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ORIGINAL ARTICLE

Non-ST Elevation Myocardial Infarction with Occluded Artery and its Clinical Implications

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Background This study aimed to determine the prevalence and differences between Non-ST elevation Myocardial Infarction (NSTEMI) with an occluded culprit artery (NSTEMIOA) and NSTEMI with a patent culprit artery (NSTEMIPA).

Methods We conducted a retrospective observational study on NSTEMI patients admitted between 01/01/2010 to 30/06/2010. The inclusion criteria were diagnosis of NSTEMI and inpatient coronary angiogram. Patients were followed up for 12 months. The primary endpoints of interest were the differentiating characteristics between NSTEMIOA and NSTEMIOA. The secondary endpoints of interest were clinical outcomes in 12 months and the effect of delay in percutaneous coronary intervention on the extent of myocardial damage.

Results Of 143 NSTEMI patients, 34 (24%) patients had NSTEMIOA. NSTEMIOA patients had higher rates of hypercholesterolaemia (85.3% vs. 64.2%, p = 0.015), ST-depression abnormality on ECGs (32.4% vs. 11.9%, p = 0.008), multi-vessel disease on coronary angiogram (76.5% vs. 48.6%, p = 0.004) and LV dysfunction on echo (75% vs 48%, p = 0.016). At 12 months post-discharge, there was a trend of higher heart failure rate in NSTEMIOA subgroup but otherwise no difference between the two cohorts in death, myocardial infarction, revascularisation, arrhythmia, and re-admission for angina. There was no correlation between the peak CK level and the timing of percutaneous revascularisation in both cohorts.

Conclusions A quarter of NSTEMI patients had an occluded culprit coronary artery. They were more likely to have hypercholesterolaemia, ECG abnormalities, multi-vessel disease and LV dysfunction.

Keywords Myocardial infarction • Acute Coronary Syndrome • Coronary occlusion • Angioplasty • Coronary artery

Introduction

Non-ST Elevation Myocardial Infarction (NSTEMI) is often thought to be due to incomplete occlusion of the culprit artery whilst ST-Elevation Myocardial Infarction (STEMI) is often thought to be due to complete occlusion of the culprit artery [1–6]. However, studies have shown that about a quarter of NSTEMI are actually due to complete occlusion of the culprit artery, not dissimilar to the findings of STEMI on coronary angiography [7–9]. Nonetheless, NSTEMIOA is

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often treated as less urgent than STEMI. There had been minimal data on the differences between NSTEMI with occluded artery (NSTEMIOA) and NSTEMI with patent artery (NSTEMIPA) in terms of clinical characteristics and outcomes, particularly in the context of early versus late percutaneous revascularisation. The objectives of this study were to investigate the demographics, clinical risk profile, angiographic differences between these two cohorts, and also to investigate the outcomes of NSTEMIOA and NSTEMIPA in terms of the timing of percutaneous revascularisation.

Material and Methods

Inclusion and Exclusion Criteria

This study was a single-centre, retrospective, observational study conducted at a community based tertiary hospital with a primary PCI facility. The inclusion criteria for this study were:

1. Acute myocardial infarction (AMI) cases between 01/01/2010 and 30/06/2010 presenting to the Emergency Department or Coronary Care Unit. AMI was defined as the presentation of Acute Coronary Syndrome (ACS) with peak troponin I of >0.03 μg/L and peak CK of >200 IU/L.
2. Diagnosis of NSTEMI in medical record with confirmation of no ST elevation or new left bundle branch block on ECG.
3. Inpatient invasive angiogram during the hospitalisation for NSTEMI

The exclusion criteria of this study were:

1. Patients who had previously undergone CABG,
2. Patients who had an outpatient instead of inpatient coronary angiogram for their NSTEMI presentation,
3. Patients with no data in the database systems (i.e. missing medical records or missing angiograms).

We defined NSTEMIOA as AMI with presence of elevated levels of troponin I or CK, an angiographic evidence of an occluded or sub-totally occluded culprit artery with a TIMI flow <3, but with no ST-elevation or new LBBB on ECG.

