ORIGINAL RESEARCH PAPER

Cardiology

PREDICTION OF MYOCARDIAL VIABILITY -BY PRESSURE WIRE GUIDED ASSESSMENT OF MICRO VASCULAR RESISTANCE USING A CALIBRATED UPSTREAM BALLOON OBSTRUCTION AND ITS CORRELATION WITH CARDIAC MRI IN PATIENTS WITH ST ELEVATION MYOCARDIAL INFARCTION

KEY WORDS: Myocardial viability, FFR, Myocardial viability

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BACKGROUND:

Patients presenting with STEMI are candidates for reperfusion (either pharmacological or catheter based) to restore flow in occluded coronary arteries. Primary percutaneous coronary intervention (PCI) is recommended in PCI capable hospital in patients with STEMI of less than 12 hours duration, including those who have contraindications to fibrinolytic therapy or patients with cardiogenic shock or with acute severe heart failure , irrespective of the time delay from first medical contact[1].

After PCI microvascular damage can be assessed by angiographic TIMI grade flow. Patients with angiographic no-reflow (absent of angiographic flow)/ slow flow(inadequate angiographic flow) usually have micro vascular dysfunction.

Micro vascular integrity can be assessed by measuring the change in Fractional Flow Reserve before and after vasodilatation with adenosine by using a calibrated balloon obstruction[2].

NO REFLOW – Defined as absence of myocardial perfusion (TIMI 0) through a given segment of coronary circulation without angiographic evidence of mechanical vessel obstruction (Dissection, spasm, thrombus, significant residual stenosis)3

SLOW FLOW -Defined as inadequate myocardial perfusion (TIMI 1-2) through a given segment of coronary circulation without angiographic evidence of mechanical vessel obstruction (Dissection, spasm, thrombus, significant residual stenosis)3

Overall mechanisms for micro vascular dysfunction are:

1. Coronary vasospasm.
2. Distal embolization of thrombus and other debris.
3. Oxygen free radical mediated injury.
4. Capillary plugging by erythrocytes, neutrophils , platelets and fibrin.
5. Intra cellular/interstitial edema with intra mural haemorrhage.

Techniques for demonstration of no reflow:

1. Myocardial scintigraphy.
2. Myocardial contrast echocardiography.
3. Nuclear magnetic resonance.
4. Positron emission tomography.
5. Coronary angiogram.
6. **Intra coronary pressure measurement.**
7. Intra coronary doppler.
8. Intravascular ultrasound and ECG.

FRACTIONAL FLOW RESERVE

DEFINITION:

Pressure drop distal to a stenosis to that of its proximal part.

FFR=Pd-Pv/Pao-Pv

(Pd is distal pressure to stenosis , Pao is aortic pressure and Pv is myocardial venous pressure)

Which is approximately Pd/Pao (Low myocardial venous pressures are usually excluded).

FFR is a better predictor of physiological significance of epicardial coronary artery obstruction than angiographic severity .

FFR of < 0.8 indicate physiologically significant coronary obstruction[4,5]

FFR does not necessarily predict preservation of contractile reserve in the distal myocardium[6]

Distal microvascular integrity can be assessed by a pressure transducer. Conventional pressure wire measurement does not reflect microvascular function because transducer is upstream of the subtended myocardium.

As hypothesized in the previous study[2], resting Pd/Pao is set to 0.8 by partially obstructing the balloon, and downstream pressure drop during the hyperaemia is calculated as

$$\Delta FFR\ 0.8 = Pd(b)/Pao(b) - Pd(h)/Pao(h)$$
$$= 0.8 - Pd(h)/Pao(h).$$

Where Pd(b) and Pd(h) indicate distal coronary artery pressure at baseline and during hyperemia and Pao(b) and Pao(h) indicate aortic pressure at baseline and during hyperemia,

Resting Pd/Pao was set to 0.8 by partially obstructing the balloon, and downstream pressure drop during the hyperaemia i.e ΔFFR 0.8 inversely corresponds to the extent of infarction in the territory subtended by the lesion (trans mural extent of infarct in MRI) and is directly proportional to the viability in that territory. (Myocardial resistance is fixed if segment is nonviable and variable if segment is viable).

