DOI: https://doi.org/10.53350/pjmhs221651407

ORIGINAL ARTICLE

Comparison of the Outcomes Between Coronary No-Reflow and Slow-Flow Phenomenon in Non-Stemi Patients

UMAIR ABRAR[1], JAVED KHURSHED SHAIKH[2], IFTIKHAR AHMED[3], AHSAN RAZA[4], GULZAR ALI BURIRO[5], SYED NADEEM HASSAN RIZVI[6]

[1]*MBBS, FCPS (Cardiology) Cardiologist Orthopedic and Medical Institute (OMI) Karachi,*
[2]*MBBS, FCPS (General Cardiology), MSPH Assistant Professor Department of Cardiology, National Institute of Cardiovascular Disease (NICVD), Karachi.*
[3]*MBBS, FCPS (Cardiology) Fellowship in Interventional Cardiology Assistant Professor Department of Cardiology, National Institute of Cardiovascular Disease, Hyderabad Centre*
[4]*MBBS, FCPS (Cardiology) Assistant Professor Department of Cardiology, National Institute of Cardiovascular Disease (NICVD), Hyderabad*
[5]*MBBS, FCPS (Cardiology) Post Fellow (Interventional cardiology) Assistant Professor Department of Cardiology, National Institute of Cardiovascular Disease, (NICVD), Karachi*
[6]*MBBS, DIP, CARD, MRCP, FRCP, FACC, FAIC (Supervisor), Professor Interventional Cardiology (NICVD) Karachi*
Corresponding author: Umair Abrar, Email: drumair.baig@hotmail.com, Cell: 03453051598

ABSTRACT

Background: Coronary slow flow phenomenon (CSFP) and coronary no-reflow phenomenon (CNP) have the potential to raise the risk of severe cardiovascular adverse events (MACE).

Objectives: This study's goal was to evaluate and contrast the clinical outcomes after a year for CNP and CSFP patients who received PCI for a non-ST elevation myocardial infarction (NSTEMI).

Methods: In this research, 95 patients had NSTEMI and had PCI within 24 hours after symptoms started. An angiographic characteristic of the infarct-related artery's TIMI flow was used to divide patients into two groups: the CSP group (n=85) and the CNP group (n=10). Patients were tracked for a full year. To be statistically significant, the p-value needed to be <0.05.

Results: There were 95 patients with NSTEMI included in this research (66 males; mean age: 62.71±13.70). CNP was seen in 10.5% (n = 10) and CSFP in 89.4% (n = 85) of NSTEMI patients, respectively. we provide the results of our demographic analysis.

Conclusion: When comparing CNP and CSFP patients with NSTEMI, the clinical results and risk of stroke are worse for CNP individuals.

Keywords: Coronary no-reflow Phenomenon, Slow-Flow Phenomenon, non-ST-elevation myocardial infarction.

INTRODUCTION

Despite advancements in treatment and prevention, the prevalence of acute coronary syndromes is still high in developed nations and rising in developing nations.[1] Non-ST elevation myocardial infarction had worse long-term results for people with acute coronary syndromes (NSTEMI).[2] However, few studies report outcomes in NSTEMI, and even fewer have simplified the differences between the coronary slow flow phenomenon (CSFP) and the coronary no-reflow phenomenon (CNP) in clinical practice, both in the hospital and in long-term follow-up.[3,4] Coronary artery disease without obstruction is considered by coronary flow and delayed opacification (CSFP).[5] TIMI 0-I flows without dissection, mechanical blockage, considerable residual stenosis, spasms, or coronary thrombus are also included in angiographic CNP.[6] The root causes of CNP and CSFP include inflammation, atherothrombotic microembolization, neutrophil activation, and endothelial damage. These factors lead to the generation of oxygen-free radicals, proteolytic enzymes, and pro-inflammatory mediators, which result in tissue and endothelial damage.[5,6]

Additionally, it is unclear how long NSTEMI patients will continue to have different clinical features and consequences. Furthermore, the literature does not mention how slow flow might affect NSTEMI outcomes. The results of CSFP versus CNP in patients with NSTEMI are not compared in the existing research. According to our hypothesis, non-TIMI III flow in the coronary arteries is significantly related to the worse clinical outcomes in NSTEMI, especially in the CNP group subgroup. This research aimed to compare the major cardiovascular outcomes of patients with NSTEMI who had either CSFP or CNP care throughout a 12-month follow-up period.

