Original Research

Incidence of Reversible Defect Seen on Myocardial Perfusion Scintigraphy Using Dipyridamole Pharmacologic Test Early After Primary Percutaneous Coronary Intervention: How Safe Is It to Perform This Protocol?

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Introduction: The purpose of this study was to investigate the safety of performing a dipyridamole stress test and to explore the incidence of reversible perfusion defects on myocardial perfusion imaging, five to six days after primary percutaneous coronary intervention (PCI).

Methods: Forty-one patients underwent myocardial perfusion imaging using a dipyridamole stress test, five to six days after primary PCI.

Results: Headache, chest pain, and dizziness were the most common side effects seen after dipyridamole administration. All occurrences were mild and short lasting. ST changes on the electrocardiogram were also seen in 12% of patients. Reversible perfusion defects occurred in 17%.

Conclusions: This is one of the few studies to investigate patients using a dipyridamole stress test early after primary PCI. We conclude that it is safe to perform myocardial perfusion imaging under dipyridamole administration, just a few days after primary PCI. Additionally, a high incidence (17%) of myocardial perfusion defects was seen in this group of patients. According to our investigational protocol, a second myocardial perfusion imaging examination is scheduled for six months later, in order to clarify how many of these patients suffer from restenosis, or whether the finding was merely due to early endothelial dysfunction.

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Pharmacological testing using vasodilator agents accounts for almost 40% of the total stress myocardial perfusion studies performed.[1] Dipyridamole is a pyrimidine based agent that increases interstitial adenosine by inhibiting adenosine reuptake across the cellular membranes of vascular endothelium.[2] Adenosine interacts with various receptors and produces vasodilatation in most vascular beds. The presence of stenosis in an epicardial artery progressively induces dilatation in the arteriolar bed, in proportion to the stenosis severity, in order to maintain normal flow to the myocardium. Consequently, under conditions of hyperemia, a normal artery is able to dilate to a greater extent compared to a stenosed one. This difference in myocardial blood flow between a normal and a narrowed artery is the basis of myocardial perfusion imaging.

European and American Guidelines recommend that patients with ST-segment

elevation myocardial infarction (STEMI) should be treated with primary percutaneous coronary intervention (PCI) if the duration of symptoms is <12 h.[3,4] The use of drug-eluting stents (DES) has now become the standard of care, because of its considerable survival benefit.[5-8] DES were developed to solve the problem of in-stent restenosis seen in patients who underwent bare-metal stent implantation.[6-8] Nevertheless the incidence of reocclusion in DES is still considerable, ranging from 7% to 9%.[9]

The purpose of this study was to investigate the presence of reversible defects on myocardial perfusion imaging, as a marker of ischemia, in patients early after primary PCI, as well as to study the safety of performing a dipyridamole pharmacological stress test early after the procedure.

Methods

Between January 1 and December 31, 2010, 28 men (age 37-73 years) and 13 women (age 46-70 years) underwent rest/dipyridamole pharmacological stress myocardial perfusion imaging using Tc-99m Sestamibi, five to six days after primary PCI. The entire patient cohort fulfilled the following criteria:

1. Symptom duration between 30 min and 12 h.
2. Cumulative ST-segment elevation of 2 mV in 2 contiguous leads or 1 mV in 2 or more standard leads.
3. Elevation of serum cardiac enzymes (creatine-kinase MB) or biomarkers (troponin T or I).
4. One-vessel disease.

Exclusion criteria were:

1. Previous coronary bypass grafting.
2. Need for mechanical ventilation.
3. Treatment with a thrombus aspiration catheter.
4. Patients in cardiogenic shock.
5. Patients presenting with restenosis in a vessel previously treated with angioplasty.
6. Second- or third-degree AV block without functioning pacemaker.
7. Systolic blood pressure <90 mmHg.
8. Hypersensitivity to dipyridamole.
9. Multi-vessel disease.