A simplified coronary hydrodynamic model assumes two serial resistance in atherosclerotic coronary artery, one in the obstructed epicardial coronary segment and second in the distal coronary microvasculature. Nonviable or dysfunctional myocardium has impaired distal microvascular flow reserve[7-10].Successful stent therapy of an isolated culprit conductance coronary artery stenosis essentially normalizes pressure based fractional flow reserve (the ratio of distal coronary to aortic pressure during the hyperaemia), fixes the local cross sectional area despite pulsatile flow, and abates the pressure contribution of collateral arteries[11-15].

The epicardial coronary artery distal to the stent is still upstream the microvascular circulation and contributes minimally to the pressure loss during vasodilatation. Partially inflating an angioplasty balloon inside the stent, calibrated to an arbitrary resting pressure, imposes a resistance proximal to the pressure transducer. This resistance creates additional pressure loss only if pharmacological vasodilatation augments blood flow, i.e, in a preserved myocardial territory. Thus the pressure transducer is rendered sensitive to microvascular flow change.

In acute MI, the differentiation of stunned and necrotic tissue of dysfunction myocardium is possible with gadolinium enhanced cardiovascular magnetic resonance imaging using a inversion recovery gradient echo technique. Necrotic area exhibits a higher signal intensity 15-20 min after contrast injection.

Due to spatial resolution of CMR, the transmural extent of infarct can be determined and is a predictor of contractile recovery. Late gadolinium enhancement was defined as signal activity greater than two standard deviations from remote normal myocardium [16-18]summed from all slices.

The risk segment area was defined as the transmural area extent bounded by the lateral margins of the late enhancement area.

PURPOSE OF PRESENT STUDY:

The extent of myocardial salvage after primary percutaneous transluminal coronary angioplasty (PTCA) in acute myocardial infarction (AMI) is variable and cannot be predicted on the basis of either vessel patency or early regional wall motion assessment.

However, fractional flow reserve alone does not necessarily predict preservation or recovery of contractile reserve in states of distal myocardial disease, whether infarct, myopathy, or hypertrophy.

A test of distal microvascular integrity using only a pressure transducer would be a valuable tool. Purpose of our study was to predict the viability of the target territory subtended by the lesion and overall outcome during the primary angioplasty itself by using a pressure wire and correlating the above results with cardiac MRI for myocardial viability.

AIMS AND OBJECTIVES:

1) To predict myocardial viability by a measuring change in FFR and correlating it with cardiac MRI for transmural extent of infarct in patient with ST elevation MI presenting within 12hrs of onset of chest pain.
2) Correlation of FFR 0.8 with serial ECG changes, troponin T levels, LV ejection fraction, TIMI angiographic myocardial blush score and Killip's class.

MATERIAL AND METHODS:

Study site

Study was carried out in **CARE HOSPITAL,** HYDERABAD, INDIA

Study population

All patients presenting to Cardiology Department , CARE hospital, Banjara Hills for treatment of STEMI were evaluated as follows.

History and physical examination.
ECG
Cardiac markers, RFT
2D echo
Coronary angiography

After the coronary angiography, patients satisfying the inclusion criteria were included in the study.

Type Of study – Prospective observational study.

Study procedure

After the culprit lesion was successfully treated with a coronary stent, a coronary guide wire incorporating a pressure transducer was positioned 2-3 cm distal to the implanted stent. An undersized balloon shorter (8-12 mm length) and narrower (0.5mm less than the nominal stent delivery balloon diameter) was positioned inside the stent lumen. The undersized balloon was then inflated to achieve a mean distal coronary pressure 80% of mean aortic pressure at rest. Subjects were excluded if Pd/Pao fluctuated more than 0.01 at baseline. Coronary hyperaemia was then induced using intravenous adenosine 140mcg/kg/min and FFR calculated after 2 min. Change in FFR was calculated by

ΔFFR 0.8=Pd(b)/Pao(b)-Pd(h)/Pao(h)
=0.8-Pd(h)/Pao(h).