MATERIAL AND METHODS

In this single-center prospective study, 95 patients aged 18 to 90 years with NSTEMI and given early PCI within 24 hours of developing symptoms were enrolled from January 2021 to June 2021 at Orthopedic and Medical Institute (OMI) Karachi. Additionally, TIMI III flow, coronary artery bypass graft (CABG), and moderate-to-severe chronic renal disease are risk factors, as are cardiogenic shock and the presence of lung edema, acute symptoms of left ventricular dysfunction, as well as the presence of thrombus aspiration during the index event. A total of 10 patients with angiographically proven CNP and 85 patients with angiographically proven CSFP were divided into two groups based on the results of the angiographic features of the treated culprit artery's TIMI flow. All of the patients were given 300 mg of acetylsalicylic acid, 600 mg of clopidogrel, and 100 mg/kg of UF heparin during the PCI procedure. Hospital records were obtained as well as follow-up information about patients during their visits after 1, 3, 6, and 12 months. The families of the individuals were contacted via phone, and hospital records and death certificates were obtained from them. Major adverse cardiovascular events (MACE) are characterized as the occurrence of myocardial re-infarction, cardiovascular death, stroke, and all-cause mortality combined.

Before percutaneous coronary intervention (PCI) was conducted, blood was collected from the antecubital vein shortly after the patient arrived at the hospital. The emergency room received an electrocardiogram with 12 leads when the patient was hospitalized. The HR was also recorded. Patients' estimated glomerular filtration rates (eGFR) were determined using an equation developed from the Chronic Kidney Disease Epidemiology Collaboration equation. In order to calculate BMI, weight (kg)/ height2 (m2) was used. Regular blood chemistry, lipid parameters, and heart Troponin-I levels were measured using a conventional auto-analyzer. An auto-analyzer such as the Sysmex K-1000 (Block Scientific, Bohemia, NY, USA) was used to measure blood counts. The samples were spun at 3000 rpm for 10 minutes using a centrifuge, after which the supernatant and serum were divided and frozen at -80° C. Following the administration of contrast medium (CM), the serum creatinine level was measured again at 72 hours. An absolute rise of 0.5 mg/dL in serum creatinine level above baseline within 72 hours after being exposed to CM, or a relative increase of ≥25% from the patient's pre-CM serum creatinine level, is the clinical diagnosis of contrast-induced nephropathy.

Based on criteria from clinical practice standards, NSTEMI was diagnosed when certain symptoms were present.[7] Patients with NSTEMI had typical chest pain or discomfort at rest or with

minimal exertion, for at least 10 minutes, and their initial electrocardiogram (ECG) showed normal ECG or ischemic changes, such as ST-depression or T-wave inversion, and an elevated cardiac troponin-I level with at least 1 value above the 99th percentile of the upper reference limit.

The same researcher examined each patient thoroughly and then took their medical history. Several risk factors for cardiovascular disease were recognized, including age, gender, diabetes mellitus, high blood pressure, high cholesterol, and smoking. Patients with blood pressure readings of at least ≥140/90 mm Hg on two detached incidents were classified as hypertensive and required treatment.[8] A patient was diagnosed with diabetes if their fasting blood glucose was >125 mg/dL on at least two separate occasions, or if they had been using oral antidiabetic and/or insulin treatment before.[9,10] Smokers were defined as individuals who had recently quit smoking and those who had used tobacco products when they were admitted to the emergency department.

Each patient was evaluated with transthoracic echocardiography using a 3.5 MHz transducer before being released from the hospital. Left ventricular ejection fraction was determined using Simpson's technique (LVEF).[11]

The femoral or radial approach was utilized to perform coronary angiography processes using Philips angiography equipment. According to accepted clinical standards, coronary angiography and PCI were performed using a nonionic, iso-osmolar contrast medium (iodixanol, Visipaque 320mg/100mL, GE Healthcare, Cork, Ireland). The artery connected to the infarct underwent PCI. At least 80 frames of angiographical pictures were collected, and 25 frames were captured each second. Offline evaluation of TIMI flow grade and coronary architecture was performed by at least two skilled cardiologists. Gibson et al.'s number of frame counts was used to calculate the coronary artery TIMI flow.[12] Angiographical CNP has defined as TIMI 0-I flows without coronary artery thrombus, dissection, mechanical obstruction, significant residual stenosis, spasm, or other obstructive conditions. When there is no obstructive coronary artery disease present, a person has CSFP, this is characterized by TIMI-II coronary flow that is normal and delayed opacification of the coronary arteries. Patients with CNP were given intracoronary (IC) epinephrine, adenosine, or glycoprotein IIb/IIIa inhibitors (Gp-IIb/IIIa inh.). Following the surgery, isotonic saline (1mL/kg/h) was administered intravenously (IV) to all patients for at least 12 hours.