Patients were kept free from antianginal medication and methylxanthines for at least 24 hours and a "one-day" protocol was performed, starting with the rest study, administering 296 MBq (8 mCi) of Tc-99m Sestamibi through an intravenous line. Image acquisition was started 45 min later, using a dual-head single-photon emission computed tomography (SPECT) camera (ADAC Vertex-Plus, Milpitas, USA), equipped with low-energy, high-resolution, parallel-hole collimators and linked to the Philips Jetstream workstation (DA Best, The Netherlands). The total duration of the rest study was approximately 15 min, divided into 32 frames of 30 seconds each. At the same time, the ejection fraction of the left ventricle was estimated using the gated SPECT method. Rest and stress images were gated at 16 frames per cardiac cycle. Immediately after the first acquisition, the pharmacological stress study was performed. A 12-lead electrocardiogram, blood pressure and heart rate were continuously recorded during the intravenous infusion of dipyridamole (0.56 mg/kg) over a four-minute period. At the 6th minute, 1110 MBq (30 mCi) of Tc-99m Sestamibi were injected and 5 minutes later the procedure was terminated by administrating 250 mg of aminophylline. Patients were encouraged to have a fatty meal and stress image acquisition started after 45 minutes, using the same parameters as in the rest study protocol. The acquired images were reconstructed in short-, horizontal long- and vertical long-axis tomograms. The data were analyzed using the Philips commercially available automatic quantitative perfusion SPECT and quantitative gated SPECT programs (QPS and QGS) (Cedars-Sinai Medical Center, Los Angeles, CA, USA)[10-11] and were interpreted by two experienced nuclear medicine specialists.

According to our investigational protocol, during a one-year period, all patients who underwent primary PCI in our hospital were tested by myocardial perfusion imaging 5 to 6 days after the procedure. Both myocardial perfusion imaging and coronary angiography were performed after 6 months in all the patients who exhibited a reversible defect, in order to clarify if the ischemia seen scintigraphically early after revascularization could serve as an early predictor of the in-stent restenosis.

The study protocol was in compliance with the Declaration of Helsinki and was approved by the local ethics committee.

Results

The characteristics of each lesion in the cohort of 41 patients treated by primary PCI and DES placement are given in Table 1. The post-intervention antiplatelet medication included acetylsalicylic acid (75 to 150 mg once daily) and clopidogrel, with a loading dose of 300 mg followed by maintenance dose of 75 mg daily.

The results of the gated pharmacological rest-stress

Table 1. Coronary lesion characteristics.

Left anterior descending, n (%)	20 (48.8)
Right coronary artery, n (%)	9 (21.9)
Left circumflex artery, n (%)	12 (29.3)
Lesion length (mm)	16 (12-22)
Stenosis luminal diameter	95-100%
Stent length (mm)	18 (14-20)
Max balloon pressure (Atm)	17 (15-19)
Max balloon diameter (mm)	3.5 (3.2-3.8)

myocardial perfusion imaging are presented in Table 2. Reversible perfusion defects were present in 7 of the 41 patients (17%), 3 in the left anterior descending artery, 2 in the right coronary artery, and 2 in the left circumflex artery. In these patients, the area of the defect ranged from 6% to 13% and the initial infarct area varied from 2% to 10% of the total myocardial mass. Five of the patients experienced a reduction in ejection fraction during pharmacologic stress, which ranged from 6% to 11%.

Table 2. Patients' characteristics.

