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Registry of Patients with STEMI Equivalent Electrocardiography Undergoing Percutaneous Coronary Intervention

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ABSTRACT

Background: Acute reperfusion therapy, namely primary percutaneous coronary intervention (PCI), is the mainstay of treatment for ST segment elevation myocardial infarction (STEMI). This work aimed to evaluate STEMI equivalent in patients admitted at cardiac care centre, Tanta university. **Methods:** This prospective observational registry was done on 50 patients aged from 39 to 81 years old, both genders, presented with electrocardiographic (ECG) findings of STEMI equivalent (De Winter syndrome-Wellen syndrome - elevation in aVR and global ST segment depression) undergoing PCI. Coronary angiography done to assess angiographic lesion then reperfusion through PCI for infarct related artery (IRA) done. **Results:** 74% of patients were classified as KILLIP I, positive troponin was found in 90 % of patients a. Average creatine kinase-myocardial band (CKMB) value was 64.34. 60% of patients were classified as intermediate risk on global registry of acute coronary events (GRACE) score, only 16% of study population were high risk GRACE score. Multi vessel disease was double time more than single vessel affection with Left anterior descending (LAD) artery as the most frequent culprit artery (88%) followed by left main (LM) artery (10%). Major adverse cardiac events (MACE) occurred in 18% of study population. No patients died during hospital stay or suffered from re-infarction or stroke during hospital stay. Conclusions: Identifying individuals with STEMI-equivalents, such as NSTEMI, provides an opportunity for timely intervention by rapid coronary angiography. Immediate reperfusion is crucial for these individuals, since their death rate remains elevated in the absence of therapy.

Keywords: percutaneous coronary intervention (PCI), electrocardiographic(ECG), creatine kinase-myocardial band (CKMB)

1. Introduction

Acute myocardial infarction (AMI) is a major contributor to mortality in industrialized countries. The illness has a global prevalence of over 3 million individuals, resulting in over one million fatalities in the United States each year. AMI may be classified into two distinct categories: non-ST segment elevation myocardial infarction (NSTEMI) and STEMI (Barberi and van den Hondel, 2018 ; Nascimento, 2019).

STEMI is caused by the sudden complete or almost complete blockage of a coronary artery, and its identification necessitates immediate restoration of blood flow (Mizuguchi *et al.,* 2016). Nevertheless, it has been noted that individuals with NSTEMI may sometimes have a completely blocked culprit artery. Recent extensive retrospective investigations indicate that as many as 30% of these patients had a blocked culprit artery (Ayad *et al.,* 2021).

These findings indicate that there is a significant subset of NSTEMI patients who might have advantages from primary coronary angiography, as opposed to receiving medical treatment with elective angiography (Nikus *et al.,* 2010).

In recent times, there has been a trend towards identifying new electrocardiogram (ECG) alterations that strongly indicate complete blockage of the coronary arteries, but do not show an elevation of the ST segment in adjacent leads, which would classify them as STEMI equivalents. It is

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essential to recognize these STEMI equivalents promptly in order to prevent delayed restoration of blood flow (Wall *et al.,* 2018).

Three specific patterns have been increasingly recognized as "STEMI equivalents" that need immediate attention. De Winter T waves are indicative of proximal blockage of the LAD artery. They are characterized by tall and symmetric T waves, accompanied by ST depression in leads V1-V4 (de Winter *et al.,* 2008; Verouden *et al.,* 2009). Global ST segment depression in most leads and elevation of the ST segment in lead aVR has been related with left main (LM) coronary artery blockage (Rostoff *et al.,* 2005; Kosuge *et al.,* 2015). There are two variants of Wellen syndrome that have been documented, and both indicate a serious narrowing of the LAD. Type A is distinguished by the presence of biphasic T waves in leads V2-V3, whereas type B is defined by the presence of deep T waves in V2-V3 (Merlo *et al.,* 2021).

The objective of this study was to assess STEMI equivalent in patients admitted at cardiac care center ,Tanta university.

2. Patients and Methods

This prospective observational registry was carried out on 50 patients aged from 39 to 81 years old, both sexes, presented with ECG findings of STEMI equivalent (De Winter syndrome -Wellen syndrome -global ST segment depression and elevation in aVR) undergoing PCI.

The research was conducted from March 2022 to March 2023, after clearance from the Ethical Committee of Tanta University Hospitals in Tanta, Egypt. The patient provided a well-informed written consent.

Exclusion criteria were patients with STEMI undergoing primary PCI, missed myocardial infarction who are not eligible for primary PCI, hyperkalaemia, tachycardia and pulmonary oedema with respiratory failure, as these commonly mimic ischemic ECG changes, pacemakers or those with post cardiac arrest, acute coronary syndrome (ACS) in associations with metabolic acidosis and patients on mechanical ventilation or very ill patients.

All patients were subjected to: history taking, clinical examination, laboratory investigations [serum urea and creatinine, cardiac enzymes include serum troponin and CKMB, sodium, potassium and hemoglobin level] and reperfusion [coronary angiography done to assess angiographic lesion then reperfusion through PCI for IRA done] and Local cardiac examination: abnormal pulsation, heart sounds & murmurs.

The GRACE score is used to stratify the risk of patients based on their scores, which are classified into three categories: low risk (<109 points), intermediate risk (109 to 140 points), and high risk (>140 points). Additionally, symptoms of heart failure may be assessed using the KILLIP classification for acute myocardial infarction(AMI). **Class I**: Absence of heart failure evidence, **Class II**: Indicative of mild to severe heart failure (presence of S3 gallop, lung rales below the midpoint of the posterior lung fields, or jugular venous distention). **Class III**: The patient is experiencing severe pulmonary edema, classified as **Class IV**, which is indicative of cardiogenic shock.

Resting 12 leads ECG

It was collected from all patients upon admission to the hospital within 10 minutes of first medical contact (FMC) in accordance with the 2017 criteria of the European society of cardiology (ESC). This data includes limb leads I, II, and III as well as aVR, aVL, and aVF, as well as chest leads from V1 to V6 (Ibanez *et al.,* 2017). The ECG recordings were analyzed, ischemic ECG changes other than typical criteria of ST segment elevation MI were assessed, and three ECG pattern of STEMI equivalent were addressed: In the de-winter pattern, the ST-segment depression at the J point in the precordial leads is upsloping and measures around 1 to 3 mm. A persistent hyperacute T wave is also present, similar to a STEMI, but this one does not evolve into conventional ST-segment elevation (Jimeno-Sánchez *et al.,* 2023).

