxygen free radical production, inflammation, stress, calcium overload, and other factors.[6] There are improvements in reperfusion techniques every year, but no-reflow can still cause poor outcomes.[7] The incidence of no-reflow after routine PCI ranges from 1% to 5%, and in AMI patients, it can be as high as 2.3% to 41%.[8–11] Achieving grade 3 thrombolysis in myocardial infarction (TIMI) is necessary for effective myocardial perfusion.[12] Early identification of no-reflow risk and active intervention can help prevent its occurrence. Several research studies have discovered a variety of factors that are linked to the development of no-reflow while treating AMI.[8,13] AMI patients with ST-elevation myocardial infarction (STEMI) are not differentiated from those without ST-elevation myocardial infarction (STEMI) in larger studies. The most significant risk factors associated with no-reflow in STEMI patients are a TIMI flow score of ≤1 and a heavy thrombus burden, according to a meta-analysis of studies.[14] STEMI patients who do not recover after PCI have been evaluated based on these factors and others.[15–17] However, the existing risk scoring systems have produced differing results, with some requiring costly medical equipment, being difficult to use, or causing delays in making predictions, making them unsatisfactory. The research aimed to examine the relationship between kidney dysfunction and the occurrence of slow blood flow or no-reflow phenomenon and insufficient ST-segment resolution after the percutaneous coronary intervention procedure in individuals suffering from ST-elevation myocardial infarction.

MATERIAL AND METHODS

The research carried out at the cardiology department focused on intervention at Hayatabad Medical Complex Peshawar. It retrospectively analyzed the medical files of 210 patients who had experienced ST-elevation myocardial infarction (STEMI) and underwent percutaneous coronary intervention (PCI). Patients who met certain requirements were eligible for the study. Specifically, they had to have ST-segment elevation that measured ≥0.1 mV in at least two limb leads or ≥0.2 mV in two contiguous precordial leads. In addition to these criteria, patients had to have received treatment within 12 hours of experiencing symptoms, have had a stent implanted, and have had a single new lesion that was identified as the culprit. However, certain groups of patients were excluded from the study. A list of individuals was included, including those who were receiving hemodialysis, those who underwent cardiopulmonary arrest upon entering the emergency department, people who received extracorporeal membrane oxygenation in the venous-arterial arteries, and those who had ischemic heart disease, left main artery issues, or collateral artery problems.

The term slow-flow/no-reflow referred to an angiogram result of TIMI Grade <3 after the implantation of a stent, despite a residual stenosis of less than 50%, and without any major damage or visible blood clots. This definition also included instances where blood flow temporarily worsened after the stent was implanted.

Before the patients participated in the study, they provided informed consent. The institutional review board had approved the study.

Statistical Analysis: SPSS Statistics 22.0 was used to analyze the data. For categorical variables, frequencies and percentages were used, while medians and interquartile ranges were calculated for continuous variables. Slow flow, no-reflow, and low STR were investigated by multivariate and univariate logistic regression. Significant results were determined by a p-value of ≤0.05.

RESULTS

The study included 210 patients, with 185 (88.0%) having normal flow and 25 (11.9%) having slow flow/no-reflow after PCI. Comparison between patients with normal flow and those with slow flow or no-reflow. The statistical significance (P value) is also provided for each variable.

In contrast to patients who have regular blood flow, those with slow flow or no reflow had a considerably higher average age (70 years vs 62 years), lower systolic blood pressure (120 mmHg vs 129 mmHg), higher diastolic blood pressure (71 mmHg vs 82 mmHg), and a greater percentage of systolic blood pressure less than or equal to 90 mmHg (20% vs 7%). They also had a higher proportion of NYHA classification III-IV (24% vs 9%) and a history of cerebrovascular disease (24% vs 5%).

Patients with an LVEF <50% were older than those with an LVEF ≥50% (mean age of 71 vs. 62 years, p<0.001).

Male sex was more common in the LVEF ≥50% group compared to the LVEF <50% group (84.2% vs. 74.0%, p=0.043).

There was no notable variation observed among the groups in terms of their systolic or diastolic blood pressure. The LVEF <50% group had a higher proportion of patients in NYHA classification II or higher (22.0% vs. 12.9%, p=0.06).

The two groups had similar body mass index (BMI), rates of hypertension, diabetes, cerebrovascular disease, and primary ventricular fibrillation.

Current smokers were more common in the LVEF ≥50% group compared to the LVEF <50% group (50.9% vs. 32.4%, p=0.01).