Patient	Lesion	Rest HR	Rest BP (mmHg)	Rest EF	% MI*	Stress HR†	Stress BP (mmHg)	Stress EF	Reversible defect	Stress %‡
1	LAD	56	130/80	45%	26%	82	120/80	49%	no	-
2	LAD	67	130/80	36%	30%	81	110/80	34%	no	-
3	LAD	56	125/80	64%	3%	90	120/80	68%	no	-
4	RCA	80	125/90	58%	6%	105	110/70	70%	no	-
5	LCX	80	150/80	70%	12%	112	120/70	72%	no	-
6	RCA	50	140/75	52%	8%	83	110/70	66%	no	-
7	LAD	68	140/90	58%	13%	91	130/80	63%	no	-
8	RCA	70	140/100	45%	19%	99	120/80	47%	no	-
9	LCX	64	130/80	63%	4%	101	100/70	76%	no	-
10	LCX	62	110/70	67%	1%	107	100/60	76%	no	-
11	LAD	58	105/70	52%	3%	71	95/65	77%	no	-
12	LAD	69	135/90	41%	21%	81	130/90	51%	no	-
13	RCA	71	135/95	60%	5%	90	120/80	62%	yes	7%
14	LAD	56	100/65	58%	7%	89	90/60	64%	no	-
15	RCA	63	120/85	57%	8%	102	100/70	53%	no	-
16	LCX	55	130/90	63%	2%	74	120/80	52%	yes	11%
17	RCA	67	140/80	65%	11%	82	130/80	76%	no	-
18	LAD	67	120/75	49%	13%	86	110/70	60%	no	-
19	LAD	71	115/80	70%	1%	98	100/60	76%	no	-
20	LAD	86	120/80	61%	3%	100	120/80	66%	no	-
21	LCX	84	120/70	63%	4%	96	120/80	72%	no	-
22	LAD	81	130/80	55%	9%	98	115/80	50%	no	-
23	LAD	73	150/70	41%	14%	86	130/70	43%	no	-
24	LCX	61	140/70	48%	24%	70	110/65	50%	no	-
25	RCA	59	110/70	62%	7%	73	100/60	51%	yes	10%
26	LCX	60	120/70	57%	2%	81	110/70	50%	yes	14%
27	LCX	62	130/90	72%	5%	93	110/80	75%	no	-
28	LAD	58	160/80	58%	7%	74	130/90	65%	no	-
29	LAD	64	150/70	60%	2%	87	140/70	71%	no	-
30	LAD	66	120/80	51%	5%	78	110/70	52%	yes	6%
31	LAD	65	130/70	57%	7%	81	120/60	51%	yes	7%
32	RCA	66	115/75	61%	15%	93	120/80	68%	no	-
33	RCA	71	120/80	63%	6%	104	130/90	72%	no	-
34	LCX	59	120/90	62%	9%	87	120/80	70%	no	-
35	LAD	60	130/85	41%	21%	82	120/80	43%	no	-
36	LCX	53	140/75	47%	27%	69	120/80	45%	no	-
37	LAD	56	160/80	50%	10%	70	130/70	40%	yes	13%
38	LCX	58	120/80	64%	5%	82	110/75	76%	no	-
39	LAD	74	125/90	39%	26%	78	105/85	39%	no	-
40	LCX	68	140/80	61%	3%	83	105/70	67%	no	-
41	LAD	69	130/90	55%	6%	94	120/80	70%	no	-

*Estimated infarction area as a percentage of the total myocardial mass. †Maximum heart rate during pharmacological stress. ‡Estimated area of reversible defect as a percentage of the total myocardial mass. BP – blood pressure; EF – ejection fraction; HR – heart rate; LAD – left anterior descending; LCX – left circumflex; MI – myocardial infarction; RCA – right coronary artery.

Side effects occurred in 41% of the patients receiving dipyridamole. These were short-term and well-tolerated, and they were rapidly reversed using 250 mg of aminophylline. All the side effects, analytically for each individual, are listed in Table 3. The most common adverse events included headache, chest pain, ST-T changes on the electrocardiogram, and dizziness. All patients were discharged from the hospital uneventfully 2 days after the examination, on medical treatment. The individuals who were positive for ischemia will be re-evaluated after 6 months by both myocardial perfusion imaging and coronary angiography, according to the previously mentioned investigational protocol.

Discussion

This is a preliminary report, part of a more extensive protocol investigating the value of myocardial scintigraphy early (5-6 days) after primary PCI. The aim of

Table 3. Side effects from dipyridamole administration.