The ECG shows pronounced T-wave inversions in leads V2 and V3, and possibly in leads V1, V4, V5, and V6. Alternatively, there may be biphasic T waves with initial positivity and terminal negativity in leads V2 and V3. The precordial R-wave progression is preserved and there are no precordial Q waves, indicating the absence of any previous anterior wall heart attack (Udechukwu *et al.,* 2018). There is widespread ST segment depression (more than 0.1 mV) in about eight leads, accompanied by ST segment elevation in lead aVR (Wagner *et al.,* 2009).

PCI for IRA

A loading dose of dual anti platelet (Aspirin 300mg chewable) plus P2Y12 inhibitor (ticagrelor 180 mg or clopidogrel 600mg), plus IV unfractionated heparin (UFH) or low molecular weight heparin (LMWH) were used before the. Glycoprotein IIb/IIIa inhibitors (eptifipatide or tirofiban) were used during or after the procedure in selected cases (Sarmiento, 2016). In this study, both femoral and radial arterial approaches were used. Femoral approach: An infiltration of local anesthetic is administered in a region of 3 to 4 cm in diameter, located 3 to 4 cm below the inguinal ligament. The expected location for the puncture should be directly above the bone, ensuring sufficient compression of the blood vessels when the sheaths are taken out. The side arm port enables uninterrupted pressure monitoring and infusion while catheters are being pushed through the sheath to reach the heart (Sarmiento, 2016) [16]. Radial approach: Radial access was done in selected patients with normal Allen's test and no previous history of abnormal anatomy, generally the choice of the arterial access was up to the operators to decide (Valgimigli *et al.,* 2015, Jolly *et al.,* 2009).

Left coronary imaging: A contrast injection in the left coronary cusp is a reasonable first step to define the ostium of the LM coronary artery, an antero-posterior (AP) view or a shallow right anterior oblique (RAO) caudal view may be useful to evaluate middle and distal LM coronary artery stenosis. A shallow left anterior oblique (LAO) or LAO cranial view is usually best to visualize osteal LM stenosis. Adequate visualization of the left coronary system commonly requires five or more views: The LAO view, the RAO view, the AP cranial view, the AP caudal view, the spider view (Sarmiento, 2016).

Right coronary imaging: The Right coronary artery (RCA) should be approached in the 30-degree LAO projection. The Judkin Right 4 (JR4) is advanced to the aortic valve level and is slowly withdrawn approximately 2 cm while clockwise rotation is applied to rotate the catheter anteriorly to the right sinus of Valsalva then the catheter should sit in the RCA ostium (Sarmiento, 2016). Usually, two or three views of the RCA are obtained: The LAO view is useful to evaluate the proximal and mid-RCA, the AP view with 30-degree cranial angulation is often the best for evaluating the RCA bifurcation, ostia of the Peripheral arterial disease (PDA) and posterolateral branches and a shallow RAO view is useful to show the entire PDA (Sarmiento, 2016).

Reperfusion success: TIMI (Thrombolysis in Myocardial Infarction) grading system: TIMI grade 3 flow should be the goal for achieving reperfusion of the epicardial infarct artery (Appelbaum *et al.,* 2009; Sarkar *et al.,* 2023). TIMI 0 flow indicates the complete absence of blood flow beyond a blockage in the coronary artery. TIMI 1 flow refers to a weak flow of blood beyond the blockage, with incomplete filling of the area supplied by the artery. TIMI 2 flow indicates a delayed or slow flow of blood, but with complete filling of the affected area. TIMI 3 flow represents normal blood flow that completely fills the area supplied by the artery.

2-D Echocardiography was performed with the patient lying partially on their left side to evaluate the left ventricle's ability to contract and pump blood. This was done using Simpson's method, which involves analysing images from the apical 4 and apical 2 views. The ejection fraction, which measures the percentage of blood pumped out of the left ventricle during each heartbeat, was calculated using a specific equation based on the volume of the ventricle during systole and diastole**.** (Lo and Thomas, 2009). EF is equal to the difference between LVEDV and LVEDV divided by LVEDV, multiplied by 100. to find the percentage change in diameter between the left ventricle end diastolic diameter and the left ventricle end systolic diameter, or fractional shortening. The size decrease may be expressed as a percentage using the following equation: (Chengode, 2016) is a Subtract the value of LVEDD from the value of FS, then divide the result by LVEDD, then multiply the result by 100. By applying the Teichholz equation to the volume measurements obtained from the diastolic and systolic diameters, the ultrasound machine is able to calculate the ejection fraction (Chengode, 2016).

Assess segmental wall motion abnormalities and global wall motion

In order to assess regional wall motion anomalies, a 17-segment model for LV segmentation was used. In order to assess regional wall motion anomalies, the left ventricle was split into 17 segments: 6 basal, 6 mid-ventricular, and 5 apical. The 17-segment model of the left ventricle in parasternal long axis, parasternal short axis, apical 2 chamber, and apical 4 chamber perspectives was used to evaluate RWMA by TTE in each patient of the research. Assuming RWMA was present in at

least one segment, the findings were documented as normal, hypokinetic, akinetic, or dyskinetic wall motion. Each of the seventeen segments had their RWMA evaluated (Lo and Thomas, 2009).

Statistical analysis

The statistical analysis was conducted using SPSS v26 (IBM Inc., Chicago, IL, USA). The quantitative variables were expressed as the mean and standard deviation (SD). The qualitative variables were shown in terms of frequency and percentage (%).

3. Results

The demographic data and baseline characteristics were presented in table 1.