Both groups had significantly different medication profiles on admission, with a higher percentage of patients receiving ACEI (15.6%), ARB (4.3%), -blockers (25.9%), statins (11.8%), and insulin (2.1%). Procedural characteristics, including the use of IVUS, predilatation, post-dilatation, and thrombectomy, were not significantly different between the groups. However, there was a significantly higher percentage of patients in the slow flow/no-reflow group who received temporary pacemaker support (16.7%) and IABP (60.0%). (Table 1)

A multivariate and univariate analysis of stroke patient outcomes is presented in Table 2. Univariate analysis examines the relationship between a single factor and the outcome, whereas multivariate analysis considers multiple factors simultaneously to determine their independent association with the outcome.

In the multivariate analysis, only low SBP decreased eGFR, and a history of cerebrovascular disease remained independently associated with worse outcomes, while age, male sex, insulin use, and time from onset to recanalization were not significant predictors.

Overall, this suggests that low SBP, decreased eGFR, and a history of cerebrovascular disease are important factors to consider in predicting outcomes in stroke patients and that these factors should be taken into account when developing treatment plans. (Table 2)

Table 3 presents the demographic, clinical, and laboratory characteristics of two groups of patients with acute myocardial infarction (AMI) who were divided based on their level of strength (STR). Regarding demographic factors, the two groups exhibited similarities in blood pressure, hypertension, diabetes, and past cerebrovascular illness. Nevertheless, the group with STR equal to or above 50% demonstrated a considerably lower average age (62 years) than the group with STR below 50% (71 years) (p<0.001). Furthermore, the male population was more prevalent in the group with STR equal to or above 50% (84.2%) as compared to the group with STR below 50% (74.0%) (p=0.043).

Concerning the clinical features, both sets of subjects exhibited comparable NYHA classifications, with most individuals belonging to NYHA grade I or II in each group. However, there was a trend towards more patients in NYHA class II or higher in the group with STR less than 50% (22.0%) compared to the group with STR greater than or equal to 50% (12.9%) (p=0.06). The two groups also had similar BMIs, but there were more current smokers in the group with STR greater than or equal to 50% (50.9%) compared to the group with STR less than 50% (32.4%) (p=0.01).

In terms of laboratory parameters, the two groups had similar WBC counts, neutrophil counts, lymphocyte counts, and NLR. However, the group with STR greater than or equal to 50% had a significantly higher mean Hb level (14.6 g/dL) compared to the group with STR less than 50% (14.1 g/dL) (p=0.02), and a significantly higher mean platelet count (221 x 1000/µg) compared to the group with STR less than 50% (203 x 1000/µg) (p=0.041). (Table 3)

Table 4 presents the results of univariate analysis and a multivariate analysis for several variables. During a univariate analysis, each variable is assessed separately, whereas a multivariate analysis controls for the other variables in the model while reviewing the relationship between each variable and the outcome variable. Based on the univariate analysis, age, eGFR, BNP, and LAD are all significantly related (with p-values of less than 0.05) to the outcome variable. The outcome variable is not significantly affected by male sex or haemoglobin level (p values >0.05). (Table 4)

Table 1: Patients with Normal Flow compared with Slow Flow and No Reflow (n= 210)

	Normal flow n=185 (88.0%)	Slow flow/no-reflow n=25 (11.9%)	P-value
Age (years)	62 [50–68]	70 [58–75]	0.007*
Male sex	146 (78.9%)	19 (76.0%)	0.56
SBP (mmHg)	129 [110–157]	120 [102–139	0.02*
DBP (mmHg)	82 [71–94]	71 [60–90]	0.05*
SBP ≤90 mmHg	13 (7.0%)	5 (20.0%)	0.01*
NYHA classification			
I II III IV	155 (83.7%) 9 (4.8%) 3 (1.6%) 18 (9.7%)	19 (76.0%) 5 (20.0%) 0 1 (4.0%)	0.02*
NYHA classification ≥II	31 (16.7%)	6 (24.0%)	0.2
BMI (kg/m^2)	23.6 [21.5–25.7]	22.8 [20.7–24.9]	0.24
Hypertension	142 (76.7%)	18 (72.0%)	0.35
Diabetes	57 (30.8%)	9 (36.0%)	0.51
CVA	9 (4.8%)	6 (24.0%)	<0.001
Smoking			
smoking Ex-smoker Never smoked	81 (43.7%) 46 (24.8%) 58 (31.3%)	8 (32.0%) 7 (28.0%) 10 (40.0%)	0.37
White blood cell	9.4 [8.2–11.5]	8.8 [8.3–11.1]	0.54
NEUT	6.2 [4.1–8.5]	6.8 [5.1–7. 3]	0.35
Lymphocytes	2.3 [1.4–3.7]	1.6 [1.2–2.8]	0.07
neutrophil-to-lymphocyte ratio	2.4 [1.2–5.2]	4.2 [1.6–6.3]	0.16
Haemoglobin	14.5 [13.2–15.5]	14.2 [12.1–15.1]	0.04*
Platelets (×1,000/µg)	215 [182–251]	226 [177–268]	0.45
Mean platelet volume (fL)	10.2 [9.5–10.8]	10 [9.6–10.6]	0.31
Triglyceride level	82 [50–131]	66 [50–97]	0.15
High-density lipoprotein cholesterol	48 [42–56]	44 [38–53]	0.23
Low-density lipoprotein cholesterol	122 [97–142]	118 [92–143]	0.14
HbA1c (%)	5.7 [5.4–6.2]	5.7 [5.7–6.6]	0.73
BG	158 [137–198]	168 [146–222]	0.31
B-type natriuretic peptide	38 [14–101]	75 [25–148]	0.04*
Medication on admission			

