

Original Research Article

Hemoglobin Level as A Predictor of Major Adverse Cardiovascular Events and Short-Term Outcomes in Stemi Patient Treated with Pharmacoinvasive Strategy Versus Primary PCI

Abstract

Background: Anaemia is prevalent among cases with acute coronary syndrome (ACS) and has been linked to poor clinical prognosis. Guidelines for cases with ST-segment elevation myocardial infarction (STEMI) recommend timely primary percutaneous coronary intervention (pPCI) as the preferred reperfusion strategy. If timely pPCI cannot be performed, a pharmacoinvasive strategy (PI) is recommended within 12 hours of symptom onset. The aim of this work was to study and assess the impact of hemoglobin level as a predictor of MACE and short-term outcomes in cases treated with Primary PCI vs pharmacoinvasive strategy.

Methods: This prospective case-control observational study was conducted on 100 cases that were divided into 2 groups. Group I consisted of 50 anemic cases & group II consisted of 50 cases that were not anaemic. Both groups were subdivided into A subgroups that underwent revascularization by pPCI and B subgroups that underwent revascularization by pharmacoinvasive strategy.

Results: There was no significant difference in LVEF, infarct site and final TIMI flow, the anaemic groups showed statistically significant more total MACE than non-anaemic groups whether revascularized by pPCI or pharmacoinvasive strategy. As expected, anaemic cases tended to have higher bleeding complications especially those undergoing pharmacoinvasive strategy. The anaemic cases also were less likely to be discharged on RAAS and beta blockers.

Conclusions: Anaemic cases whether revascularized with pPCI or pharmacoinvasive strategy tend to have higher incidence of MACE and major bleeding with no significant difference in mortality. There was no significant difference between LVEF between the study groups.

Keywords: Haemoglobin, Major Adverse Cardiovascular Events, Stemi Patient, Pharmacoinvasive Strategy, Primary PCI.

UNDER PEER REVIEW

Introduction:

Anaemia is common in individuals with acute coronary syndrome (ACS) and has been linked to a poor outcome. Although some studies [1, 2] have demonstrated that a low admission haemoglobin level is an independent predictor of in-hospital, early and late death in cases with ST-elevation myocardial infarction (STEMI), others [3] have found no such connection. The therapy of anaemia in STEMI cases having primary percutaneous intervention (pPCI) is not definitively recommended by clinical guidelines [3]. Importantly, individuals with anaemia are typically excluded from clinical trials, and there is a dearth of data about their best therapy. In ordinary clinical practise, cases with anaemia are often treated conservatively [4].

The optimal reperfusion method for individuals with STEMI is timely pPCI. If pPCI cannot be conducted within 12 hours of symptom onset, a pharmacoinvasive approach (PI) is advised for individuals without contraindications [2].

In a not insignificant number of anaemic cases, intensive therapy are rejected due to the belief of a short life expectancy and fear of consequences [4].

The purpose of this research was to investigate and evaluate the influence of haemoglobin level as a predictor of MACE and early outcomes in cases treated with pPCI versus pharmacoinvasive (PI) approach.

Patients and Methods:

This prospective case-control observational study was carried out on 100 cases, aged from 34 to 85 years old, with STEMI within 24 hours and treated with 1ry PCI or pharmacoinvasive technique at cardiovascular medicine department Tanta university hospitals within 6 months starting from October 2020.

The study was done after being approved from the Ethical Committee Tanta University Hospitals. An informed written consent was obtained from the cases.

Exclusion criteria were missing admission haemoglobin data, Cases with history of CABG, Cases with contraindications to both forms of treatment.

Cases were categorized into two groups: Group I: 50 cases anaemic (men with HB level below 13 and women with HB level below 11.5). Group II: 50 cases non-anaemic (normal haemoglobin) group. Group I were further subdivided to group IA: treated with primary PCI and group IB: treated with pharmacoinvasive strategy. Group II subdivided to group IIA: treated with primary PCI, group IIB: treated with pharmacoinvasive strategy.

The cases were subjected to full history taking, full clinical examination, routine laboratory tests, resting 12 leads ECG, Right pericardial leads (V3R, V4R, V5R, V6R) and posterior chest leads (V7 to V9) to detect posterior wall and right ventricular infarction ^[2].

