Original Article

Observed Influence of Nitroglycerine on Myocardial Perfusion Scintigraphy in Patients with Multiple Vessel Coronary Artery Disease and Well-developed Collaterals

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Abstract

The objective of this scientific work was to evaluate the extent and severity of perfusion abnormalities on myocardial perfusion scintigraphy (MPS) at rest and with sublingual nitroglycerine, in relation to the presence and anatomical location of collaterals demonstrated by selective coronary angiography (SCA). Twenty-eight patients with unstable angina underwent selective coronary angiography. Eighteen of them were diagnosed with myocardial infarction (MI) 2–15 days prior to examination. Presence or absence of collaterals was noted, with anatomical depiction of donor and recipient arteries as well as evaluation of degree of collateral flow. As an inclusion criterion, collateral flow had to be grade 2 (partial epicardial filling of the occluded artery) or 3 (complete epicardial filling of the occluded artery) in accordance with the Rentrop collateral flow classification. Flow was noted as follows: Complete antegrade (CA), complete retrograde (CR), partial antegrade (PA), and partial retrograde (PR). Myocardial perfusion scintigraphy using Tc-99m Sestamibi at rest and after sublingual administration of nitroglycerine was performed according to a 2-day protocol. Perfusion abnormalities, which were quantified using the 20-segments model and visual 5-point system (0, normal perfusion; 4, absent perfusion), were analyzed according to donor's and recipient's territories, as well as territories with limited or without collateral flow (PA/PR, grade 0–1 flow). A total of 84 arteries were analyzed, with stenosis in 79 of them. Arteries were divided into three groups: Donors (group I), recipients (group II), and arteries with limited or without collaterals (group III). In group I, there were 28 donor arteries, with mean severity of stenosis 71.3 ± 0.65%. In group II, there were 36 recipient arteries and mean severity of stenosis was 94.8 ± 0.26%. In group III, there were 20 arteries, and all of them had either no or poorly developed collaterals (mean severity of stenosis 60.4 ± 2%). In 3 cases, 2 donor arteries gave collaterals to 1 recipient artery, while in 11 cases, a single donor artery gave collaterals to 2 recipient arteries, and in 11 cases there was 1 donor to 1 recipient artery. A total of 1120 MPS segments were analyzed (560 at rest and 560 after nitroglycerine). The number of segments in groups I, II, and III were 204, 242, and 144, respectively. Mean number of segments per donor artery was 7.2 ± 0.7, mean number of segments per recipient artery was 7.0 ± 0.3, and mean number of segments in the territory of arteries without collaterals was 5.5 ± 0.5. In the territory of donor arteries, the mean number of segments with normal, decreased, and absent perfusion at rest was 1.6 ± 0.07, 5.67 ± 0.07, and 0.6 ± 0.03, respectively. After nitroglycerine administration, the mean number of above-mentioned segments was 1.2 ± 0.07, 6.07 ± 0.06, and 2.3 ± 0.06, respectively. There was no significant difference in the mean number of segments with normal and decreased perfusion at rest and after nitroglycerine administration ($P = 0.4$). However, the increase of mean segments with absent perfusion that appeared after nitroglycerine administration in donor arteries was statistically significant in comparison to MPS at rest ($P < 0.0001$). In the territory of recipient arteries, there was statistically significant increase in the mean number of segments with normal perfusion from 0.5 ± 0.02 at rest to 2.7 ± 0.06 with nitroglycerine ($P < 0.0001$), decrease in mean number of segments with decreased perfusion from 6.5 ± 0.06 at rest to 4.19 ± 0.06 with nitroglycerine ($P < 0.0001$), and decrease in the mean number of segments with absent perfusion from 2.3 ± 0.06 to 0.7 ± 0.03 ($P = 0.003$). In Group III, there was increase in mean segments with normal perfusion from 2.4 ± 0.5 to 3.2 ± 0.5, decrease in mean segments with decreased perfusion from 3.15 ± 0.5 to 2.35 ± 0.5, and absent