Prasugrel (20 mg loading) Clopidogrel (300 mg loading) ACEI ARB β-blockers Calcium channel blockers Statin Insulin	68 (36.7%) 113 (61.0%) 3 (1.6%) 29 (15.6%) 8 (4.3%) 48 (25.9%) 22 (11.8%) 4 (2.1%)	10 (40.0%) 14 (56.0%) 0 8 (32.0%) 2 (8.0%) 7(28.0%) 4 (16.0%) 2 (8.0%)	0.75 0.62 0.37 0.02* 0.51 0.63 0.37 0.02*
Culprit lesion			
LAD RCA LCX	93 (50.2%) 78 (42.1%) 14 (7.5%)	14 (56.0%) 10 (40.0%) 1 (4.0%)	0.54
Syntax score	14.7 [9–21.6]	15.6 [10–22]	0.8
Procedural characteristics			
Use of IVUS Predilatation Post-dilatation Temporary pacemaker support Use of IABP Use of thrombectomy	98 (52.9%) 137 (74.0%) 39 (21.0%) 26 (14.0%) 31 (16.7%) 35 (18.9%)	15 (60.0%) 22 (88.0%) 4 (16.0%) 4 (16.0%) 15 (60.0%) 4 (16.0%)	0.51 0.21 0.45 0.61 <0.001* 0.56
Time from onset to recanalization (min)	176 [136–268]	246 [146–323]	0.13
Time from onset to recanalization ≥4 h	57 (3.8%)	13 (52.0%)	0.01*

CVA = cerebrovascular, NEUT = Neutrophils, BG = Blood Glucose

Table 2: Slow Flow and No Reflow Prediction by Univariate and Multivariate Logistic Regression Analysis

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P-value	OR (95% CI)	P-value
Age	1.04 (1.02–1.08)	0.007*	1.03 (0.87–1.06)	0.26
Male sex	0.78 (0.25–2.17)	0.68	1.38 (0.41–4.20)	0.27
SBP ≤90 mmHg	4.35 (1.17–9.02)	0.01*	3.34 (1.01–11.5)	0.035*
eGFR	0.85 (0.83–0.87)	<0.001 *	0.86 (0.83–0.88)	0.006*
Insulin (on admission)	5.43 (1.28–31.6)	0.03*	2.84 (0.4–16.4)	0.13
Cerebrovascular disease	5.63 (2.05–14.8)	<0.001 *	4.54 (1.41–13.2)	0.005*
Time from onset to recanalization ≥4 h	2.26 (1.12–5.03)	0.02*	2.65 (1.14–6.5.52)	0.012*

Table 3: ST Segment Resolution (STR) in Patients with High and Low Clinical Characteristics (n=185)