Statistical Analysis: The statistical software program SPSS version 22.0 was used to conduct data analysis. If the continuous variable was regularly distributed, its mean and standard deviation were reported, whereas if it was not, its median and 25th and 75th percentiles were given. Statistical analyses were performed using numeric representations of categorical variables (percentage). Categorical variables were compared using the Chi-square test. An examination of the Receiver Operating Characteristic (ROC) curve was done to determine the predictive value of BMI and HR for CNP. Multivariate logistic regression analysis was conducted after univariate logistic regression analysis was conducted on the statistically relevant variables ($p<0.1$). Each independent variable's odds ratio and 95% confidence interval were determined. In this study, statistical significance was defined as a p-value of <0.05.

RESULTS

There were 95 patients with NSTEMI included in this study (66 males; mean age: 62.71±13.70). CNP was seen in 10.5 % (n = 10) and CSFP in 89.4 % (n = 85) of NSTEMI patients, respectively. In terms of the total study population, the CNP group comprised 10 patients (10.5%) whereas the CSFP group had 85 patients (89.4%). In **Table 1** we provide the results of our demographic analysis. Furthermore, there was a significant ($p<0.04$) correlation between Euro SCORE-II and NYHA class, heart rate, hospital duration of stay, score, and eGFR. **Table-2**. The findings of the clinical follow-up research are shown in **Table 3**. There were no incidences of hemorrhagic stroke throughout the time we followed up on patients. **Figure- 2A and Figure 2 B** depict the Kaplan-Meier estimates for the prevalence of stroke and major adverse cardiovascular events, respectively CNP was shown to be independently predicted by both body mass index and heart rate in a conditional logistic regression model **(Table 4).**

Table-1: Demographic and laboratory details of the patients (n=95)

Characteristics	Coronary no-reflow phenomenon n=10 (10.5%)	coronary slow-flow phenomenon n=85 (89.4%)	p-value
Age, years	67.29±15.13	62.16±13.35	0.046
Female gender, n (%)	5 (50)	24 (28.2)	0.054
BMI, kg/m²	31.52±3.98	27.35±4.54	0.023
HT, n (%)	7 (70)	50 (58.8)	0.076
DM, n (%)	4 (40)	27 (31.7)	0.500
HL, n (%)	4 (40)	37 (43.5)	0.405
Smoker, n (%)	6 (60)	51 (60)	0.867
Family History, n (%)	3 (30)	28 (32.9)	0.826
peripheral arterial disease, n (%)	2 (20)	5 (5.88)	0.020
chronic obstructive pulmonary disease, n (%)	2 (20)	12 (14.1)	0.312
LVEF, %	51±8.50	51.28±7.18	0.215
Glucose, mg/dl	114 (91.51-186)	107 (95-147)	0.618
Uric acid, mg/dl	5.60 (4.54-6.24)	6.81 (5.21-7.91)	0.204
Creatinine, mg/dl	0.87 (0.76-1.22)	0.86 (0.75-1.04)	0.164
eGFR, mL/min per 1.73 m²	80.70±34.84	73.75±30.90	0.031
Triglycerides, mg/dL	154 (126-196)	146 (112.6-181)	0.264
LDL, mg/dL	136 (115-172)	126 (97-150)	0.041
HTC, %	41.61 (36.81-41)	42 (36.11-42.16)	0.253
Platelets, 10³/uL	221 (184-267)	226 (191-277.51)	0.537
Peak Troponin-I, pg/ml	815 (157-5794.51)	147 (117-2212)	0.026
high-sensitivity C-reactive protein , mg/dL	0.11 (0.02-0.46)	0.17 (0.03-0.60)	0.727
HR, bpm	73.70±18.75	87.61±12.35	<0.001
Hospital length of stay, d.	4.30±0.84	4.00±0.77	0.014
Score	8.45±7.20	6.15±5.81	0.015
CIN development, n (%)	2 (20)	7 (8.23)	0.327
NYHA class	3.27±0.40	3.02±0.50	<0.001
EuroSCORE II, %	2.87±2.86	3.15±3.41	<0.001
Medications, n (%)			
Ace inh	7 (70)	42 (49.41)	0.075
ARB	3 (30)	29 (34.11)	0.641
B-blocker	9 (90)	82 (96.47)	0.875
CCB	3 (30)	20 (23.52)	0.162
Statin	10 (100)	75 (88.23)	0.073
Nitrate	3 (30)	28 (32.94)	0.162
OAD	4 (40)	26 (30.58)	0.256
Diuretic	5 (50)	27 (31.76)	0.036
Intracoronary Gp-IIb/IIIa inh.	10 (100)	3 (3.52)	<0.001
Intracoronary adenosine	10 (100)	1 (1.17)	<0.001