LV wall affected ST segment elevated in the following leads: Septal (V1, V2), Anterior (V1 to V6), Antro-septal (V1 to V4), Antro-lateral (V3 to V6 & aVL), Extensive anterior (V1 to V6, I & aVL), Inferior (II, III, aVF), Right (V3R, V4R, V5R), Posterior (V7 to V9).

PCI Femoral approach: Three to four centimetres in diameter are injected three to four centimetres below the inguinal ligament. When the sheaths are removed, the predicted puncture location should allow for appropriate compression of the vessels. A needle of 18-gauge is inserted through the skin and into the femoral artery lumen. Once blood is freely flowing through the needle, a Teflon-coated guide wire is inserted into the lumen of the perforated vessel. As the needle is withdrawn and the wire is cleaned to eliminate blood and thrombi, the wire is retained securely in place. The wire is then withdrawn and a sheath with a side arm port is advanced over the wire into the vessel lumen. As catheters are progressed through the sheath to the heart, the side arm port enables continuous pressure monitoring and infusion ^[5].

PCI Radial approach: cases 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. The

ideal position of entry is approximately 2 cm proximal to the radial styloid, the operator can elect to use a double wall through-and-through approach with an Angiocath, or a single-wall anterior puncture with a micro puncture needle ^{[6] [7]}.

Imaging:

Left coronary imaging: A contrast injection in the left coronary cusp is a reasonable first step to define the ostium of the left main (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 ostial 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 ^[5]

Right coronary imaging: The 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^[5]. 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 PDA and posterolateral branches and a shallow RAO view is useful to show the entire PDA ^[5]. The infarcted related artery (IRA) was identified. An interventional cardiologist identified the culprit lesion on the basis of the infarct location on the admission ECG and the angiographic findings (target vessel, lesion characteristics) ^[5].

Reperfusion success is measured by TIMI blood flow grade: Reperfusion was considered successful (TIMI 3) or abnormal (TIMI 0-1-2) ^[5, 8].

Pharmacoinvasive technique: cases received thrombolytic therapy followed by coronary angiography either immediately in case of failed thrombolytic or within 3-24 hours after sign

of successful reperfusion ^[2]. The used type of thrombolytic in Tanta university hospital CCU is Streptokinase (1.5 million units IV over 30-60 minutes) according to availability in CCU and the accepted time for starting the infusion according to ESC guidelines 2017 IV bolus of thrombolytic therapy should start within 10 minutes; however thrombolytic therapy can be given within 12 hrs from onset of chest pain ^[2]. A loading dose of dual anti-platelets; Aspirin (300 mg) & Clopidogrel (300 mg) was given if the patient's age was below 75 years old and half-loading dose if the patient's age was ≥ 75 years old was given ^[2].

Assessment of thrombolytic success: ^[9] Chest discomfort alleviation, reduction in STEMI by 50% relative to the baseline ECG, reperfusion arrhythmia, and cardiac enzyme elevation. Coronary angiography was done either immediately after thrombolytic treatment failed or within 3 to 24 hours after thrombolysis success criteria were met [2].

All research was done utilising echocardiography (a GE vivid seven Cardiac ultrasound phased array system with tissue Doppler imaging using M4S transducer 4 M.HZ.). Two-Dimensional echocardiographic assessment by M-mode and modified Simpson method were done during admission after PCI. Echocardiography was performed in a posture of semi left lateral position to: Assess LV systolic function with Simpson's technique in the apical 4 and apical 2 views, as well as Left ventricular volumes. (End diastolic and end systolic volume) $EF = (EDV - ESV) / EDV$ [10]. Analyze anomalous segmental wall motion and global wall movement Utilizing a 17 segment model for LV segmentation, regional wall movement anomalies were evaluated. Each segment's wall motion was rated between 1 (normal) and 4 (abnormal) (dyskinetic). M-mode assessment of LV systolic function through getting the long parasternal axis view and directing the M-mode cursor across the LV & it is measured also in the parasternal short view with directing the M-mode cursor across the mid LV.

Follow up : Follow up of all cases included in the study as regard in hospital incidence of MACE (Major adverse cardiovascular events) defined as recurrent AMI, heart failure, stroke

and in hospital death due to cardiac cause and bleeding complication. Detection of mortality, heart failure, re-infarction, stroke and assessment of clinical condition & symptomatology of the cases within this month.

