

Angiographic Predictors of Viability During Intervention for a ST Elevation Myocardial Infarction

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ABSTRACT: Objectives: This study aimed to identify angiographic features that would predict myocardial viability after coronary intervention for ST elevation myocardial infarction (STEMI). **Methods:** This retrospective study included patients who attended Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 with a STEMI. **Results:** A total of 72 patients (61 male; mean age = 54.9 ± 12.7 years) were included in the study; 11 patients had evidence of non-viability on echocardiography. There were 13 patients with viable myocardium and 3 with non-viable myocardium who had a myocardial blush grade (MBG) of 2 or lower. Similarly, 10 patients with viability and 1 with non-viable myocardium had thrombolysis in myocardial infarction (TIMI) flow of 2 or lower in the infarct related artery (IRA). However, none of these were statistically significant. The TIMI flow in the IRA at the end of the procedure correlated with the MBG. **Conclusion:** There were no clear angiographic features during primary angioplasty that could predict myocardial viability.

Keywords: Myocardial infarction; Coronary Angiography; Viability; Oman.

THE PHRASE “TIME IS MUSCLE” IS OFTEN-QUOTED during the management of a ST elevation myocardial infarction (STEMI) to emphasise the need for timely reperfusion and restoration of flow in the occluded artery.¹ Studies have demonstrated that rapid restoration of flow in the occluded artery, either by thrombolysis or by primary percutaneous coronary intervention (PPCI), is associated with lesser myocardial damage, resulting in improved mortality and morbidity and decreased incidence of long term complications such as heart failure and ventricular arrhythmias.² Restoration of brisk flow (thrombolysis in myocardial infarction [TIMI]-3 flow) in the infarct related artery (IRA) has been previously demonstrated to be associated with good long-term prognosis following a STEMI.^{3,4}

Studies have conclusively demonstrated that PPCI is better than thrombolysis in restoring epicardial flow with more than 95% of patients achieving TIMI 2–3 flow compared to 60–80% with thrombolysis.^{5–8} However, studies have also demonstrated that although a commonly accepted indicator of reperfusion, brisk flow in an epicardial vessel might not always be associated with myocardial viability as flow in the epicardial vessel does not always reflect flow at the microvasculature or perfusion at the cellular level.^{9,10} For late presenters, irreversible damage might have already been done and restoring flow does not restore myocardial viability or improve mortality.¹¹ Even for those where the artery is opened within the guideline-recommended time frame, the myocardium might

have been rendered non-viable due to other factors.¹² The IRA in a STEMI is occluded with thrombus and although routine aspiration of the thrombus during the PPCI has not been demonstrated to be useful in preserving myocardium or improving prognosis, it is inevitable that during the coronary intervention, some of the thrombus can embolise downstream and block the microvasculature.^{13,14}

One potential way to assess flow in the microvasculature is by the myocardial blush grade (MBG) which is the presence of contrast in the myocardium.^{15,16} MBG has been found to be a predictor (independent of the TIMI flow in the vessel) of both in-hospital and long-term mortality in patients with STEMI who underwent primary angioplasty.¹⁶ It is a qualitative visual assessment of the amount of contrast medium filling a territory supplied by an epicardial coronary artery and correlates with tissue perfusion and microvascular patency. This is a variable that is often noticed by the operator, but not routinely commented on or documented. Similarly, the slow-flow, no-reflow phenomenon can also reflect poor myocardial perfusion as can sluggish flow (less than TIMI 2 flow) in the IRA.^{16,17} There is limited and largely conflicting data correlating these angiographic findings and myocardial viability.^{17,18}

Assessment of these findings during a PPCI procedure is important and could guide the operator to optimise angiographic reperfusion end points, which could be a surrogate of myocardial viability. This study aimed to assess the different angiographic

findings at the time of a PPCI and examine whether any of these features, especially the MBG, predict myocardial viability after reperfusion.

Methods

This retrospective study included all patients who attended Sultan Qaboos University Hospital, Muscat, Oman, between January and December 2019 with a STEMI. Patients were included in the study if full data on pre- and post-angiography electrocardiogram (ECG) and post procedure echocardiogram to assess viability were available. Those who did not have a full set of investigations and patients who were transferred from other institutions where information was incomplete were excluded.