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tracer uptake from 1.1 ± 0.5 to 0.45 ± 0.3. However, these changes were not statistically significant ($P = 0.3$, $P = 0.4$, and $P = 0.2$, respectively). There was also statistically significant improvement of perfusion in the recipient territories from mean severity score at rest of 2.67 ± 0.08 to 1.6 ± 0.09 with nitroglycerine ($P < 0.0001$), in territories of poorly collateralized arteries from mean severity score at rest of 1.5 ± 0.14 to 0.8 ± 0.12 with nitroglycerine ($P < 0.0008$), as well as significant deterioration of myocardial perfusion in donor artery territories from mean severity score at rest of 1.7 ± 0.06 to 2.4 ± 0.06 with nitroglycerine ($P < 0.0001$). Based on the results of the study, we concluded that nitroglycerine administration in patients with multiple vessel coronary artery disease and well-developed collaterals can reduce myocardial perfusion to the areas supplied by donor arteries, even resulting in apparent absent perfusion, probably due to "steal syndrome," although these arteries were less stenosed angiographically and deemed viable on MPS at rest. It appears that MPS in patients on nitroglycerine medication may result in an inappropriate decision by interventionists and surgeons to forgo revascularization. Hence, in cases where large and severe perfusion abnormalities are noted, MPS should be repeated after omitting nitrates.

Key words: Collaterals, myocardial perfusion scintigraphy, nitroglycerine, selective coronary angiography, unstable angina

Introduction

The selection of patients with coronary artery disease for revascularization is based on detection of the affected myocardium that is potentially viable, since this is a necessary condition for successful revascularization.[1] Nitroglycerine causes marked relaxation of all components of the vascular system and dramatically decreases pulmonary vascular pressure, intraventricular pressure, chamber size, and cardiac output. Nitroglycerine improves myocardial perfusion via a reduction in wall tension and myocardial oxygen demand.[2-4] Numerous studies have demonstrated that ^{99m}Tc Sestamibi myocardial perfusion scintigraphy (MPS) with nitroglycerine has similar results with nitrate-enhanced ^{201}Tl rest–redistribution for estimation of myocardial viability, prediction of functional improvements, remodeling, and survival after revascularization in patients with ischemic heart disease.[3-6] Some authors have concluded that nitroglycerine infusion during ^{201}Tl imaging is a useful technique for detecting underperfused, viable myocardium, requires less time to perform than rest/redistribution imaging, and may allow detection of viable myocardium with a single ^{201}Tl single-photon emission computed tomographic (SPECT) study. They concluded that ^{201}Tl SPECT with nitrate-augmented redistribution has significant logistical and economic advantages over traditional delayed redistribution with ^{201}Tl reinjection.[6,7] However, according to literature,[8,9] in 18–26.4% of patients, there was deterioration of myocardial perfusion defects after administration of nitroglycerine in comparison to the rest ^{99m}Tc Tetrofosmin. The authors did not find significant correlation between degree of coronary artery stenosis, number of involved vessels, and history of myocardial infarction (MI).

Coronary collaterals play a protective role in patients with occluded vessels. This has been shown by smaller infarct size, less ventricular aneurysm formation, improved ventricular function, and better survival, compared to patients in whom collaterals were not visualized.[10] However, whether collaterals can play a role in deterioration of myocardial perfusion with nitroglycerine has not been studied before.

The purpose of this study was to evaluate the extent and severity of perfusion abnormalities assessed by MPS at rest and with nitroglycerine in relation to degree of existence of collaterals and anatomical location of donor and recipient arteries demonstrated by selective coronary angiography (SCA).

Materials and Methods

Twenty-eight patients (25 males, 3 females; mean age: 55.7 ± 8.2 years; range: 43–73 years) with unstable angina (UA) were referred for revascularization to Republic Specialized Center of Surgery, Tashkent, between February and May 2012, and underwent diagnostic evaluation at Republic Specialized Center of Surgery and at Department of Angiography Interventional Radiology, Republican Research Medical Centre of Emergency Medicine. Eighteen of them had history of MI 2–15 days prior to the examination. The diagnostic work-up included clinical assessment, SCA, and MPS using ^{99m}Tc Sestamibi.

Mean ejection fraction in this group was 43 ± 12.8% (range: 16–58%). Clinical status was assessed by Braunwald classification[11] and is presented in Table 1.