Statistical analysis

Data were inputted into the computer and analysed using IBM SPSS version 20.0 software programme (Armonk, NY: IBM Corp). Quantitative and percentage descriptions were provided for qualitative data. The Kolmogorov-Smirnov test / Shapiro-Wilk test was utilised to confirm the distribution's normality. The range (minimum and maximum), mean, standard deviation, median, and interquartile range were used to characterise quantitative data (IQR). At the 5% significance level, the acquired findings were deemed significant.

Results:

There was no statistically significant difference between the studied groups according to demographic data, different risk factors, STEMI location and different parameters. According to Killip class, there was statistically significant difference between group IIA & group IIB (P2 value = 0.005). Table 1

Table 1: Comparison between the different studied groups according to demographic data, different risk factors, Killip class, STEMI location and different parameters

	Group IA (n = 25)	Group IB (n = 25)	Group IIA (n = 25)	Group IIB (n = 25)	p
	No (%)	No (%)	No (%)	No (%)	
Male	19 (76.0%)	11 (44.0%)	19 (76.0%)	16 (64.0%)	0.057
Female	6 (24.0%)	14 (56.0%)	6 (24.0%)	9 (36.0%)	
Age Mean ± SD.	58.60 ± 13.64	56.28 ± 9.71	56.08 ± 11.68	59.92 ± 8.88	0.555
Diabetes	9 (36.0%)	17 (68.0 %)	9 (36.0%)	12 (48.0%)	0.076
Hypertension	8 (32.0 %)	16 (64.0 %)	12 (48.0%)	11 (44.0%)	0.154
Smoking	15 (60.0 %)	11 (44.0%)	19 (76.0%)	14 (56.0 %)	0.144
Family History	2 (8.0 %)	5 (20.0 %)	7 (28.0%)	3 (12.0 %)	^{MC} p=0.285
IHD	12(48.0 %)	8 (32.0 %)	6 (24.0%)	8 (32.0 %)	0.336
Chronic kidney disease	3 (12.0 %)	8 (32.0%)	2 (8.0%)	4 (16.0 %)	^{MC} p=0.176

Killip class	1	21 (84.0%)	15 (60.0%)	24 (96.0%)	16 (64.0%)	0.008* p ₁ =0.059 p ₂ =0.005* p ₃ =0.157 p ₄ =0.771
	2-4	4 (16.0%)	10 (40.0%)	1 (4.0%)	9 (36.0%)	
STEMI location						
Anterior		18 (72.0%)	15 (60.0%)	16 (64.0%)	17 (68.0%)	0.903
Inferior		6 (24.0%)	8 (32.0%)	6 (24.0%)	5 (20.0%)	
Lateral		1 (4.0%)	2 (8.0%)	3 (12.0%)	3 (12.0%)	
Infarction related artery						
LAD		18 (72.0%)	15 (60.0%)	16 (64.0%)	16 (64.0%)	MC p=0.974
RCA		4 (16.0%)	6 (24.0%)	4 (16.0%)	5 (20.0%)	
LCX		3 (12.0%)	4 (16.0%)	5 (20.0%)	4 (16.0%)	
Number of diseased vessels						
Single vessel		13 (52.0%)	12 (48.0%)	11 (44.0%)	12 (48.0%)	0.956
Multi vessel		12 (48.0%)	13 (52.0%)	14 (56.0%)	13 (52.0%)	
Final TIMI Flow						
<3		1 (4.0%)	4 (16.0%)	2 (8.0%)	2 (8.0%)	MC p=0.616
3		24 (96.0%)	21 (84.0%)	23 (92.0%)	23 (92.0%)	

p1: p value for comparing between Group IA and Group IB; p2: p value for comparing between Group IIA and Group IIB; p3: p value for comparing between Group IA and Group IIA; p4: p value for comparing between Group IB and Group IIB; *: Statistically significant at $p \leq 0.05$. c2: Chi square test; MC: Monte Carlo.

There was no statistically significant difference between 4 groups regarding the duration and vital sign. Table 2

Table 2: Comparison between the different studied groups according to symptoms duration and Vital signs

	Group IA (n = 25)	Group IB (n = 25)	Group IIA (n = 25)	Group IIB (n = 25)	p
Symptoms duration	9.44 ± 8.43	4.94 ± 2.62	6.30 ± 5.17	4.96 ± 2.62	0.384
Vital signs "clinical"					
Systolic (mmHg) blood pressure	124.0 ± 21.79	118.0 ± 20.21	120.0 ± 22.55	114.0 ± 25.17	0.467
Diastolic (mmHg) blood pressure	77.20 ± 12.42	72.80 ± 12.75	74.40 ± 12.61	70.40 ± 14.85	0.326
Pulse (bpm)	88.60 ± 21.58	87.60 ± 27.08	83.0 ± 14.58	90.80 ± 21.73	0.634

Data was presented in Mean ± SD.