	STR ≥50% (n=108)	STR <50% (n=77)	P-value
Age (years)	62 [52–69]	71 [60–77]	<0.001*
Male sex	91 (84.2%)	57 (74.0%)	0.043
SBP (mmHg)	136 [112–155]	132 [116–155]	0.83
DBP (mmHg)	81 [68–95]	87 [71–97]	0.25
SBP ≤90 mmHg	10 (9.2%)	3 (3.8%)	0.12
NYHA classification			
I II III IV	94 (87.0%) 5 (4.6%) 0 9 (8.3%)	60 (77.9%) 4 (5.1%) 3 (3.8%) 10 (12.9%)	0.07
NYHA classification ≥II	14 (12.9%)	17 (22.0%)	0.06
BMI (kg/m^2)	23.5 [21.6–25.8]	23.5 [21.4–24.8]	0.31
Hypertension	80 (74.0%)	62 (80.2%)	0.16
Diabetes	32 (29.6%)	25 (32.4%)	0.51
CVA	4 (3.7%)	5 (6.4%)	0.41
Smoking status			
Current smoker Ex-smoker Never smoked	55 (50.9%) 25 (23.1%) 28 (25.9%)	25 (32.4%) 22 (28.5%) 30 (38.9%)	0.01*
White blood cell	9.5 [8.1–12.2]	9.2 [7.8–11.5]	0.14
NEUT	6.2 [4.3–8.7]	6.2 [3.8–7.8]	0.14
Lymphocytes	2.3 [1.6–3.7]	2.5 [1.6–3.7]	0.41
neutrophil-to-lymphocyte ratio	2.7 [1.3–5.6]	2.2 [1.2–4.2]	0.17
Haemoglobin	14.6 [13.4–15.7]	14.1 [12.7–15.5]	0.02*
Platelets (×1,000/μg)	221 [191–257]	203 [176–248]	0.041*
Mean platelet volume (fL)	10.2 [9.5–10.8]	10.2 [9.8–10.8]	0.36
Triglyceride level	81 [52–134]	78 [48–122]	0.37
High-density lipoprotein cholesterol	46 [41–57]	46 [41–55]	0.45
Low-density lipoprotein cholesterol	123 [98–143]	122 [98–142]	0.45
HbA1c (%)	5.6 [5.3–6.4]	5.8 [5.5–6.6]	0.06
BG	155 [136–188]	163 [137–226]	0.17
B-type natriuretic peptide	27 [12–92]	56 [22–146]	<0.001*
Medication on admission			
Prasugrel (20 mg loading) Clopidogrel (300 mg loading) ACEI ARB β-blockers Calcium channel blockers Statin Insulin	40 (37.0%) 67 (62.0%) 2 (1.8%) 13 (12.0%) 2 (1.8%) 24 (22.2%) 14 (12.9%) 2 (1.8%)	28 (36.3%) 47 (61.0%) 1 (1.2%) 16 (20.7%) 7 (9.0%) 25 (32.4%) 8 (10.3%) 2 (2.5%)	0.86 0.78 0.41 0.041* 0.004* 0.06 0.43 0.61
Culprit lesion			
LAD RCA LCX	43 (39.8%) 53 (49.0%) 11 (10.1%)	50 (64.9%) 24 (31.1%) 3 (3.8%)	<0.001*
Syntax score	10 [9–21]	16.6 [10–18.6]	0.07
Procedural characteristics			
Use of IVUS Predilatation using an additional balloon Post-dilatation using an additional balloon Temporary pacemaker support Use of IABP Use of thrombectomy	53 (49.0%) 80 (74.0%) 24 (22.2%) 15 (13.8%) 17 (15.7%) 24 (22.2%)	46 (59.7%) 58 (75.3%) 15 (19.4%) 10 (12.9%) 15 (19.4%) 12 (15.5%)	0.07 0.81 0.42 0.61 0.53 0.13
The time between 2 ECGs (min)	124 [104–141]	128 [107–151]	0.07

CVA = cerebrovascular, NEUT = Neutrophils, BG = Blood Glucose

Table 4: Predicting low ST-segment resolution using a multivariate logistic regression model

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	P-value	OR (95% CI)	P-value
Age	1.03 (1.03–1.06)	<0.001*	1.00 (0.87–1.02)	0.73
Male sex	0.44 (0.18–1.01)	0.07	0.61 (0.23–1.38)	0.25
Haemoglobin	0.83 (0.76–1.03)	0.14		
eGFR	0.84 (0.83–0.86)	<0.001	0.93 (0.82–0.85)	<0.001 *
BNP pg/mL >18.4	2.23 (1.21–4.13)	0.003*	1.92 (0.86–3.72)	0.07
LAD	2.6 (1.5–4.45)	<0.001*	4.48 (2.25–8.41)	<0.001 *