SCA was performed for five vessels, namely, right coronary artery (RCA), left anterior descending (LAD), obtuse marginal (OM), first diagonal branch (D1), and left circumflex artery (LCx). Images were interpreted by an experienced interventional cardiologist in multiple views, and stenosis was calculated by computer-assisted quantitative angiography as percentage of cross-sectional area of coronary artery lumen and its hemodynamic significance.[12] Severity of stenosis was categorized as follows: (1) less than

50% = normal; (2) 50–75% = insignificant stenosis; (3) 76–95% = significant stenosis; (4) 96–99% = critical stenosis; and (5) 100% = occlusion. Presence of collateral flow, donor, recipient artery, and type of flow were estimated. The collaterals to the occluded coronary artery were assessed by contrast injection into the donor artery and were graded according to the classification of Rentrop *et al*.[13] As an inclusion criterion, collateral flow was of grade 2 (partial epicardial filling of the occluded artery) or 3 (complete epicardial filling of the occluded artery) with complete antegrade (CA), complete retrograde (CR), partial antegrade (PA), and partial retrograde (PR) flow. The grading was performed independently by two experienced investigators, and in case of discordance, consensus was obtained with a third investigator.

MPS was performed with ^{99m}Tc Sestamibi using a 2-day protocol. Image acquisition was performed by using a Mediso Nuclear Spirit dual-head SPECT gamma camera (18 patients) and Siemens E.CAM, e.soft single-head SPECT gamma camera (10 patients).

Acquisition protocol

The following acquisition protocol was used. Low energy high resolution (LEHR) collimator, matrix size 64 × 64, rotation 90° (dual head) or 180° (single head) CCW, time/view 30 s, zoom 1.28, and energy photopeak 140 keV. Image acquisition was performed 45 min after injection of 15 mCi ^{99m}Tc Sestamibi at rest and in the next day after sublingual administration of nitroglycerine, and approximately 40 min after a fatty meal. Image interpretation was performed using 20-segment analysis of the left ventricle where the myocardium was divided into three sections: Short axis, vertical long axis, and horizontal long axis. Each of these sections was further divided into apical, mid, and basal segments. Thus, semi-quantitative analysis was performed on 20 segments of the left ventricle.[14] For each segment, assessment was performed using five points on a continuous color scale, and graded as follows: Normal uptake =0; mildly reduced =1; moderately reduced =2; severely reduced =3; and absent uptake =4. Myocardial viability was estimated by the following principle: If color saturation was within 50% of the maximum range, myocardium was considered viable; if color saturation was within 30–50%, then mixed (partly viable and partly necrotic); and if less than 30%, then it was considered necrotic myocardial tissue. The extent of perfusion abnormality was defined by the number of segments involved, as follows: (1) up to 5 segments = small; (2) 5–10 segments = intermediate; and (3) more than 10 segments = large. Estimation of myocardial perfusion abnormalities was done according to territory of donor, recipient artery, and artery without or with limited collateral flow [Figure 1].

Statistical analysis

The acquired results have been expressed as standard error of the mean (SEM) for each index. Comparison of data amongst various groups has been performed with Student's unpaired *t*-test for normal distributed values. A *P* value of <0.05 was considered statistically significant.

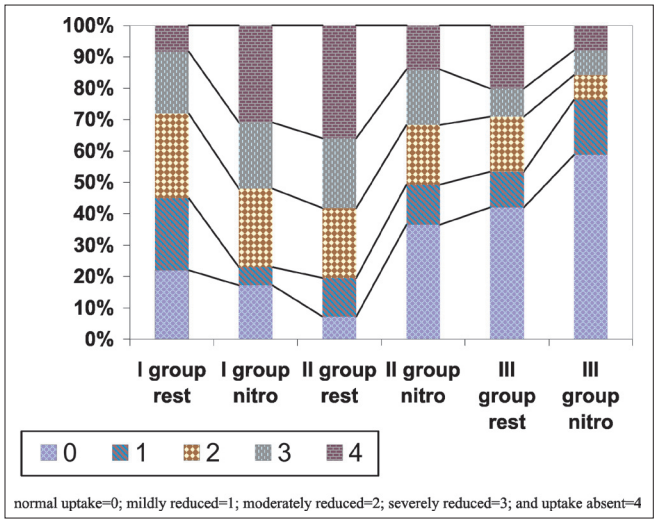

Figure 1: Myocardial perfusion abnormalities in territories of donor (group I), recipient (group II) arteries, and in arteries without or with limited collateral flow (group III), at rest and after nitroglycerine administration

Table 1: Clinical status assessment by Braunwald classification[11] in 28 patients included in the study