According to door to balloon, there was no statistically significant difference between IA and IIA groups (P value = 0.465). Table 3

Table 3: Comparison between the two studied groups according to door to balloon

Door to balloon (hours)	Group IA	Group IIA	p
--------------------------------	-----------------	------------------	----------

	(n = 25)	(n = 25)	
Mean ± SD.	1.90 ± 2.24	1.68 ± 0.76	0.465

Group IA: Anemia with primary PCI; **Group IIA:** Non anemia with primary PCI

There was no statistically significant difference between the four groups regarding MACE and total MACE during the hospital stay and follow up including death, stroke, reinfarction, CHF and major bleeding. LVEF was insignificantly different between the four groups. Clopidogrel was significantly elevated in IB group compared to group IA group. Ant failure medication on discharge and Contrast induced nephropathy were insignificantly different between the studied groups. Table 4

Table 4: Comparison between the different studied groups according to different complications, MACE, Total MACE, different parameters contrast induced nephropathy

	Group IA	Group IB	Group IIA	Group IIB	^{MC} p
	(n = 25)	(n = 25)	(n = 25)	(n = 25)	
	No (%)	No (%)	No (%)	No (%)	
In Hospital Death	0 (0.0%)	2 (8.0%)	0 (0.0%)	1 (4.0%)	0.612
In Hospital Stroke	0 (0.0%)	1(4.0%)	0 (0.0%)	1(4.0%)	1.000
In-Hospital Reinfarction	1 (4.0%)	2 (8.0%)	0 (0.0%)	0 (0.0%)	0.612
In-Hospital CHF	6 (24.0%)	6 (24.0%)	1 (4.0%)	4 (16.0%)	0.192
In hospital major bleeding	2 (8.0%)	3 (12.0%)	1(4.0%)	3 (12.0%)	0.869
Follow-up Stroke	3 (12.0%)	1 (4.3%)	0 (0.0%)	0 (0.0%)	0.121
Follow-up Death	2 (8.0%)	2 (8.7%)	1 (4.0%)	3 (12.5%)	0.753
Follow-up Reinfarction	1 (4.0%)	1 (4.3%)	0 (0.0%)	1 (4.2%)	0.805
Follow-up CHF	4 (16.0%)	3 (13.0%)	1 (4.0%)	2 (8.3%)	0.519
Follow-up Major bleed	3 (12.0%)	9 (39.1%)	1 (4.0%)	2 (8.3%)	0.006*
MACE	9 (36.0%)	11 (44.0%)	2 (8.0%)	4 (16.0%)	0.012* p ₁ =0.564 ^{FE} p ₂ =0.667 p ₃ =0.017 p ₄ =0.031

Total MACE					
In hospital stay	7 (28.0%)	11 (44.0%)	1 (4.0%)	6 (24.0%)	0.013* p ₁ =0.239 FE p ₂ =0.098 FE p ₃ =0.049* p ₄ = 0.136
Follow up	10 (40.0%)	7 (28.0%)	2 (8.0%)	6 (24.0%)	0.072
LVEF					
Below 40%	8 (32.0%)	7 (28.0%)	4 (16.0%)	9 (36.0%)	0.565
From 40% to 49%	13 (52.0%)	13 (52.0)	12 (48.0%)	10 (40.0%)	
Over 50 %	4 (16.0%)	5 (20.0%)	9 (36.0%)	6 (24.0%)	
P2Y12 inhibitor					
Clopidogrel	18 (72.0%)	25 (100.0%)	23 (92.0%)	25 (100.0%)	MC p= 0.002* FE p ₁ =0.010* FE p ₂ =0.490 FE p ₃ =0.138 p ₄ = -
Ticagrelol	7 (28.0%)	0 (0.0%)	2 (8.0%)	0 (0.0%)	
Ant failure medication on discharge	17 (68.0%)	16 (64.0%)	22 (88.0%)	18 (72.0%)	0.240
Contrast induced nephropathy	3 (12.0%)	4 (16.0%)	2 (8.0%)	2 (8.0%)	0.899

c2: Chi square test; MC: Monte Carlo; FE: Fisher Exact; p: p value for comparing between the studied groups
p1: p value for comparing between Group IA and Group IB; p2: p value for comparing between Group IIA and Group IIB; p3: p value for comparing between Group IA and Group IIA; p4: p value for comparing between Group IB and Group IIB; *: Statistically significant at p ≤ 0.05