Table-2: Demographic characteristics significantly associated with European System for Cardiac Operative Risk Evaluation II (n=95)

Characteristics	r	p-value
New York Heart Association class	0.480	<0.001
HR	0.183	0.002
Hospital length of stay	0.579	<0.001
Score	0.872	<0.001
Estimated Glomerular Filtration Rate	-0.581	<0.001

Table-3: Clinical follow-up (n=95)

Characteristics	Coronary no-reflow phenomenon n=10 (10.5%)	Coronary slow-flow phenomenon n=85 (89.4%)	p-value
All-Cause Mortality	1 (10)	11 (12.94)	0.598
Cardiovascular Death	1 (10)	9 (10.58)	0.487
Stroke	2 (20)	2 (2.35)	<0.001
Myocardial re-infarction	1(10)	10 (11.76)	0.817
MACE	4 (40)	20 (23.52)	0.031

Table-4: Independent predictors of CNP

Characteristics	Odds ratio	95% Confidence interval	p-value
Body Mass Index	1.12	1.00-1.25	0.027
Heart Rate	0.914	0.87-0.97	<0.001

DISCUSSION

In this study, high BMI levels and low Heart Rate were recognized as two determinants of CNP in NSTEMI patients. Finally, CNP was strongly linked to worse outcomes in individuals with NSTEMI. The presence of CNP in NSTEMI can be assumed by BMI values >28.42 kg/m2. Additionally, an HR of <66.31 bpm suggests that NSTEMI has CNP. CNP patients with NSTEMI who have higher BMI also have lower heart rates. This is the first report of its kind in the literature. In comparison to the control group, the CNP group had a substantially greater incidence of stroke and MACE one year following the research. The outcomes of NSTEMI patients were worsened by CNP in this study. In coronary angiography, coronary slow flow phenomenon and coronary no-reflow phenomenon are rare events, with an occurrence of approximately 1%; however, published data indicate that CNP and CSFP occur in acute coronary syndrome at a frequency ranging from 1% to 60%.[13,14] It was found that CNP occurred in 3.1% of the study population, whereas CSFP occurred in 25.2% of the population. The short- and long-term clinical consequences of CSFP and CNP are significantly correlated with poor patient outcomes.[3] NSTEMI patients with CNP have a particularly poor cardiac outcome.[13,15] According to published data, the worst outcomes were found in the coronary no-reflow phenomenon group. According to our research, CNP patients had significantly higher MACE and stroke outcomes over their one-year follow-up. Strokes were 8.88-fold more likely to occur in the CNP group than in the CSFP group. A further finding was that the probability of MACE in the CNP group was 1.90-fold greater than that in the coronary slow flow phenomenon group. In retrospective and prospective studies, cardiac troponin has been associated with adverse outcomes following NSTEMI in previous meta-analyses.[16] This study found that the CNP group had significantly higher peak troponin-I levels compared to the other groups, which is consistent with the literature. Meanwhile, stroke was related to thrombus burden. Research has identified thrombus activation after an index event as the associated mechanism causing this adverse event, which may contribute to stroke risk. The CNP patients had a significantly greater incidence of stroke than NSTEMI patients who received antithrombotic treatment regularly. Such patients should thus be closely observed following discharge. In addition, the Body Mass Index is the most common tool for assessing cardiovascular risk and obesity.[17]

Among NSTEMI patients, Bakirci et al.[18] There is an association between epicardial fat, which increases in obese patients, and coronary artery disease.[18] According to a recent study, CNP is related to hyperglycemia, hypercholesterolemia, and mild to moderate renal impairment more frequently.[19] In the present research, we discovered that CNP patients had lower eGFR scores than CNP patients, consistent with literature findings. Moreover, we observed that CNP group patients had significantly higher BMI, which might account for increased stroke risk. Therefore, determining body mass index (BMI) may help evaluate cardiac outcomes in individuals suffering from NSTEMI and CNP.