The culprit artery of the NSTEMI was determined by the cardiologist performing the coronary angiography based on the findings of ECG changes, angiography and left ventriculography or echocardiography.

Data Collection

Data were collected from three sources of databases: medical records, Clinical Information System (pathology database) and Centricity (General Electric database of cardiac catheterisation laboratory). Data collected were inclusive of patient demographic details (gender, age, weight, ethnicity), medical history and risk factors for ischaemic heart disease, investigation findings (ECG, cardiac biomarkers), angiographic and revascularisation details, and clinical outcomes.

The primary endpoints of interest were the differentiating characteristics between NSTEMIOA and NSTEMIPA in terms of the patient's medical history, biomarkers, clinical and angiographic findings. The secondary endpoints of interest were the clinical outcomes of NSTEMIOA and NSTEMIPA and the effect of the timing of percutaneous revascularisation on the extent of myocardial damage (peak CK level vs the time delay to percutaneous coronary intervention). Patients' clinical outcomes in 12 months were based on the review of the aforementioned databases. The clinical outcomes of interest were, death, MI, heart failure, cardiac arrhythmia, hospital re-admission for angina and coronary revascularisation.

Statistical Methods

All NSTEMI patients were categorised into either occluded or patent culprit artery cohorts. All data were analysed using Minitab-15 (Minitab Inc, State College, PA, USA). Data were first analysed by descriptive analysis. Categorical data were expressed in number and count, whereas continuous data were expressed in mean and standard deviation. Differences between the two groups were analysed with chi-squared test for categorical data and student t-test for continuous variables. Two-proportion test was used to compare the number of endpoints reached by each cohort. Results were considered statistically significant if p-value was < 0.05. The effects of the timing of revascularisation on myocardium damage (as indicated by the level of peak CK) was analysed with linear regression analysis and Pearson correlation coefficient.

Ethics approval was obtained from Melbourne Health Human Research Ethics Committee to certify that the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee. As this study involved only audit data, the consent from the patients to participate in this study had been waived by the aforementioned ethics committee.

Results

Study Population

During the period between 01/01/2010 and 30/06/2010 we identified 192 NSTEMI patients who underwent angiograms. A total of 49 patients were excluded based on exclusion criteria of previous CABG (18), incomplete medical records (20), peak troponin I less than 0.03mcg/L (4) and outpatient instead of inpatient coronary angiography (7). Hence, 143 patients were included in the study. Of these, 46 patients were female (32%) and 97 were male (68%). The mean age of the patients was 64 years old. We identified that 34 patients or 23.8% of our NSTEMI study population had an occluded culprit artery and 109 patients (76.2%) had a patent culprit artery.

Clinical Findings

The differences between NSTEMIOA and NSTEMIPA in terms of medical history, clinical findings and biochemical markers are summarised in Table 1. NSTEMI patients with occluded arteries were more likely to have hypercholesterolaemia (85.3% vs. 64.2%, $p = 0.015$) and the presence of ST depression or the combination of T-wave inversion (TWI) and ST depression on admission ECG (32.4% vs. 11.9%,

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Table 1 Patient medical history, clinical findings and biochemical markers.

