Original paper

Non-ST elevation myocardial infarction related to total coronary artery occlusion – prevalence and patient characteristics

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Abstract

Introduction: Acute coronary occlusion (ACO) may also present as non-ST elevation myocardial infarction (NSTEMI) and thus veil the real threat.

Aim: Based on combined analysis of electrocardiography and echocardiography findings, we aimed to describe profile of NSTEMI patients at increased risk of ACO.

Material and methods: It was a retrospective study that included patients referred for cardiac catheterisation due to NSTEMI. Patients were selected into the study in two different time frames. Firstly, all consecutive NSTEMI patients were enrolled in a 12-month period to detect the prevalence of ACO (prevalence group). Secondly, all NSTEMI patients with ACO hospitalized in the previous 5 years were also enrolled (NSTEMI-ACO group). All patients had 12-lead electrocardiogram (ECG) and the transthoracic echocardiography (TTE) performed before the cardiac catheterisation.

Results: Fifty-three consecutive patients (37 males) were enrolled into the prevalence group in a 12-month period. Ten (19%) of them were diagnosed with ACO. Thirty-four consecutive patients were enrolled into the NSTEMI-ACO group. Non-ST elevation myocardial infarction patients with ACO were younger as compared to NSTEMI patients without ACO. Non-ST elevation myocardial infarction patients with ACO were less likely to have anterior wall ischaemia as detected by ECG, which was not reflected by TTE results. Combined assessment of ischaemia by ECG and impaired contractility by TTE did not reveal any significant differences between NSTEMI patients with or without ACO.

Conclusions: The identification of NSTEMI patients with ACO is challenging. Therefore, the utmost caution should be paid to prevent delay of coronary angiography in NSTEMI patients who have increased risk of ACO.

Key words: non-ST elevation myocardial infarction, total coronary occlusion, electrocardiogram, echocardiography.

Introduction

Acute coronary occlusion (ACO) is responsible for ST-segment elevation myocardial infarction (STEMI) [1]. The introduction of urgent (< 2 h) percutaneous coronary interventions (PCI), which enabled quick revascularisation of ACO, significantly decreased the mortality among STEMI patients [2]. However, ACO may also present as non-ST elevation myocardial infarction (NSTEMI) and thus veil the real threat [3].

ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation reflect the heterogeneity of NSTEMI patients [4]. They define the optimal timing for revascularisation in NSTEMI as no longer than 72 h from the presentation of symptoms or 24 h for patients with GRACE score > 140. Urgent coronary angiography is only recommended in NSTEMI patients with life-threating ventricular arrhythmias, refractory angina, and haemodynamic instability or heart failure symptoms. If these symptoms do not occur, the detection and revascularisation of potential ACO may be delayed, which increases the risk of MI complications [5].

Previous studies have reported that total occlusion of infarct-related artery increased the mortality in patients with NSTEMI [6]. The identification of NSTEMI patients at high risk of ACO may accelerate PCI and thus decrease the mortality. Appropriate evaluation of NSTEMI patients involves the assessment of clinical characteristics togeth-

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er with myocardial necrosis biomarkers, electrocardiography, and echocardiography findings.

Aim

Our study, based on combined analysis of the above parameters, aimed to describe their profile in NSTEMI patients at increased risk of ACO.

Material and methods

It was a retrospective study that included patients referred for cardiac catheterisation due to NSTEMI. Patients were selected into the study in two different time frames. Firstly, all consecutive NSTEMI patients were enrolled in a 12-months period to detect the prevalence of ACO (prevalence group). Secondly, all NSTEMI patients with ACO hospitalised in the last 5 years (NSTEMI-ACO group) were enrolled into the database to present their characteristics as compared to NSTEMI patients without ACO enrolled into the prevalence group.

Inclusion/exclusion criteria

Patients considered eligible for inclusion had to be over 18 years old and present with acute myocardial infarction type I (ESC guidelines 2012) without ST elevation confirmed within 24 h of the onset of symptoms [4]. Myocardial infarction (MI) was defined as a rise in cardiac enzyme concentration according to local laboratory reference ranges. Moreover, all patients needed to have a 12-lead electrocardiogram (ECG) and transthoracic echocardiography (TTE) performed before the cardiac catheterisation.