The angiograms were assessed for target vessel intervention, the TIMI flow in the culprit vessel before and after intervention and the TIMI blush score along with the presence or absence of collaterals to the IRA prior to intervention. The angiograms were assessed independently by 2 trained operators. Where there was a discrepancy, a third operator assessed the data, blinded to the values of the other 2 operators. Where a discrepancy still persisted, all 3 operators assessed the angiograms together to come to a consensus.

The TIMI flow in the culprit vessel was classified as described previously.¹⁹ TIMI 0 flow indicates complete occlusion of the vessel with no flow; TIMI 1 flow is slow flow within the vessel with the contrast failing to opacify of the distal end of the vessel; TIMI 2 flow is sluggish flow in the vessel with contrast opacifying the distal vessel but at a rate much slower than that of other corresponding vessels; and TIMI 3 flow indicates brisk flow in the vessel with complete opacification of the distal bed at a rate similar to other comparable vascular beds.

The MBG was scored as described earlier.¹⁵ MBG of 0 signifies no contrast entering the myocardial microvasculature; MPG of 1 suggests slow entry of contrast into the microvasculature, but it does not get washed away and persists even into the next set of images; and MPG of 2 represents delayed entry and exit of contrast from the microvasculature. There is delayed entry, and the intensity of contrast persists to more than 3 cardiac cycles from entry but is washed away before the next set of images. Lastly, a MPG of 3 represents brisk entry and exit of contrast from the microvasculature. The contrast enters briskly and the intensity quickly fades to complete washout within 3 cardiac cycles from the time of entry of the contrast into the microvasculature.

The presence of collaterals was defined by the Rentrop classification.²⁰ Grade 0 represents no collaterals; Grade 1 represents filling of just the side branch of the recipient artery without filling of the main epicardial artery; Grade 2 is partial filling of the main epicardial recipient artery; and Grade 3 is complete filling of the main epicardial recipient artery.

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS), Version 221 (IBM Corp., Armonk, New York, USA). The data are represented using percentages and mean \pm standard deviation or median (interquartile range). Analysis was performed using Chi-square test and bivariate analysis. Cohens Kappa was used to assess the level of agreement between the 2 investigators. For the binary regression analysis, presence of viability was the outcome and other angiographic features were used as the predicting variables. A *P* value of less than 0.05 was considered statistically significant.

Ethical approval was obtained prior to commencing the study.

Results

A total of 110 patients (89 male and 21 female; mean age = 55.8 ± 12.1 years) were identified as having presented with a STEMI during the study period. Of these, 72 patients (61 male; mean age = 54.9 ± 12.7 years) had the complete data set which was used in the analysis. Evidence of akinesia of the target left ventricular (LV) wall on echocardiography was found in 11 patients and was considered to have non-viable myocardium. The remaining 61 patients had either normal wall motion or hypokinesia suggesting viability. All patients received loading dose of acetylsalicylic acid (300 mg) and clopidogrel (600 mg) prior to the procedure. None of the patients in this study received a glycoprotein IIb/IIIa inhibitor. There was no difference between the 2 groups in terms of cardiovascular risk factors, location of MI, pain to balloon time or resolution of ST elevation or presence of Q waves on ECG [Table 1]. Interestingly, all patients with non-viable myocardium had Q waves on their ECG, as well as a high proportion of those with viable myocardium had Q waves (100% versus 81.9%; *P* = 0.22)

Table 2 shows the angiographic correlates of these patients with viability on echocardiography. A Cohens Kappa of 0.80 (*P* < 0.001) was observed for assessing the TIMI flow in the IRA (95% agreement in first reading) and a kappa of 0.56 (*P* = 0.02) for the MBG score (85% agreement in first reading). Although there was good agreement in assessing both parameters by the

Table 1: Baseline characteristics of the patients (N = 72)