Medical history, clinical findings and biochemical markers				
Variables	**Total NSTEMI patient = 143 (%)**	**NSTEMIOA = 34 (%)**	**NSTEMIPA = 109 (%)**	**p-value**
Medical history				
Past history of IHD	57 (39.86)	18 (52.94)	39 (35.78)	0.074
Family history of IHD*	33 (23.08)	8 (23.53)	25 (22.94)	0.943
Diabetes	40 (27.97)	13 (38.24)	27 (24.77)	0.127
Hypertension	94 (65.73)	27 (79.41)	67 (61.47)	0.054
Hypercholesterolaemia	99 (69.23)	29 (85.29)	70 (64.22)	0.015
Smoking				0.452
Current:	45 (33.58)	8 (23.52)	37 (33.94)	
Ex-smoker:	38 (28.36)	11 (32.35)	27 (24.77)	
Non-smoker:	51 (38.06)	14 (41.18)	37 (33.94)	
Unknown:	9 (6.29)	1 (2.94)	8 (7.33)	
Peripheral vascular disease	5 (3.50)	1 (2.94)	4 (3.67)	1
Cerebrovascular accident	15 (10.49)	1 (2.94)	14 (12.84)	0.120
Presentation and clinical findings				
Syncope at admission	4 (2.80)	0 (0.00)	4 (3.67)	
ECG findings				0.008
ST-depression	24 (16.78)	11 (32.35)	13 (11.93)	
LBBB	8 (5.59)	2 (5.88)	6 (5.50)	
T-Wave inversion (TWI)	58 (40.56)	10 (29.41)	48 (44.04)	
ST depression and TWI	10 (6.99)	5 (14.71)	5 (4.59)	
No ischaemic changes	43 (30.07)	6 (17.65)	37 (33.94)	
Cardiac arrhythmia*				
Total number	14 (9.79)	2 (5.88)	12 (11.01)	0.519
Killip Class				0.748
I	100 (69.93)	22 (64.71)	78 (71.56)	
II	36 (25.17)	10 (29.41)	26 (23.85)	
III	7 (4.90)	2 (5.88)	5 (4.59)	
IV	0 (0.00)	0 (0.00)	0 (0.00)	
Hypotension	7 (4.90)	1 (2.94)	6 (5.50)	1
TIMI risk score (Grade)				0.267
Low (0 – 2)	57(39.86)	10 (29.41)	47 (43.12)	
Medium (3 – 4)	52 (36.36)	13 (38.24)	39 (35.78)	
High (5 – 7)	34 (23.78)	11 (32.35)	23 (21.10)	
Echo-LV function				0.016
Normal	50 (45.05)	7 (25.00)	43 (51.81)	
Abnormal Mild	34 (30.63)	12 (42.86)	22 (26.51)	
Moderate	17 (15.32)	4 (14.29)	13 (15.66)	
Severe	10 (9.01)	5 (17.86)	5 (6.02)	
Cardiac biochemical markers				
Troponin I: (μg/L)				
At admission	1.404 ± 3.377	1.741 ± 3.778	1.298 ± 3.253	0.541
Max. < 24 hrs	5.785 ± 10.26	5.54 ± 9.31	5.86 ± 10.58	0.868
Peak	6.312 ± 10.75	6.20 ± 9.48	6.35 ± 11.17	0.941
CK: (IU/L)				
At admission	194.3 ± 208.6	212.2 ± 188.0	188.7 ± 215.1	0.541
Peak	354.5 ± 462.4	433.0 ± 700.0	330.0 ± 359.1	0.414
Creatinine: (μmol/L)				
At admission	96.43 ± 70.49	99.31 ± 66.52	95.52 ± 71.95	0.777
Peak	107.02 ± 94.55	109.32 ± 76.85	106.31 ± 99.75	0.855

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Table 2 Patient coronary angiographic findings and percutaneous revascularisation data.