Severity		Clinical circumstances		
		A	B	C
		Develops in the presence of extra-cardiac condition that intensifies myocardial ischemia (secondary UA)	Develops in the absence of extra-cardiac condition (primary UA)	Develops within 2 weeks after acute myocardial infarction (post-infarction UA)
I	New onset of severe angina or accelerated angina; no rest pain	No of patients: 0	No of patients: 2	No of patients: 0
II	Angina at rest within past month, but not within preceding 48 h (angina at rest, subacute)	No of patients: 0	No of patients: 2	No of patients: 10
III	Angina at rest within 48 h (angina at rest, acute)	No of patients: 0	Troponin negative: *n* = 0 Troponin positive: *n* = 3	No of patients: 11

Results

Eighty-four arteries were analyzed, and 79 of them were stenosed. Results of SCA are given in Table 2. Arteries were divided into three groups: I, donors; II, recipients; and III, arteries with limited or without collaterals. Mean degree of severity of stenosis and number of dominant arteries are presented in Table 3.

Table 2: Results of selective coronary angiography in group I,II, and III coronary arteries

Age	Sex	Donor artery (group I)				Recipient artery (group II)				Flow	Without or with limited collaterals (group III)				
		Site	Type	% stenosis	Level	Site	Type	% stenosis	Level		Site	Type	% stenosis	Level	Flow
46	F	RCA	d	80	Prox	LAD		100	Med	CR3	LCX		0	0	
48	M	LAD	d	50	Med	RCA		100	Prox	CR3	LCX		0	0	
60	M	LAD	d	95	Prox	RCA		100	Med	CR3					
		LCX		95	Dist										
70	F	LAD	d	80	Prox	RCA		100	Med	CR3					
		LCX		70	Med										
49	M	LAD	d	50	Med	RCA		100	Prox	CR3					
						LCX		100	Prox	CR3/PA2					
71	M	RCA	d	70	Med	LAD		98	Med	PA2/PR2					
						LCX		100	Med	CR3					
73	M	RCA	d	98	Med	LAD		100	Med	CA3	LCX		0	0	
43	M	RCA	d	50	Prox	LAD		99	Prox	CR3/CA3					
						LCX		100	Prox	PA3					
69	M	RCA		70	Med	LAD	d	100	Prox	PA2					
						LCX		100	Prox	PR2					
59	M	RCA	d	50	Med	LAD		100	Prox	PR2	LCX		90	Med	PAO
58	M	RCA	d	50	Prox	LAD		70	Prox	CA3					
						LCX		90	Prox	CA3					
55	M	RCA	d	98	Med	LAD		100	Prox	CR3	LCX		50	Dist	0
57	M	RCA		90	Prox	LAD	d	70	Multiple	CR3	LCX		70	Dist	0
47	M	RCA		60	Med	LAD	d	100	Prox	CR3	LCX		0	0	
50	M	LAD	d	70	Med	LCX		100	Prox	CR3/CA3					
		RCA		100	Med										
56	M	LAD		70	Med	RCA	d	100	Prox	PR2	LCX		80	Med	PA1
61	M	RCA		70	Dist	LAD	d	99	Prox	CR3					
						LCX		99	Dist	CR3					
56	M	LAD		60	Prox	RCA	d	100	Prox	PR2	LCX		90	Med	0
44	M	RCA	d	50	Dist	LAD		85	Prox	PR2					
						LCX		75	Dist	CR2					
63	M	LCX		60	Dist	LAD	d	100	Prox	CR3					
						RCA		90	Prox	PR2					
53	M	LAD	d	50	Prox	RCA		100	Prox	PR2					
						LCX		80	Prox	CR3					
59	M	RCA		80	Med	LAD	d	80	Dist	CR3	LCX		99	Dist	PA1
50	M	LAD		100	Dist	RCA	d	80	Dist	PR2					
						LCX		100	Med	PR2					
52	M	LCX		70	Med	LAD	d	100	Prox	PR2					
						RCA		100	Prox	PR2					
52	M	RCA		60	Prox	LAD	d	100	Med	CR3	LCX		50	Prox	0
50	M										LAD	d	100	Med	CA3
											RCA		60	Med	PA1
											LCX		60	Med	PA1
55	F										LAD		50	Med	0
											RCA	d	100	Prox	PA1
											LCX		0		
52	M										LAD	d	75	Prox	0
											RCA		50	Med	0
											LCX		100	Prox	0

C: Complete, P: Partial, A: Antegrade flow, R: Retrograde flow, d: Dominant.