The diagnosis of NSTEMI required troponin T (TnT) concentration above 0.10 ng/ml accompanied by at least one of the following: angina, ECG evidence of acute ischaemia, or presumed new regional wall motion abnormalities on echocardiography.

Electrocardiography

For the purposes of this study, ECG changes were classified as reflecting the anterior (leads V2, V3, V4), the lateral (leads I, aVL, V5, V6), or the inferior wall ischaemia (leads II, III, and aVF). The diagnosis of acute ischaemia required new horizontal or down-sloping ST depression ≥ 0.5 mm in two contiguous leads and/or ≥ 0.1 mV T-wave inversion in two contiguous leads with prominent R wave, or an R/S ratio of 1. In case of ST depression in V1–V3 and positive T-waves, additionally V7–9 were analysed, and patients with ≥ 0.5 mm ST elevation in these leads were excluded. Other exclusion criteria included ST-elevation myocardial infarction; left bundle branch block (LBBB) and troponin T rise following PCI/coronary artery bypass graft surgery (CABG).

Left ventricle contractility

Transthoracic echocardiography was performed after ECG and prior to angiography using standard parasternal and apical views. The contractility was assessed according to AHA guidelines and defined as normo-, hypo-, a-, or dyskinesis [7]. To enable direct comparison of ischaemia location between ECG and TTE, regional wall motion abnormalities were classified as affecting the anterior/apical, inferior, or posterior wall of the left ventricle. The left ventricle ejection fraction was calculated using Simpson's rule [8].

Angiography and revascularisation

The cardiac catheterisation of NSTEMI patients was performed according to the ESC guidelines for myocardial revascularisation and NSTEMI diagnosis and treatment [4].

To identify the culprit lesion responsible for MI three main coronary arteries (right – RCA, left anterior descending – LAD, and circumflex – Cx) and, when appropriate, coronary grafts were assessed. Vessel stenosis evaluation was based on visual assessment. An ACO was defined as coronary vessel occlusion with angiographically determined thrombus – containing lesion and no distal flow. The decision on PCI of the infarct-related artery vs. CABG referral was left to the operator's discretion.

Statistical analysis

The Kolmogorov-Smirnov test was used to analyse the continuous data distribution. Normally distributed values were presented as mean with standard deviation. Non-normally distributed values were presented as median with 25^{th} and 75^{th} percentile (interquartile range – IQR). One-way ANOVA was used to compare normally distributed data and the Mann-Whitney test was used to compare non-normally distributed data. The categorical data were compared using Fischer's exact test or χ^2 test. Value of $p < 0.05$ was considered as statistically significant.

Results

Seventy-three patients were enrolled into the study, 43 with NSTEMI without ACO and 34 with ACO.

Prevalence group

Fifty-three consecutive patients (37 males) were enrolled into the prevalence group during a 12-month period. Ten (19%) of them were diagnosed to have ACO and 3 (6%) of them died before discharge from hospital. The patients enrolled into the prevalence group had mean age 79 (59, 74) years and had body mass index (BMI) 28.1 ±4.3 kg/m^2. Forty-six of those patients (87%) suffered from hypertension and 20 (38%) from diabetes. Ten (19%) patients had a history of previous CABG and 7 (13%) of PCI. The laboratory results were as follows: haemoglobin 13.9 (13.2, 15.1) g/dl, glomerular filtration rate 74 ±25 ml/min/1.73 m^2, TnT was 1.03 (0.27, 2.88) ng/ml, and creatine kinase MB (CK-MB) 28 (17, 66) IU/l.

NSTEMI-ACO group

In a 5-year time frame, 34 consecutive NSTEMI patients with ACO were included into the database. Four of them died before the discharge from hospital. The patients' characteristics are presented in Table I.