Coronary angiographic findings				
Variables	**NSTEMI (%)**	**NSTEMIOA (%)**	**NSTEMIPA (%)**	**p-value**
Culprit vessel*				0.316
LAD	68 (47.55)	16 (47.06)	52 (47.71)	
Non LAD				
LCx	31 (21.68)	8 (23.53)	23 (21.10)	
RCA	25 (17.48)	10 (29.41)	15 (13.76)	
Normal	21 (14.69)	0 (0.00)	21 (19.27)	
Number of diseased vessels (total < 143 as some were normal arteries)				
Single	43 (30.07)	8 (23.53)	35 (32.11)	0.341
Double	45 (31.47)	13 (38.24)	32 (29.36)	0.330
Triple	34 (23.78)	13 (38.24)	21 (19.27)	0.023
Multi-vessel (Double or Triple)	79 (55. 24)	26 (76.47)	53 (48.62)	0.004
Collateral supply	32 (28.83)	26 (76.47)	6 (5.50)	<0.001
TIMI flow pre-PCI				<0.001
0	21 (14.69)	24 (61.76)	0 (0.00)	
1	9 (6.29)	9 (26.47)	0 (0.00)	
2	14 (9.79)	4 (11.76)	10 (9.17)	
3	99 (69.23)	0 (0.00)	99 (90.83)	
TMP pre-PCI**				<0.001
0	13 (9.09)	13 (38.24)	0 (0.00)	
1	16 (11.19)	14 (41.18)	2 (1.83)	
2	17 (11.89)	7 (20.59)	10 (9.17)	
3	97 (67.83)	0 (0.00)	97 (88.99)	
Percutaneous revascularisation data				
Variables	**NSTEMI (%)**	**NSTEMIOA (%)**	**NSTEMIPA (%)**	**p-value**
Total of patients underwent PCI	69 (48.25)	14 (41.18)	55 (50.46)	0.344
Admission-to-PCI time				
Hours	53.11 ± 41.61	47.3 ± 68.1	54.6 ± 32.3	0.703
Symptom to PCI time:				
Hours	60.89 ± 42.59	56.8 ± 73.6	61.9 ± 31.9	0.820
Successful PCI	67 (97.10)	13 (92.86)	54 (98.18)	0.367
Balloon Pre-dilation	38 (55.07)	10 (71.43)	28 (50.91)	0.168
Export catheter	5 (7.25)	2 (14.29)	3 (5.45)	0.266
Stent Post-dilation	33 (47.83)	6 (42.86)	27 (49.09)	0.677
TIMI flow post-PCI				
0 – 2:	2 (2.90)	1 (7.14)	1 (1.82)	0.454
3:	67 (97.10)	13 (92.86)	54 (98.18)	
TMP post-PCI				
0 – 2:	5 (7.25)	3 (21.43)	2 (3.64)	0.114
3:	64 (92.75)	11 (78.57)	53 (96.36)	
CABG**	0 (0.00)	0 (0.00)	0 (0.00)	N/A

*Culprit vessel-percentage will exceed 100% as left main artery stenosis was considered as LAD + LCx

**TMP-TIMI myocardial perfusion grade

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p = 0.008 and 14.7% vs. 4.6%, p = 0.008 respectively). Other clinical risk factors for ischaemic heart disease (IHD) such as family history, diabetes, smoking, peripheral vascular disease and cerebrovascular accidents were not statistically significant (Table 1). In terms of clinical presentation, there was no significant difference between NSTEMIOA and NSTEMIPA in their Killip class and TIMI risk score. However, NSTEMIOA cohort appeared to have a higher incidence of LV systolic dysfunction on echocardiography (p = 0.038).

Angiographic Findings

The angiographic data are presented in Table 2. The main culprit artery identified in our study was the LAD at 47.6%. We identified that NSTEMIOA cohort had a higher incidence of multi-vessel disease (76.5% vs. 48.6%, p = 0.004). NSTEMIOA cohort was more likely to have triple vessel disease than NSTEMIPA cohort (38.2% vs. 19.3%, p = 0.023). NSTEMIOA cohort was also more likely to have collateral supply to the culprit artery than NSTEMIPA cohort (76.5% vs. 5.5%, p < 0.001). As expected, NSTEMIOA cohort had a much higher incidence of poor TIMI flow (TIMI flow ≤2) pre-PCI than NSTEMIPA cohort (88.2% vs. 0.00%, p < 0.001). NSTEMIOA cohort also had a higher number of TIMI myocardial perfusion (TMP) grade ≤2 pre-PCI (79.4% vs. 1.8%, p < 0.001) (Table 2).

The revascularisation data is also presented in Table 2. The results showed that there was no significant difference between NSTEMIOA and NSTEMIPA in the PCI rate (41.2% vs. 50.5%, p = 0.344). There was also no significant difference between the symptom onset and admission to PCI time between the two groups. There was no significant

Table 3 The clinical outcomes of NSTEMI patients in twelve 12 months.