Pressure wire –Boston
FFR machine- St Jude -Model 3059833

Cardiac MRI was done for all the subjects and transmural extent of infarct was measured.
Average TEI=LGE area/Infarct segment area.
TEI-Transmural extent of infarct.
LGE-Late gadolinium enhancement.

Area –Measured from in a stack of six late gadolinium enhancement MRI images.

Cardiac MRI used – Siemens 1.5 Tesla

- Killip's class was noted at the time of admission.
- Daily ECGs were taken for 3 days.
- Troponin levels were sent after 8 hrs of onset of pain/symptoms.
- Left ventricular ejection fraction was calculated at presentation and at discharge.

ECG was done after PTCA at 2hrs, 24 hrs, 48 hrs and 72 hrs. If post PTCA ECG showed ST segment resolution of 50%, then it was considered as significant ECG resolution.

The sample size required for the correlation coefficient of 0.806 to show the significance at 99.9 significance level and 95% power was 21 cases.

Time frame of study

Necessary approval of scientific committee and hospital ethics committee was obtained. Study was carried out over a duration of one and a half year beginning from September 2013 to March 2015.The data was analysed in detail in the next 60 days and observations and conclusions of study were established by June 2015.

INCLUSION CRITERIA

1) Patients who were undergoing percutaneous coronary intervention for STEMI.
2) Culprit lesion should be in proximal and middle segments of a major epicardial coronary arteries with a reference diameter of 2.75 and 4 mm which could be successfully treated with a coronary stent.

EXCLUSION CRITERIA

1) Significant obstructive coronary artery lesion (>50%) in the target vessel distal to culprit site.
2) Previous infarct other than in culprit vessel
3) CKD patients requiring renal replacement therapy.
4) Left ventricular hypertrophy with wall thickness >12mm or hypertrophic cardiomyopathy.
5) Cardiogenic shock or requiring catecholamine infusion.
6) Collateral flow to the target vessel more than angiographic grade1.
7) Excessive baseline variability of distal coronary artery pressure during investigational balloon obstruction.

STUDY ANALYSIS

Continuous parameters were compared using a student *'t'* test. Categorical and ordinal parameters were compared using a chi square test. The association between ΔFFR 0.8 and other parameters was determined using a univariate logisticregression models.

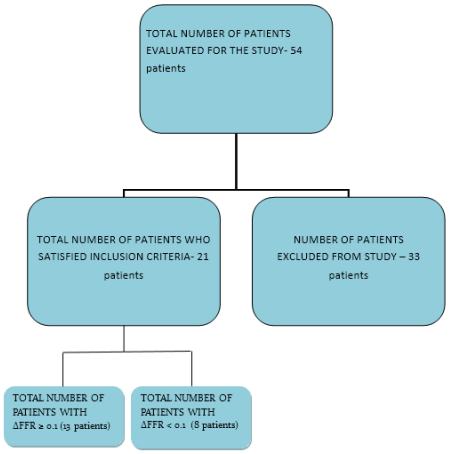

FIG 1: Flow Chart Of The Study

RESULTS:

A total of 54 people were evaluated for the study, out of that 21 cases were included into study. Out of 21 cases, 1(5%) case was in 31-40 years group, 2 (9%) cases were in 41-50 years group,10(48%) cases were in 51-60 years group, 2(9%) cases were in 61-70 years group and 1(5%) case was in >70 years group . Out of 21 patients, 16 (76%) cases were males and 5(24 %) cases were females.

Out of 21 cases all patients had chest pain as their presenting complaint, 16 patients (76%) had shortness of breath, 2 patients(9%) had palpitations, no patient had syncope or any other symptoms.