Characteristic	n (%)		P value
	No akinesia on echo (n = 61)	Akinesia on echo (n = 11)	
Age in years \pm SD	55 \pm 12	47 \pm 10	0.04*
Sex			0.53
Male	51 (83.6)	10 (90.9)	
Female	10 (16.4)	1 (8.9)	
Diabetes	25 (40.9)	7 (63.6)	0.16
Hypertension	31 (50.8)	6 (54.5)	0.052
Dyslipidaemia	20 (32.7)	3 (27.2)	0.052
Smoker	17 (27.8)	3 (27.2)	0.96
Location of STEMI			0.08
Anterior	13 (21.3)	5 (45.4)	
Inferior/inferoposterior	35 (57.3)	2 (18.1)	
Lateral	13 (21.3)	4 (36.3)	
Pain to balloon time in minutes	230 (165–318)	285 (153–442)	0.60†
50% resolution of ST segment	47 (77.1)	9 (81.8)	0.53
Q waves on ECG post-procedure	50 (81.9)	11 (100)	0.22
Peak troponin	2593 (1321–5076)	3590 (2955–6387)	0.06†
Peak creatinine	82 (67–99)	73 (67–84)	0.30†
Lowest GFR \pm SD	75 \pm 19	83 \pm 11	0.17*

SD = standard deviation; STEMI = ST segment elevation myocardial infarction; ECG = electrocardiogram; GFR = glomerular filtration rate.

*Using students *t*-test. †Using Mann-Whitney test.

2 investigators, these findings show greater consensus for the assessment of TIMI flow rather than the MBG. There was no difference between the 2 groups in terms of TIMI flow in the IRA pre- or post-PCI, or even the MBG, or the presence or absence of collaterals on presentation. None of the angiographic features could predict the presence of viable myocardium on echocardiography. Although 7 of the 61 patients with TIMI 2 flow or greater in the vessel post intervention had a MBG of 2 or less, there was however a significant correlation between the TIMI flow in the vessel at the end of the procedure and the MBG (fishers exact test: $P < 0.001$). Binary regression analysis was performed using viability on echocardiogram as the dependent variable. All other angiographic and clinical features were used as the independent variables. None of the independent variables were predictive of myocardial viability.

Discussion

Preserving myocardial viability is the main outcome desired of timely intervention in PPCI. The aim of the cardiac interventionalist therefore, is to achieve an

optimal angiographic appearance post-intervention that would increase the likelihood of myocardial viability. These include getting an optimal result in the occluded vessel and achieving TIMI 3 flow in the IRA.²¹ If there were additional factors that could be identified on the angiogram that could predict better long term outcomes, the interventionalist would aim to achieve these optimal features.

The current study found that clinical factors such as cardiovascular risk factors and the ischaemic time (pain to balloon time) did not predict viability. It is well documented that the longer the delays in revascularisation, the higher the chance of complete myocardial necrosis and non-viability.¹ However, the documented ischaemic times are often unreliable as the patient might have ignored the pains or delayed seeking medical advice and recollection of the onset of pain has been demonstrated to be inaccurate.^{22–24}

This study also demonstrated that there are no angiographic features that would predict viability post-intervention. Presence of flow greater than TIMI 2 on the first angiographic image has previously been shown to be associated with improved prognosis and viability and referred to as an aborted STEMI.²⁵

Table 2: Angiographic findings of the included patients (N = 72)

Finding	n (%)		P value*
	No akinesia in infarct territory (n = 61)	Akinesia in infarct territory (n = 11)	
Culprit vessel			0.11
LAD	23 (37.7)	9 (81.8)	
Circumflex	12 (19.6)	1 (8.9)	
RCA	22 (36.1)	1 (8.9)	
Diagonal	3 (4.9)	0	
SVG	1 (1.6)	0	
TIMI flow in IRA pre- PCI			0.6
0	43 (70.4)	7 (63.6)	
1	4 (6.5)	1 (8.9)	
2	4 (6.5)	2 (18.1)	
3	9 (14.7)	1 (8.9)	
TIMI flow pre-PCI ≥ 2	13 (21.2)	3 (27)	0.3
TIMI flow in IRA post-PCI			0.53
0	0	0	
1	0	0	
2	10 (16.4)	1 (8.1)	
3	51 (83.6)	10 (91.9)	
MBG post-PCI			0.8
0	1 (1.6)	0	
1	2 (3.2)	1 (8.2)	
2	10 (16.4)	2 (18.1)	
3	48 (70.4)	8 (72.7)	
MBG of 2 or 3	58 (86.8)	10 (90.8)	0.58
Presence of collaterals	18 (29.5)	2 (18.1)	0.44
Collateral grade			0.77
0	43 (70.4)	9 (81.8)	
1	9 (14.7)	1 (8.9)	
2	5 (8.1)	1 (8.9)	
3	4 (6.5)	0	

LAD = left anterior descending artery; RCA = right coronary artery; SVG = saphenous vein graft; TIMI = thrombolysis in myocardial infarction; IRA = infarct related artery; PCI = percutaneous coronary intervention; MBG = myocardial blush grade.