In group I, there were 28 donor arteries; in 9 (32.2%) there was proximal stenosis, in 5 (17.8%) distal stenosis, and in 14 (50%) cases, stenosis was located in the mid artery.

In group II, there were 36 recipient arteries. Twenty-three (63.8%) had proximal stenosis, 8 (22.2%) had stenosis in the middle, and 4 (11.1%) cases had distal stenosis. In 1 case (2.7%), there were multiple stenoses. Fifteen arteries had CR3 (flow/Rentrop grade) filling; 11 had PA2, 3 had CA3, 2 had CR3/CA3, and there was one combination each with PA2, PA3, CR3/PA2, CR2, PA2, PA2/PR2 filling. Four arteries (20%) had proximal stenosis, 8 (40%) had stenosis in middle part of artery, and 3 (15%) had distal stenosis.

In group III, there were 20 arteries which had no or poorly developed collaterals, 5 of them without stenosis (25%) and 15 stenosed (75%). Out of 15 stenosed arteries, 4 (26.6%) had proximal stenosis, 8 (53.3%) had stenosis in middle part of artery, and 3 (20%) in the distal part of artery.

In 3 cases, 2 donors were giving collaterals to 1 recipient artery; in 11 cases, 1 donor artery was giving collaterals to 2 recipient arteries; and in 11 cases, 1 donor artery was giving collaterals to 1 recipient artery. Number of dominant arteries, and degree and level of stenosis are presented in Table 2.

Using MPS, a total of 1120 segments were analyzed (560 at rest and 560 after nitroglycerine administration). In territory of donor, recipient, and arteries without or poorly developed collaterals, there were 204, 242, and 144 segments, respectively. Number of segments per artery varied, according to which the artery was dominant. Thus, when RCA is dominant, eight segments (including inferolateral) belong to RCA territory and three segments to LCX. When left is dominant, six segments belong to LCX territory and five to RCA.

Mean number of segments per territory of donor arteries was 7.2 ± 0.7 (range: 5–9); mean number of segments in territory of recipient's arteries was 7.0 ± 0.3 (range: 3–9); and mean number of segments in the territory of arteries without collaterals was 5.5 ± 0.46 (range: 3–9). Myocardial perfusion abnormalities seen in territory of three groups are presented in Figure 1.

In the area of donor arteries, mean number of segments with normal, decreased perfusion, and absence of tracer uptake at rest were 1.6 ± 0.07 (range: 0–9), 5.67 ± 0.07 (range: 0–9), and 0.6 ± 0.03 (range: 0–4), respectively. After nitroglycerine administration, mean number of above-mentioned segments were 1.2 ± 0.07 (range: 0–9), 6.07 ± 0.06 (range: 3–9), and 2.3 ± 0.06 (range: 0–6), respectively. There were no significant changes in the mean number of segments with normal and decreased perfusion at rest and after nitroglycerine administration (P = 0.4). However, the increased number of segments with absence of tracer uptake after nitroglycerine administration was statistically significant in comparison to MPS at rest (P < 0.0001). In the area of recipient's artery, there was statistically significant increase in the number of segments with normal perfusion (0.5 ± 0.02 at rest vs. 2.7 ± 0.06 with nitroglycerine, P < 0.0001), decreased number of segments with deteriorated perfusion (from 6.5 ± 0.06 at rest to 4.19 ± 0.06 with nitroglycerine, P < 0.0001), and decreased number of segments with absence of tracer uptake (from 2.3 ± 0.06 to 0.7 ± 0.03, P = 0.003).

There was increase in segments with normal perfusion (from 2.4 ± 0.5 to 3.2 ± 0.5), and decreasing number of segments with deteriorated perfusion (from 3.15 ± 0.5 to 2.35 ± 0.5) and absence of tracer uptake (from 1.1 ± 0.5 to 0.45 ± 0.3) in territory of arteries with limited or without collaterals; however, these changes were not statistically significant: P = 0.3 for segments with normal perfusion, P = 0.4 for segments with decrease of tracer uptake, and P = 0.2 for segments with absence of tracer uptake.