Out of 21 patients , 57 % patients had diabetes, 67% patients had hypertension, 52 % patients had dyslipidemia, 57 % patients had smoking history and 48% patients had a family history of coronary artery disease.

TABLE 1 Baseline Clinical And Angiographic Characteristics

CLINICAL	**N=21**
AGE (YEARS)	56±17
SEX (MALE/FEMALE)	16/5
CORONARY RISK FACTORS(n,%)	
DIABETES	12(57)
HYPERTENSION	14(67)
SMOKING	11(52)
HYPERLIPIDEMIA	12(57)
FAMILY HISTORY	10(48)
MEAN LV EJECTION FRACTION AT ADMISSION (%)	36±7
TIME INTERVAL BETWEEN MRI AND PCI IN DAYS	2.9±.3
CULPRIT VESSEL n(%)	
LAD	13(62)
LCX	5(24)
RCA	3(14)
LOCATION OF LESION n(%)	
PROXIMAL	16(76)
MID	5 (24)
REFERENCE VESSEL SIZE (mm)	3.25±0.50
TIMI FLOW AFTER PCI n(%)	
TIMI 1	2(9)
TIMI 2	5(24)
TIMI 3	14(67)
TIMI ANGIOGRAPHIC MYOCARDIAL BLUSH GRADE AFTER PCI n(%)	
GRADE 0/1	2(9)
GRADE 2	6 (29)
GRADE 3	13 (62)

Total 21 patients were taken up for study. Mean ejection fraction at the time of admission was 36±7%. At the time of admission, 71 % patients were in Killip class 1, 24% patients were in Killip class 2 and 5% patients were in Killip class 3.

After coronary angiogram, 62 % patients had LAD lesion, 24% patients had LCX lesion and 24 % had RCA lesion. 76% patients had lesion in the proximal segment and 24 % patients had mid segment lesion. Reference vessel size was 3.25 ±0.50 mm. 13 patients had TIMI flow grade 0 before PCI, 4 patients had TIMI flow grade 1, 2 patients had TIMI flow grade 2 and 2 patients had TIMI flow grade 3.After primary PCI and stenting, ΔFFR 0.8 was calculated for all patients. 14 patients had ΔFFR 0.8 >0.1 and 8 patients had ΔFFR 0.8 <0.1. Mean time for reperfusion time for all patients was 5.6±3.8 hours.

TIMI flow after PCI – 2 patients had TIMI flow 1, 5 patients had TIMI flow 2 and 14 patients had TIMI flow grade 3. Myocardial blush score after PCI - 2 patients had grade 0/1, 6 patients had grade 2 and 13 patients had grade 3.Time interval between cardiac MRI and PCI was 2.9 ±0.3 days.

TABLE 2. Clinical And Lab Parameters According To Δffr 0.8 With Threshold Of 0.1

	ΔFFR0.8 >0.1 N=13	**ΔFFR0.8 <0.1 N=8**	**p value**
LV EF %	49±6	37±5	t (19)= 4.75, p=0.001
PEAK TROPONIN T	0.21±0.24	0.63±0.3	t (19)= 3.54, p=0.002
Time to reperfusion time in STEMI (In hours)	4.2±1.8	7.8±1.2	t (19)= 4.75, p=0.001
Time interval of MRI and PCI (In Days)	2.9±0.3	3±0.3	t (19)= 0.75, p=0.46
Culprit vessel, n (LAD/LCX/RCA)	7/3/3	6/2/0	Chi-sq(2)=2.2, p=0.33
Location of lesion(PROXIMAL/ MID)	9/4	7/1	Chi-sq(1)=1.8, p=0.67
TIMI Flow Grade before PCI , n Grade 0/1/2/3	7/3/1/2	6/1/1/0	Chi-sq(3)=2.0, p=0.78
TEI by MRI(%)	33.7±14.9	72.6±7.4	t (19)= 6.85, p=0.001
TIMI Angiographic Myocardial Blush score Grade 0/1 (n,%) Grade 2(n,%) Grade 3(n,%)	0 2 11	3 3 2	Chi-sq(2)=8.74, p=0.013
Significant ECG resolution (Present/absent)	9/4	3/5	Chi-sq(1)=0.94, p=0.33

Out of 21 patients ,13 patients had ΔFFR 0.8 >0.1 and 8 patients had ΔFFR 0.8 <0.1. There was significant change in LVEF at discharge in 2 groups, patients with ΔFFR 0.8 >0.1 had ejection fraction 49±6 and patients with ΔFFR 0.8 <0.1 had 37±5 and p value was 0.001.