Clinical outcomes				
Variables	**NSTEMI (%)**	**NSTEMIOA (%)**	**NSTEMIPA(%)**	**p-value**
Death				
Within 12 months	0 (0.00)	0 (0.00)	0 (0.00)	NS
Total endpoints	0 (0.00)	0 (0.00)	0 (0.00)	
MI				
<30 days	4 (2.80)	1 (2.94)	3 (2.75)	
<3 months	6 (4.20)	1 (2.94)	5 (4.59)	
<6 months	7 (4.90)	1 (2.94)	6 (5.50)	
<12 months	8 (5.59)	2 (5.88)	6 (5.50)	1.000
Heart Faillure*				
<30 days	3 (2.10)	3 (8.82)	0 (0.00)	
<3 months	4 (2.80)	3 (8.82)	1 (0.92)	
<6 months	5 (3.50)	3 (8.82)	2 (1.83)	
<12 months	5 (3.50)	3 (8.82)	2 (1.83)	0.087
Recurrent chest pain				
<30 days	4 (2.80)	2 (5.88)	2 (1.83)	
<3 months	8 (5.59)	2 (5.88)	6 (5.50)	
<6 months	13 (9.09)	3 (8.82)	10 (9.17)	
<12 months	16 (11.19)	4 (11.76)	12 (11.01)	1.000
Arrhythmia**				
<30 days	3 (2.10)	0 (0.00)	3 (2.75)	
<3 months	5 (3.50)	0 (0.00)	5 (4.59)	
<6 months	5 (3.50)	0 (0.00)	5 (4.59)	
<12 months	7 (4.90)	0 (0.00)	7 (6.42)	0.198
Revascularisation (PCI or CABG)				
<30 days	74 (51.75)	17 (50.00)	57 (52.29)	
<3 months	80 (55.94)	19 (55.88)	61 (55.96)	
<6 months	80 (55.94)	19 (55.88)	61 (55.96)	
<12 months	81 (56.64)	19 (55.88)	62 (56.88)	0.918

*Heart failure as documented in the clinical notes or discharge summary

**Arrhythmia defined as one of the following: atrial fibrillation/flutter, ventricular tachycardia/fibrillation, second degree or third degree heart block, or asystole.

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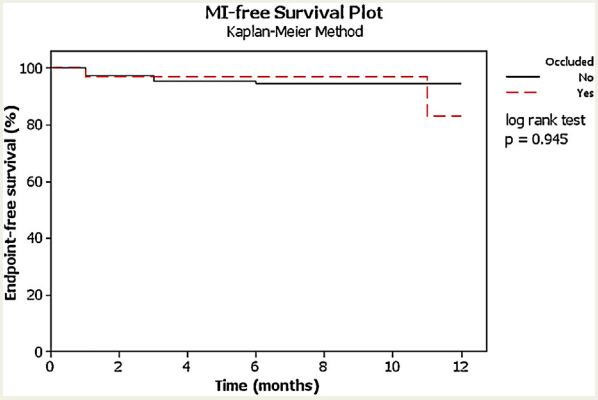

Figure 1 Myocardial infarction-free Kaplan-Meier survival plot over twelve months.

Figure 2 Heart-failure free Kaplan-Meier survival plot over twelve months.

difference in the angiographic findings of post PCI TIMI flow and TMP between these two cohorts (Table 2).

Clinical Outcomes

The data of clinical outcomes are presented in Table 3. In our study cohort no patients reached the end point of death within 12-month follow up period. Overall, our study cohort had a low rate of recurrent MI within 12 months (5.6%). There was no significant difference in the recurrent MI rate between NSTEMIOA and STEMIPA cohorts and their MI-free Kaplan Meier survival plot (Table 3 and Figure 1). There was a trend showing that NSTEMIOA cohort had a higher incidence of heart failure post-discharge than the NSTEMIPA cohort (8.82% vs. 1.83%, $p = 0.087$). However, the difference between the two cohorts was not statistically significant (Table 3 and Figure 2). There was no statistical difference in terms of cardiac arrhythmia, readmission for angina and revascularisation rate at 12 months (Table 3).

Timing of Revascularisation

Linear regression analysis showed that there was no correlation between the time delay to PCI and the peak CK level for the whole NSTEMI patient cohort (Pearson Correlation = -0.157, $p = 0.199$) or for both NSTEMIOA and NSTEMIPA subgroups (Figure 3a and 3b).

Disscussion

Our retrospective observational study has shed light on the prevalence and characteristics of NSTEMI patients with occluded arteries as well the effect of the timing of revascularisation on these patients in accordance with our objectives.