With regard to the severity of perfusion defects, there was statistically significant improvement of myocardial perfusion in the territory of recipient arteries (mean severity score at rest 2.67 ± 0.08 and with nitroglycerine 1.6 ± 0.09, P < 0.0001) and in territories of arteries without or with poorly developed collaterals (at rest 1.5 ± 0.14 to 0.8 ± 0.12 with nitroglycerine, P < 0.0008) and statistically significant deterioration of myocardial perfusion in the territory of donor arteries (mean severity score at rest 1.7 ± 0.06 to 2.4 ± 0.06 with nitroglycerine, P < 0.0001). These changes were more severe when one donor artery gave collaterals to one recipient artery than in other donor–recipient combinations. This is due to the fact that when one donor artery collateralized two recipient arteries, the stenosis in the donor artery was non-significant; and when two donor arteries gave collaterals to one recipient artery, these changes were more moderated [Tables 3 and 4]. Clinical examples of the more common combinations are presented: Donor LAD–recipient RCA and donor RCA–recipient LAD are presented in Figures 2 and 3, respectively.

Discussion

Collaterals can maintain viability or provide a minimum nutritional supply for the myocardium distal to occluded coronary arteries.[15] Functional, hemodynamic, or biophysical aspects of well-grown collateral arteries relate to the fact that they constitute a network within the coronary circulation. Such connections between adjacent vascular territories, together with spatially varying vascular resistances

Table 3: Degree of stenosis in donor and recipient arteries, and in arteries without or with limited collaterals

	Group I	Group II	Group III
	71.3±0.63% (range=50–100%)	94.8±0.26% (min-70%, max-100%)	60.4±2.0% (range=0–100%)
Right coronary artery	15 (8 dominant)	10 (3 dominant)	3 (1 dominant)
Left anterior descending	9 (6 dominant)	16 (8 dominant)	3 (1 dominant)
Left circumflex artery	4	10	9
Without stenosis			5 (LCX)

Table 4: Characteristics of arteries in three combinations of "donor–recipient" arteries

	1 donor–1 recipient		1 donor–2 recipients		2 donors–1 recipient	
Number of cases	11		11		3	
Mean degree of severity of stenosis	75.1 ± 4.7% (range: 50–98%)	95.3 ± 3.1% (range: 70–100%)	60 ± 4.6% (range: 50–100%)	93.5 ± 2.2% (range: 70–100%)	85 ± 0.13% (range: 70–100%)	100 ± 0% (min, max-100%)
Number of dominant arteries	4	6	7	4	3	0
Levels of stenosis						
Proximal	3	6	4	15	2	1
Medial	7	3	4	5	3	2
Distal	1	1	3	2	1	0
	1 Multiple stenoses					

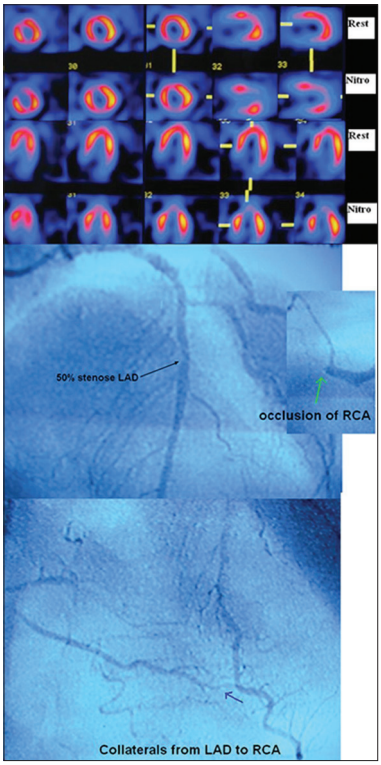

Figure 2: Panel 1 (top): Polar map showing markedly decreased perfusion of the anterior wall and apex at rest. After nitroglycerine administration, there is marked decrease in perfusion of the inferior and inferolateral walls with significant improvement to anterior wall perfusion, illustrating "steal" of flow from the right coronary artery (RCA) to the left anterior descending (LAD) territory. Panels 2 and 3 (middle and bottom, respectively): LAD is occluded; RCA donor is dominant with 80% stenosis. Collateral flow is demonstrated from RCA to LAD