Table 1: Distribution of the studied cases according to demographic data, patient characteristics, risk factors, clinical presentation and GRACE score

		N=50
Age (years)		57.16 ± 9.53
Sex	**Male**	36(72.0%)
	Female	14(28.0%)
BMI (kg/m^2)		25.82 ± 4.40
	Risk Factors	
Hypertension		28(56.0%)
DM		24(48.0%)
Dyslipidemia		21(42.0%)
Smoking		31(62.0%)
Cerebrovascular disease		3(6.0%)
Renal		3(6.0%)
Hepatic		4(8.0%)
COPD		2(4.0%)
Chemo or radio		1(2.0%)
Previous CVD		15(30.0%)
Family history of CVD		27(54.0%)
	Clinical presentation	
HR		83.02 ± 11.59
ECG Rhythm	**SR**	49(98.0%)
	AF	1(2.0%)
SBP at admission		127.80 ± 22.79
DBP at admission		80.0 ± 16.32
KILLIP	**I**	37(74.0%)
	II	8(16.0%)
	III	5(10.0%)
ECG ischemic changes	**Wellen syndrome**	27(54.0%)
	De winter syndrome	4(8.0%)
	Most leads ST dep.	19(39.0%)
	GRACE score	
Low risk		12(24.0%)
Intermediate risk		30(60.0%)
High risk		8(16.0%)

Data are presented as mean ± SD or frequency (%). BMI: Body mass index, DM: Diabetes mellitus, COPD: Chronic obstructive pulmonary disease, CVD: Cardiovascular disease, CVA: cerebro-vascular accidents, TIA: transient ischemic attacks, HR: heart rate, ECG: Electrocardiogram, AF: Atrial fibrillation, SR: sinus rhythm, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, GRACE: Global registry of acute coronary events.

The mean of troponin was positive in 45(90%) patients of the study population. The mean of CK-MB was 64.34 ± 26.45 U/L. 28(56%) patients had coronary angiography performed through femoral artery access and 22 (44%) through radial access. 17(34%) patients had single vessel disease and 33(66%) patients had multi-vessel disease. There were no patients with normal coronary vessels. LAD was the culprit artery in 44(88%) patients, LM was the culprit vessel in 5(10%) patients while RCA was the culprit vessel in 1(2%) patient. 45(90%) patients had severe grade of stenosis in the culprit vessel while 5(10%) patients had total occlusion of the culprit vessel. Culprit segment was the

proximal segment in 34(68%) patients, mid segment was affected in 11(22%) patients, while distal segment was affected in 5(10%) patients. LM was affected in 1(2%) patient, LAD was affected in 4(8%) patients, Diagonal was affected in 3(6%) patients, LCX was affected in 17(34%) patients, OM was affected in 2(4%) patients of cases and RCA was affected in 20(40%) patients of the study population (Table 2).

Table 2: Descriptive analysis of the studied cases according to laboratory data, troponin and PCI findings

		N=50
Hb level		12.06 ± 1.57
Creatinine		1.06 ± 0.33
Urea		52.82 ± 17.97
LDL		120.0 ± 25.45
HDL		36.73 ± 6.44
Triglyceride		156.2 ± 22.62
K		4.01 ± 0.40
Na		143.98 ± 5.32
RBS		176.1 ± 74.01
CKMB		64.34 ± 26.45
Troponin		45(90.0%)
	PCI findings	
Access	**Femoral**	28(56.0%)
	Radial	22(44.0%)
Number of diseased vessels	**Single vessel disease**	17(34.0%)
	Multiple vessel disease	33(66.0%)
Culprit vessel	**LM**	5(10.0%)
	LAD	44(88.0%)
	Diagonal	0(0.0%)
	LCX	0(0.0%)
	OM	0(0.0%)
	RCA	1(2.0%)
Culprit lesion stenosis grading	**Minimal (< 25% stenosis)**	0(0.0%)
	Mild (25-49% stenosis)	0(0.0%)
	Moderate (50-69%)	0(0.0%)
	Severe (70-99%)	45(90.0%)
	Totally occluded (100%)	5(10.0%)
Culprit segment location	**Proximal segment**	34(68.0%)
	Mid segment	11(22.0%)
	Distal segment	5(10.0%)
Non culprit lesion	**LM**	1(2.0%)
	LAD	4(8.0%)
	Diagonal	3(6.0%)
	LCX	17(34.0%)
	OM	2(4.0%)
	RCA	20(40.0%)

Data are presented as mean ± SD or frequency (%), LDH: Lactate dehydrogenase, HDL: high-density lipoprotein, RBS: Random blood sugar, CKMB: creatine kinase-myocardial band, PCI: Percutaneous coronary intervention, LAD: Left anterior descending artery, LM: left main coronary artery, LCX: Left circumflex artery, OM: Obtuse marginal branch, RCA: Right coronary artery.

TIMI flow 0 presented in 5(10 %) of patients, 44(88%) patients had TIMI I flow, and 1(2%) patient had TIMI flow II. 4(8%) patients had TIMI flow < 3, and 46(92 %) patients had TIMI III flow. 17(34%) patients were treated with tirofiban either during or after PCI. The mean of ESD was 4.99 ± 0.53 mm. The mean of EDD was 3.90 ± 0.42 mm. The mean of EF was 50.72 ± 7.09 %. 48(96.0%) patients had SWMA, and 2(4.0%) patients had no SWMA (Table 3).

Table 3: Distribution of the studied cases according to TIMI flow, need for tirofiban, echocardiographic parameters assessment before discharge

		N=50
T1M1 flow before PCI	**0**	5(10.0%)
	1	44(88.0%)
	2	1(2.0%)
	3	0(0.0%)
T1M1 flow after PC1	**0**	0(0.0%)
	1	0(0.0%)
	2	4(8.0%)
	3	46(92.0%)
Need for tirofiban		
Need for tirofiban		17(34.0%)
Echocardiographic parameters		
EF		50.72 ± 7.09
ESD		4.99 ± 0.53
EDD		3.90 ± 0.42
SWMA		48(96.0%)

Data are presented as mean ± SD or frequency (%), PCI: Percutaneous coronary intervention, TIMI: thrombolysis in myocardial infarction, Echo: echocardiogram, EF: Ejection fraction, ESD: end systolic diameter, EDD: end diastolic diameter, SWMA: Segmental wall motion abnormality.

Significant involvement of basal anterior, basal anteroseptal, mid inferoseptal, or mid anterolateral segments indicates typical involvement of LAD territory; over 80% of the subject's exhibited predominance of involvement in either the mid anterior, mid anteroseptal, apical anterior, or apical septal walls. Other segments show varying degrees of regional wall motion abnormalities, suggesting that the LAD-RCA and LAD-LCX (left circumflex) arteries are involved in the overlapping zones of supply (Table 4)

Only individuals admitted to the hospital had MACEs documented. A MI, CHF, stroke, or death occurred while the patient was in the hospital; these events were referred to as MACE. No patients died during hospital stay (0%). No patients suffered from re-infarction during hospital stay (0%). 9(18%) patients suffered from CHF, 9(18%) patients needed IV diuretics and 4(8%) patients needed noninvasive ventilation. 4(8 %) patients suffered from bleeding complications. 2(4%) patients of the study population suffered from CIN. No patients suffered from stroke (0%). No patients suffered from cardiogenic shock (0%). 3(6%) patients suffered from atrial arrhythmia during hospital stay and 2(4%) patients of the population study suffered from ventricular arrhythmia during hospital stay. The duration of the hospital stay was 1.8 ± 1.18 days (Table 5).