NSTEMI-ACO vs. NSTEMI-non-ACO – patients' characteristics

There were no significant differences between patients except that NSTEMI patients with ACO were younger as compared to NSTEMI patients without ACO. The patient's characteristics are summarised in Table I.

NSTEMI-ACO vs. NSTEMI-non-ACO – culprit lesion and procedure details

Non-ST elevation myocardial infarction patients with ACO were more likely to have a culprit lesion located in saphenous vein graft (SVG) as compared to NSTEMI patients without ACO. The door-to-balloon time was also shorter in NSTEMI patients with ACO, and they were treated with BMS implantation more often. The PCI details are summarised in Table II.

NSTEMI-ACO vs. NSTEMI-non-ACO – electrocardiographic and echocardiographic results

Non-ST elevation myocardial infarction patients with ACO were less likely to have anterior wall ischaemia as detected by ECG. Surprisingly, this was not reflected by TTE, which did not show any differences in the location of impaired left ventricle wall contractility. Combined assessment of ischaemia by ECG and impaired contractility by TTE did not reveal any significant differences between NSTEMI patients with or without ACO. The ECG and TTE results are summarised in Table III.

Table I. Patients' characteristics

Parameter	NSTEMI without ACO (n = 43)	NSTEMI with ACO (n = 34)	Value of p
Age [years]	67.7 ±9.9	64.8 ±9.8	0.038
Male, *n* (%)	30 (69.8)	26 (76.5)	0.512
BMI [kg/m^2]	27.8 ±4.4	28.2 ±4.24	0.105
Hypertension, *n* (%)	39 (91)	26 (77)	0.087
Diabetes type 2, *n* (%)	15 (35)	16 (47)	0.279
Insulin therapy, *n* (%)	5 (12)	6 (18)	0.479
Previous CABG, *n* (%)	8 (19)	6 (18)	0.914
Previous PCI, *n* (%)	6 (14)	10 (29)	0.097
Haemoglobin [mg/dl]	13.9 (13.0, 15.1)	13.6 (12.4, 14.9)	0.457
GRF [ml/kg/1.73 m^2]	75.57 ±25.59	75.27 ±34.12	0.965
CK-MB	27.5 (16, 54)	34 (23, 94)	0.214
Troponin	0.86 (0.25, 2.32)	1.12 (0.46, 3.1)	0.231

Table II. Culprit lesion and revascularisation details

Parameter	NSTEMI without ACO (n = 43)	NSTEMI with ACO (n = 34)	Value of p
Culprit lesion:			
LM	4 (9%)	0	
LAD	11 (26%)	5 (15%)	
RCA	13 (30%)	7 (21%)	
Cx	12 (28%)	16 (47%)	
SVG	1 (2%)*	5 (15%)*	
IM	0	1 (3%)	0.037
%DS	90 (90, 99)	100	< 0.001
Door-to-balloon [min]	240 (76, 388)	120 (64, 159)	0.022
PCI	33 (77%)	24 (71%)	0.541
POBA	10 (23%)	10 (29%)	
1 stent	25 (58%)	22 (65%)	
2 stents	5 (12%)	2 (6%)	
3 stents	3 (7%)	0	0.324
BMS	3 (7%)	14 (41%)	< 0.001
CABG	0	1 (3%)	0.258

*$p < 0.05$.

Discussion

Prior clinical observations of NSTEMI patients mainly focused on ECG changes and biomarkers of myocardial necrosis [3, 9]. This is the first study to add echocardiography imaging with the intention of enhancing ACO detection in NSTEMI patients. Nevertheless, our results seem to confirm that identification of NSTEMI patients with ACO may be challenging.

The prevalence of NSTEMI with ACO (19%) in our study group was lower as compared to earlier studies. Acute coronary occlusion occurrence was formerly reported as being between 25% (Wang *et al.*) and 29% (Bahrmann *et al.*) inpatients with NSTEMI [3, 9]. This discrepancy may result from the low number of patients included in our study. The previous two retrospective studies were much larger observations and enrolled almost 2000 and 450 patients, respectively.