*Using Chi-squared test.

However in the current study, there was no difference between the groups.

The MBG has been assessed previously with regards to myocardial viability. Bertomeu-González *et al.* demonstrated only a very weak correlation between the MBG and perfusion and LV function in patients who had been thrombolysed for their STEMI.²⁶ Kampinga *et al.* studied the prognostic value of

MBG as scored by the operator during PPCI in 2,118 consecutive patients with STEMI. They found a strong correlation between the MBG at the end of the PPCI procedure and mortality and they recommended that MBG be documented at the end of each procedure.¹⁸ They however did not correlate this with myocardial viability. Similarly, Henriques *et al.* also found a strong correlation between an MBG of greater than 2 with

improved mortality and long-term outcomes in their study on 924 patients. They demonstrated that approximately 1 in 10 patients with TIMI 3 flow in the IRA had MBG of 0 or 1 and these patients had a worse long term prognosis and lower LV ejection fraction.¹⁶ However, the current study could not find any correlation between higher MBG grades and viability, although patients were not followed-up to assess outcomes.

Medications given during the procedure as well as during follow-up can affect myocardial viability. Locuratolo *et al.* followed-up 2,476 patients after an acute coronary syndrome and demonstrated clearly that good medical therapy in the acute phase and during follow-up is associated with good outcomes.²⁷ The role of antiplatelet therapy in improving hospital outcomes and long term morbidity and mortality are well known and recommended by all major guidelines.² The newer antiplatelets such as ticagrelor and prasrel have been demonstrated to improve myocardial microcirculation after infarction and limit infarct size.^{28,29} In the current study, all patients were given dual antiplatelet therapy prior to the procedure. None of the patients were given a glycoprotein IIb/IIIa inhibitor. Hence, it was not possible to assess the effect of medications on viability.

It was previously demonstrated that the presence of collaterals in patients with STEMI does not indicate viability in case of a chronic occlusion.³⁰ In this study as well, it was found that the presence of collaterals prior to the procedure did not indicate or predict viability. The current study highlights that myocardial viability is the result of a complex interaction between various factors and there was no single factor that the authors could identify on angiography that could predict viability.

The retrospective nature of this study was the main limitation as the authors had to rely on recorded information from the case notes for the clinical information. Prospective data on the long term outcomes of the patients as well as follow up echocardiography to see if there was any improvement in wall motion abnormalities was not available. This study relied on akinesia on echocardiography to diagnose non-viability rather than viability tests. The authors did not have nuclear scans or magnetic resonance imaging to look for definitive signs of infarction and loss of viability. The sample size was small which could also have influenced the results.

Conclusions

There are no definite angiographic features during a PPCI that can predict viability. Despite this, however,

every effort should be made to achieve optimal end result at the end of an intervention for STEMI.

CONFLICTS OF INTEREST

The authors declare no conflict of interests.

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AUTHORS' CONTRIBUTION

SA and YA collected data. HA, AA, SKN analysed data, supervised the data collection and wrote the manuscript. All authors approved the final version of the manuscript.