Before time of admission, 16(32%) patients were on aspirin, 3(6%) patients were on P2Y12 receptor inhibitor, 8(16%) patients were on statins, 11(22%) patients were on B-blockers, 14 (28%) patients were on ACE inhibitors, 12(24%) patients were on ARBs, 2(4%) patients were on oral diuretics, 9(18%) patients were on nitrates, 1(2%) patient was on oral anticoagulation. Upon discharge, 48 (96%) patients were on aspirin, 50(100%) patients were on P2Y12 receptor inhibitor, 44(88%) patients were on statins, 43(86%) patients were on B-blockers, 28(56%) patients were on ACE inhibitors, 17(34%) patients were on ARBs, 11(22%) patients were on oral diuretics, 4(8%) patients were on ivabradine, 2(4%) patients were on MRAs, 16(32%) patients were on nitrates, 3(6%) patients was on oral anticoagulation (Table 6).

Table 4: Segmental wall motion pattern corresponding to the respective echocardiographic left ventricular segments

LV segment	Wall motion abnormality	N=50
Basal anterior 1	**Hypokinesia**	34(68.0%)
	Akinsia	1(2.0%)
Basal anteroseptal 2	**Hypokinesia**	35(70.0%)
	Akinsia	1(2.0%)
Basal inferoseptal 3	**Hypokinesia**	10(20.0%)
	Akinsia	0(0.0%)
Basal inferior 4	**Hypokinesia**	13(26.0%)
	Akinsia	0(0.0%)
Basal inferolatreal 5	**Hypokinesia**	9(18.0%)
	Akinsia	0(0.0%)
Basal anterolateral 6	**Hypokinesia**	11(22.0%)
	Akinsia	0(0.0%)
Mid anterior 7	**Hypokinesia**	44(88.0%)
	Akinsia	2(4.0%)
Mid anteroseptal 8	**Hypokinesia**	45(90.0%)
	Akinsia	1(2.0%)
Mid inferoseptal 9	**Hypokinesia**	27(54.0%)
	Akinsia	0(0.0%)
Mid inferior 10	**Hypokinesia**	12(24.0%)
	Akinsia	0(0.0%)
Mid inferlateral 11	**Hypokinesia**	7(14.0%)
	Akinsia	0(0.0%)
Mid Anterolateral 12	**Hypokinesia**	23(46.0%)
	Akinsia	0(0.0%)
Apicoanterior 13	**Hypokinesia**	40(80.0%)
	Akinsia	2(4.0%)
Apicoseptal 14	**Hypokinesia**	41(82.0%)
	Akinsia	2(4.0%)
Apicoinferior 15	**Hypokinesia**	4(8.0%)
	Akinsia	0(0.0%)
Apicolateral 16	**Hypokinesia**	8(16.0%)
	Akinsia	0(0.0%)
Apical 17	**Hypokinesia**	17(4.0%)
	Akinsia	0(0.0%)

Data are presented as frequency (%).

Table 5: Distribution of the studied cases according to in-hospital follow up of MACE, duration of hospital stay

	N=50
Reinfarction	0(0.0%)
HF	9(18.0%)
Stroke	0(0.0%)
Mortality	0(0.0%)
bleeding complications	4(8.0%)
Atrial arrhythmias	3(6.0%)
Ventricular arrhythmias	2(4.0%)
CIN	2(4.0%)
Need for diuretics	7(14.0%)
Noninvasive ventilation	3(6.0%)
Duration of hospital stay	
Hospital stays (days)	1.8 ± 1.18
1	24(48.0%)
2	21(42.0%)
>2	5(10.0%)

Data are presented as mean ± SD or frequency (%), MACE: major adverse cardiac events, HF: Heart failure , CIN: Cervical intraepithelial neoplasia.

Table 6: Distribution of the studied cases according to medications

	N=50	
	Preadmission	**At discharge**
Aspirin	16(32.0%)	48(96.0%)
P2Y12 receptor inhibitor	3(6.0%)	50(100.0%)
Statins	8(16.0%)	44(88.0%)
B – Blockers	11(22.0%)	43(86.0%)
ACE-I	14(28.0%)	28(56.0%)
ARBS	12(24.0%)	17(34.0%)
Diuretics	2(4.0%)	11(22.0%)
Ivabradine	0(0.0%)	4(8.0%)
MRAs	0(0.0%)	2(4.0%)
Nitrates	9(18.0%)	16(32.0%)
Anticoagulations	1(2.0%)	3(6.0%)

Data are presented as frequency (%), ACE: Angiotensin converting enzymes inhibitors, ARBs: Angiotensin receptor blockers, MRAs: Mineralo-corticoid antagonists.

Cases

Case 1

Male patient aged 47 years old, known to be diabetic and active shisha smoker, not hypertensive patient, not known to be cardiac before, negative family and drug history, BMI was 18. He presented to cardiology department, Tanta university hospital complaining of typical angina pain. Vital signs: B/P 120/70 mmHg, Heart rate 75 bpm, Killip class: I, and ECG: sinus rhythm with ST segment depression and hyper-acute T wave from V1 – V4 (De winter pattern). Figure 1, He initially received 4 tablets of chewable acetyl salicylic acid 75 mg (300mg), 300 mg clopidogrel, 80 mg atorvastatin and He also received 5000 IU of UFH intravenously. Laboratory investigations were within normal except for positive troponin with CKMB value 57 (Reference up to 25). Then patient entered the catheterization laboratory where coronary angiography was performed through a femoral approach. The coronary angiography revealed that the LM was normal, bifurcating into LAD and LCX, the LAD has proximal subtotal thrombotic occlusion with TIMI 1 flow, while left circumflex artery (LCX) and RCA showed no significant lesions. Full loading with additional 300 mg Clopidogrel was given, then engagement to left system was done, wiring through subtotal lesion. Pre-dilatation was done with multiple balloon inflation then a DES was deployed to LAD with final TIMI III flow. Figure 2

The patient returned to the CCU for follow up and monitoring. IV tirofiban was given as bail-out therapy. No in-hospital MACE was documented and the ECHO before discharge showed an EF about 50-55%. He was discharged from CCU after 2 days on anti-ischemic medications Figure 3.