There was no significant change in the time interval between PCI and MRI done in both the groups, patients with ΔFFR 0.8> 0.1 had time interval of 2.9±0.3 hours between PCI and MRI and in patients with ΔFFR 0.8 <0.1 had time interval of 3±0.3 hours and p value was 0.46.

There was significant change in peak troponin levels in 2 groups , patients with ΔFFR 0.8 >0.1 had peak troponin level of 0.21 whereas patients with ΔFFR 0.8 <0.1 had peak troponin levels of 0.63 and p value was 0.002.

There was significant change in time to reperfusion in 2 groups, patients with ΔFFR 0.8> 0.1 had time to reperfusion of 4.2 ±1.8 hours and patients with ΔFFR 0.8 <0.1 had time to reperfusion of 7.8 ±1.2 hours and p value was 0.001.

Out of 21 patients LAD territory was involved in 13 patients, in

those 7 patients had ΔFFR 0.8> 0.1 and 6 patients had ΔFFR 0.8 <0.1. 5 patients had LCX territory involved, in those 3 patients had ΔFFR 0.8> 0.1 and 2 patients had ΔFFR 0.8< 0.1. 3 patients had RCA territory involved , all 3 patients had ΔFFR 0.8> 0.1

Out of 21 patients 16 patients had proximal lesion, in those 9 patients had ΔFFR 0.8> 0.1 and 7 patients had FFR 0.8< 0.1 . 5 patients had distal lesion, in those ΔFFR 0.8> 0.1 was seen in 4 patients and 1 patient had ΔFFR 0.8< 0.1. There was no statistical significant difference in both groups.

69 % of patients in ΔFFR 0.8> 0.1 group had significant ST resolution whereas only 37 % patients in ΔFFR 0.8< 0.1 group had significant ST resolution but there was no statistical significant difference noted in both groups.

Grade 3 myocardial blush was seen in 13 patients, 11 patients were in ΔFFR 0.8> 0.1 group and 2 patients were in ΔFFR 0.8< 0.1 group. Myocardial blush score 2 was seen in 5 patients out of which 2 patients were in ΔFFR0.8>0.1 group and 3 patients ΔFFR 0.8< 0.1 group. Myocardial blush score 0/1 was seen in 3 patients, all 3 patients belonged to ΔFFR 0.8< 0.1 group. There was a significant change in myocardial blush score in both groups and p value was 0.013

Based on gadolinium enhancement on cardiac MRI , average transmural extent of infarct (TEI%) in patients with ΔFFR 0.8> 0.1 was 34 % whereas in patients with ΔFFR 0.8< 0.1 was 73%. This indicated that there was a significant change in mean transmural extent of infarct in both groups and p value was 0.001.

Patients with FFR 0.8> 0.1 had significantly less transmural extent of infarct compared to infarcted territory and most of the patients had significant viable myocardium.

DISCUSSION:

We had conducted this study in a tertiary care centre. Required approval of Hospital ethics committee and scientific committee was taken before study. In the study period (September 2013 to March 2015), we recruited 54 patients in study after informed written consent. As per inclusion criteria only 21 people were finally included in the study.

There were 16 males (76%), 5 females (24%) in the study . According to age distribution, 1(5%) patient was in the 31-40 years group, 2(9%) patients were in 41-50 years group, 10(48%) patients were in 51-60 years group, 2(9%) patients were in 61-70 years group and 1(5 %) patient was in >70 years group. Mean age of study population was 56 years.