References

1. De LG, Suryapranata H, Ottervanger JP, Antman EM. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: every minute of delay counts. *Circulation* 2004; 109:1223–5. <https://doi.org/10.1161/01.CIR.0000121424.76486.20>.
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. <https://doi.org/10.1093/eurheartj/ehx393>.
3. Ndrepepa G, Mehilli J, Schulz S, Iijima R, Keta D, Byrne RA, et al. Prognostic significance of epicardial blood flow before and after percutaneous coronary intervention in patients with acute coronary syndromes. *J Am Coll Cardiol* 2008; 52:512–17. <https://doi.org/10.1016/j.jacc.2008.05.009>.
4. Simes RJ, Topol EJ, Holmes DR, Jr, White HD, Rutsch WR, Vahanian A, et al. Link between the angiographic substudy and mortality outcomes in a large randomized trial of myocardial reperfusion. Importance of early and complete infarct artery reperfusion. GUSTO-I Investigators. *Circulation* 1995; 91:1923–8. <https://doi.org/10.1161/01.cir.91.7.1923>.
5. Grines CL, Cox DA, Stone GW, Garcia E, Mattos LA, Giambartolomei A, et al. Coronary angioplasty with or without stent implantation for acute myocardial infarction. Stent Primary Angioplasty in Myocardial Infarction Study Group. *N Engl J Med* 1999; 341:1949–56. <https://doi.org/10.1056/NEJM199912233412601>.
6. Stone GW, Brodie BR, Griffin JJ, Costantini C, Morice MC, St Goar FG, et al. Clinical and angiographic follow-Up after primary stenting in acute myocardial infarction: the Primary Angioplasty in Myocardial Infarction (PAMI) stent pilot trial. *Circulation* 1999; 99:1548–54. <https://doi.org/10.1161/01.cir.99.12.1548>.
7. Bode C, Smalling RW, Berg G, Burnett C, Lorch G, Kalbfleisch JM, et al. Randomized comparison of coronary thrombolysis achieved with double-bolus reteplase (recombinant plasminogen activator) and front-loaded, accelerated alteplase (recombinant tissue plasminogen activator) in patients with acute myocardial infarction. The RAPID II Investigators. *Circulation* 1996;94:891–8. <https://doi.org/10.1161/01.cir.94.5.891>.
8. Gokhroo RK, Gupta S, Bisht DS, Padmanabhan D. A study of coronary artery patency in relation to the index event in patients with myocardial infarction thrombolysed with streptokinase. *Heart Asia* 2014; 6:55–8. <https://doi.org/10.1136/heartasia-2014-010494>.