The hemoglobin level was significantly lower in group IA compared to group IIA and was significantly lower in group IB compared to group IIB (p<0.001*) with no significant difference between the studied groups regarding platelets and creatinine level. Table 5

Table 5: Comparison between the different studied groups according to haemoglobin, platelets and creatinine

	Group IA (n = 25)	Group IB (n = 25)	Group IIA (n = 25)	Group IIB (n = 25)	p
Hemoglobin (gm/dl)	10.04 ± 1.09	9.76 ± 0.91	14.68 ± 1.41	14.36 ± 0.76	<0.001* p ₁ =0.806 p ₂ =0.717 p ₃ <0.001* p ₄ <0.001*
Platelets (number/mm³)	234.1 ± 58.85	212.0 ± 57.01	232.4 ± 68.67	230.0 ± 67.97	0.585
Creatinine (mg/dl)	1.25 ± 0.36	1.26 ± 0.36	1.19 ± 0.44	1.23 ± 0.25	0.556

SD: Standard deviation; H: H for Kruskal Wallis test; F: F for One way ANOVA test, pairwise comparison bet. each 2 groups were done using Post Hoc Test (Tukey); IQR: Inter quartile range; p: p value for comparing between the studied groups; p₁: p value for comparing between Group IA and Group IB; p₂: p value for

comparing between Group IIA and Group IIB; p₃: p value for comparing between Group IA and Group IIA; p₄: p value for comparing between Group IB and Group IIB; *: Statistically significant at p ≤ 0.05

Discussion

Reperfusion treatment in acute myocardial infarction aims at early and sustained reperfusion of the myocardium at risk. Traditionally, Reperfusion can be obtained by thrombolysis or by primary percutaneous coronary intervention (PCI) ^[11].

Anaemia is a frequently encountered comorbidity among cases presenting with ACS. Previous studies of the effect of the haemoglobin level on the prognosis of cases with coronary heart disease have mostly concentrated in ACS cases, but few studies have been conducted for STEMI patients in particular also the effect of the haemoglobin level on the prognosis of STEMI patients remains in dispute ^[12].

As expected, the anemic group tended to have more comorbidities numerically relative to non anemic group as regard hypertension (48% VS 46%), Diabetes Mellitus (52% VS 42%), IHD (40% vs 28 %) and CKD (22% vs 12%).

This goes along with what Mamas **MA et. al.** ^[13] The UK Myocardial Ischemia National Audit Project Registry revealed that anaemic individuals presenting with STEMI were more likely to have concomitant illnesses. The anaemic cohort had a history of hypertension (59% versus 48%, P<0.001), angina (43% versus 27%, P<0.001), myocardial infarction (38% versus 23%, P<0.001), prior heart failure (12% versus 4%, P<0.001), stroke (14% versus 7%, P<0.001), peripheral vascular disease (8% versus 3%, P<0.001), COPD (18% versus 14%, P<0.001).

As regard age there was no statistically significant difference between the 2 groups with anemic mean age 57.44 ± 11.77 years old & non anemic 58.0 ± 10.45 years old (P value = 0.802)

This is unlike what was described by **Mamas MA et. al.** ^[13] showed that anemic group was older (76 ± 12 years old) vs (66 ± 14 years old).

As regard sex females represent larger portion of anemic group (40 %) than non-anaemic group (30%) which goes along with other studies showing more prevalence of anaemia in female presenting with ACS and STEMI per se ^[3, 13].