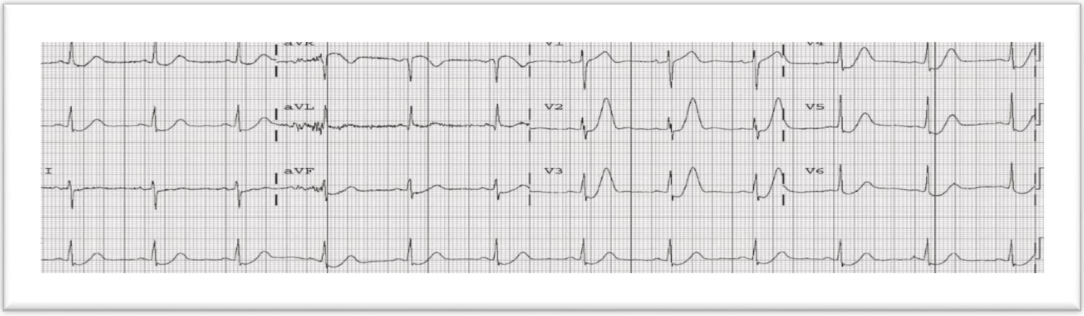

Fig. 1: ECG showed ST depression and hyper-acute T wave in anterior chest leads

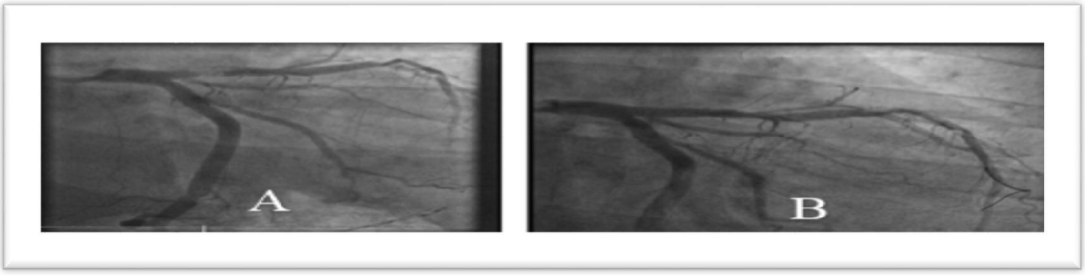

Fig. 2: A. RAO view showed subtotal lesion in the proximal LAD. B.LAD coronary artery in RAO view showing result after stent placement.

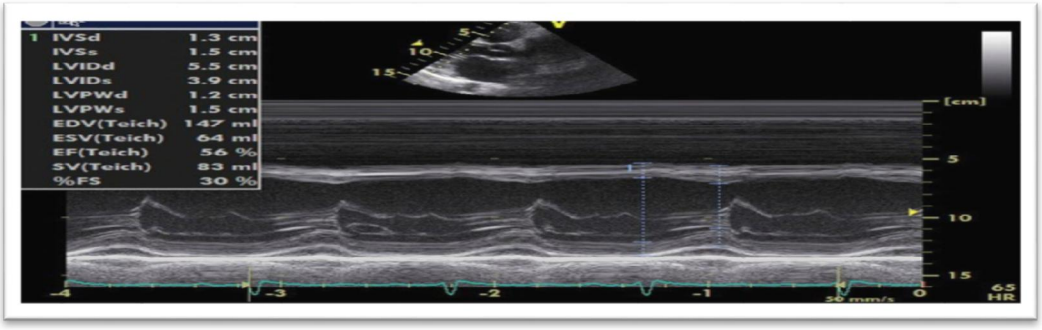

Fig. 3: Measurement of LV systolic function by M-mode in the parasternal long view

Case 2

Female patient aged 78 years old, known to be hypertensive and diabetic, not known to be cardiac before, positive family history of CAD, BMI was 30, presented to cardiology department, Tanta university hospital complaining of typical angina pain. Vital signs were B/P 160/100 mmHg, Heart rate 80 bpm, Killip class: II, and ECG: sinus rhythm with ST segment elevation in aVR with diffuse ST segment depression Figure 4.

He initially received 4 tablets of chewable acetyl salicylic acid 75 mg (300mg), 300 mg clopidogrel, 80 mg atorvastatin and He also received 5000 IU of UFH intravenously. Laboratory investigations were within normal except for positive troponin with CKMB value 69 (Reference up to 25). Then patient entered the catheterization laboratory where coronary angiography was performed through femoral approach. The coronary angiography revealed that the LM had distal significant lesion, bifurcating into LAD and that had no significant lesions, while RCA showed mid segment significant stenosis. Full loading with additional 300 mg Clopidogrel was given, then engagement to left system was done, wiring through total lesion. Pre-dilatation was done with multiple balloon inflation then a DES was deployed to LAD with final TIMI III flow Figure 5.

The patient returned to the CCU for follow up and monitoring. MACE was documented in form of heart failure symptoms, managed with IV diuretics. Patient developed contrast induced nephropathy that resolved later. ECHO before discharge showed an EF about 30-35%. She was discharged from CCU after 5 days on anti-ischemic and anti- failure medications Figure 6.