Patient	Chest pain	Headache	Dizziness	Hypotension	Dyspnea	AV block (degree)	Arrhythmia	ST-T changes*	Time to subside (min)†
1	No	Yes	No	No	No	No	No	No	4
2	No	Yes	No	No	Yes	No	No	No	5
3	Yes	No	No	No	No	No	No	No	3
4	No	No	No	No	No	No	No	Yes	3
5	No	No	No	No	No	No	No	No	-
6	No	No	No	No	No	No	No	No	-
7	No	No	No	No	No	No	No	No	-
8	No	Yes	No	No	No	No	No	No	1
9	No	Yes	No	No	No	No	No	No	4
10	No	No	No	No	No	No	No	No	-
11	No	Yes	No	Yes	No	No	No	No	5
12	Yes	No	No	No	No	No	No	No	3
13	Yes	No	No	No	No	No	No	Yes	3
14	No	No	No	Yes	No	No	No	No	3
15	No	No	No	No	No	No	No	No	-
16	No	No	No	No	No	No	No	No	-
17	Yes	No	No	No	No	No	No	No	2
18	Yes	No	No	No	No	No	No	No	2
19	No	No	No	No	No	No	No	No	-
20	No	No	No	No	No	No	No	No	-
21	No	No	No	No	No	No	No	No	-
22	No	No	No	No	No	No	No	Yes	3
23	No	No	No	No	No	No	No	Yes	3
24	No	Yes	No	No	No	No	No	No	2
25	Yes	No	No	No	No	No	No	No	2
26	No	No	No	No	No	No	No	Yes	3
27	No	No	No	No	No	No	No	No	-
28	No	No	No	No	No	No	No	No	-
29	No	No	No	No	No	No	No	No	4
30	No	Yes	Yes	No	No	No	No	No	5
31	Yes	Yes	No	No	No	1st	No	No	4
32	No	No	No	No	No	No	No	No	-
33	Yes	No	No	No	No	No	No	No	6
34	No	No	No	No	No	No	No	No	-
35	No	Yes	No	No	No	No	No	No	2
36	No	Yes	Yes	No	No	No	No	No	5
37	No	No	No	No	No	No	No	No	-
38	Yes	No	No	No	No	No	No	No	3
39	No	No	No	No	No	No	No	No	-
40	No	No	No	No	No	No	No	No	-
41	No	Yes	Yes	No	No	No	No	No	3
Total	9 (21%)	11 (27%)	3 (7.5%)	2 (5%)	1 (2.5%)	1 (2.5%)	0 (0%)	5 (12%)	

*ST segment depression <2 mm. †Minutes after aminophylline administration.

this protocol was to evaluate the safety of dipyridamole as a cardiac stressor in these patients, the severity and frequency of adverse effects, and finally, to clarify whether the ischemia seen on the early myocardial scintigraphic study represented a real phenomenon due to in-stent restenosis, or was merely an observation attributable to other causes.

To date, many studies have confirmed the safety of dipyridamole stress testing in patients who undergo successful percutaneous coronary angioplasty, 48 hours after the intervention.[12,13] In addition, an adequate number of publications support the safety of performing a dipyridamole stress test early after myocardial infarction.[14-16] In all the above studies, individuals experienced mild side effects, which subsided after aminophylline administration. This report additionally confirms the safety of dipyridamole use in patients who undergo successful primary PCI after an acute cardiac event. The noted incidence of hypotension, ST-T changes, chest pain and nausea was similar to that in previous studies, and only headache occurred more often.[17]

Some doubt has been expressed concerning the appropriateness of performing stress testing within a few days after coronary stent implantation, based on previous case reports that documented stent thrombosis within hours after stress testing.[18-19] On the other hand, a large and randomized study showed that myocardial scintigraphy using adenosine early after myocardial infarction is safe.[20] In order to avoid any stent thrombosis, we used only a pharmacological and not a treadmill stress test, and we examined our patients 5-6 days after the intervention, while all of them were on antiplatelet therapy.