In our study we found that about a quarter of our NSTEMI cohort had occluded arteries, similar to previously

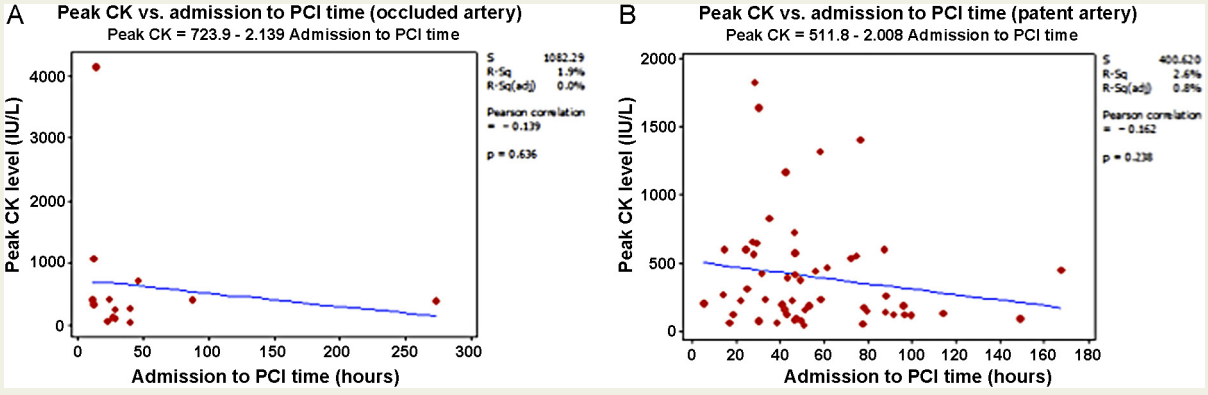

Figure 3 (A) Peak CK level versus time delay in the PCI of NSTEMIOA subgroup. (B) Peak CK level versus time delay in the PCI of NSTEMIPA subgroup.

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conducted studies [7,8]. From the data that we gathered regarding the patients' demographic and clinical presentation data we were able to identify that NSTEMI patients with occluded arteries were more likely to have hypercholesterolaemia and ECG changes such as ST depression and T-wave inversion on ECG and LV systolic dysfunction on echocardiography. These findings may help us to suspect NSTEMIOA in our first encounter with NSTEMI patients so that we keep a low threshold to investigate such patients with coronary angiography at the earliest time possible logistically. The longer the occlusive thrombus is left in situ, the harder it becomes to perform PCI subsequently. The information obtained from our study may help in identifying NSTEMIOA patients for future study comparing very early vs delayed PCI for NSTEMIOA patients.

In our study we did not have enough evidence to show that there were any significant differences between NSTEMIOA and NSTEMIPA in terms of patient demographics, biochemical markers, past medical history of coronary artery disease (CAD) and clinical findings. Their TIMI risk scores were not statistically significant as well, although numerically there were more NSTEMIOA with moderate to high TIMI risk score as compared to the TIMI risk score of patients with NSTEMIPA. Our small sample size could have probably resulted in non-statistical significance of the results.

In terms of clinical outcomes, our findings showed no statistical difference between NSTEMIOA and NSTEMIPA cohorts. Nonetheless, there was a trend showing that NSTEMIOA patients were more likely to develop heart failure at 12 months post-admission. Such difference was not statistically different probably due to the small sample size.

Our findings regarding clinical outcomes were significantly different to the findings of Wang et al. [7]. They found that NSTEMIOA patients had a higher unadjusted rate of death at six months follow-up as compared to patients with patent arteries [7]. In our study, we found a zero mortality rate. This difference could be due to several factors. Firstly, the data used by Wang et al was the data gathered in the PARAGON B study which finished patient enrolment in 1999 [7,10]. Since 1999, the treatment of NSTEMI patients has improved significantly and hence could help to explain the difference between our findings. In our study we also found a higher rate of collateral supply in our NSTEMIOA cohort. This could help to explain the zero death rate as the presence of collateral supplies might have improved the outcome of these patients. Lastly, the difference could also be due to our inclusion criterion of the presence of an invasive angiogram on index admission. Due to this criterion, older patients presenting with multiple co-morbidities who were not investigated with coronary angiogram would be excluded. Such a subset of NSTEMI patients was likely to have coronary occlusions and poorer prognosis and yet they would be excluded from the study.