In our study anemic group tended to present with statistically significant worse Killip class (P value = 0.008)

in the subgroups: Primary PCI subgroups: 16 % of anemic group presented with Killip II to IV vs 4 % only in non anemic

Pharmacoinvasive subgroups: there was no significant difference in Killip class presentation This goes along with what **Wester A et. al.** showed, Cases with anemia also more frequently presented with STEMI and Killip class 3 or 4 than cases without anemia (P<0.001). (15)

In the primary PCI groups on comparing Door to balloon time there was no statistically significant difference between group IA & group IIA as regard door to balloon time. similar data were concluded by **Moghaddam N et. al.** ^[3] as there were no major differences in reperfusion times among anaemic STEMI cases, particularly those presenting to PCI-capable hospitals.

As regard site of infarction: There was no statistically significant difference between the 4 subgroups regarding site of infarction with anterior STEMI being most common which also resembles what **Moghaddam N et. al.** ^[3] found in their trial.

As regard final TIMI flow: There was no statistically significant difference between the four subgroups as regard final TIMI flow

Wang X et. al. ^[14] found that unlike our results Anaemic cases were less likely to have a successful procedure, defined as a thrombolysis in myocardial infarction III flow and residual stenosis less than 30%.

Total in hospital and follow up MACE: the anemic group tended to have statistically significant MACE relative to non anemic group (40% vs 12%) (P value = 0.001). Group IA

had statistically significant more MACE than Group IIA (36% vs 8%) (P value=0.017) Group IB had also statistically significant MACE than Group IIB (44% vs 16 %) (P value=0.031). As regard heart failure the anaemic groups tended to have numerically higher incidence of in hospital CHF whether they have undergone pPCI (24% vs 4 %) or pharmacoinvasive strategy (24% vs 16%). Also, in follow up same findings were observed (16% vs 4%) and (12% vs 8%) **Tang C et. al.** ^[12] showed that the incidence of MACCE increased significantly with the decrease of haemoglobin level within 30 days, 6 months, and 1 year after pPCI.

Mamas MA et. al. ^[13] anemia is independently linked to an ~50% increased risk of mortality in the short and long term and that this prognostic impact is observed in both men and women and persist even after exclusion of bleeding complications in pPCI treated cases. **Wang X et. al.** ^[14] cases with anaemia undergoing pPCI had significantly worse survival compared with the cases with normal Hb levels. **Wester A et. al.** ^[15] showed that anemia in cases undergoing pPCI linked to a large excess risk of mortality at 180 days.

Interestingly there was no associated increase in stent thrombosis in anemic group – the mechanism of reinfarction was not investigated in our study.

Moghaddam N et. al. ^[3] After multivariable adjustment, anemia was not significantly linked to the occurrence of all-cause mortality, congestive HF, or cardiogenic shock in STEMI cases treated with pPCI.

As regard major bleeding the anemic group tended to have statistically significant higher incidence of major bleeding (24% vs 6%) (P value=0.015). In both pPCI and pharmacoinvasive the anemic subgroups had statistically significant higher incidence of bleeding. This goes along with what was mentioned by **Moghaddam N et. al.** ^[3] and **Wester A et. al.** ^[15] that anemia is linked to higher incidence of major bleeding which was usually non access site related bleeding – this was not investigated in our study.

As regard LVEF There was no statistically significant difference between the LVEF after PCI between the subgroups. Unlike what was mentioned by **Wester A et. al.** ^[15] left ventricular ejection fraction of cases with anaemia throughout their hospital stay was shorter versus that of cases without anaemia, which could be suggestive of a bigger infarct size or region at ischemia risk. But **Moghaddam N et. al.** ^[3] found that there was no statistically significant difference between LVEF in STEMI cases treated with pPCI.

Anemic cases were generally less likely to receive optimal medical treatment including RAS blocker and beta blockers (66% vs 80%). This goes along with what **Wester A et. al.** ^[15] found in their study the anemic group was statistically significant less likely to be discharged on RAAS blocker and beta blocker.

The study had some potential limitations such as; small size of study population, which was due to many factors, one of them that some cases missed follow up after one month, also a lot of cases were missing haemoglobin data during presentation. Another limitation was that the effect of chronicity of anaemia was not investigated during this study also the relation between degree of anaemia and outcomes. Another limitation was the short period assigned for follow up which didn't allow the appearance of results for mortality, re-infarction & re-hospitalization.

Conclusions:

Anaemia was linked to a non-statistically significant higher incidence of comorbidities namely DM, HTN, CKD and ischemic heart disease. Anaemic cases whether revascularized with pPCI or pharmacoinvasive strategy tend to have higher incidence of MACCE and major bleeding with no significant difference in mortality. Anaemic cases are less likely to be discharged on GDMT. There was no significant difference between LVEF between the study groups.