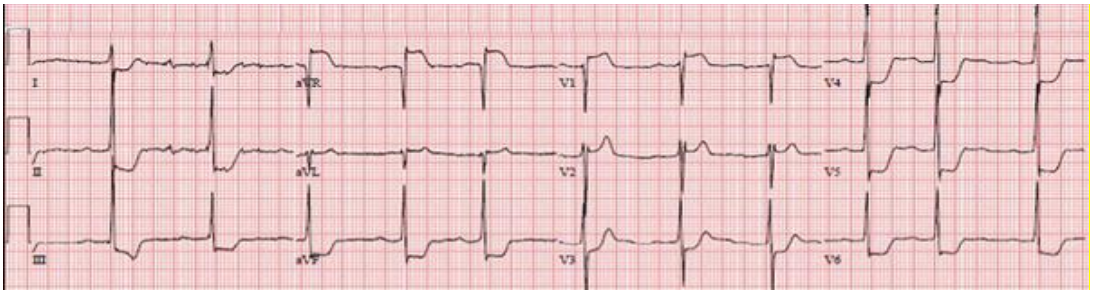

Fig. 4: ECG showed ST segment elevation in aVR with diffuse ST depression

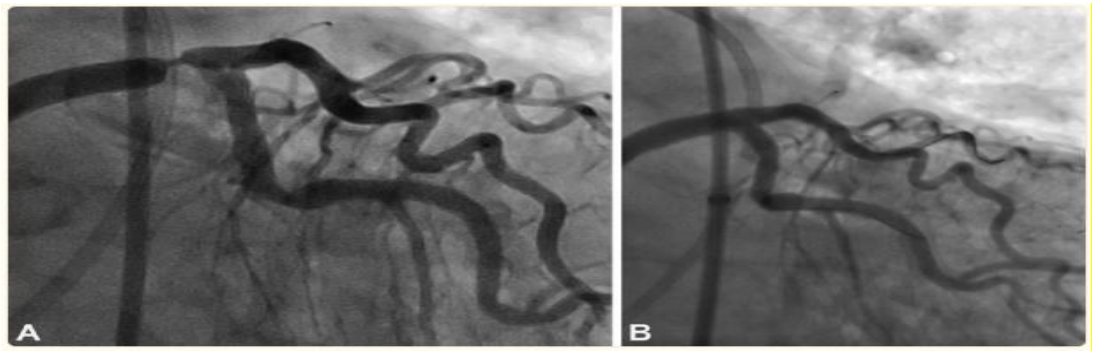

Fig. 4: A**.** Severe stenosis of the distal LM coronary artery with no significant lesions in LAD and LCX. B**.** Angiogram following successful placement of a DES in the distal LM coronary artery

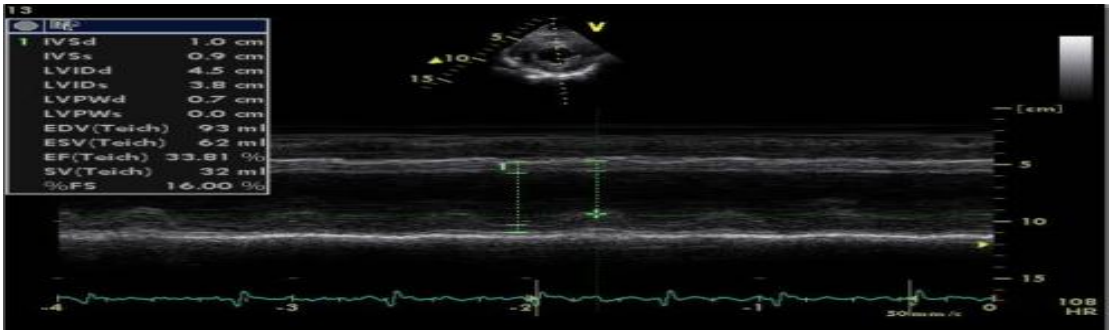

Fig. 5: Measurement of LV systolic function using M-mode in the parasternal short view

4. Discussion

According to the present study, 27 of the study population presented by wellen syndrome (54%), 19 patients presented by global ST segment depression with elevated ST segment in aVR (38%) and 4 patients presented by de Winter syndrome (8%) also, 74% of patients in this study presented by killip class I while 16% presented by killip class II and only 10% presented by killip class III. These findings came in agreement to the study of Polonski *et al.* (2011) reported 87% of NSTEMI patients had KILLIP classes I and II at time of presentation. Another study conducted by Lupu *et al.* (2022) found that 76.2% of very high risk NSTEMI patients were Killip I and II while 19.2 % were Killip III class .

Most of the patients had a normal heart rate with average 83.02 ± 11.59, systolic blood pressure with average 127.80 ± 22.79 and diastolic blood pressure with average 80.0 ± 16.32. Similar results were noticed by Abdelhameed *et al.* (2021) about pattern and outcomes of patients presented with high risk NSTEMI .

In this study, 12(24%) patients of the study population were low risk GRACE score, 30(60%) patients were intermediate risk GRACE score and 8(16%) patients were high risk GRACE score. Compared to a study conducted by Lupu *et al.* (2022) that found high GRACE score was observed in 36.4% of very high risk NSTEMI patients and 13.5% of high risk NSTEMI patients.

It was found that 33(66%) of the patients had multi-vessel coronary disease and 17 (34%) patients had single culprit vessel disease. Multi-vessel disease was defined as presence of ≥ 1 lesion with > 50 % stenosis in ≥ 1 major epicardial coronary artery or its major branches remote from the IRA (Boden *et al.,* 2007). In agreement with a study , Lupu *et al.* (2022) in which percentage of MVD in very high risk patients was 76.3% and in high risk NSTEMI patients was 63% while, the percentage of single vessel disease in very high risk patients was 21% and in high risk NSTEMI patients was 31%.

LAD was the prevalent culprit artery in 44(88%) patients, LM was the culprit in 5(10%) patients, while RCA was the culprit artery in 1(2 %) patients of our study population. This came in agreement with a study, Deharo *et al.* (2017) in which LAD was the most affected culprit artery (32%) in high-risk patients With NSTEMI patients managed invasively, followed by RCA (22.5%)

then LCX (19.6%) and LMA (5%) , also Lupu *et al.* (2022) found that LAD was the most affected culprit artery in very high risk patients (61.2%) and in high risk patients (48.9%) followed by LCX with affection percentage (32%) and (35%) respectively, LM was affected in (15%) and (3%) respectively.

Percentage of culprit stenosis in this study ranged between severe degree of stenosis (70-99%) and total occlusion (100%) with a percentage of 90 % and 10 % respectively. These findings were comparable to the study of Deharo *et al.* (2017) who reported 52% of patients with high risk NSTEMI who underwent coronary angiography had (50-90% luminal stenosis) while 45.4% of patients had (90-100% luminal stenosis).

Regarding location of culprit segment, proximal segment was the most affected in 34 patients (68%) then mid segment in 11 patients (22%) followed by distal segment in 5 patients (10%) of study population. Our findings were similar to the study of Ayad *et al.,* 2021 [4] who reported 50% of culprit vessels of totally and near totally occluded vessels in NSTEMI patients had proximal segment affection, 35.7% at mid segment and 14.3% at distal segment. Also, de Winter *et al.* (2019) found that proximal LAD was the most affected segment in de Winter patients of STEMI equivalents cases with either total or subtotal occlusion.

Our study Final 2 and 3 TIMI flow grades in the culprit artery were 8% and 92% respectively. similar to the study conducted by Terlecki *et al.* (2021) about Impact of acute total occlusion of the culprit artery on outcome in NSTEMI based on the results of a large Polish national registry (Dudek *et al.* (2019) that found final TIMI flow 3 in NSTEMI patients with and without total culprit occlusion were 83.4% and 95.6% respectively, and final TIMI flow 2 in both groups were 5.1% and 2.6 % respectively.