However, recent randomized studies have shown that Microvascular perfusion and long-term outcomes may be improved with manual thrombus aspiration catheter usage as compared to controls.[20] Therefore, we decided to exclude the patients (n=2) who had thrombus aspiration catheters during the index surgery so that the thrombus aspiration would not affect the stroke endpoint. There have been positive effects on myocardial perfusion with the use of platelet inhibitors (Gp-IIb/IIIa inh., abciximab, tirofiban), nicorandil, nitroprusside, and adenosine.[21] A beneficial effect of intracoronary epinephrine on CNP was also found by Aksu et al.[22] Moreover, Skelding et al. found that epinephrine may also promote tachycardia and increase coronary blood pressure.[23] In our investigation of NSTEMI patients, a lower HR was found to be an independent predictor of CNP, following the literature. The lower HR of NSTEMI patients could be an indicator of CNP caused by slow microcirculation. The operative is essential aware of the patient's heart rate and considers a patient with a lower heart rate for CNP before starting the PCI. Our study found a lower HR despite encouraging results, but randomized trials should be used to explain these findings.

CONCLUSION

Patients with NSTEMI whose CNP was determined by low HR and high BMI. Our study showed that CNPs had a significantly higher stroke and MACE incidences after one year of clinical follow-up. The study shows CNP harms NSTEMI patients' outcomes.

REFERENCES

1. Fox K. The ENACT study: a pan-European survey of acute coronary syndromes. Eur Heart J [Internet]. 2000 Sep 1;21(17):1440–9. Available from: https://academic.oup.com/eurheartj/article-lookup/doi/10.1053/euhj.2000.2185
2. Savonitto S. Prognostic Value of the Admission Electrocardiogram in Acute Coronary Syndromes. JAMA [Internet]. 1999 Feb 24;281(8):707. Available from: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.281.8.707
3. Jaffe R, Dick A, Strauss BH. Prevention and Treatment of Microvascular Obstruction-Related Myocardial Injury and Coronary No-Reflow Following Percutaneous Coronary Intervention. JACC Cardiovasc Interv [Internet]. 2010 Jul;3(7):695–704. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1936879810003225
4. Brosh D, Assali AR, Mager A, Porter A, Hasdai D, Teplitsky I, et al. Effect of No-Reflow During Primary Percutaneous Coronary Intervention for Acute Myocardial Infarction on Six-Month Mortality. Am J Cardiol [Internet]. 2007 Feb;99(4):442–5. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002914906021412
5. Hawkins BM, Stavrakis S, Rousan TA, Abu-Fadel M, Schechter E. Coronary Slow Flow. Circ J [Internet]. 2012;76(4):936–42. Available from: https://www.jstage.jst.go.jp/article/circj/76/4/76_CJ-11-0959/_article
6. Rezkalla SH, Stankowski R V., Hanna J, Kloner RA. Management of No-Reflow Phenomenon in the Catheterization Laboratory. JACC Cardiovasc Interv [Internet]. 2017 Feb;10(3):215–23. Available from: https://linkinghub.elsevier.com/retrieve/pii/S1936879816323366
7. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth Universal Definition of Myocardial Infarction (2018). Circulation [Internet]. 2018 Nov 13;138(20). Available from: https://www.ahajournals.org/doi/10.1161/CIR.0000000000000617
8. Armstrong C, Joint National Committee. JNC8 guidelines for the management of hypertension in adults. Am Fam Physician [Internet]. 2014 Oct 1;90(7):503–4. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25369633
9. Gavin JR, Alberti KGMM, Davidson MB, DeFronzo RA, Drash A, Gabbe SG, et al. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care [Internet]. 2003 Jan 1;26(SUPPL. 1):s5–20. Available from: https://diabetesjournals.org/care/article/26/suppl_1/s5/21693/Report-of-the-Expert-Committee-on-the-Diagnosis
10. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and T of HBC in A (Adult TPI. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in

Adults (Adult Treatment Panel III) final report. Circulation [Internet]. 2002 Dec 17;106(25):3143–421. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12485966