Although our study was purely an observational study as compared to the OAT trial which was an interventional trial investigating the effects of PCI to the occluded infarct related artery versus medical therapy, our findings were not too dissimilar to the OAT trial in terms of the clinical outcomes and patency of infarct related artery [11]. In the OAT trial, successful PCI to the infract-related occluded artery was not associated with improved outcomes in death, re-infarction and heart failure. Hence, the outcomes of the OAT trial appear to negate the necessity of determining the patency of the infarct related artery. That said, the majority of the patients in the OAT trial were STEMI patients (two-thirds of the recruited patients) and the PCI was delayed for at least three days post infarction.

Previous studies investigating invasive strategy (coronary angiography and PCI within 48 hours of hospitalisation) versus conservative management strategy (medical therapy first) in the management of NSTEMI patients showed invasive strategy is more likely to have better outcomes in terms of lower rates of all cause mortality, non-fatal MI and re-hospitalisation for unstable angina [12–26]. Nonetheless, the optimal timing of PCI for invasive strategy is still controversial i.e. before (early) or after 24 hours (delayed) of presentation. Navarese et al reviewed all major PCI trials of NSTEMI and concluded that there was insufficient evidence in favour of or against an early PCI [27]. Similarly, in a meta-analysis of randomised controlled trials of early (<24 hours) versus delayed (>24 hrs) PCI for NSTEMI patients, Rajpurohit et al reported that early PCI did not reduce the odds of the composite endpoint of death or non-fatal MI in 30 days [28]. However, there was no sub-analysis on the effect of early versus delayed PCI in the subset of NSTEMI with occluded infarct-related artery. In trying to answer this question, we performed a linear regression analysis and Pearson correlation coefficient of the peak CK level versus admission/symptom onset to PCI. Disappointingly, our analysis showed no evidence of early PCI resulting in a lesser extent of myocardial damage. Nonetheless, our study was purely observational. The factors influenced the timing of PCI would be patient's progress and the logistics of the cardiac catheterisation laboratory rather than the status of infarct related artery. With the evidence available so far, the patency status of infarct related artery of NSTEMI is unlikely to influence the risk stratification of ACS although current technology such as coronary CT angiography is capable of diagnosing an acute coronary occlusion [29].

There were some limitations in our study. Firstly, our study was subjected to the usual limitations associated with a retrospective observational study including missing records (9.4%), selection bias as well as being uncontrolled in nature.

Secondly, our study population was small and hence it was difficult to achieve statistical significance for the differences in clinical outcomes between NSTEMIOA and NSTEMIPA cohorts. Our study was also single-centred and hence the results we obtained cannot be generalised to other populations.

Thirdly, our study had a relatively short follow up time of one year. As a result, there might have been late accrual of superior treatment effect relating to the timing of revascularisation well as the outcome of NSTEMIPA patients as

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compared to NSTEMIOA patients. To ascertain the long-term effect of the timing of PCI, a study with a longer follow up period is required.

Fourthly, our definition of clinical outcomes was based on the specific findings in medical records of patients. Therefore some instances of clinical outcomes, such as heart failure, may be overlooked if not specifically recorded. This would have affected the count of outcomes and hence our results and analysis. There was also a lack of telephone or interview follow-up with patients and we relied only on internal medical records to show any further admissions or outcomes. This means that other hospitalisation or incidences of clinical outcomes at other hospitals was potentially neglected and may have affected our results.

Conclusion

We found that around a quarter of NSTEMI patients present with occluded arteries. We identified that NSTEMI patients with occluded arteries were more likely to have hypercholesterolaemia, ST changes on admission ECG, multi-vessel disease and the presence of collateral supplies on coronary angiography. Otherwise, there were no other clear differentiating risk profiles, biochemical markers and presentations between the two groups. There were also no significant differences in the clinical outcome at 12 months follow up between the two groups. We found no clear association between the timing of revascularisation and extent of myocardial damage in both cohorts.

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