34% of our study population were treated with tirofiban either during or after PCI. This finding was in agreement with the study of Ayad *et al.* (2021). about impact of totally occluded culprit coronary artery in patients with NSTEMI , where use of GP IIa–IIIb antagonists was 23% of NSTEMI patients with totally occluded culprit vessel . Our findings were greater than the Deharo *et al.* (2017) who reported that 1% of high risk NSTEMI patients managed invasively needed bailout use of GpIIb/IIIa inhibitor. Matching these data, STEMI equivalents represent highly thrombotic field that may cause thrombotic procedural complications.

Echocardiographic study was done for all the study population during the hospital stay, ejection fraction ranged from 37.0 – 70.0 with the mean ejection fraction 50%. Only 1 patient have impaired LV systolic function (<40%), 21 patients (42%) had mild reduced EF (40-49%), and most of patients 28 (56%) had EF levels >50%. Similarly, among the high risk NSTEMI group.

A staggering 96% of the participants exhibited abnormalities in segmental wall motion. Of those, over 80% exhibited predominance of involvement in the mid anterior, mid antero-septal, apical anterior, or apical septal walls. Moreover, a considerable portion of the participants exhibited involvement in the basal anterior, basal antero-septal, mid infero-septal, and mid anterolateral segments, suggesting the typical involvement of LAD territory. Variable degree of regional wall motion abnormality was seen in other segments indicating involvement of overlapping zones of LAD-RCA and LAD-LCX (left circumflex) artery supply. In agreement with Okobi *et al.* (2022); de Winter *et al.* (2019) and Kossaify *et al.* (2013) where LAD was the most affected culprit vessel in the 3 STEMI equivalent pattern: Wellens syndrome, De Winter T waves, and ST-Segment Elevation in Lead aVR With global ST-Segment Depression discussed in our study.

MACE occurred in 18% of study population. No patients died during hospital stay or suffered from re-infarction or stroke during hospital stay. Nine patients suffered from in hospital congestive heart failure (18%), 9 patients needed IV diuretics (18%) and 4 patients needed non-invasive ventilation (8%). No patients suffered from cardiogenic shock. In-hospital bleeding complications was observed in 4 patients (8%) of study population. 2 patients of the study population suffered from contrast induced nephropathy (4%). 3 patients suffered from atrial arrhythmia during hospital stay (6%) and 2 patients of the population study suffered from ventricular arrhythmia during hospital stay (4%).

The incidence of MACE in our study was compared to the study conducted by Lupu *et al.* (2022) among patients with very high risk and high risk NSTEMI patients managed with early invasive strategy that had a MACE in 28% of very high risk patients while MACE was detected in 5.8% in high risk patients. These variations might be due to exclusion of CHF form MACE and

duration for MACE follow up was 30 days, unlike our study that observed in-hospital MACE including CHF.

All patients in our study underwent coronary angiography within 24 h of presentation ranging from 5.5 - 21.0 hours with a mean of 12.97 ± 4.05, compared to another study conducted by Mahendiran *et al.* (2020) showed rate of MACE in high risk NSTEMI group with GRACE score >140 managed with PCI within 24 h of admission was 22.2% with no statically significant difference between patients receiving invasive angiography within 12 hours to those treated between 12 and 24 hours. This could shed better light on severity of STEMI equivalent patterns discussed in our study, being mostly of moderate GRACE risk group but carries equal rate of MACE with other high GRACE risk groups. Compared to another study conducted by Deharo *et al.* (2017) about timing of angiography and outcomes in high-risk patients With NSTEMI patients managed invasively, the rate of MACE at 7 days following invasive intervention within 24 h of presentation was 13.5% .

The limitations of our study were small size of study population, which was due to short study duration in a single-centre experience and some patients refused doing PCI at our centre due to logistic or cultural issues as well and the short period for follow up the patients (in hospital stay) which didn't allow the appearance of results for mortality, re-infarction and re-hospitalization.

Conclusions

Identifying individuals with STEMI-equivalents, such as NSTEMI, provides an opportunity for timely intervention by rapid coronary angiography. Immediate reperfusion is crucial for these individuals, since their death rate remains elevated in the absence of therapy.

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References

Abdelhameed, M., O. Hakim, A. Mohamed, and E. Gadour, 2021. Pattern and Outcome of Acute Non-ST-Segment Elevation Myocardial Infarction Seen in Adult Emergency Department of Al-Shaab Teaching Hospital: A prospective Observational Study in a Tertiary Cardiology Center. Cureus., 13:e17981.

Appelbaum, E., A.J. Kirtane, A. Clark, Y.B. Pride, E.V. Gelfand, C.J. Harrigan, et al., 2009. Association of TIMI myocardial perfusion grade and ST-segment resolution with cardiovascular magnetic resonance measures of microvascular obstruction and infarct size following ST-segment elevation myocardial infarction. Journal of Thrombosis and Thrombolysis, 27:123-9.

Ayad, S.W., T.H. El Zawawy, M.I. Lotfy, A.M. Naguib, and A.M. El Amrawy, 2021. Incidence and impact of totally occluded culprit coronary artery in patients with non-ST segment elevation myocardial infarction acute coronary syndrome. Egypt Heart J., 73:36.

Barberi, C., and K.E. van den Hondel, 2018. The use of cardiac troponin T (cTnT) in the postmortem diagnosis of acute myocardial infarction and sudden cardiac death: A systematic review. Forensic science international, 292:27-38.

Boden, W.E., R.A. O'Rourke, K.K. Teo, P.M. Hartigan, D.J. Maron, W.J. Kostuk, et al., 2007. Optimal medical therapy with or without PCI for stable coronary disease. New England journal of medicine, 356:1503-16.

Chengode, S., 2016. Left ventricular global systolic function assessment by echocardiography. Ann. Card Anaesth., 19:S26-s34.

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de Winter, R.J., N.J. Verouden, H.J. Wellens, and A.A. Wilde, 2008. A new ECG sign of proximal LAD occlusion. New England Journal of Medicine, 359:2071-3.

de Winter, R.W., R. Adams, G. Amoroso, Y. Appelman, L. Ten Brinke, B. Huybrechts, et al., 2019. Prevalence of junctional ST-depression with tall symmetrical T-waves in a pre-hospital field triage system for STEMI patients. J Electrocardiol., 52:1-5.