11. Acquatella H, Asch FM, Barbosa MM, Barros M, Bern C, Cavalcante JL, et al. Recommendations for Multimodality Cardiac Imaging in Patients with Chagas Disease: A Report from the American Society of Echocardiography in Collaboration With the InterAmerican Association of Echocardiography (ECOSIAC) and the Cardiovascular Imaging Depa. J Am Soc Echocardiogr [Internet]. 2018 Jan;31(1):3–25. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0894731717307599
12. Gibson CM, Cannon CP, Daley WL, Dodge JT, Alexander B, Marble SJ, et al. TIMI Frame Count. Circulation [Internet]. 1996 Mar;93(5):879–88. Available from: https://www.ahajournals.org/doi/10.1161/01.CIR.93.5.879
13. Durante A, Camici PG. Novel insights into an “old” phenomenon: the no reflow. Int J Cardiol [Internet]. 2015 May;187:273–80. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0167527315006178
14. Chaudhry MA, Smith M, Hanna EB, Lazzara R. Diverse Spectrum of Presentation of Coronary Slow Flow Phenomenon: A Concise Review of the Literature. Cardiol Res Pract [Internet]. 2012;2012:1–6. Available from: http://www.hindawi.com/journals/crp/2012/383181/
15. Ndrepepa G, Tiroch K, Fusaro M, Keta D, Seyfarth M, Byrne RA, et al. 5-Year Prognostic Value of No-Reflow Phenomenon After Percutaneous Coronary Intervention in Patients With Acute Myocardial Infarction. J Am Coll Cardiol [Internet]. 2010 May;55(21):2383–9. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0735109710010582
16. Nienhuis MB, Ottervanger JP, Bilo HJG, Dikkeschei BD, Zijlstra F. Prognostic value of troponin after elective percutaneous coronary intervention: A meta-analysis. Catheter Cardiovasc Interv [Internet]. 2008 Feb 20;71(3):318–24. Available from: https://onlinelibrary.wiley.com/doi/10.1002/ccd.21345
17. Bergman RN, Kim SP, Catalano KJ, Hsu IR, Chiu JD, Kabir M, et al. Why Visceral Fat is Bad: Mechanisms of the Metabolic Syndrome. Obesity [Internet]. 2006 Feb;14(2S):16S–19S. Available from: http://doi.wiley.com/10.1038/oby.2006.277
18. Bakirci EM, Degirmenci H, Duman H, Inci S, Hamur H, Buyuklu M, et al. Increased Epicardial Adipose Tissue Thickness is Associated With Angiographic Thrombus Burden in the Patients With Non-ST-Segment Elevation Myocardial Infarction. Clin Appl Thromb [Internet]. 2015 Oct 6;21(7):612–8. Available from: http://journals.sagepub.com/doi/10.1177/1076029614558113
19. Ipek G, Onuk T, Karatas MB, Gungor B, Osken A, Keskin M, et al. CHA 2 DS 2 -VASc Score is a Predictor of No-Reflow in Patients With ST-Segment Elevation Myocardial Infarction Who Underwent Primary Percutaneous Intervention. Angiology [Internet]. 2016 Oct 19;67(9):840–5. Available from: http://journals.sagepub.com/doi/10.1177/0003319715622844
20. Vlaar PJ, Svilaas T, van der Horst IC, Diercks GF, Fokkema ML, de Smet BJ, et al. Cardiac death and reinfarction after 1 year in the Thrombus Aspiration during Percutaneous coronary intervention in Acute myocardial infarction Study (TAPAS): a 1-year follow-up study. Lancet [Internet]. 2008 Jun;371(9628):1915–20. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0140673608608338
21. Amit G, Cafri C, Yaroslavtsev S, Fuchs S, Paltiel O, Abu-Ful A, et al. Intracoronary nitroprusside for the prevention of the no-reflow phenomenon after primary percutaneous coronary intervention in acute myocardial infarction. A randomized, double-blind, placebo-controlled clinical trial. Am Heart J [Internet]. 2006 Nov;152(5):887.e9-887.e14. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0002870306004583
22. Aksu T, Guler TE, Colak A, Baysal E, Durukan M, Sen T, et al. Intracoronary epinephrine in the treatment of refractory no-reflow after primary percutaneous coronary intervention: a retrospective study. BMC Cardiovasc Disord [Internet]. 2015 Dec 19;15(1):10. Available from: https://bmccardiovascdisord.biomedcentral.com/articles/10.1186/s12872-015-0004-6
23. Skelding KA, Goldstein JA, Mehta L, Pica MC, O’Neill WW. Resolution of refractory no-reflow with intracoronary epinephrine. Catheter Cardiovasc Interv [Internet]. 2002 Nov;57(3):305–9. Available from: https://onlinelibrary.wiley.com/doi/10.1002/ccd.10303