9. Ito H, Okamura A, Iwakura K, Masuyama T, Hori M, Takiuchi S, et al. Myocardial perfusion patterns related to thrombolysis in myocardial infarction perfusion grades after coronary angioplasty in patients with acute anterior wall myocardial infarction. *Circulation* 1996; 93:1993–9. <https://doi.org/10.1161/01.cir.93.11.1993>.
10. Kaul S. Coronary angiography cannot be used to assess myocardial perfusion in patients undergoing reperfusion for acute myocardial infarction. *Heart* 2001 Nov;86(5):483–4. <https://doi.org/10.1136/heart.86.5.483>.
11. Hochman JS, Lamas GA, Buller CE, Dzavik V, Reynolds HR, Abramsky SJ, et al. Coronary intervention for persistent occlusion after myocardial infarction. *N Engl J Med* 2006; 355:2395–407. <https://doi.org/10.1056/NEJMoa066139>.
12. Busk M, Kaltoft A, Nielsen SS, Bottcher M, Rehling M, Thuesen L, et al. Infarct size and myocardial salvage after primary angioplasty in patients presenting with symptoms for <12 h vs. 12–72 h. *Eur Heart J* 2009; 30:1322–30. <https://doi.org/10.1093/eurheartj/ehp113>.
13. Frobert O, Lagerqvist B, Olivecrona GK, Omerovic E, Gudnason T, Maeng M, et al. Thrombus aspiration during ST-segment elevation myocardial infarction. *N Engl J Med* 2013; 369:1587–97. <https://doi.org/10.1056/NEJMoa1308789>.
14. Jolly SS, Cairns JA, Yusuf S, Rokoss MJ, Gao P, Meeks B, et al. Outcomes after thrombus aspiration for ST elevation myocardial infarction: 1-year follow-up of the prospective randomised TOTAL trial. *Lancet* 2016; 387:127–35. [https://doi.org/10.1016/S0140-6736\(15\)00448-1](https://doi.org/10.1016/S0140-6736(15)00448-1).
15. van 't Hof AW, Liem A, Suryapranata H, Hoorntje JC, de Boer MJ, Zijlstra F. Clinical presentation and outcome of patients with early, intermediate and late reperfusion therapy by primary coronary angioplasty for acute myocardial infarction. *Eur Heart J* 1998; 19:118–23. <https://doi.org/10.1053/euhj.1997.0746>.
16. Henriques JP, Zijlstra F, van 't Hof AW, de Boer MJ, Dambrink JH, Gosselink M, et al. Angiographic assessment of reperfusion in acute myocardial infarction by myocardial blush grade. *Circulation* 2003; 107:2115–19. <https://doi.org/10.1161/01.CIR.0000065221.06430.ED>.
17. Ito H, Maruyama A, Iwakura K, Takiuchi S, Masuyama T, Hori M, et al. Clinical implications of the 'no reflow' phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. *Circulation* 1996; 93:223–8. <https://doi.org/10.1161/01.cir.93.2.223>.
18. Kampinga MA, Nijsten MW, Gu YL, Dijk WA, de Smet BJ, van den Heuvel AF, et al. Is the myocardial blush grade scored by the operator during primary percutaneous coronary intervention of prognostic value in patients with ST-elevation myocardial infarction in routine clinical practice? *Circ Cardiovasc Interv* 2010; 3:216–23. <https://doi.org/10.1161/CIRCINTERVENTIONS.109.916247>.
19. The Thrombolysis in Myocardial Infarction (TIMI) trial. Phase I findings. *N Engl J Med* 1985; 312:932–6. <https://doi.org/10.1056/NEJM198504043121437>.
20. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. *J Am Coll Cardiol* 1985; 5:587–92. [https://doi.org/10.1016/s0735-1097\(85\)80380-6](https://doi.org/10.1016/s0735-1097(85)80380-6).
21. Kristensen SD. Primary PCI: how can we improve outcome? *EuroIntervention* 2013; 8:1115–17. <https://doi.org/10.4244/EIJV8110A172>.
22. Everts B, Karlson B, Wahrborg P, Abdou N, Herlitz J, Hedner T. Pain recollection after chest pain of cardiac origin. *Cardiology* 1999; 92:115–20. <https://doi.org/10.1159/000006958>.
23. Gartner C, Walz L, Bauernschmitt E, Ladwig KH. The causes of prehospital delay in myocardial infarction. *Dtsch Arztebl Int* 2008; 105:286–91. <https://doi.org/10.3238/arztebl.2008.0286>.
24. Almashari S, Al-Malki Y, Al-Riyami A, Nadar SK. Delays in Presentation by Patients with ST Elevation Myocardial Infarction: A single centre experience from Oman. *Sultan Qaboos Univ Med J* 2022; 22:283–7. <https://doi.org/10.18295/squmj.4.2021.069>.
25. Prech M, Bartela E, Araszkievicz A, Kutrowska A, Janus M, Jeremicz I, et al. Initial TIMI flow ≥ 2 and pre-angiography total ST-segment resolution predict an aborted myocardial infarction in patients undergoing primary percutaneous coronary intervention. *Kardiol Pol* 2014; 72:223–30. <https://doi.org/10.5603/KP.a2013.0250>.
26. Bertomeu-Gonzalez V, Bodi V, Sanchis J, Nunez J, Lopez-Lereu MP, Pena G, et al. [Limitations of myocardial blush grade in the evaluation of myocardial perfusion in patients with acute myocardial infarction and TIMI grade 3 flow]. *Rev Esp Cardiol* 2006; 59:575–81. <https://doi.org/10.5603/KP.a2013.0250>.
27. Locuratolo N, Scicchitano P, Antoncecechi E, Basso P, Bonfantino VM, Brescia F, et al. [Follow-up of patients after an acute coronary event: the Apulia PONTE-SCA program]. *G Ital Cardiol (Rome)* 2022; 23:63–74. <https://doi.org/10.1714/3715.37064>.
28. Aytakin A, Ndrepepa G, Neumann FJ, Menichelli M, Mayer K, Wohrle J, et al. Ticagrelor or Prasugrel in Patients With ST-Segment-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. *Circulation* 2020; 142:2329–37. <https://doi.org/10.1161/CIRCULATIONAHA.120.050244>.
29. Vilahur G, Gutierrez M, Casani L, Varela L, Capdevila A, Pons-Llado G, et al. Protective Effects of Ticagrelor on Myocardial Injury After Infarction. *Circulation* 2016; 134:1708–19. <https://doi.org/10.1161/CIRCULATIONAHA.116.024014>.
30. Shaikh MM, Sadiq MA, Nadar SK. Q-Waves Associated With Postinfarct Chronic Total Occlusion Arteries Predict Non-Viable Myocardium Even in the Presence of Collaterals. *J Invasive Cardiol* 2020; 32:E213–15.