References:

1. Billett HH. Hemoglobin and Hematocrit. In: Walker HK, Hall WD, Hurst JW, editors. Clinical Methods: The History, Physical, and Laboratory Examinations. Boston: Butterworths Copyright © 1990, Butterworth Publishers, a division of Reed Publishing.; 1990.
2. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, 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). *Eur Heart J*. 2018;39:119-77.
3. Moghaddam N, Wong GC, Cairns JA, Goodman SG, Perry-Arnesen M, Tocher W, et al. Association of Anemia With Outcomes Among ST-Segment-Elevation Myocardial Infarction Patients Receiving Primary Percutaneous Coronary Intervention. *Circ Cardiovasc Interv*. 2018;11:7-17.
4. Lorente V, Aboal J, Garcia C, Sans-Roselló J, Sambola A, Andrea R, et al. Anemia in patients with high-risk acute coronary syndromes admitted to Intensive Cardiac Care Units. *J Geriatr Cardiol*. 2020;17:35-42.
5. Valgimigli M, Bueno H, Byrne RA, Collet JP, Costa F, Jeppsson A, et al. 2017 ESC focused update on dual antiplatelet therapy in coronary artery disease developed in collaboration with EACTS: The Task Force for dual antiplatelet therapy in coronary artery disease of the European Society of Cardiology (ESC) and of the European Association for Cardio-Thoracic Surgery (EACTS). *Eur Heart J*. 2018;39:213-60.
6. Valgimigli M, Gagnor A, Calabró P, Frigoli E, Leonardi S, Zaro T, et al. Radial versus femoral access in patients with acute coronary syndromes undergoing invasive management: a randomised multicentre trial. *Lancet*. 2015;385:2465-76.

7. Jolly SS, Amlani S, Hamon M, Yusuf S, Mehta SR. 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. *Am Heart J.* 2009;157:132-40.
8. Boden WE, O'Rourke RA, Teo KK, Hartigan PM, Maron DJ, Kostuk WJ, et al. Optimal medical therapy with or without PCI for stable coronary disease. *N Engl J Med.* 2007;356:1503-16.
9. Hancock JE, Cooke JC, Chin DT, Monaghan MJ. Determination of successful reperfusion after thrombolysis for acute myocardial infarction: a noninvasive method using ultrasonic tissue characterization that can be applied clinically. *Circulation.* 2002;105:157-61.
10. Hole T, Vegsundvåg J, Skjaerpe T. Estimation of left ventricular ejection fraction from Doppler derived myocardial performance index in patients with acute myocardial infarction: agreement with echocardiographic and radionuclide measurements. *Echocardiography.* 2003;20:231-6.
11. Mega JL, Morrow D. ST-elevation myocardial infarction: management. Mann D, Zipes D, Libby P, Bonow R Braunwald's heart disease: a textbook of cardiovascular medicine 10th ed Philadelphia: Elsevier. 2015:1095-147.
12. Tang C, Luo E, Wang D, Yan G, Qiao Y, Zhu B, et al. Usefulness of Haemoglobin Level Combined with CAMI-STEMI Score for Predicting MACCE in Patients with Acute ST-Elevation Myocardial Infarction after PCI. *Biomed Res Int.* 2019;2019:8-52.
13. Mamas MA, Kwok CS, Kontopantelis E, Fryer AA, Buchan I, Bachmann MO, et al. Relationship Between Anemia and Mortality Outcomes in a National Acute Coronary Syndrome Cohort: Insights From the UK Myocardial Ischemia National Audit Project Registry. *J Am Heart Assoc.* 2016;5.

14. Wang X, Qiu M, Qi J, Li J, Wang H, Li Y, et al. Impact of anemia on long-term ischemic events and bleeding events in patients undergoing percutaneous coronary intervention: a system review and meta-analysis. *J Thorac Dis.* 2015;7:2041-52.

15. Wester A, Attar R, Mohammad MA, Andell P, Hofmann R, Jensen J, et al. Impact of Baseline Anemia in Patients With Acute Coronary Syndromes Undergoing Percutaneous Coronary Intervention: A Prespecified Analysis From the VALIDATE-SWEDEHEART Trial. *J Am Heart Assoc.* 2019;8:12-41.

UNDER PEER REVIEW