The incidence of ischemic defects soon after successful coronary balloon angioplasty was notably higher (30-50%) during the early days of PCI.[21-23] Whether these reversible perfusion defects were caused by real intracoronary anatomic anomalies, such as residual restenosis, intraluminal debris, or elastic recoil, is still a matter of debate, since their incidences ought to be significantly reduced in the era of coronary stenting – a fact that has not yet been definitely proven. Rodés-Cabau et al[24] reported that coronary angioplasty with stent implantation is associated with a 17% rate of reversible perfusion defects early after the procedure. However, Kim at al[25] observed reversible perfusion defects in 36% of lesions after stent implantation, which is as high as for balloon angioplasty. Similar results have been found by other studies. Jaffe et al[26] evaluated the incidence of reversible perfusion defects after balloon angioplasty and stenting, and found that they were very similar (32% for balloon angioplasty and 36% for stenting). Nagaoka et al[27] also observed the same rate (36%) of reversible defects in patients, four days after stent implantation.

In previous studies, regional perfusion defects associated with endothelial dysfunction and diminished coronary flow reserve were more frequently observed soon after PCI.[28-30] Versaci et al[31] demonstrated similar findings using adenosine thallium-201 scintigraphy after balloon angioplasty and stent implantation. Kern et al[32] measured coronary flow velocity reserve in arteries receiving a stent and in an angiographically normal reference vessel, and found that at least half of the functional abnormalities after stent implantation were caused by global microvascular disease. Other authors[25] expected that perfusion defects occuring soon after stent implantation would reflect the regional perfusion of stented arteries more precisely. Thus, they imaged patients within 24 h of stent implantation and found that myocardial perfusion imaging predicts late restenosis, since the incidence of late restenosis was significantly higher in patients with a reversible perfusion defect (47%) than in those without (18%).

On the other hand, none of the abovementioned reports dealt with patients who underwent primary PCI. The only available information dealing with myocardial scintigraphy early after primary PCI is in the studies of Kaltoft et al,[33,34] but these authors merely examined the size of the infarcted area using only rest Sestamibi myocardial scintigraphy. As far as we know, this is the first prospective study to deal with this group of patients. The observed incidence of reversible perfusion defects was 17% in our patients. Many theories have been posited in order to explain this phenomenon. Impairment of endothelial function after the dilatation of the vessel, distal micro-embolization from the targeted lesion and endothelial dysfunction due to coronary stenting have been postulated.[35-40]

The safety of performing myocardial scintigraphy 5-6 days after primary PCI has been proven by the fact that all our patients were discharged from the hospital with no anginal pain or any other symptoms. We do not know if the reversible defects seen in 7 of these patients corresponded to real ischemia, or were due to endothelial dysfunction. According to our protocol, and as far as they remain asymptomatic, a new myocardial perfusion study in all patients who were positive for ischemia, combined with coronary angi-

ography, will clarify the real cause of the reversible defects.

We conclude that myocardial perfusion imaging using dipyridamole stress testing is a safe procedure for evaluating patients who undergo primary PCI, 5-6 days after the procedure. A high incidence of reversible perfusion defects (17%) was noted in our patient cohort. This phenomenon will be fully explored after six months, in order to clarify whether this was due to real in-stent restenosis, or was merely attributable to microvascular dysfunction, which is commonly seen after intracoronary interventions. If the ischemia seen in the 5-6 days post-revascularization study was due to in-stent restenosis, then myocardial scintigraphy performed early can be used as a predictor of stent occlusion, gaining valuable time and resources.

Limitations of the study

The two main limitations of the study are that the number of patients was borderline for drawing firm conclusions and that there is a lack of evidence to support the performance of an imaging test in asymptomatic patients with coronary artery disease, according to both the European and American Guidelines and appropriateness criteria. It must be considered, however, that the cohort of patients enrolled was somewhat unique. All of them suffered an acute myocardial infarction, underwent primary PCI, were imaged early after the intervention, and only the patients with an abnormal study (symptomatic or not) will be re-evaluated after 6 months, in order to clarify whether the scintigram early after primary PCI may predict future restenosis. This group is not included in any guidelines, nor in any appropriateness criteria. Additionally, this patient group was a part of a larger cohort, in the context of a PhD thesis of one of the authors, investigating whether perfusion abnormalities seen on myocardial perfusion imaging 6 days after primary PCI are a predictive factor for stent restenosis.

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