The commonest presenting symptom in our study group was chest pain (n=21, 100%), followed by shortness of breath (n=16, 76%) and palpitations (n=2, 9%). No patients had presyncope or syncope.

Regarding co-morbidities, 57% patients had diabetes, 67% patients had hypertension, 57% patients had dyslipidemia, 52% patients had a history of smoking and 48% patients had a family history of coronary artery disease.

Mean ejection fraction at the time of admission was 36 ± 7%. At the time of admission 71% patients were in Killip class 1 , 24% patients were in Killip class 2 and 5% patients were in Killip class 3. After coronary angiogram, 62 % patients had LAD lesion, 24% patients had LCX lesion and 24 % had RCA lesion. 76% patients had lesion in the proximal segment and 24 % patients had mid segment lesion. 13 patients had TIMI flow grade 0 before PCI, 4 patients had TIMI flow grade 1, 2 patients had TIMI flow grade 2 and 2 patients had TIMI flow grade 3.

After primary PCI and stenting, ΔFFR 0.8 was calculated for all patients. 14 patients had ΔFFR 0.8 >0.1 and 8 patients had ΔFFR 0.8 <0.1.TIMI flow after PCI – 2 patients had TIMI flow 1, 5 patients had TIMI flow 2 and 14 patients had TIMI flow grade 3. Myocardial blush score after PCI – 2 patients had grade 0/1, 6 patients had grade 2 and 13 patients had grade 3.

Based on our study we found that there was a significant change in LV ejection fraction at discharge in two groups (p value of 0.001), patients with ΔFFR 0.8 >0.1 had LV ejection fraction at discharge of 49±6% compared with 37±5% in patients with ΔFFR 0.8 <0.1. This finding suggested that a pressure drop of >0.1 after intravascular adenosine indicated good microvascular integrity.

There was a significant change in peak troponin levels in both groups (p value of 0.002). Patients with ΔFFR 0.8 >0.1 had mean troponin level of 0.21 compared with 0.63 in patients with ΔFFR 0.8 <0.1. This showed that pressure drop of <0.1 indicated significant loss of microvascular integrity and more myocardial injury within that territory.

Time to reperfusion for all patients was 5.6±3.8 hours. There was a significant change in time to reperfusion in both groups (p value of 0.001); patients with ΔFFR 0.8 >0.1 had a mean reperfusion time of 4.2 hrs compared with 7.8 hrs in patients with ΔFFR 0.8 <0.1. This finding suggested that as the time to reperfusion increases, there is an increase in myocardial injury and loss of microvascular integrity.

There was a significant change in the transmural extent of infarction in both groups(p value of 0.001); patients with ΔFFR 0.8 >0.1 had TEI of 34% as compared to 73% in those with ΔFFR 0.8 <0.1. This indicated that patients with ΔFFR 0.8 >0.1 had good microvascular integrity and that segment was viable by cardiac MRI.

69% of the patients in ΔFFR 0.8 >0.1 group had proximal lesion whereas 88 % of patients had proximal lesion in ΔFFR 0.8 <0.1group. There was no statistically significant difference in both groups (p value of 0.67).

69 % of patients in ΔFFR 0.8 >0.1 group had significant ST resolution whereas only 37 % patients in ΔFFR 0.8 <0.1 group had significant ST resolution but there was no statistically significant difference between both groups.

54 % of patients with ΔFFR 0.8> 0.1 group had LAD territory involvement compared to 75 % in FFR0.8<0.1 group.

Grade 3 myocardial blush was seen in 11 out of 13 patients in ΔFFR 0.8 >0.1 group and 2 out of 8 patients in ΔFFR 0.8 <0.1 group (p value of 0.013). This indicated that patients with ΔFFR 0.8 <0.1 had significant loss of microvascular integrity leading to decrease in myocardial blush score.

There was a linear relation between ΔFFR 0.8 and extent of viable myocardium by cardiac MRI subtended by the index coronary artery lesion.