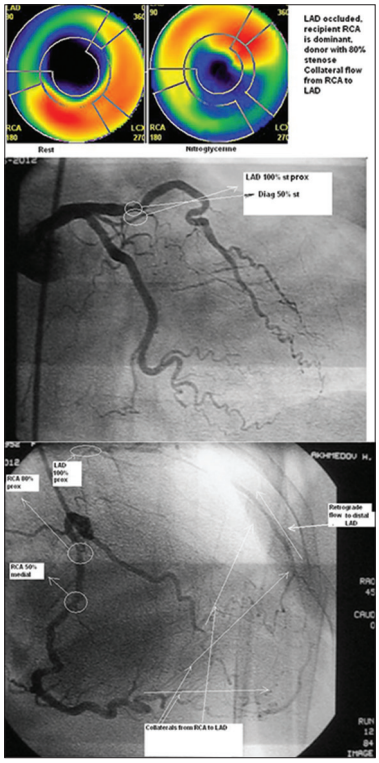

Figure 3: Panel 1 (top): Myocardial perfusion scintigraphy at rest and with nitroglycerine. The rest images show severe decrease in perfusion to the inferior wall [right coronary artery (RCA) territory]. With nitroglycerine, there is improvement in flow to the inferior wall, but with markedly decreased perfusion to the anterior wall and apex [left anterior descending (LAD) territory], illustrating "coronary steal" from LAD to RCA territory. Panel 2 (middle): Angiographic view showing neartotal occlusion of the RCA and 50% stenosis in the midLAD. Panel 3 (bottom): Collaterals can be seen from LAD to RCA

to blood flow are the bases for pathophysiological aspects of collaterals rarely considered, such as the redistribution of blood during vasodilatation away from a region in need (i.e. coronary steal).

Physical exercise induces a more than twofold perfusion increase in collateral-dependent myocardium via β-adrenergic and nitric oxide mediated mechanisms. In up to 50% of patients with chronic total coronary artery occlusions, there may be no infarcted myocardium within the vascular territory supplied by the blocked vessel.[16]

Previous studies have shown that coronary collaterals are usually not visualized until the coronary obstruction is more than 90%;[17] however, in more recent publications, it has been reported that in humans with angiographically normal coronary arteries, there are functional collateral vessels to the extent that one-fifth to one-quarter of them do not show signs of myocardial ischemia during brief vascular occlusions.[18] In our study, mean severity of stenosis of recipient arteries was from 93.5 to 100%. However, in seven cases, stenoses were less than 90% (from 70% involving proximal part of the artery or the whole artery to 85%). Mean degree of stenosis for donor arteries was from 60 to 85%.

Many studies have emphasized the importance of collateral blood flow in coronary artery disease.[19,20] Pijls *et al*.[21] described a method that enables the assessment of recruitable collaterals at coronary artery occlusion by estimating the index called the pressure-derived collateral fraction flow reserve (FFRcall) or pressure-derived collateral fraction flow index (CFp). Hitoshi Matsuo *et al*.[22] calculated FFRcall from coronary pressure during balloon occlusion. ^{99m}Tc Sestamibi was injected during balloon inflation and the occlusion continued for 3 min after injection of ^{99m}Tc Sestamibi. They observed that there is high correlation with the extent and severity of defect at myocardial perfusion of the territory of the occluded artery and that MPS can be used for quantitative assessment of collateral blood flow.

Many studies confirmed that MPS with nitroglycerine can improve sensitivity in the detection a viable myocardium.[1,23,24] Nitroglycerine is known to increase blood flow in the hypoperfused myocardium and reduce regional ischemia of the heart muscle in coronary artery disease.[2,4,5,8,9] Our study confirms these findings; we found statistically significant improvement of myocardial perfusion after nitroglycerine administration in hypoperfused areas seen at rest myocardial perfusion study. However, previous studies did neither pay much attention nor analyze areas with decreased tracer uptake at MPS with nitroglycerine *vis-à-vis* rest MPS. According to our study, such areas belong to those supplied by donor arteries. On the other hand, areas with increasing tracer uptake on MPS with nitroglycerine belong to recipient arteries. Antianginal medication can alter both resting and hyperemic myocardial perfusion and might affect the ability to detect flow-limiting stenosis.[4]

Vasodilator drugs such as nitrates enhance Sestamibi uptake within viable areas of recipient arteries and may engineer a coronary steal in areas of donor arteries.

Conclusion

Nitroglycerine administration in patients with multiple vessel coronary artery disease and well-developed collaterals can reduce myocardial perfusion to areas supplied by donor arteries, even resulting in apparent absent perfusion, probably due to "steal syndrome," even though these arteries were less stenosed angiographically and deemed viable on myocardial perfusion study at rest. Hence, MPS while patients are taking nitroglycerine may result in an inappropriate decision by the interventionists and surgeons to forgo revascularization. In cases where large and severe perfusion abnormalities are seen, MPS should be repeated after withdrawing nitrates.

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