No difference in location of the culprit lesion was observed in our study within native coronaries. Only in patients who had previously undergone CABG was the ACO of SVG more often observed. These results are in contrast to previous observations stating that in NSTEMI patients with ACO the infarct-related artery was typically other than LAD [3]. Nevertheless, we have also observed that NSTEMI patients with ACO more frequently presented with ECG changes other than anterior wall, usually nourished by LAD.

The PCI in NSTEMI patients with ACO was performed faster as compared to patients without ACO. This was probably due to patients' symptoms being more indicative for urgent PCI. Interestingly, the NSTEMI patients with ACO received BMS more often. However, it should be noted that it is a 5-year retrospective registry from

Table III. The ECG and TTE results

Parameter	NSTEMI without ACO (n = 43)	NSTEMI with ACO (n = 34)	Value of p
Ischaemia detected by ECG:			
Inferior wall	6 (14%)	8 (23%)	0.279
Posterior wall	1 (2.3%)	4 (11.8%)	0.095
Anterior wall	17 (40%)	6 (18%)	0.037
Lateral wall	21 (50%)	17 (50%)	0.919
Impaired contractility detected by TTE:			
LVEF	50 (45, 60)	55 (35, 60)	0.817
EDD	51 (50, 53)	52 (49, 56)	0.397
ESD	36 (32, 40)	36 (31, 41)	0.531
Inferior wall	22 (51%)	21 (62%)	0.352
Posterior wall	5 (12%)	7 (21%)	0.282
Anterior wall	6 (14%)	7 (21%)	0.440
Lateral wall	13 (30%)	9 (26%)	0.717
IVS	7 (16%)	5 (15%)	0.850
APEX	15 (35%)	9 (27%)	0.429
LA area	22 (19, 26)	21 (20, 24)	0.663
Ischaemia detected by ECG with simultaneous impaired contractility identified by TTE:			
Inferior wall	10 (24%)	11 (32%)	0.408
Posterior wall	0	0	
Anterior wall	0	0	
Lateral wall	6 (14%)	3 (9%)	0.464

the period when DES implantation in acute coronary syndrome (ACS) was not so widely established [10].

The clinical characteristics revealed that NSTEMI patients with ACO were younger as compared to those without ACO. That is in line with the observed age differences between STEMI and NSTEMI patients [11]. Further analysis, however, did not reveal any differences in terms of patients' co-morbidity, level of cardiac biomarkers, and the location of contractility abnormalities by echocardiography. The combined analysis of ischaemia detected by ECG and echocardiography did not produce any ACO-indicative profile in NSTEMI patients either. These results are in contrast to the previous studies were inferolateral ischaemia was more often observed in NSTEMI patients with ACO [3].

The identification of NSTEMI patients with ACO remains difficult, and utmost caution is mandatory not to omit those requiring urgent coronary angiography. Introduction of new, non-invasive imaging modalities, including magnetic resonance imaging (MRI) may prove helpful in diagnosing such patients more accurately. Not only does the MRI visualise the contractility of the heart but also enables the assessment of the coronaries [12]. Since it does not increase the radiation dose, it may pose a valuable alternative to echocardiography, before planned coronary angiography in NSTEMI patients [13].

There are several study limitations that have to be addressed. Firstly, a small number of patients were enrolled into the study. Secondly, NSTEMI patients with and without ACO were enrolled in different time frames, which might have influenced the results. Thirdly, it was a retrospective and non-randomised study with potentially biased patient selection. Finally, there was no reference angiography core laboratory involved to identify the infarct related artery, and its detection relied only on the operator's assessment.

Conclusions

The identification of NSTEMI patients with ACO brings a lot of difficulties. Although there are tools that enable quick detection of myocardial ischaemia, NSTEMI patients with ACO may still be omitted in a daily clinical basis. Therefore, extreme caution should be paid so as not to delay coronary angiography in those patients.

Conflict of interest

The authors declare no conflict of interest.

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