Based on our results,ΔFFR 0.8 correlated well with level of troponin T elevation , reperfusion time, Post PCI TIMI angiographic myocardial blush score, Killip's class at the time of admission and LV ejection fraction at discharge.

Overall, patients with ΔFFR 0.8 >0.1 had less peak troponin elevation, early presentation i.e time to reperfusion was less, less transmural extent of infarction (TEI%) by cardiac MRI, good post PCI myocardial blush score (score 2,3) and improved LV ejection fraction at discharge. However, ΔFFR 0.8 did not correlate significantly with ECG resolution, arterial territory involvement (LAD vs non LAD) and location of lesion (proximal vs distal).

In the study conducted by Kim H et al[2], microvascular function was measured immediately after coronary artery stenting by a simple pressure wire (by proximally occluding with balloon) and this correlated well with myocardial viability by MRI. Moreover, they identified a convenient threshold value ΔFFR 0.8 of 0.1 that corresponded to a transmural extent of infarction of 50%, which predicted recovery of myocardial contractile function after revascularization. These findings appeared consistent irrespective of coronary artery territory or clinical syndrome. They also observed a linear relation between ΔFFR 0.8, and extent of viable

myocardium subtended by the index coronary artery lesion.

Both studies showed that ΔFFR 0.8 is a simple predictor of downstream myocardial viability immediately after percutaneous stenting of patients with ST elevation myocardial infarction and this correlated fairly with cardiac MRI for myocardial viability.

CONCLUSIONS:

Based upon our study we conclude that by using simple pressure wire, microvascular integrity can be well assessed in patients with ST elevation myocardial infarction after primary PCI and stenting.

Change in pressure >0.1 induced after pharmacological vasodilator stress indicated significant myocardial reserve i.e there was a significant viability in that territory .

Patients with ΔFFR 0.8 >0.1 usually have
1) Less peak troponin elevation.
2) Early presentation i.e time to reperfusion time is less.
3) Less transmural extent of infarct (TEI%).
4) Good post PCI myocardial blush score.
5) Improved LV ejection fraction at discharge.

ΔFFR 0.8 significantly correlated with level of troponin elevation, time to reperfusion, Post PCI TIMI angiographic myocardial blush score, Killip's class at the time of admission and LV ejection fraction at discharge, whereas ΔFFR 0.8 did not significantly correlate with ECG resolution, arterial territory involvement (LAD vs non LAD) and location of lesion (proximal vs distal).

LIMITATIONS:

1. Drawback of our study is failure to obtain baseline and hyperemic pressure gradients and matching thermodilution transit time, for comparative ''delta FFR'' and Index of Microvascular Resistance measurements.
2. We measured infarct area by MRI late gadolinium (average transmural extent of infarction) and we assessed infarcted area using a patient- and vessel-Specific index which is different from standard American Society of Echocardiography segmentation.
3. Our approach is unsuitable for patients not undergoing PCI.

RECOMMENDATIONS:

Based upon our study results we recommend that in patients with ST elevation myocardial infarction after primary PCI and stenting, ΔFFR0.8 is a simple predictor of downstream myocardial viability which fairly correlates with viability on cardiac MRI.

Patients with ΔFFR 0.8 >0.1 after vasodilator stress usually have good microvascular integrity and usually have viable myocardium in that territory.