DISCUSSION

A study was conducted on the no-reflow condition in patients with STEMI who underwent direct PCI. Coronary microvascular dysfunction and obstruction can be evaluated using the well-known parameters of reflow and STR impairment. STR is a prognostic tool for AMI that is non-invasive, widely accessible, and low-cost.[18] Assessing microvascular dysfunction and obstruction with STR requires careful consideration of three factors. Typically, there are two distinct approaches to determining STR. Most research has used the sum of STR following reperfusion therapy to forecast various outcomes, such as the extent of the infarction, the condition of the left ventricle, the openness of the epicardial vessels, and the probability of death.[19–21] Measuring ST segment elevations from all leads associated with the location of a heart attack can be time-consuming. Evaluating a single-lead STR that displays the highest ST elevation at the starting point may be equally effective as summing up multiple STR measurements.[22] The study at hand used single-lead STR. There are two methods available for assessing the extent of STR: one proposes a threshold of less than 50%, while the other suggests less than 70%. Either of these measurements shows compromised STR, as their predictive capabilities were established during the early phase of pharmacological reperfusion and continue to hold in the present era of mechanical reperfusion.[18] To assess STR easier, the binary method was employed, which categorized STR as either <50% or ≥50%. As for when the ECG recordings were taken, most studies conducted the recordings at either 60 or 90 minutes or 3 hours after the occurrence of thrombotic and pPCI.[23–25] The current research involved conducting an ECG on patients immediately upon their admission to the intensive care unit following pPCI. The timing of the second ECG measurement after coronary recanalization did not vary between the two groups (STR <50% vs. ≥50%).[5,26,27] Chronic kidney disease (CKD) is a significant contributor to poorer cardiovascular outcomes, according to research.[28] In a study by Kurtul et al.,[29] they investigated the potential causes of no-reflow across three groups with varying

eGFR levels.[29] The study revealed that eGFR was an independent risk factor for no-reflow. However, it is still uncertain whether there is a connection between CKD and STR. CKD has a potential link to coronary microvascular dysfunction and blockages in three ways, which are pre-existing issues with the microvascular function of the heart, a difference in white blood cell count, and changes in platelet activity. The normal response of coronary blood flow is to increase from its resting level to its peak level automatically when there is an increase in the demand for myocardial oxygen.[30] The term CFR is used to describe a modification in the flow of blood through the coronary arteries. A number of investigations have shown that there is a strong connection between CFR and impaired kidney function, indicating that patients who suffer from reduced renal capabilities are more likely to have limited coronary vasodilation, particularly in cases where no blockages are present in the arteries.[31,32] An increase in the number of white blood cells (leukocytosis) and changes in the proportions of different types of white blood cells are typical indicators of inflammation and are closely linked to the risk of developing cardiovascular disease.[33] Scientists have studied the neutrophil/lymphocyte ratio (NLR), which is calculated based on the white blood cell count, and discovered that it is a significant indicator of inflammation and a useful marker for assessing cardiovascular risk.[34,35]A prior investigation found that the NLR was linked to a heightened death rate and unfavourable outlook in acute coronary syndrome, particularly in cases involving ST-segment elevation.[36] Additionally, Machado and colleagues determined that a high NLR upon admission independently predicted no-reflow following pPCI in STEMI patients.[37] Unexpectedly, Sevencan and colleagues discovered that patients with Stage 3 CKD had a higher NLR than those with Stage 1 or 2 CKD.[37] In STEMI patients with high MPV upon admission, Machado et al. revealed it to be an independent predictor of no-reflow following PCI.[37] Verdoia et al discovered that patients with chronic kidney disease (CKD) had larger platelets and a weak correlation between mean platelet volume (MPV) and the decay of renal function (measured as eGFR).[38] This may be due to CKD leading to platelet activation and increased coagulability, which can cause the slow-flow/no-reflow phenomenon. The MPV, as well as the neutrophil-to-lymphocyte ratio (NLR), did not differ between those groups with the slow flow or no reflow compared with those with the normal flow in the present study. Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) have been associated with CKD and slow-flow/no-reflow diagnostic imaging techniques. According to the research conducted by Soeda and colleagues,[39] the rupture of plaque was identified as the underlying factor for the morphological indicators leading to no-reflow after percutaneous coronary intervention (pPCI) among patients suffering from ST-segment elevation myocardial infarction (STEMI).[40] A higher lipid index and greater plaque burden were also found among patients with CKD in non-culprit lesions, which can contribute to no-reflow.[41]

CONCLUSION

The study found that patients with STEMI who have renal dysfunction are more likely to experience SFR and ISR after undergoing PCI. Renal dysfunction should be regarded as a significant factor that increases the risk of these complications. Treatment may need to be more intensive to achieve better results in these patients.

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