Deharo, P., G. Ducrocq, C. Bode, M. Cohen, T. Cuisset, S.R. Mehta, et al., 2017. Timing of Angiography and Outcomes in High-Risk Patients With Non–ST-Segment–Elevation Myocardial Infarction Managed Invasively. Circulation, 136:1895-907.

Dudek, D., Z. Siudak, M. Grygier, A. Araszkiewicz, M. Dąbrowski, J. Kusa, et al., 2019. Interventional cardiology procedures in Poland in 2018. Summary report of the Association of Cardiovascular Interventions of the Polish Cardiac Society (AISN PTK) and Jagiellonian University Medical College. Advances in Interventional Cardiology/Postępy w Kardiologii Interwencyjnej, 15:391-3.

Ibanez, B., S. James, S. Agewall, M.J. Antunes, C. Bucciarelli-Ducci, H. Bueno, et al., 2017. ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). European heart journal, 39:119-77.

Jimeno-Sánchez, J., M. Javierre-Loris, D.L. Cuevas-León, and E. Bonasa-Alzuria, 2023. "Wellens" and "de Winter" electrocardiographic patterns as equivalents of ST-segment elevation myocardial infarction. Arch Cardiol Mex., 93:120-3.

Jolly, S.S., S. Amlani, M. Hamon, S. Yusuf, and S.R. Mehta, 2009. Radial versus femoral access for coronary angiography or intervention and the impact on major bleeding and ischemic events: a systematic review and meta-analysis of randomized trials. American heart journal, 157:132-40.

Kossaify A., 2013. ST segment elevation in aVR: Clinical significance in acute coronary syndrome. Clinical Medicine Insights: Case Reports, 6:CCRep. S11261.

Kosuge, M., K. Uchida, K. Imoto, S. Isoda, N. Karube, T. Ebina, et al., 2015. Prognostic value of ST-segment elevation in lead aVR in patients with type A acute aortic dissection. Journal of the American College of Cardiology, 65:2570-1.

Lo, Q., and L. Thomas, 2009. Echocardiographic evaluation of systolic heart failure. Australas J Ultrasound Med., 12:21-9.

Lupu, L., L. Taha, A. Banai, H. Shmueli, A. Borohovitz, S. Matetzky, et al., 2022. Immediate and early percutaneous coronary intervention in very high-risk and high-risk non-ST segment elevation myocardial infarction patients. Clinical Cardiology, 45:359-69.

Mahendiran, T., D. Nanchen, D. Meier, B. Gencer, R. Klingenberg, L. Räber, et al., 2020. Optimal Timing of Invasive Coronary Angiography following NSTEMI. J Interv Cardiol., 8513257.

Merlo, A.C., F. Pescetelli, P. Ameri, and I. Porto, 2021. Not Every ST-Segment Elevation Is a STEMI. JACC Case Rep., 3:283-5.

Mitsis, A., and F. Gragnano, 2021. Myocardial Infarction with and without ST-segment Elevation: a Contemporary Reappraisal of Similarities and Differences. Curr. Cardiol. Rev., 17:e230421189013.

Mizuguchi, Y., S. Hashimoto, T. Yamada, N. Taniguchi, S. Nakajima, T. Hata, et al., 2016Percutaneous coronary intervention for nonagenarian patients with ST-segment elevation myocardial infarction: Experience of a single Japanese center. J. Cardiol., 67:331-4.

Nascimento, B.R., L.C.C. Brant, B.C. Marino, L.G. Passaglia, and A.L.P. Ribeiro, 2019. Implementing myocardial infarction systems of care in low/middle-income countries. Heart, 105:20-6.

Nikus, K., O. Pahlm, G. Wagner, Y. Birnbaum, J. Cinca, P. Clemmensen, et al., 2010. Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology. J. Electrocardiol., 43:91-103.

Okobi, O.E., I.O. Bakare, E.O. Evbayekha, A. Olawoye, C.C. Umeh, and A. Sowemimo, 2022. Wellens Syndrome: A Possible Precursor. Cureus.;14:e31963.

Polonski, L., M. Gasior, M. Gierlotka, T. Osadnik, Z. Kalarus, M. Trusz-Gluza, et al., 2011. A comparison of ST elevation versus non-ST elevation myocardial infarction outcomes in a large registry database: are non-ST myocardial infarctions associated with worse long-term prognoses? International journal of cardiology,152:70-7.

Rostoff, P., W. Piwowarska, E. Konduracka, A. Libionka, M. Bobrowska-Juszczuk, K. Stopyra, et al., 2005. Value of lead aVR in the detection of significant left main coronary artery stenosis in acute coronary syndrome. Kardiologia Polska (Polish Heart Journal). 62:132-5.

Sarkar, A., W.S. Grigg, and J.J. Lee, 2023.TIMI Grade Flow. StatPearls. Treasure Island (FL): StatPearls Publishing

Sarmiento R.A., 2016. Grossman & Baim's Cardiac Catheterization, Angiography, and Intervention. Revista Argentina de Cardiología, 84:502-3.

Terlecki, M., W. Wojciechowska, D. Dudek, Z. Siudak, K. Plens, T.J. Guzik, et al., 2021. Impact of acute total occlusion of the culprit artery on outcome in NSTEMI based on the results of a large national registry. BMC Cardiovascular Disorders, 21:297.

Udechukwu, N., P. Shrestha, M.Z. Khan, A.A. Donato, 2018. Wellens' syndrome: a close call. BMJ Case Rep., 2018.

Valgimigli, M., A. Gagnor, P. Calabró, E. Frigoli, S. Leonardi, T. Zaro, et al., 2015. Radial versus femoral access in patients with acute coronary syndromes undergoing invasive management: a randomised multicentre trial. Lancet., 385:2465-76.

Verouden, N.J., K.T. Koch, R.J. Peters, J.P. Henriques, J. Baan, R.J. van der Schaaf, et al., 2009. Persistent precordial "hyperacute" T-waves signify proximal left anterior descending artery occlusion. Heart, 95:1701-6.

Wagner, G.S., P. Macfarlane, H. Wellens, M. Josephson, A. Gorgels, D.M. Mirvis, et al., 2009. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part VI: acute ischemia/infarction: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J. Am. Coll. Cardiol., 53:1003-11.

Wall, J., L.D. White, and A. Lee, 2018. Novel ECG changes in acute coronary syndromes. Would improvement in the recognition of 'STEMI-equivalents' affect time until reperfusion? Intern Emerg. Med.,13:243-9.