REFERENCES

1. O 'Gara et al. 2013 ACHF/AHA Guideline for management of ST Elevation myocardial infarction: Executive summary: A Report of American college of Cardiology foundation/ American Heart Association Task Force on Practice Guidelines.
2. Kim H, Park H et al. Pressure-Wire Based Assessment of Microvascular Resistance Using Calibrated Upstream Balloon Obstruction: A Predictor of Myocardial Viability. Catheterization and cardiovascular intervention journal 80:581-589(2012).
3. Rezkella SH, Kloner et al.No reflow phenomenon. Circulation 2002:105(5):656-62.
4. Kern MJ, Lerman A, Bech JW, De Bruyne B, Eeckhout E, et al. Physiological assessment of coronary artery disease in the cardiac catheterization laboratory: A scientific statement from the American Heart Association Committee on Diagnostic and Interventional Cardiac Catheterization, Council on Clinical Cardiology.Circulation 2006;114:1321–1341.
5. Tonino PA, De Bruyne B, Pijls NH, Siebert U, Ikeno F.Fractional flow reserve versus angiography for guiding percutaneous coronary intervention. N Engl J Med 2009;360:213–224.
6. Kocaman S, Sahinarslan A, Arslan U, Timurkaynak T. The delta fractional flow reserve can predict lesion severity and long-term prognosis. Atherosclerosis 2009;203:178–184.
7. De Bruyne B, Pijls N, Bartunek J, Kulecki K, Bech J, De Winter H, Van Crombrugge P, Heyndrickx G, Wijns W. Fractional flow reserve in patients with prior myocardial infarction. Circulation 2001;104:157–162.
8. Kern MJ, Samady H. Current concepts of integrated coronary physiology in the catheterization laboratory. J Am Coll Cardiol 2010;55:173–185.
9. Marques K, Knaapen P, Boellaard R, Lammertsma A, Westerhof N, Visser F. Microvascular function in viable myocardium after chronic infarction does not influence fractional flow reserve measurements. J Nucl Med 2007;48:1987–1992.
10. Marques K, Knaapen P, Boellaard R, Westerhof N, Lammertsma A, Visser C, Visser F. Hyperaemic microvascular resistance is not increased in viable myocardium after chronic myocardial infarction. Eur Heart J 2007;28:2320–2325.
11. Fearon W, Aarnoudse W, Pijls N, De Bruyne B, Balsam L, Cooke D, Robbins R, Fitzgerald P, Yeung A, Yock P. Microvascular resistance is not influenced by epicardial coronary artery stenosis severity: Experimental validation. Circulation 2004;109:2269–2272.
12. Aarnoudse W, Fearon W, Manoharan G, Geven M, van de Vosse F, Rutten M, De Bruyne B, Pijls N. Epicardial stenosis severity does not affect minimal microcirculatory resistance. Circulation 2004;110:2137–2142.
13. Ng M, Yeung A, Fearon W. Invasive assessment of the coronary microcirculation: Superior reproducibility and less hemodynamic dependence of index of microcirculatory resistance compared with coronary flow reserve. Circulation 2006;113:2054–2061.
14. Fearon W, Shah M, Ng M, Brinton T, Wilson A, et al. Predictive value of the index of microcirculatory resistance in patients with ST-segment elevation myocardial infarction. J Am Coll Cardiol 2008;51:560–565.
15. Lim HS, Yoon MH, Tahk SJ, Yang HM, Choi BJ, Choi SY,Sheen SS, Hwang GS, Kang SJ, Shin JH. Usefulness of the index of microcirculatory resistance for invasively assessing myocardial viability immediately after primary angioplasty for anterior myocardial infarction. Eur Heart J 2009;30:2854–2860.
16. Berry C, Kellman P, Mancini C, Chen MY, Bandettini WP,Lowrey T, Hsu LY, Aletras AH, Arai AE. Magnetic resonance imaging delineates the ischemic area at risk and myocardial salvage in patients with acute myocardial infarction. Circ Cardiovascular Imaging 2010;3:527–535.
17. O'Regan D, Ahmed R, Neuwirth C, Tan Y, Durighel G, Hajnal J,Nadra I, Corbett S, Cook S. Cardiac MRI of myocardial salvage at the peri-infarct border zones after primary coronaryintervention.Am J Physiol Heart Circ Physiol 2009;297 :H 340–H346
18. Kim RJ, Wu E, Rafael A, Chen EL, Parker MA, Simonetti O,Klocke FJ, Bonow RO, Judd RM. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. N Engl J Med 2000;343:1445–1453.